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Fundamentals and Perspectives

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Preface

In 2002, with its *World Report on Violence and Health* (Geneva 2002), the World Health Organization (WHO) presented the first comprehensive review of violence committed by man against man. Violence represents the leading cause of death in the 15- to 45-year age group: 14 % of all male and 7 % of all female deaths in this age group are of a violent nature. A total of 520,000 homicides occurring in individual instances of non-wartime violence were recorded worldwide for the year 2000. To this figure can be added approximately 815,000 suicides. The number of cases of serious physical assault is considerably higher, whilst the psychological effects of experiencing violence are all but impossible to quantify. Other causes of traumatic death include fatal traffic accidents, domestic and occupational accidents, as well as fatalities and bodily injury associated with the use of alcohol, drugs, and medication. A proportion of serious physical assaults is accounted for by domestic and family violence (including child abuse) and also includes sex offences (rape, child sexual abuse), not least as a systematic feature of organized crime (e.g., child sex tourism).

At least in principle, all countries worldwide undertake formal procedures to investigate fatalities and crimes. To this end, the investigative activities of the police and judicial authorities require the support of medical expertise, be it at the scene of body discovery, at autopsy, in the form of scientific medical investigations, or the physical examination of victims of violence and suspects for the purposes of reconstructing a crime. The goal of these forensic medical investigations is to establish objective facts and subject these to expert interpretation, enabling the organs of the law (police, public prosecution department, and law courts) to dutifully reach decisions and judgements. At the same time, it is important to remember that much of the most threatening violence in quantitative terms is committed by state organs (police, secret service, paramilitary organizations, military, etc.). The (forensic medical) expert therefore requires a post that falls under administrative protection in order to function and prevail, in the face of other state or non-state entities, with the necessary objectivity and neutrality, and without breaching his or her obligation to tell the truth or violating basic ethical and moral principles, as demanded not least by the international code of professional conduct for medical practitioners.

The present book is intended to promote this goal and impart the basic principles of forensic medical appraisal across all fields of our specialty, whilst at the same time highlighting diagnostic and methodological perspectives.

Indeed, considerable advances in detection methods have been made over the last 10–15 years in, for instance, toxicological analysis, most notably in terms of sensitivity, specificity, selectivity, and practicability. Modern computers have made screening procedures previously considered impossible a reality and enabled global literature research. Equipment once found only in large centers and institutions is now available in smaller laboratories. These advances have been made necessary by the relentless appearance of new substances on the market, e.g., pep, crystal ice, crack, spice, crocodile, bath salts, newer benzodiazepines, Z-drugs, pesticides, plant toxins, and liquid ecstasy (GHB/GBL). Other novel methods include immunochemical screening, congener analysis, breath alcohol measurement, hair analysis, the measurement of alcohol consumption markers, as well as recent scientific findings on substance interactions. New rapid methods are being used in the emergency setting. The questions of quality control and plausibility of analysis have also assumed greater importance.

However, the limited length of the book precluded an in-depth discussion of rarer and more specialized issues, which have therefore been addressed only by way of example. Having said that, meeting the quality standards for forensic medical practice set out in this book can in itself be seen as a major achievement, particularly against the background of considerable differences prevailing not only across the globe but even between European countries. In addition to presenting the basic principles of our specialty, we offer the reader a selection of references for further guidance at the end of each chapter. Given the multitude of publications relating to the individual disciplines within the specialty of forensic medicine, only a limited bibliography is offered. In their selection of references, the authors have sought to ensure that suggested works are current and up-to-date and at the same time cover the broad spectrum of topics in the field. While making no claim to exhaustiveness, primarily contributions to the English-language literature and publications in forensic medical journals have been cited for further reading. The appendix includes additional information, useful tables, and sample documentation. All the information contained in this book, including data given in tables, has been carefully checked. It should be noted, however, that the authors cannot assume responsibility for expert opinions formed on the basis of information contained in this book.

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Contents

Abbreviations

A	Ampere
AAP	American Academy of Pediatrics
ACP	American Academy of Pathology
ADH	Alcohol dehydrogenase
ADH	Anti-diuretic hormone
ADHS	Attention deficit hyperactivity syndrome
ADR	Adverse drug reaction
AIDS	Acquired immunodeficiency syndrome
ALDH	Aldehyde dehydrogenase
ALTE	Apparent life-threatening event
AMIA	Ascent multi-immunoassay
APA	American Psychiatric Association
approx.	Approximately
ARVC	Arrhythmogenic right ventricular cardiomyopathy
ASD	Atrial septal defect
ASL	Above sea level
ATP	Adenosine triphosphate
AV	Adenovirus
AWMF	Association of the Scientific Medical Societies
ß-APP	ß-Amyloid precursor protein
B.A.D.S.	German Association against Alcohol and Drugs in Road Traffic
BAC	Blood alcohol concentration
BMI	Body mass index
bp	Base pairs
BrAC	Breath alcohol concentration
BS	Blood sample
BSA	Body surface area
BVerfG	German Federal Constitutional Court
°C	Celsius
C	Child
C5b-$9_{(m)}$	Monoclonal complement factor C5b-$9_{(m)}$ terminal complement complex
CAD	Cervical acceleration–deceleration
CAT	Committee Against Torture
CBD	Cannabidiol
CBN	Cannabinol
CCT	Cerebral computed tomography

CCT	Craniocerebral trauma
CCTV	Closed-circuit television video
CD	Cluster of differentiation
cDNA	Complementary DNA
CD-ROM	Compact disc–read only memory
CDT	Carbohydrate deficient transferring
CEDIA	Cloned-enzyme donor immunoassay
Chap.	Chapter
CHD	Coronary heart disease
CHL	Crown-heel length
CIF	Cannabis influence factor
CIRS	Critical Incident Reporting System
CK	Creatine kinase
CLSM	Confocal laser scanning microscopy
cm	Centimeter
CMV	Cytomegalovirus
CNS	Central nervous system
CO	Carbon monoxide
CO_2	Carbon dioxide
COC	Cocain
COHb	Carboxyhemoglobin
COPD	Chronic obstructive pulmonary disease
CPT	Committee for the Prevention of Torture
CPTSD	Complex post-traumatic stress disorder
CRL	Crown-rump length
CRS	Cambridge Reference Sequence
croc	Crocodile (Desomorphine)
CT	Computed tomography
CVB	Coxsackie virus group B
Da	Dalton
DAI	Diffuse axonal injury
DCM	Dilated cardiomyopathy
DCMi	Dilated cardiomyopathy – inflammatory type
DD	Differential diagnosis
DDT	Dithiothreitol
DGHS	German Society for Humane Death
DGRM	Deutsche Gesellschaft für Rechtsmedizin
DICOM	Digital Imaging and Communications in Medicine
DIP	Deletion/insertion polymorphism
DNA	Desoxyribonucleic acid
DS	Diffusion drop
DSM	Diagnostic and Statistical Manual of Mental Disorders
DVI	Disaster-Victim Identification
E-605	Parathion
EBV	Epstein-Barr virus
ECG	Electrocardiogram
Ed.	Edition
EDX	Energy dispersive microanalysis

EEG	Electroencephalogram
ELISA	Enzyme-linked immunoassay
EMIT	Enzyme-multiplied immunoassay technique
Epo	Erythropoietin
EtG	Ethylglucuronide
Ets	Ethylsulfate
EU	European Union
EV	Enterovirus
EvG	Elastica van Gieson
FAEE	Fatty acid ethyl esters
FCG	Female genital cutting
FDA	Food and Drug Administration
FGM	Female genital mutilation
Fig.	Figure
Figs.	Figures
FPIA	Fluorescence polarization immunoassay
FWD	Fresh water drowning
g	Gram
GABA	Gamma amino butyric acid
GBL	γ-butyrolactone
GC	Gas chromatography
GC-MS	Gas chromatography/mass spectroscopy
GCS	Glasgow Coma Scale
GenDG	Genetic Diagnostics Act
GGT	γ-glutamyl transferase
GHB	Gamma hydroxybutyric acid
GI	Gastrointestinal
GLORIA	Gold labeled optically read rapid immunoassay
GLP	Good laboratory practice
GP	Gréhant plateau
GTFCh	German Society for Toxicological and Forensic Chemistry
h	Hours
H&E	Hematoxylin-Eosin
Hb	Hemoglobin
HBA1c	Glycated hemoglobin A1c
HCL	Hydrochloride
HCM	Hypertrophic cardiomyopathy
HCN	Hydrogen cyanide
HCV	Hepatitis C virus
HE	Hematoxylin-eosin
HHSV	Human herpes simplex virus
HIV	Human immunodeficiency virus
hpf	High-power field
HPLC	High pressure liquid chromatography
hpm	Hours post mortem
HPV	Human papilloma virus
HV	Hypervariable
HVR	Hypervariable region

Hz	Hertz
ICAM	Intracellular adhesion molecule
ICC	International Criminal Court
ICD	International Classification of Diseases
ICN	Inflicted childhood neurotrauma
IHSS	Idiopathic hypertrophic subaortic stenosis
Il	Interleukin
IPPNW	International Physicians for the Prevention of Nuclear War
ISPID	International Society for the Prevention of Sudden Infant Death
ITBI	Inflicted traumatic brain injury
ITP	Immune thrombocytopenic purpura
J	Joule
KCN	Potassium cyanide
Kg	kilogram
kHz	Kilohertz
km/h	kilometer/hour
kV	Kilovolt
L	Liter
LC	Liquid chromatography
LCA	Leucocyte common antigen
LC-MS	Liquid chromatography/Mass spectroscopy
LFB	Luxol Fast blue
LIA	Luminescence immunoassay
LSD	Lysergide
LVNC	Left ventricular non-compaction cardiomyopathy
m	Meter
M	Mother
m/s	meter/seconds
mA	Milliampere
MCV	Mean corpuscular erythrocyte volume
MDA	Methylendioxyamphetamin
MDE	3,4-Methylenedioxyethylamphetamine
MDEA	3,4-Methylenedioxyethylamphetamine
MDMA	Methylenedioxymethamphetamine
MDPV	Methylenedioxypyrovalerone
MEIA	Microparticle enzyme immunoassay
MEOS	Microsomal ethanol-oxidizing system
mg	milligram
MHC	Major histocompatibility complex
min	Minutes
MIP	Maximum intensity projection
mm	Millimeter
MOF	Multi-organ failure
MRI	Magnetic resonance imaging
MRT	Magnetic resonance tomography
MS	Mass spectroscopy
MSbP	Munchhausen syndrome by proxy
MSCT	Multi-slice CT

mt-DNA	mitochondrial-DNA
NAD	Nicotinamide dinucleotide
NAHI	Non-accidental head injury
NAI	Non-accidental injury
NCCM	Non-compaction cardiomyopathy
NJW	Neue Juristische Wochenschrift
No.	Number
OECD	Organisation for Economic Co-operation and Development
OI	Osteogenesis imperfecta
OPCAT	Optional Protocol to the Convention against Torture
OPI	Opiate
OPSI	Overwhelming postsplenectomy infection
p.m.	Postmortem
PCISME	Primary Care International Study on Medical Errors
PCR	Polymerase chain reaction
PEM	Protein-energy malnutrition
PF	Putative Father
pmi	Postmortem-interval
PSA	Prostate-specific antigen
PSS	Poisoning Severity Score
PTSD	Post-traumatic stress disorder
PVB19	Parvovirus B19
RAD	Reflex anal dilatation
REM	Raster electron microscopy
RIA	Radioimmunoassay
RNA	Ribonucleic acid
rt-PCR	Reverse transcriptase-PCR
s	Seconds
S&W	Smith & Wesson
SAC	Saliva alcohol concentration
SBS	Shaken baby syndrome
SCD	Sudden cardiac death
SDH	Subdural hemorrhage
SEM	scanning electron microscopy
SIDS	Sudden infant death syndrome
SIRS	Systemic inflammatory response syndrome
SNP	Single nucleotide polymorphism
SRF	Serial rib fractures
STA	Systematic toxicological analysis
STD	Sexual transmitted disease
STR	Short tandem repeat
SUDEP	Sudden unexpected death in epilepsy
SWD	Salt water drowning
TBI	Traumatic brain injury
TCA	Tricyclic antidepressant
TDM	Therapeutic drug monitoring
THC	Tetrahydrocannabinol
TIA	Transitoric ischemic attacks

TNF-α	Tumor necrosis factor-α
TURP	Transurethral resection of the prostate
UAC	Urine alcohol concentration
UDHR	Universal Declaration of Human Rights
UK	United Kingdom
UN	United Nations
USA	United States of America
V	Volt
vitr	Vitreous
Vol.	Volume
VSD	Ventricular septal defect
WADA	World Anti Doping Agency
WaffG	Waffengesetz
WFS	Waterhouse-Friderichsen syndrome
WGAD	Working Group on Arbitrary Detention
WHO	World Health Organization
WMA	World Medical Association
WWII	World War II
γ-GT	γ-glutamyl transferase

Introduction 1

From a historical perspective, "forensic"—or "medicolegal"—medicine has grown as a scientific medical specialty primarily to assist police and legal authorities by providing expert appraisals in the fact-finding and adjudication process. In many, although not all countries, forensic medicine is traditionally a university-based discipline. The investigative and appraisal activities inherent to forensic medicine are required far beyond the purposes of criminal prosecution and thus make an important contribution in terms of legal certainty and a functioning rule of law. The tasks of forensic medicine include a broad spectrum of activities.

Firstly, forensic medicine is traditionally concerned foremost with the investigation of sudden and unexpected deaths. The focus here is on determining the mode of death on the basis of the established cause of death. In this context and besides unnatural deaths apparent at first glance, sudden natural deaths as well as a large group of unexplained deaths need to be considered.

Clinical forensic medicine is concerned with the investigation of trauma victims and alleged victims of trauma inflicted by a third party. This group includes victims of various forms of physical injury, including sex offenses, child abuse, and road traffic accidents, as well as special groups of trauma victims such as victims of domestic violence, violence against the elderly, and violence against individuals held in official custody (homes, schools, hospitals, prisons, etc.).

The results of forensic medical investigations (autopsy findings, injury patterns, trace evidence analysis, chemical toxicological analysis, etc.) need to be reported and interpreted in court partly in writing and partly verbally by the forensic pathologist in his or her capacity as an expert witness in criminal proceedings. The forensic medical expert is accountable to all parties involved (the court, the counsel for the defense, the defendant, the public prosecutor's office, and the joint plaintiff; in civil cases, the plaintiff and the defendant) and must assume an impartial and issue-specific stance. In cases where investigations are carried out on behalf of private individuals, e.g., the relatives of a deceased person in order to establish the cause of death, these private individuals receive the forensic medical report. In Germany, it is mandatory for the expert's report to remain impartial and issue-specific even when compiled on behalf of a private individual; other countries such as the USA, in contrast, permit "partisan" expert reports.

Important: **Forensic medicine is the application of medical knowledge for the scientific investigation of facts and causal relationships**, **as well as the analysis and interpretation thereof in the service of the law in its broadest sense**; **moreover**, **it addresses all legal aspects of the practice of medicine during teaching**, **medical training**, **and specialist training**.

Over the years, there have been significant shifts within this broad definition of the spectrum of forensic medical tasks. Modern forensic medicine continues to deal with all forms of violence against individuals, as well as what has grown to become a multitude of traffic medicine issues,

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such as impaired driving safety due to disease, alcohol, drugs, and medication. Traffic accident traumatology has gained considerably in importance. Forensic DNA analysis has revolutionized the collection of evidence. Forensic pathologists are needed the world over in the event of disasters involving large numbers of dead (wars, civil wars, or natural disasters such as tsunamis). In their capacity as experts, they may be called upon, e.g., by the International Court of Justice in The Hague or to inspect state prisons for the purposes of examining inmates for signs of abuse (torture).

The German Society of Forensic Medicine (*Deutsche Gesellschaft für Rechtsmedizin*, DGRM), founded in 1904 as the German Society of Legal Medicine, is one of the oldest specialist medical societies. Forensic medicine has been recognized by medical associations as a specialty in the former German Democratic Republic since 1956 and in the Federal Republic of Germany since 1976. Specialist training comprises 3.5–4 years in a forensic medical institute, 0.5–1 year in pathology, and 0.5 years in psychiatry.

Important: **In contrast to many other clinical specialties**, **efforts to introduce forensic medicine as a speciality on a European level have hitherto failed**. **Nevertheless**, **it is imperative that this endeavor be pursued and intensified**.

Expert medical appraisals may be requested by the authorities or by private individuals for the purposes of protecting rights in civil, social, and criminal inquiries, as well as in administrative law. Claims made by patients, victims of criminal violence, as well as parties aggrieved due to other forms of injury, such as occupational accidents, sometimes need to be substantiated and verified in an expert appraisal. Forensic medicine thereby plays an expert advisory role at the interface between medicine and the law. Although this is predominantly the case in criminal proceedings, forensic medicine's remit can extend to all fields of law. While on the one hand the requirements made of an expert appraiser may vary depending on the field of law, advances in forensic medical methods, diagnosis, and analysis on the other always need to be taken into account when formulating an appraisal opinion. Only in this way can the findings and results of an expert appraisal be adequately recognized in terms of their validity as evidence according to current medical knowledge. Thus, the spectrum of forensic medical activities is extensive.

Important: **A sound forensic medical knowledge** (**forensic medicine and medical law**) **is essential for any physician wishing to practice this specialty**.

The practicing physician needs to recognize which legal environment he or she is working in. In addition to knowing the rights and duties of a licensed physician (professional code of medical conduct), he or she needs to be aware of the particular risks inherent to failing to observe, e.g., a physician's duty to inform or duty of documentation.

A basic knowledge of documentation is essential for medical findings to meet the minimum requirements of legal proceedings. Since no country is able to guarantee forensic medical services on a nationwide scale, it is necessary to have recourse to medical documentation compiled at the time of a patient's/victim's admission to an emergency trauma department in, e.g., many cases of criminal prosecution for bodily harm.

External postmortem examinations can and in many countries must be carried out by all physicians. It is the duty of forensic medicine—both during medical studies and in the context of advanced training in routine practice—to impart the basic knowledge required for this, in particular the skills needed to recognize signs of unnatural death.

Applying medical expertise for the purposes of crime clearance, decision-making, establishing the truth, and ensuring legal peace, as well as for the legislature (in an advisory capacity), the executive authorities (in judicial inquiries), and the judiciary (for adjudication), is a task that traditionally falls to forensic medicine.

The range of expert knowledge called upon by police and judicial bodies, as well as other authorities (youth welfare services, care home inspectorates, asylum authorities, statutory and private insurance institutions, driving license

authorities, highway authorities, etc.) and private individuals, is so extensive that no one expert can reasonably be expected to have expert qualifications spanning the entire field. As a result, fields of specialization have evolved within the specialty of forensic medicine:

- *Forensic Medicine and Pathology*. This field includes the traditional tasks of carrying out external postmortem examinations (Chap. 2), assessing postmortem changes (Chap. 3), and performing autopsies (Chap. 4) for the purposes of, among others, establishing cause of death on the basis of pathological anatomical findings and determining the postmortem interval in, e.g., cases of homicide. Post-exhumation findings (Chap. 5), issues relating to identification (Chap. 6), as well as the issue common to all cases of trauma resulting in death—differentiating between ante- and postmortem injuries, i.e., wound vitality (Chap. 7)—need to be considered separately. Forensic pathologists are called by the investigating authorities to the scene of body discovery to perform a preliminary examination of the body for injuries and to classify these findings in terms of the overall circumstances (position of the body, distribution of livor mortis, time of death estimation, and identification issues). A postmortem examination (forensic autopsy) may be ordered for the purposes of establishing cause of death, mode of death, or identity. Where no indications of either a natural death or disease symptoms are found, an unnatural death may be suspected purely on the grounds of the acuteness of death. In addition to autopsies called for by the police or courts, forensic pathologists (or, depending on the legal framework of the respective country, specially trained physicians) are tasked with performing a second official external postmortem examination (e.g., prior to cremation). This procedure involves reexamining a body and verifying the plausibility of the information given on cause and mode of death in the death certificate. It thereby serves to ensure legal certainty, given that there are always isolated cases of unnatural death in which the first certifying physician has incorrectly classified death to be natural.
- *Forensic (General) Traumatology and Forensic Neurotraumatology*. Forensic traumatology deals with the typical traumatological and biomechanical sequelae of trauma (relevant to an expert appraisal) in living and deceased individuals, most notably as a result of blunt force trauma (Chap. 8), sharp force trauma (Chap. 9), gunshot and blast injuries (Chap. 10), particular forms of trauma such as neck trauma (Chap. 11), thermal injury (Chap. 12), as well as the effects of electricity, lightning, and gases (Chap. 13). To these can be added the various forms of death due to asphyxia (Chap. 14), water-related deaths (Chap. 15), and deaths resulting from starvation and dehydration (Chap. 16). In addition to road traffic accidents, it is concerned with all forms of trauma to the human body incurred in the context of criminal violence, accidents, and suicides. From the perspective of neurotraumatology, injuries of varying intensity following craniocerebral trauma (including shaken baby syndrome in infants and small children), as well as injuries to the spine and spinal cord are of particular relevance.
- *Clinical Forensic Medicine*. Clinical forensic medicine deals with the examination of living individuals for the purposes of collecting evidence and documenting injuries (Chap. 17). The majority of examinations are carried out on victims of trauma [including child abuse (Chap. 18) and child sexual abuse (Chap. 19)] resulting from domestic violence, trauma occurring in association with alcohol and drugs, as well as trauma to particular groups of individuals. It is important to remember that cases of simulated trauma are not uncommon, sometimes involving discernible signs of self-harm. Not only victims of trauma but also the accused undergo forensic examination in the process of establishing the type and severity of injury and for the purposes of reconstructing an incident. Commissioning agents besides the police often include youth welfare services, (adult) trauma victims who reserve the right to take legal steps at some later point, as well as other (official) bodies or

institutions. Causing death in neonates, infants, and children is dealt with in a separate chapter (Chap. 20).

- *(Forensic) Road Traffic Medicine*. Expert appraisals on driving safety not only take into consideration the immediate impairments to driving fitness caused by alcohol, drugs, medication, or other toxins but also relate to long-term fitness to drive in the setting of a number of diseases (cardiovascular diseases, epilepsy, metabolic diseases such as diabetes, etc.) or against the background of, for example, narcotic substitution therapy (methadone). In road traffic accident victims, polytrauma injury patterns typical of road accidents sometimes need to be established in order to reconstruct the accident: velocity of impact, sites of impact, roles played by those involved in the accident (pedestrians, cyclists, drivers, and passengers) as evidenced, for example, by safety belt marks or rollover injuries. It is also important to detect or exclude preexisting disease as a possible cause of an accident (Chap. 21).
- *Forensic Molecular Genetics*. This field of forensic medicine is tasked with identifying genetic fingerprints in trace evidence collected from crime scenes for the purposes of comparing it with the DNA of individuals implicated in a crime, most notably suspects (Chap. 22). Molecular genetic testing for identification purposes is carried out in the DNA laboratories of forensic medical institutes; short tandem repeat (STR) analysis has become the method of choice in this regard. DNA is obtained from a wide range of biological trace evidence. Besides its criminological applications, this method is also used to determine paternity (paternity testing). Saliva samples are taken from suspects or parties involved in paternity testing (mother, child, and putative father). STR profiles obtained from trace material are compared with those of the individuals in question. Furthermore, comparative analyses using, for example, police databases can be performed. Continual improvements to the method have made it possible to analyze tiny volumes of DNA from what formerly represented challenging forms of trace evidence, such as single cells or telogenic hairs.
- *Forensic Paternity Testing*. Although serology was the method of choice up until recently, paternity testing today falls within the remit of forensic molecular genetics. The objective here is to compare DNA for the purposes of determining paternity, sometimes even maternity, on behalf of, e.g., a family court. The admissibility of "private paternity tests" is controversial, for instance when carried out solely on behalf of the putative father without the mother's consent. Investigations of this kind are often not recognized by the courts, while forensic institutes regularly refuse to carry out secret paternity testing and in some countries, e.g., Germany, test results are not admissible in court.
- *Forensic Osteology*. Insofar as osteological diagnosis is possible, forensic osteology is concerned with the classification of bone and skeletal findings in terms of human specificity, age, sex, stature, origin, postmortem interval, preexisting diseases, and injuries (Chap. 23). Specialist knowledge and methods are required in cases where, due to postmortem changes, the human remains to be analyzed consist (almost) only of bone tissue. The first question to be answered on finding bones is whether or not they are of human origin. Where this is the case, the next important question relates to postmortem interval—historical bones are of no relevance to the investigating authorities. As a general rule, the time limit is set at a postmortem interval in excess of 50 years. Determining the postmortem interval more precisely may provide early indications as to identity. Additional information is gained by constructing a biological profile: using a variety of methods and depending on the bone remains available and their condition, it is possible to estimate age, sex, stature, and ancestry. It is also important to identify fresh or healed evidence of bone injury, which may in turn afford further insight into identity and cause of death.
- *Forensic Radiology*. This particular field of forensic medicine is concerned with the use of radiological diagnosis to answer forensically relevant questions relating, e.g., to injuries,

age estimation, and cause of death determination (Chap. 24). Ever since the discovery of X-rays, postmortem X-ray diagnosis has played an important role in the documentation of primarily bone injuries as well as in autopsy planning, e.g., in order to localize projectiles. Moreover, the number of postmortem CT and MRI scans has increased since the turn of the millennium, making "virtual autopsies" a reality by using three-dimensional data sets. However, numerous studies have dispelled initial beliefs that the virtual autopsy would replace the "real" autopsy. Nevertheless, combining virtual and real autopsies produces diagnostically conclusive findings. Comparative X-ray analysis is an old and effective method of determining identity. In cases where antemortem X-ray images of a missing person are available, these can be compared with postmortem images made of the region in question on an unidentified body.

In clinical forensic medicine, X-rays are often included in the expert appraisal of fresh or healed bone fractures, while CT or MRI data sets are used increasingly in the assessment of soft tissue injuries. Radiological examinations form the principal basis of age determination in living individuals.

- *Forensic Determination of Cause of Death in Natural Deaths*. The forensic determination of cause of death relates primarily to deaths involving, e.g., suspicious circumstances in which a body is found or any other circumstances that may require the authorities to determine the cause of death. In such cases, the spectrum of diseases causally linked to (sudden) death is broad: myocardial infarct, myocarditis, coronary atherosclerosis with acute heart failure, ruptured aneurysm, pneumonia, meningitis, etc. Death is also often caused by relatively rare diseases which, due to acute death and failure of the patient to seek medical advice, remained undiagnosed during the individual's lifetime (Chap. 25).
- *Forensic Medical Examination of Torture Victims*. The central focus here is on identifying injuries and injury sequelae, with forensic medicine making its contribution in terms of differentiating between fresh and old injuries, scars, and (healed) fractures. As such, forensic medicine is seen as one part of the broader diagnostic work-up and treatment of torture victims. Regular spot inspections of, e.g., state prisons, including the examination of inmates, can have a preventive effect, assuming that medical examinations are carried out by impartial and independent (forensic) physicians. A sound knowledge of torture methods and the injuries they produce is a prerequisite in this field (Chap. 26).
- *Forensic Psychopathology*. Although legislation may differ from country to country, acquiring a basic knowledge of psychiatry forms a mandatory part of the forensic pathologist's specialist medical training. A major focus in this field is the expert appraisal of the effects psychotropic substances (alcohol, drugs, medication, as well as other toxins) have on an individual's capacity for insight into their wrongdoing at the time of an offense and/or their ability to act on this insight, i.e., the question of criminal responsibility (Chap. 27). The objective here is to facilitate the court's decision in terms of enforcing a penalty that is appropriate to the crime or to ensure that the existence of significantly impaired criminal responsibility is taken into account and reflected in a milder punishment. In addition to determining the presence of an impairing substance, any expert appraisal of criminal responsibility—which can often only be carried out retrospectively—needs to evaluate an individual's behavior "before, during, and after" the offense. In specific cases, a psychiatric expert with forensic experience may be required in the case of, e.g., severe neuroses and personality disorders or for the purposes of assessing the risk posed by convicted (sex) offenders pending their release from prison. Occasionally, expert assessments may be needed in order to establish an individual's fitness to undergo questioning, be held in custody, serve a custodial sentence, stand trial, or make a will. Expert assessments on credibility or witness psychology require particular expertise and experience.

- *Assessing Medical Malpractice Claims*. Subject to significant national variations, forensic medical experts are called upon to formulate expert appraisals on causality relating to a possible treatment error (Chap. 28). Thus, an expert forensic medical report of this kind must address the issue of causality between a specific treatment error and existing damage or death. Expert reports such as these are often called for as a result of claims brought by relatives or due to information given on the death certificate by the certifying physician that may arouse suspicion of unnatural death due to a treatment error. Examples of claims include insufficient decubitus ulcer prophylaxis, insufficient venous thromboembolism prophylaxis, undetected myocardial infarction, delayed identification of postoperative peritonitis, foreign objects retained in the surgical area, as well as medication errors, among others. In the case of a patient's death, the autopsy examination and subsequent analysis of, e.g., medical records form the basis of the expert forensic report. In the rarer cases of medical malpractice claims in living patients, the forensic pathologist is often the first port of call for the legal authorities, whose main questions include: "is there any indication of a treatment error?" and "to which speciality does the treatment error, and hence the examination of the patient, belong?" The forensic pathologist is often asked to nominate a medical expert witness.
- *Forensic Alcohology*. Measuring blood alcohol concentrations by determining alcohol levels in blood, urine, and organ or tissue specimens makes the most important contribution to determining an individual's fitness to drive, as well as to the expert assessment of their (criminal) responsibility (Chap. 29). In quantitative terms, ethyl alcohol (ethanol) plays the leading role among noxae that impair psychophysical fitness to drive. Evaluating the effects of alcohol and validating the methods used to analyze congener alcohols (fusel alcohol) have long formed an integral part of forensic medical alcohol analysis. The results of forensic medical research have helped to shape legislation and jurisdiction on limit values and alcohol-related unfitness to drive, while claims relating to alcohol volumes and types of alcohol can be verified by means of congener analysis.
- *Forensic Toxicology*. Forensic toxicology is concerned with the analytical determination of organic and anorganic toxins, as well as the interpretation of analysis results for the purposes of answering forensically relevant questions, such as cause of death, fitness to drive, and criminal responsibility (Chap. 30). In the case of suspected intoxication, forensic medical institutes are able to analyze specimens obtained at autopsy (e.g., cardiac blood, femoral vein blood, brain tissue, hepatic tissue, liver tissue, gallbladder, cerebrospinal fluid, bone, hair, finger- and toenails), while in the case of living patients, specimens are sent for analysis (clinical toxicology). Depending on national laws, chemical toxicological analyses also need to be carried out as part of brain death diagnosis prior to organ transplantation in order to rule out central nervous impairment. Most investigations involve extensive blood and urine sample analyses in cases of abnormal driver behavior to detect drugs and medication with a central nervous effect (heroin, cocaine, amphetamines, psychotropic drugs, fungal poisons, cannabinoids, inhalants, etc.). However, blood and urine sample analysis is also carried out in trauma victims for the detection of, e.g., knockout drugs in association with a sex offense.

In addition to the fields of forensic medical activity mentioned above, smaller specialist areas of activity that are nevertheless worthy of note in their own right are discussed within chapters, including:

- *Forensic Odontostomatology*. This particular field of activity encompasses dental development, treatment, and injury/disease of the oropharyngeal cavity, teeth, and jaws, insofar as these are required to answer legally relevant questions. The chief tasks here include recording dental status to assist in the identification process and comparing ante- and postmortem dental status to establish identity. Moreover,

dental development in children and dental wear in adults can provide important insights in terms of age estimation. The evaluation of injuries resulting from trauma to facial bones is yet another important area of investigation and also includes dental injuries relevant in the setting of child abuse.

- *General Forensic Histopathology and Forensic Neurohistopathology*. Microscopy plays an important diagnostic role in cause of death determination, trace evidence detection (e.g., sperm), and wound age estimation [including tissue analysis by immunohistochemistry, cytology, and scanning electron microscopy (SEM)]. Within the scope of routine diagnostic procedures, histological analysis of tissue samples often reveals (natural) causes of death, such as pneumonia, myocarditis, amyloidosis, early meningitis, ascending cholangitis, among many other diseases that are difficult, if not impossible, to detect macroscopically. Histology is often indispensible in the forensic assessment of cause of death, on the one hand, in its capacity to confirm and complement diagnoses suspected macroscopically and, on the other, to rule out competing causes of death. Specific findings consistent with the medical history of the deceased (e.g., long-term drug abuse) or with the intake of toxic substances (e.g., ethylene glycol) are often made. Histology is also able to detect numerous infections, such as malaria, echinococcus cysts, or fungal infections.
- *Forensic Anthropology*. Many English-speaking countries equate forensic anthropology with forensic osteology. According to the understanding of biological anthropology in Europe, man's external appearance is as relevant as his bony structure, thereby extending the spectrum of forensic anthropological tasks to the identification of individuals on pictorial documents. This deals with the question, for example, of whether a specific person under suspicion has been captured on film by a security camera. In Germany, identity is often reported following speeding offenses or other road traffic contraventions, making it the object of forensic anthropological expert appraisal.
- *Forensic Entomology*. This field of forensic medicine applies the analysis of insects to the task of determining the postmortem interval of a body. This approach is based on identifying the species of insect from eggs, larvae (maggots), or adult insects found on the body, having a good understanding of the species' course of development, and taking ambient temperatures and their effect on the speed of development into account. Microscopy and DNA analysis are used to identify the species of insect. It is sometimes necessary to observe development to adulthood under laboratory conditions.
- *Forensic Wound Ballistics*. Knowledge of firearms and ballistics is used here to classify (fatal) gunshot wounds; to determine range of fire, type of weapon, and whether or not death was caused by suicidal shooting; as well as to identify unusual wound tracks and gunshot wounds. This field also deals with how different types of projectile behave within the body of the victim after firing (e.g., bullet fragmentation).
- *Forensic Research*. Forensic medical research activities relate to the abovementioned fields using widely varying approaches in terms of method and content: epidemiological studies, case–control studies, clinical forensic studies using various analytical methods (in line with current social developments, such as the emergence of new drugs), basic forensic research, population genetics studies, and microscopic analysis (histology, immunohistochemistry, cytology, etc.). Forensic medical investigations and findings are essential prerequisites to assessing and resolving expert lines of inquiry and affect not only the decision-making process in the higher courts but also the laws set down by the legislator and the public administration.
- *Forensic Teaching*. As one of the largest medical specialties tasked with diagnosis and expert appraisal, the teaching of forensic medicine encompasses all the above mentioned topics. Within medical specialties in most countries, knowledge of medical law is taught only by representatives of forensic medicine

in lectures and seminars that can cover a broad spectrum of topics: medical and public health law (insurance medicine, infection prevention laws, transplantation, transfusion, drug, and autopsy laws, sterilization and castration, and the professional code of medical conduct), duty to inform, duty of documentation, confidentiality, physician liability law, legal issues relating to beginning- and end-of-life care (e.g., preimplantation and antenatal diagnosis, euthanasia), treating minors, refusal of treatment and patient rights, and handling of treatment errors. In most countries, external postmortem medical examinations are carried out by all physicians rather than by trained specialists. Since the external postmortem examination plays an important role in determining whether or not an investigation procedure is opened, it is essential for a physician to recognize signs of trauma and evidence of an unnatural death.

As a multi- and interdisciplinary specialty, forensic medicine can only answer the questions put to it if the scientists active in the field keep abreast of social changes through continuous further development and regular advanced training. This means, for example:

- Improving analysis techniques in forensic medical laboratories as new drugs emerge
- Establishing and applying new analysis methods, e.g., mt-DNA, in order to resolve old (sometimes dating back decades) as well as recent crimes
- Implementing higher standards of routine evidence collection, as in part explicitly specified mainly by the police and judicial authorities, e.g., in relation to the accreditation of forensic laboratories according to fixed standards
- Ensuring that forensic medical appraisals meet the high standards of proof set by the criminal prosecution system. This relates to the qualifications of expert appraisers, the diagnostic and analytical means at their disposal, the means available to them to document objective findings, as well as the extent to which they can carry out literature research when formulating an expert appraisal

Moreover, all expert appraisers active in forensic medical institutes need to be prepared to communicate with the police and legal representatives, a fact which presupposes knowledge of numerous legal norms on the one hand and adherence to the standards of personal integrity required of court-approved experts on the other.

In addition to its activities on behalf of national authorities, forensic medicine traditionally has an international focus, despite the fact that numerous countries do not recognize the forensic pathologist as belonging to an independent medical specialty with defined and protected legal status as an expert. Nevertheless, forensic medical activities on an international level include collaboration in:

- Identification commissions (e.g., of the German Federal Office of Criminal Investigation Police)
- Assignments in crisis zones to solve criminal acts (e.g., mass grave excavations)
- Fulfilling forensic medical tasks in disasters involving mass deaths (e.g., tsunami disasters, identifying thousands of dead)
- Providing expert testimony at the International Criminal Court in The Hague, The Netherlands

It is also often the case that too little attention is paid to the fact that forensic pathologists—subject to official protected status as independent experts—are involved in establishing the truth about violations committed by state bodies. Deaths in police custody or prison are worthy of note in this regard, as are deaths among asylum seekers undergoing deportation and cases of torture committed on detainees by official (or semi-official) bodies. Duties also include the inspection of prisons and detention conditions as well as physical examinations performed on detainees to identify injuries. These last two points need to be considered against the background of guaranteed inalienable human rights, the observance of which requires continuous monitoring.

Given forensic medicine's broad spectrum of activities and its purpose not only in terms of ensuring a functioning state of law and adherence to minimum human rights standards, it is essential for the following requirements to be met:

1. The state must guarantee the forensic medical expert's independent status; this needs to be institutionally embedded in public law and not subject to legal or disciplinary directives, in particular from the criminal prosecution system and/or the judiciary (e.g., the ministry of justice). Disciplinary law for officials needs to be viewed independently; however, responsibility needs to be taken by authorities independent of the criminal prosecution system, such as the ministry of science. Furthermore, the expert's financial autonomy must be ensured in order to preclude any suspicion that a commissioning agent or financially strong defendant could influence the content of an expert report.
2. Forensic medical institutes should be established foremost as university institutes, not least since the freedom to teach and research enjoyed by such institutes underpins their autonomy and provides financing solutions independent of the departments of justice and the interior, thereby also making it more difficult to exert politically motivated influence.
3. While on the one hand the personnel and equipment in forensic medical institutes need to meet research and teaching requirements, they must be configured on the other in such a way that these services can be quantitatively and qualitatively supported by the police, judiciary, and other authorities. It should be pointed out here that the courts demand work at a medical specialist level, which requires an adequate number not only of medical specialists but also of advanced training places in order to ensure specialist medical services. Moreover, financing of the necessary facilities, which range from calibrated cadaver weighing scales to much more costly equipment for toxicological analysis, needs to be regulated.
4. For the above reasons, at both a European and an international level, calls should be made for all countries to introduce a qualified, independent field of forensic medicine as an integral component of the rule of law. This step presupposes the introduction of the "specialist in forensic medicine" or a comparable qualification.
5. Wherever personnel and qualitative requirements have been met, forensic medical knowledge should be disseminated more readily and in a variety of contexts. Examples include incorporating forensic medicine in the education and training of physicians, police, and state prosecutors (at least with regard to capital offenses, criminal road traffic offenses, and medical malpractice claims), child service workers, social workers, family midwives, juvenile court judges, and family court judges—most notably on the issues of "child abuse" and "domestic violence." Furthermore, forensic medical knowledge on "medical malpractice" and "medical malpractice prevention" should be incorporated in advanced training as a way of promoting patient safety.
6. In teaching, calls should be made for content on forensic medicine—beyond what is currently offered—to be included in training catalogues for medical students, as well as in the training of prospective members of the legal profession, possibly also biologists (forensic anthropology and forensic histopathology) and dentists (forensic odontostomatology). In addition to this, more time should be devoted during student instruction to issues relating to medical law.

Points 5 and 6 presuppose allocation of the necessary personnel resources, which leads to the purely political question: how much does a state want to spend on forensic medicine in order to finance the abovementioned tasks? In this context, caution is advised against the false belief that transferring investigation assignments to private suppliers permanently guarantees high-quality forensic medical services. Indeed, the opposite is true, since fragmenting investigation tasks or dividing them up between multiple institutes and people precludes in the medium term the possibility of a specialist-level expert appraisal geared to the requirements of a court from being formulated on the basis of a meaningful bringing together of findings in one institute. A significant level of organization is already required when, e.g., the autopsy is performed at one institute, the analysis of blood collected

ante-mortem is carried out by a private company, trace evidence from the crime scene is analyzed by the police, and further investigations are handed out in an uncoordinated manner in various directions. Quite apart from the considerable loss of time, the administrative and financial burden associated with this fragmented approach is ultimately greater than if all investigations were concentrated in one forensic medical institute.

A further aspect of forensic medicine is the potential influence it could have on statistics. One is tempted to conclude here that it is not worth investing in forensic medicine. This is best illustrated using the forensic investigation of death as an example: if more forensic pathologists were called to death scenes or if the autopsy rate increased, one can assume that otherwise undetected homicides would come to light. These homicides, however, are comparatively difficult to solve. If, on the other hand, a husband phones the police with a knife still in his hand, confesses to having just stabbed his wife, and gives himself up for arrest without resistance, this represents a clear case of homicide with a 100 % crime clearance rate. Intensifying forensic medical activities in death investigations would be reflected in criminal statistics in that the number of homicides would rise, while the crime clearance rate would fall. This kind of statistic could have a negative impact on the population's subjective sense of safety.

The importance of the forensic medical expert's independent status becomes all the more apparent in the context of violations committed by state authorities (police, secret service, military personnel, etc.) against detainees (claims relating to prohibited interrogation methods or torture). An objective, neutral, and fact-based expert appraisal aimed at establishing the truth is only possible if the forensic medical expert has protected status as well as personal and material autonomy. However, not all official organs around the world guarantee this status; indeed, one is more inclined to assume that the prevailing relationships of dependency permit (forensic) medical specialists to be put under pressure to submit the desired appraisal. It is for precisely this reason that the status of the forensic medical expert should be embedded in and protected by law. This step would achieve the following:

1. The forensic medical expert would not be bound by instructions from investigating bodies (police, judiciary, or other official organs).
2. Expert appraisals could, at the forensic pathologist's discretion, be compiled by one or more forensic medical specialists (of his/her choice).
3. Claims of partiality made against a forensic medical expert would need to be presented and substantiated in public proceedings. Only a court that is not involved in the proceedings in question would be qualified to judge claims of partiality.
4. The lawmaker would specify the grounds deemed sufficient to determine partiality in a forensic medical expert.
5. Forensic medical experts could decide autonomously, prior to submitting their expert testimony, which questions they wish to pose to the parties involved in the proceedings (defendant, witnesses); they would also be permitted to present the court with unsolicited facts and interpretations insofar as these, in their opinion, were of relevance to the proceedings.
6. All forensic medical experts would be entitled in specific cases to call upon external expertise, even beyond national borders. This would include entrusting external colleagues with documentation and information as deemed necessary by the forensic medical expert responsible.
7. All forensic medical experts should be provided with the means to communicate in a professional capacity with colleagues both nationally and internationally (including via the Internet) and should be granted access to the national and international literature required to compile an expert report.
8. Participation in state measures of repression and prohibited interrogation methods (e.g., torture) would not be reconcilable with medical ethics or with the position of an independent, neutral, and objective forensic medical expert.

Forensic medical experts exposed to measures of repression due to their "undesirable" expert reporting in their own countries must be granted asylum in a neutral, third country along with their

families. The observations and recommendations made here are based on an awareness that by no means all forensic medical experts worldwide maintain the necessary distance from and impartiality to state authority.

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The External Postmortem Examination

2

Case Study

A schoolboy found the lifeless body of an elderly woman lying on the pavement beside a busy main road in a built-up area in the early hours of the morning. The emergency physician who was summoned by mobile phone pronounced the victim dead but was forced to leave immediately thereafter to attend another emergency. Having arrived at the scene, the police requested an internist, whose medical practice was located in the immediate vicinity, to undertake the external postmortem examination and issue a death certificate. The internist stated that the woman was not known to him, that he was unable to undress and examine the body in public, and that it would be preferable to locate the deceased's family practitioner and allow him or her to perform the external postmortem examination. When asked, the internist also declined to perform the examination in his practice with the argument that his patients were waiting for him. Meanwhile, the police officers were able to identify the woman from an identity card they found in her coat pocket; however, they were forced to conclude that she was visiting an unknown person. They suggested to the internist that the body be transported to an undertaker's where it could then be examined. Mindful of his waiting patients, the internist proposed performing the external postmortem examination in the early evening—that being the earliest opportunity he would have for the examination.

Legislation governing the performance of external postmortem examinations varies from country to country. However, in many countries, an external postmortem examination may only be carried out by a physician, subsequent to which a death certificate should be issued. The structure of death certificates has changed somewhat over the years (Fig. 2.1). The purpose of the (medical) external postmortem examination includes the following:

Important: **It is mandatory for all bodies to be (medically) examined in order to pronounce death and establish time of death, mode of death, and cause of death. Findings, including medical findings, should be recorded on the death certificate. Depending on the legal framework, on-duty emergency physicians can limit their tasks to pronouncing death and filling out a provisional death certificate. The World Health Organization (WHO) already proposed an international death certificate form many years ago (Fig. 2.2) as a complement to national death certificates.**

In the absence of clear signs of death, resuscitation efforts need to be undertaken. Failure to achieve cardiac resuscitation is considered the criterion for unsuccessful resuscitation. In

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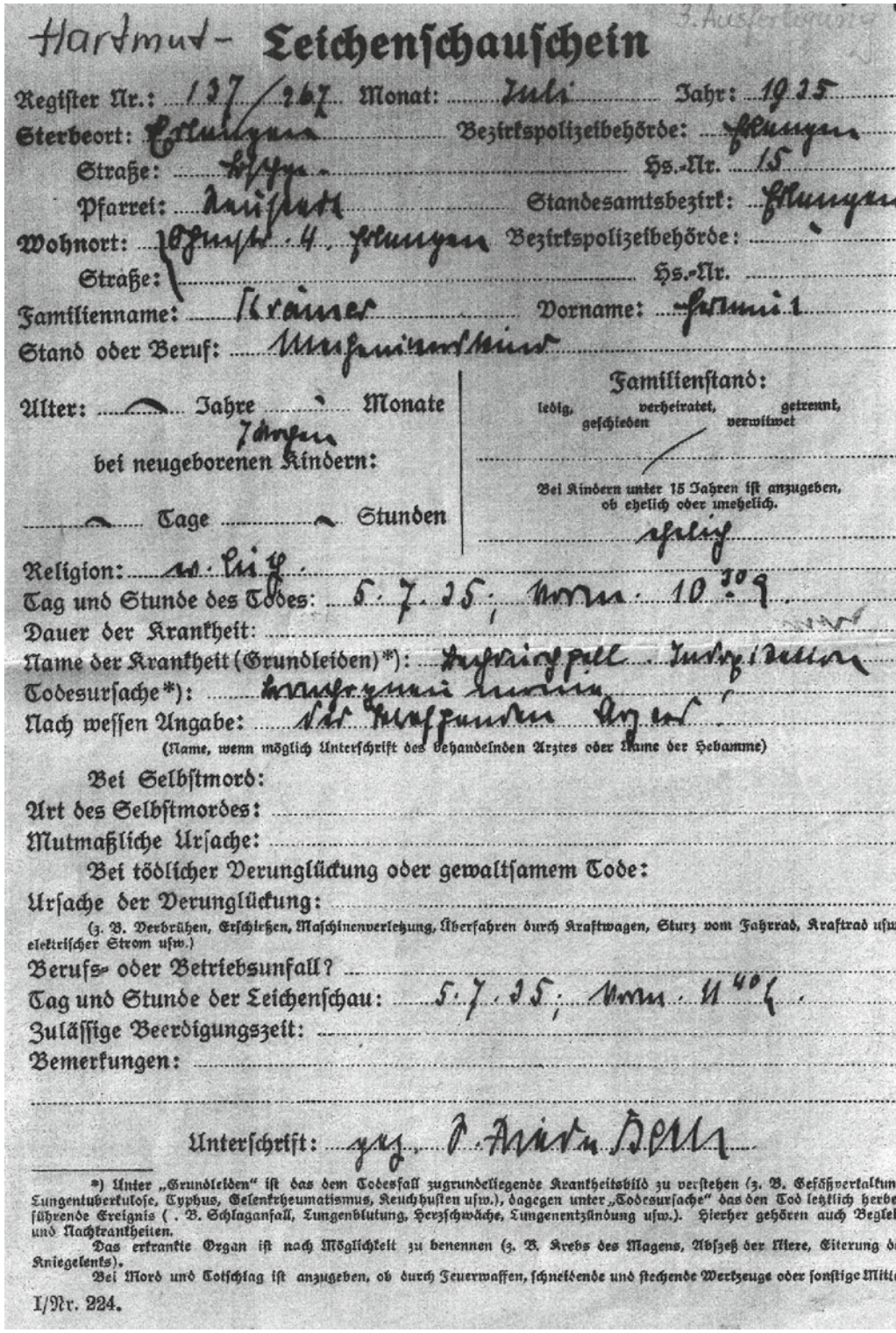


Hartmut- **Leichenschauschein**

Register Nr.: 127/267 Monat: Juli Jahr: 1935
Sterbeort: Erlangen Bezirkspolizeibehörde: Erlangen
Straße: Bismarck Hs.-Nr. 15
Pfarrei: Lutherkirche Standesamtsbezirk: Erlangen
Wohnort: Nürnberg 4, Erlangen Bezirkspolizeibehörde:
Straße: Hs.-Nr.
Familienname: Kraemer Vorname: Hermann
Stand oder Beruf: Maschinenschlosser

Alter: Jahre Monate
7 Wochen
bei neugeborenen Kindern:
Tage Stunden

Familienstand:
ledig, verheiratet, getrennt, geschieden, verwitwet
Bei Kindern unter 15 Jahren ist anzugeben, ob ehelich oder unehelich.
ehelich

Religion: ev. Luth.
Tag und Stunde des Todes: 5. 7. 35; vorm. 10 30
Dauer der Krankheit:
Name der Krankheit (Grundleiden)*): Kreislaufschwäche Herzschwäche
Todesursache*): Kreislaufschwäche Herzschwäche
Nach wessen Angabe: der Hausarzt Dr. [illegible]
(Name, wenn möglich Unterschrift des behandelnden Arztes oder Name der Hebamme)

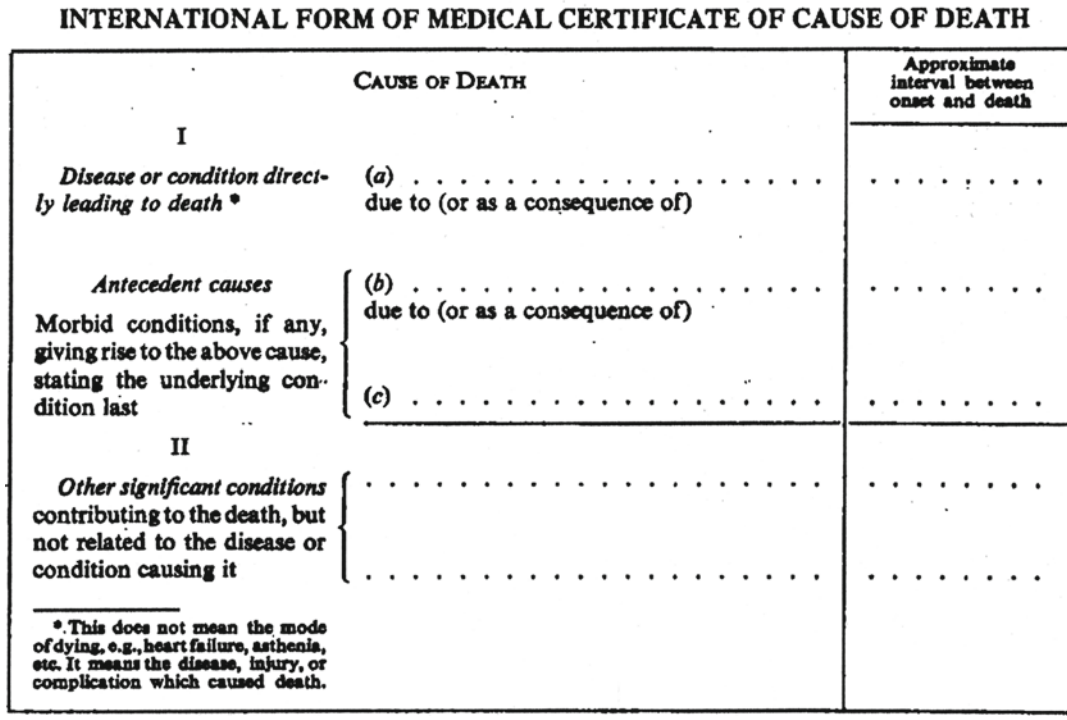
Bei Selbstmord:
Art des Selbstmordes:
Mutmaßliche Ursache:
Bei tödlicher Verunglückung oder gewaltsamem Tode:
Ursache der Verunglückung:
(z. B. Verbrühen, Erschießen, Maschinenverletzung, Überfahren durch Kraftwagen, Sturz vom Fahrrad, Kraftrad usw., elektrischer Strom usw.)
Berufs- oder Betriebsunfall?
Tag und Stunde der Leichenschau: 5. 7. 35; vorm. 11 40
Zulässige Beerdigungszeit:
Bemerkungen:

Unterschrift: gez. Dr. Arzt Stadt

*) Unter „Grundleiden" ist das dem Todesfall zugrundeliegende Krankheitsbild zu verstehen (z. B. Gefäßverkalkung, Lungentuberkulose, Typhus, Gelenkrheumatismus, Keuchhusten usw.), dagegen unter „Todesursache" das den Tod letztlich herbeiführende Ereignis (z. B. Schlaganfall, Lungenblutung, Herzschwäche, Lungenentzündung usw.). Hierher gehören auch Begleit- und Nachkrankheiten.
Das erkrankte Organ ist nach Möglichkeit zu benennen (z. B. Krebs des Magens, Abszeß der Niere, Eiterung des Kniegelenks).
Bei Mord und Totschlag ist anzugeben, ob durch Feuerwaffen, schneidende und stechende Werkzeuge oder sonstige Mittel.

I/Nr. 224.

Fig. 2.1 German death certificate dating back to 1935



INTERNATIONAL FORM OF MEDICAL CERTIFICATE OF CAUSE OF DEATH

	Cause of Death	Approximate interval between onset and death
I		
Disease or condition directly leading to death *	(*a*) . . . . . due to (or as a consequence of)	. . . . .
Antecedent causes Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last	(*b*) . . . . . due to (or as a consequence of)	. . . . .
	(*c*)	. . . . .
II		
Other significant conditions contributing to the death, but not related to the disease or condition causing it	. . . . .	. . . . .
	. . . . .	. . . . .

*This does not mean the mode of dying, e.g., heart failure, asthenia, etc. It means the disease, injury, or complication which caused death.

Fig. 2.2 World Health Organization (WHO) international death certificate

the resuscitation setting, death is pronounced if no spontaneous respiration or spontaneous cardiac activity is seen after 20 min of uninterrupted resuscitation and if irreversible cardiac arrest is proven on the basis of asystole in ECG for a prolonged period of time. This does not apply in cases of general hypothermia, intoxication, and near drowning, where longer resuscitation times are required. Recent studies show, however, that survival chances can be improved by longer resuscitation times; the median resuscitation time in a large study was 35 min, with significant variations. Older (>70 years) long-term survivors who had undergone resuscitation for longer than 60 min were also seen in the study.

Resuscitation Injuries. Alongside chest compression as part of resuscitation efforts, other measures may include aspiration, intubation, the placing of a gastric probe, possibly also a thoracic drain, and coniotomy, among others. Prolonged and intensive resuscitation can cause injury, including:

- Rib fractures, most notably the second to seventh ribs, generally with a left-sided preponderance.
- Bilateral rib fractures are seen in approximately 50 % of cases.
- The 8th–12th ribs are usually spared from fracture in the course of resuscitation attempts.
- Transverse fractures of the sternum are caused more commonly by resuscitation than by any other trauma.
- Peristernal bleeding in the absence of fractures is seen in the resuscitation setting.
- Rarer resuscitation-related injuries: Ruptured aorta (more commonly the descending aorta) and hematothorax.
- Subpericardial and intramyocardial hemorrhage can occur due to resuscitation.
- Injury to the liver and spleen is possible.
- Very rare: Ruptured diaphragm or gastric wall (resuscitation-related rupture of the gastric wall due to high-pressure ventilation usually affects the lesser curvature).

Resuscitation-related injuries are rarely so severe that they need to be considered as a possible competing cause of death.

The Certifying Physician. Where possible, a deceased's general practitioner is the physician most suited to performing an external postmortem examination, not least since he or she is aware of the patient's history, underlying diseases, and clinical symptoms, as well as the circumstances of death in many cases. However, unnatural deaths occasionally go unrecognized as such at external postmortem examinations performed in particular by community-based physicians. Insufficient thoroughness during the examination is the main reason for this failing. On the other hand, many unnatural deaths or homicides cannot be detected at external examination alone. An autopsy, as well as possible forensic toxicological analyses, is needed to establish an unnatural death. Thus, the low autopsy rate goes a long way to explain unrecognized homicides and unnatural deaths. Where an emergency physician has been called, he or she is essentially authorized to perform an external examination following unsuccessful resuscitation. However, there are a number of (legal) regulations, whereby emergency physicians are not obliged to carry out an external examination if they are called to another emergency. The family practitioner who is then often called upon is able to decide whether performing the external examination would constitute a conflict of interests. In such cases, it is advisable for the next of kin to seek a neutral physician to perform the external examination. Only in rare cases do the next of kin actually have a legal entitlement to decline the deceased's treating physician as the certifying physician.

2.1 Tasks and Duties of Care at Medical External Examination

The requirements given in Table 2.1 highlight the need for care and diligence at external postmortem examination.

A distinction is made between early postmortem changes (livor mortis, rigor mortis, and algor mortis), late postmortem changes (autolysis, putrefaction, and insect and animal predation), and conserving processes (mummification and adipocere formation) for the purposes of estimating time of death. In specific cases, checking supravital functions (see Chap. 3) may be helpful, alongside other information or indications, e.g., witness statements, newspapers in the mailbox, last telephone call, last time seen alive, or the condition of food remains.

Caution is advised when entering the time of death; restricting oneself to giving approximate

Table 2.1 The tasks of the certifying physician at external postmortem examination

Task	Relevance
Pronouncing death	In the interests of the individual In the interests of society in terms of a reliable pronouncement of death and its legal consequences (e.g., inheritance issues, insurance policy deadlines) Legal certification of death by the authorities
Determining identity	At the same time, the examining physician certifies the identity of the deceased
Determining cause of death	Information on the medical cause of death can be included in cause-of-death statistics (malignancies, cardiovascular disease, etc.) Epidemiological data on causes of death can be obtained Information on the cause of death can influence health policy decision-making on, for example, the distribution of healthcare resources The spectrum of detected medical causes of death can improve quality assurance of patient treatment
Determining time of death	Relevant for official records, possibly also for inheritance issues if the order of death is relevant to multiple legal heirs
Determining mode of death	Natural Unnatural Unknown With the relative consequences in terms of how to proceed (burial, cremation, notifying the police authorities, etc.)
Detecting contagious diseases	Infectious diseases need to be detected and reported in the interests of the public

Table 2.1 (continued)

Task	Relevance
Obligation to notify the authorities	An unknown mode of death, unknown identity of the deceased, and signs of an unnatural death should prompt notification of the police (possibly by the emergency physician or later by the pathologist) Although rare, an occupational disease may be suspected at external examination; this suspicion needs to be communicated to the authorities depending on the legal framework. (Caution: a road traffic accident may need to be reported as a commuting accident according to insurance law!) Some countries have special lists of recognized occupational diseases!

Table 2.2 Orienting data for time of death estimations (hpm, hours post-mortem)

Livor mortis	
Confluent livor mortis	ca. 1–2 hpm
Fully developed livor mortis	ca. 6–8 hpm
Livor mortis blanches under (thumb) pressure	up to ca. 20 hpm
Partial blanching on sharp-edged pressure	up to ca. 36 hpm
Livor mortis may be completely displaced	up to ca. 6 hpm
Livor mortis may be partially displaced	ca. 6–12 hpm
Rigor mortis[a]	
Rigor mortis begins (jaw)	2–4 hpm
Fully developed rigor mortis	ca. 6–8 hpm
Renewed-onset rigor mortis after breaking	up to ca. 8 hpm
Rigor mortis resolution	after 2–4 days (strongly temperature- dependent)

[a]The extent of rigor mortis is always estimated by inspecting the large and small joints

times is recommended. In some cases, a time period of death can be given. The data shown in Table 2.2 apply to the estimation of time of death.

Livor mortis needs to be inspected for intensity, extent, color, localization (consistent with the position of the body?), contour marks, and capacity to shift or blanch. Other criteria used to estimate time of death include the Zsako muscle phenomenon (idiomuscular contraction) and measuring core body temperature (deep rectal temperature) (see Chap. 3).

2.2 Legal Considerations Relating to External Examinations

As a rule, it is the duty of the next of kin to arrange for an external postmortem examination to be performed; depending on national legislation, the examination should be carried out promptly by any doctor. A thorough external examination—preferably at the scene of death if possible—includes determining death, time of death, cause of death, mode of death, and the identity of the deceased; a final death certificate is issued only once the examination has been completed. The flowchart in Fig. 2.3 shows the complex tasks involved in a medical external examination and their chronological sequence.

In addition to the mandatory external examination, some legal systems make provision in all cases of death for the option to perform an inquest when explicitly ordered by a court or, in urgent cases, by the public prosecutor's office or police.

2.2.1 The Concept of the "Dead Body" and Ordering an External Postmortem Examination

The legislator sometimes provides definitions of the concept of the "dead body," including the following:

- The body of a deceased individual on condition that tissue integrity has not been compromised by decomposition (skeletons and skeletal fragments are not dead bodies!)
- A body part without which continued life is unviable
- A stillborn child weighing >500 g (in Germany; different weights are specified in other countries)

From a conceptual point of view, a distinction is made between a spontaneous abortion, a stillbirth, and a neonate/live birth when, for example, body weight is taken into account and depending on the legal framework:

Spontaneous abortion: A dead fetus weighing <500 g and showing no signs of having lived outside the uterus

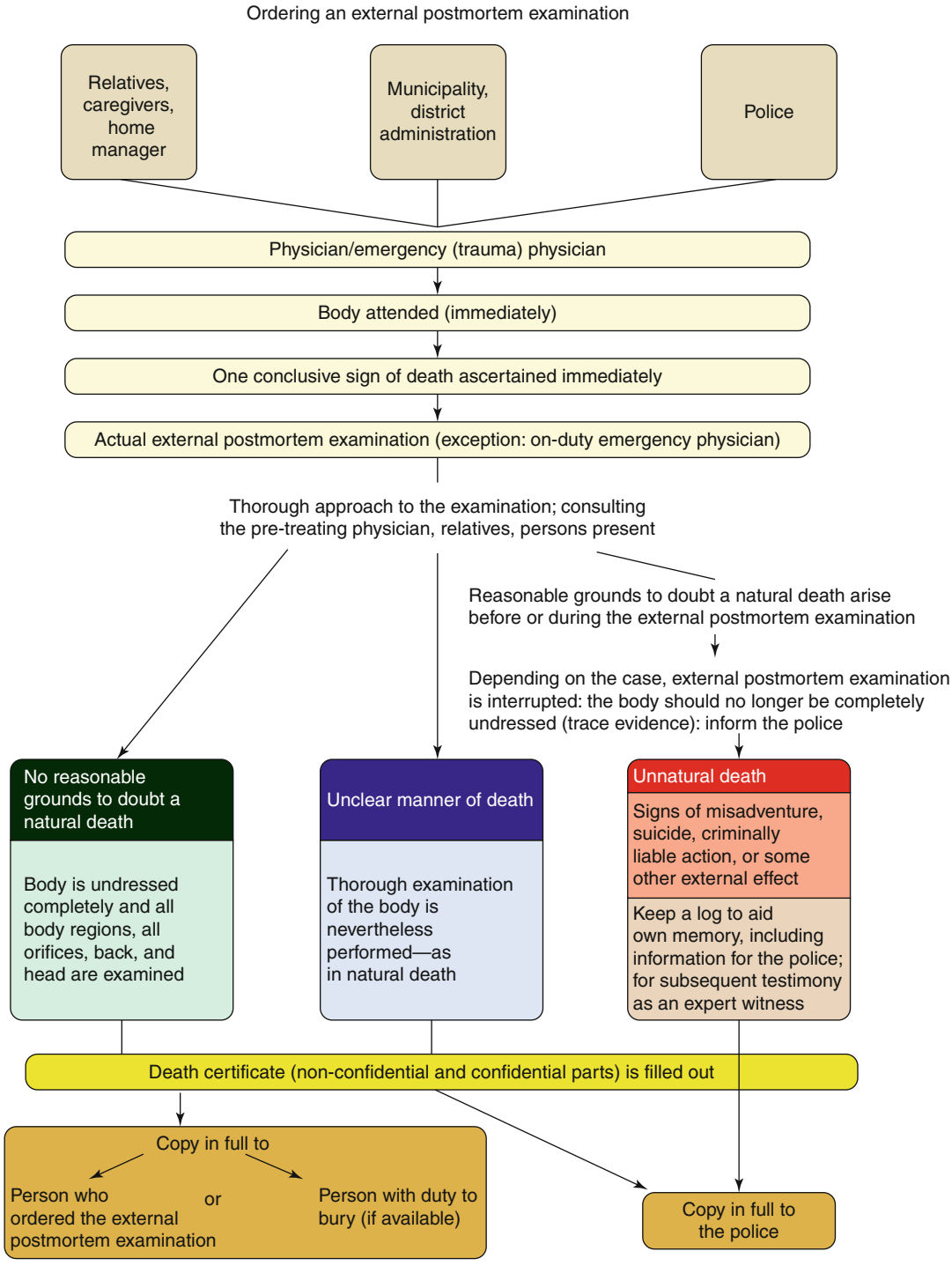


Fig. 2.3 Flowchart for performing a medical external examination (Modified from Hof (2001))

Table 2.3 Place of death and duty to instigate an external postmortem examination (depending on the legal situation)

Place of death or circumstances of death	Party obliged to instigate a postmortem external examination
Private home or private land	Relatives (spouse, partner, children, parents, other persons belonging to the domestic household) Owner or proprietor of the home or land Persons incidentally present
Hospital or clinic	Hospital or clinic management/ head physician
Homes, institutions, schools, etc.	Management of the institution
Ships, aircraft	Ship's or aircraft's captain
Live births irrespective of weight and stillbirths >500 g	Midwife or physician present at the birth or any other person present at the birth or who learnt of the birth through own findings
Discovery of a dead body	Anyone discovering a dead body has a duty to instigate a postmortem external examination; however, once informed, the police instigate the medical external examination

Stillbirth: Dead fetus weighing >500 g and showing no signs of having lived outside the uterus

Live birth: Fetus showing the following signs of having lived outside the uterus (irrespective of umbilical cord transection, placental expulsion, and birth weight)—heartbeat, pulsation of the umbilical cord, and natural pulmonary respiration

Depending on the legal situation, the place of death determines who is obliged to instigate an external postmortem examination (Table 2.3).

Important: **The place of death should also serve as the place at which the external postmortem examination is carried out**. **Where this is not possible**, **such as in a public place or a location that poses a possible hazard to the examiner**, **the examination can be continued at a more suitable location once death has been determined and documented**.

2.2.2 Timing of the External Postmortem Examination

Some countries make legal requirements relating to the time of the postmortem examination. In Germany, it should be carried out either "immediately," "promptly," or within a legally specified time period (8–12 h). Where death is unequivocal, the physician with a duty to perform the postmortem external examination should not interrupt the treatment of other patients or a surgical procedure on the basis of the examination.

Rights and Duties of the Certifying Physician. Depending on the legal framework, emergency or certifying physicians have the right to:

- Gain access to the place of death or scene at which a body was discovered (= permissible breach of the basic right of inviolability of the home)
- Request information from any persons with knowledge relating to the circumstances of death, including physicians who previously treated the deceased (= permissible breach of medical confidentiality)

If an emergency or certifying physician is refused access to the relevant site, access should be forcibly gained by the attending police officers. When filling out the death certificate, any information obtained should be noted in the fields relating in particular to underlying disease and cause of death, taking the specific circumstances of death into consideration. Physicians can refuse to provide information in the case of, for example, possible treatment errors associated with the death in question.

Undressing a Dead Body. The dead body should be examined in an undressed state under good lighting conditions (ideally in daylight); moreover, the examination should include inspection of the anterior and posterior sides of the body as well as all orifices. Any plasters or dressings should be removed unless a forensic autopsy is envisaged due to suspected unnatural death or unknown cause of death. Naturally, determining death always takes priority and making alterations at the scene of discovery, to clothing, and to the body is permitted to this end.

Table 2.4 Determining death: definite and inconclusive signs of death

Definite signs of death	Inconclusive signs of death
Livor mortis	Lack of consciousness or coma
Rigor mortis	Absence of spontaneous pulmonary respiration
Putrefaction	No pulse, no heartbeat
Mutilation of the body incompatible with life (e.g., internal organ evisceration)	Dilated, unresponsive pupils
Brain death identified under clinical conditions and in the setting of assisted ventilation (= state of irreversible loss of all cerebral, cerebellar, and brainstem functions). Depending on the legal framework, a distinction is made between complete and partial brain death!	Absence of reflexes (areflexia), loss of muscle tone

Determining Death. Death is determined on the basis of identifying reliable signs of death. In the absence of these signs during the early phase or in the case of an apparent state of death, attempts at resuscitation should be initiated. Under no circumstances should the determination of death be based on inconclusive signs of death (Table 2.4).

Death can only be conclusively determined once at least one definite sign of death has been identified or following 30 min of unsuccessful resuscitation and around 30-min asystole on ECG despite adequate resuscitation efforts in the proven absence of hypothermia or intoxication.

Important: **Marked cooling of the body and skin pallor should not be interpreted as definite signs of death**. **Incorrect determination of death is seen most notably in cases of intoxication and hypothermia**.

Determining the Time of Death. Information on time of death according to year, day, and time of day are required. Providing this information is straightforward assuming:

- Death occurred under medical supervision
- Reliable witnesses are able to report the time life expired
- An ultrashort agonal period in the case of an accident/disaster (e.g., an explosion) can be assumed
- The time of the accident/disaster corresponds to the time of death

In other cases, a period of death should be given; where applicable, an additional note stating "time of death according to information from…" is recommended. Inexperienced certifying physicians should restrict their time of death determinations to approximate time estimations based on the extent of rigor mortis, livor mortis, and body temperature (the body is still warm or has already cooled). A specialist forensic examination should be called for in cases where the time of death is of particular relevance.

Performing an External Postmortem Examination. The regulations governing how to perform external postmortem examinations and autopsies (see Chap. 4) in Germany can be found in the German Association of the Scientific Medical Societies (AWMF) Guidelines Register (Guidelines of the German Society of Forensic Medicine, DGRM), No. 054/002 (http://www.uni-duesseldorf.de/WWW/AWMF/11/054-002.htm).

Important: **An external postmortem examination should be carried out on a fully unclothed body and should include inspection of all body orifices under good light conditions**.

2.3 Collecting Evidence at the Scene of Discovery

Issues relating to the collection of evidence may be relevant to the emergency or certifying physician if indications point to an unnatural death or if the mode of death is unknown. The following applies in such cases:

- The external examination should be discontinued.
- The police should be notified immediately.
- No further alterations should be made at the scene of discovery.
- Make a record of all alterations already undertaken: Changes in position, partial undressing of the body, cutting open of clothes, aspiration,

other medical steps taken on the body, and unusual findings on the body.
- Ensure that no alterations are made to the scene of discovery by third persons present at the scene.
- As a memory aid, the certifying physician should compile a short record for himself or herself in the event questions arise possibly days or weeks later. Particularly relevant details may include those persons present and their behavior; apparent injury to persons present; indications of alcohol, drug, or medication use; indications of a physical confrontation; and whether doors and windows are locked.

When it is apparent, or later comes to light, that a crime has been committed, the emergency physician and/or certifying physician may be summoned by the investigating authorities to give evidence as an expert witness at court.

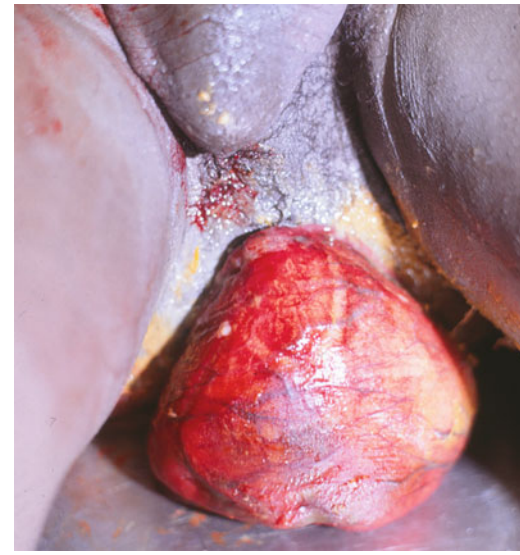


Fig. 2.4 Rectal prolapse seen at external postmortem examination

2.4 Abnormal Findings and Information at External Postmortem Examination

In the course of the mandatory external postmortem examination of a dead body, findings requiring closer inspection may be made on clothing on the body, in the immediate vicinity of the body, or in the form of information provided by relatives or witnesses. Suspicious findings can include those identified on the body (e.g., abnormal findings that in fact occurred post-mortem, findings resulting from the body being in an unusual position following death, or findings due to autolysis or putrefaction), or those found in the vicinity of the body. The following examples are taken from the broad spectrum of conceivable abnormal findings:
- Petechiae in the conjunctivae and scalp in a head-down position are interpreted as possibly the result of compression trauma to the neck.
- Vibices in areas of livor mortis in a head-down position are often misinterpreted as antemortem hemorrhage due to trauma to the neck.
- Putrefactive changes may lead to incorrect assessments [e.g., rectal prolapse (Fig. 2.4) or coffin birth due to putrefaction].
- Light-brown/ocher-colored areas of dry skin due to postmortem pressure points may be misinterpreted as antemortem injuries (Fig. 2.5).
- Skin discoloration and corresponding marks consistent with medical intervention, e.g., defibrillator marks (Fig. 2.6) or the imprint of a bite block, may be misinterpreted as due to antemortem blunt force trauma (Fig. 2.7).
- Tears in the seams of clothing, e.g., armpit seams of a shirt, may be interpreted as due to physical conflict when in reality they occurred during transportation of the body post-mortem.
- Indications of a break-in, a physical conflict in the home, or entertaining guests shortly prior to death are not considered as possible indications of an unnatural death.
- No possible causal link is made between information relating to accidents in the deceased's history and their death.
- Serious discrepancies between information on past history and previous disease fail to prompt further investigations (e.g., reporting sudden death where the cause of death has been given as hepatic coma and the body shows marked jaundice).

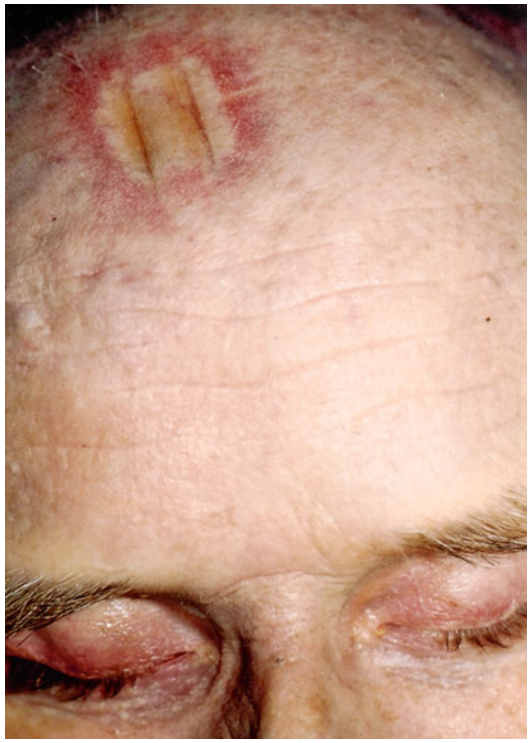


Fig. 2.5 Parallel marks above the occipital frontal circumference misinterpreted as a fatal blow to the head. In reality, the head had lain against the sections of a radiator post-mortem. No regional hemorrhage was seen at autopsy (image kindly provided by the competent police authorities)

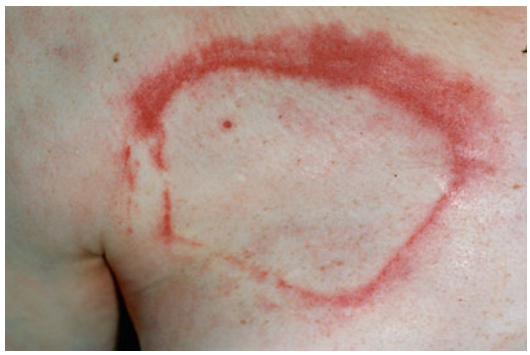


Fig. 2.6 Defibrillator marks in a typical localization, misinterpreted as blunt force trauma inflicted by a third party (image kindly provided by the competent police authorities)

There are numerous other examples seen in forensic practice that necessarily lead to uncertainty and thus, quite reasonably, result in the police being notified. It should be noted, however, that further investigation frequently yields no indication of third-party responsibility and that already overstretched police officers often urge the certifying physician to record a natural death in the death certificate.

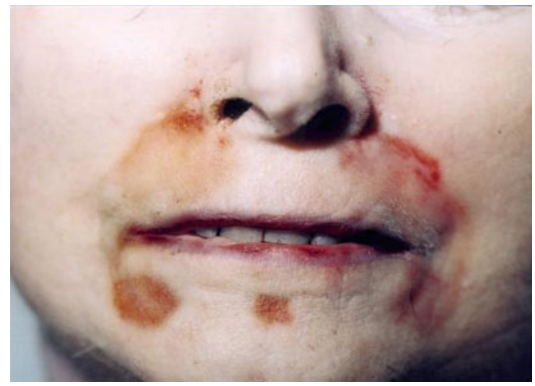


Fig. 2.7 A bite block left on the body following death in an epileptic state led in this case to postmortem pressure-related skin discoloration; the certifying physician expressed suspicion of asphyxia with a soft cover and notified the police

A systematic approach to examining the unclothed body is recommended in order to avoid overlooking important findings at external postmortem examination. Thus, a thorough examination involves careful attention to or inspection of the following:

General findings: Sex, age, type of physique, height, weight, nutritional and personal-care status, skin color, areas of swelling, asymmetries, scars, tattoos, amputations, degree of rigor mortis, extent of livor mortis, signs of putrefaction, injuries, impurities, and signs of mummification.

Head: Deformities, bony reaming, swelling, hematomas, and fluid effusion from natural orifices. Hairy scalp: hair color, hair length, swelling, transections, abnormal discoloration, and deposits on the scalp or hair. Non-hairy forehead: eyebrows, eyelids, conjunctivae, eyeball consistency, pupil width and anisocoria, facial skin, palpable fractures of the skull and facial bones, outer nostrils, upper and lower lips, corners of the mouth, (saliva) contact traces, oral vestibule and oral cavity (foam cone?), oral mucosa, dental characteristics, tongue position, checking for oral

odor (aromatic, acetonemic, uremic, or of bitter almonds/garlic) by pressing the edge of the left costal arch at the medioclavicular line, outer ear, and external auditory canals.

Neck: Mobility, findings on and in neck skin (strangulation marks, including manual and ligature strangulation marks), puncture sites, swelling, hematomas, scratches, and areas of drying due to abrasion.

Chest: Shape of the chest, symmetry, stability, and mobility, scars, defibrillation marks, injuries and discoloration, and assessment of the nipples; is skin emphysema palpable?

Abdominal walls: Above, at, or beneath chest level, curvatures, abdominal tension (gas congestion), scars, striae, puncture marks (e.g., heparin injections), pattern of hair growth, and pelvic ring mobility.

Anogenital region: Penis, scrotum, position of the testes, the major and minor labia and the hymen, deposits/trace evidence (blood, foreign bodies, secretions, feces), injuries, as well as the anal ring.

Extremities: Shape; form; mobility; edema; atrophy; lateral and circumferential differences (deep vein thrombosis!), evaluated separately according to upper and lower leg; shortening of extremities; amputations; scars; needle tracks, particularly on the flexor side of the elbow in i.v. drug abuse; and scars due to tentative wounds (previous suicide attempt?). Assessing the palms of the hands and soles of the feet: color of finger- and toenail beds, possibly collecting evidence found below fingernails by clipping the nails (take evidence separately for each finger!); applying adhesive tape to the palms of the hands (Frei's fiber test) in cases of fatal strangulation; palms and flexor side of the fingers (electrical burns?); washerwoman's skin on the palms of the hands and soles of the feet; and skin wounds on the palms of the hands and skin between the thumb and index finger (active defense wounds, seen most notably in sharp force trauma such as knife stabs, etc.).

Back: Pressure marks, swelling, discoloration, scars, injuries, and decubitus ulcers (in the buttock region, over the tips of the shoulder blades, over spinous processes, and on the heels!).

In specific cases, photodocumentation should be compiled at the time of external postmortem examination irrespective of whether or not an autopsy is foreseen. Making sketches can also be helpful, using, for example, a printed body diagram. In rare cases, it is necessary for an expert appraisal to be based purely on postmortem radiological findings; occasionally, an external postmortem examination consists solely of taking a blood sample from a femoral vein for blood alcohol determination.

2.5 Duty of Due Diligence at External Postmortem Examination

Failure to exercise due diligence at external postmortem examination can lead to unnatural deaths, including homicides, remaining undetected. Thus, misplaced consideration for the relatives of the deceased is ill-advised. Relatives should be made aware of the certifying physician's legal obligation to perform an external postmortem examination with due diligence.

Depending on the legal framework, a breach of the established rules relating to medical diligence at external postmortem examination can represent an offense punishable with a fine. Some cases may even result in criminal sanctions, such as manslaughter charges (see "Case Study" below).

Case Study

The physician facing prosecution had failed to undress the body of a 70-year-old woman found lifeless in a bathroom, thereby overlooking bright red livor mortis. Shortly thereafter, the deceased's daughter was also found dead in the same bathroom. It was subsequently revealed that both women had died of CO poisoning. Due to his failure to undress the body of the 70-year-old woman, the physician had not observed the characteristic bright red livor mortis.

Had he performed the external postmortem examination of the first victim according to regulations, the daughter's death may have been avoided. Criminal proceedings were taken against the physician and he was found guilty of manslaughter.

An official death certificate is completed once an external postmortem examination has been performed. One portion of the death certificate is intended for the official register of births, deaths, and marriages and includes the deceased's personal details, form of identification, determination of death, as well as additional information in the case of stillbirths or possible infectious diseases.

Another portion of the death certificate comprises information on the type of disease that caused death. A plausible chain of causality in terms of pathological changes that could lead to death should be given here (the medical determination of the cause of death).

Determining the Cause of Death. Information relating to cause of death is structured in the death certificate according to the WHO sample certificate, the International Form of Medical Certification of Cause of Death (Fig. 2.2). Each line is numbered as follows:

Ia = immediate cause of death
Ib = antecedent cause, i.e., morbid conditions giving rise to the immediate cause of death mentioned in 1a
Ic = underlying cause, i.e., the primary cause or underlying morbid condition
II = other significant morbid conditions contributing to death but unrelated to the underlying morbid condition mentioned in 1c

A scientifically and medically recognized pathophysiological mechanism should exist between the underlying morbid condition given and the immediate cause of death. Each field should additionally include information on the duration of the morbid condition prior to death, thus enabling its time course to be checked for plausibility. A number of sample cases are provided below by way of illustration:

Example 1:
Ia: Myocardial infarction (hours)—due to
Ib: Fresh coronary thrombosis (hours)—due to
Ic: Stenosing coronary sclerosis (years)
II: Hypertension, diabetes mellitus

Example 2:
Ia: Right ventricular decompensation (1 h)—due to
Ib: Recurrent pulmonary thromboembolism (2 h)—due to
Ic: Deep vein thrombosis (days)
II: Left-sided hypertensive intracerebral hemorrhage with right hemiparesis (6 months)

Example 3:
Ia: Cardiac tamponade (minutes)—due to
Ib: Dissecting aortic aneurysm (hours)—due to
Ic: Idiopathic medial necrosis of the aortic wall (years)
II: Hypertension, liver cirrhosis, and pyelonephritis

Example 4:
Ia: Polytrauma involving numerous body parts
Ib: Struck by a rail vehicle
Ic: Known depression
II: Diabetes mellitus

It is not always essential to complete all lines. If the cause of death (a mandatory field) is not the result of an underlying morbid condition, no further information is required, e.g., if the entry in field Ia is "narcotic poisoning," the other fields (Ib, Ic, and II) remain empty.

It is not uncommon, even after additional information has been gathered, for the medical cause of death to remain unexplained; this should be noted accordingly in the death certificate. Entering a speculative immediate cause of death, a pathophysiologically unfounded end state, or a meaningless diagnosis is not acceptable either in terms of cause of death statistics or as a basis for classifying the mode of death.

Important: **Inconclusive diagnoses**, **as well as entering the end states of any process of dying**, **should be avoided**, **e.g.**, "**old age**," "**heart failure**," "**respiratory arrest**," "**circulatory collapse**," **or** "**loss of the will to live**."

Findings are evaluated in terms of their relevance to the cause of death and weighted accord-

ingly, resulting in their differentiated classification into three groups:

Group I. Findings on the immediate cause of death and the underlying morbid condition/trauma may provide a highly plausible explanation in terms of causality and time course, as seen in "hard" causes of death: the underlying morbid condition and immediate cause of death are closely linked on a pathophysiological level, both occurring at a short time interval from one another (e.g., extensive subarachnoid hemorrhage due to a ruptured basilar artery aneurysm).

The mode of death can be clearly classified.

Group II. In contrast, a known disease can cause death by several different pathomechanisms, or several concomitant underlying diseases can converge in a final stage to cause death, so-called "soft" causes of death (e.g., stenosing coronary sclerosis and right lower lobe pneumonia in the setting of chronic lymphocytic leukemia). It is sometimes possible to identify the principal immediate cause of death from the deceased's medical history.

Classifying the mode of death may be problematic and needs to be performed on a case-by-case basis.

Group III. Lastly, there are cases of death in which, due to insufficient information on the deceased's medical history and lack of background knowledge relating to the circumstances of death, an immediate cause of death or relevant underlying disease cannot be recorded. In such cases, if the cause of death is recorded as unclear, the mode of death should likewise be recorded as unclear.

The mode of death can be classified as "unclear" or "unexplained."

Nevertheless, there are numerous medically accepted direct or indirect causes of death that are either organ-specific or non-organ-specific. Organ-specific causes of death include, for example, hepatic coma following decompensated liver cirrhosis in chronic alcohol abuse or central regulatory failure due to an epileptic state, intracerebral hemorrhage, subdural hematoma, or leptomeningitis. Non-organ-specific causes of death include embolisms or death due to sepsis.

Important: **Before filling out a death certificate**, **the certifying physician should always check whether the deceased's medical history and the circumstances of death plausibly explain final morbidity**, **in addition to whether the acuity of death can be accounted for**. **Where no plausible cause of death is found**, **this should be recorded on the form**.

Numerous studies have shown that the correspondence rate between causes of death recorded on death certificates and actual causes of death established at autopsy is 50–60 % at best. This is by no means due to a lack of diligence among certifying physicians, but rather to the unpredictability of the biological organism and its susceptibility to a multitude of possible diseases. Improving the data on actual causes of death could only be achieved by significantly increasing the number of autopsies performed.

Classifying the Manner of Death. Information on the manner of death should be recorded separately from information on the cause of death. Whereas the cause of death relates to the medical and scientific cause of death, information on the manner of death relates to the circumstances of death in terms of their criminological relevance: natural, unnatural, or unexplained. Thus, classifying the manner of death is in some cases of crucial importance in terms of the further measures taken by the authorities. In this context, the following applies:

Natural death: Death as a consequence of pathological internal causes that are in no way attributable to legally relevant external factors.

Unnatural death: Death as a consequence of an event that was caused, triggered, or influenced externally (including self-inflicted and third-party effects).

Unexplained manner of death: In the absence of evidence of a natural death, it is not possible to conclusively determine the manner of death solely on the basis of a medical external postmortem examination.

Determining whether or not a death was natural is paramount, i.e., identifying a defined internal disease for which the deceased received medical treatment ante-mortem and which, according to the given time course, could have

Table 2.5 Information or findings pointing to an unnatural cause of death

Patient history	Findings on the body
Sudden death	Congestive hemorrhage
No detectable preexisting disease	Color of livor mortis
Occupational, traffic, or other sort of accident	Odor of air from the lungs
Situation in which the body was discovered (e.g., drug paraphernalia at the scene of discovery)	Tablet remnants in the oral vestibule or mouth
Unexpected death in particularly young individuals (<40 years)	Injuries on the body

plausibly caused death, while at the same time there are no identifiable indications of an unnatural death. It is important to bear in mind here that even an individual suffering from a serious disease that could plausibly cause imminent death may be the victim of an unnatural death (poisoning, homicide to accelerate an inheritance process). Therefore, it is important to be aware of all findings that could point to an unnatural death. While legal definitions of unnatural death provide orientation, they are of only limited help in practice. One particular definition reads:

Any death resulting from suicide, an accident, an error in medical treatment, or any other external effect caused by the conduct of a third-party (death by a third party) is considered an unnatural death.

In practice, it is crucial to be able to recognize the signs of an unnatural death precisely as such. These signs may be apparent in the patient history or in findings made on the body (Table 2.5). It is sometimes necessary to verify findings in terms of whether processes associated with a natural death could be considered as the cause of death.

Since homicide can leave little or no trace, an absence of injuries is not sufficient grounds to assume a natural death. Finally, even if no indications of third-party involvement are found at the scene of discovery, this should not be taken as confirmation of a natural death. Particular attention should be paid to sequelae following (initially) survived trauma, i.e., delayed death following trauma. The following applies in this context:

Important: **Causality between the primary effect at the beginning of the causal chain resulting in death and death itself is not diminished with time—the interval may be many years**.

Incorrect death certification is seen most notably in cases with longer time intervals between the primary trauma/effect and the time of death, despite the fact that causality can be proven scientifically and medically and is recognized legally. The following examples illustrate this well:

Example 1: As a result of a road traffic accident, a cyclist suffered a lower leg fracture that could be treated successfully. The cyclist developed deep vein thrombosis in the lower leg followed by fatal pulmonary embolism 1 year later (= unnatural death).

Example 2: A female patient was left bedridden in a comatose state following hypoxic brain damage due to a mistake in anesthesia. The patient died of pneumonia after 6 years (= unnatural death).

Example 3: A patient suffered acute allergic shock following the application of a medically indicated medication (= unnatural death).

Example 4: A former metal worker died of malignant pleural mesothelioma 20 years after his final massive exposure to asbestos in an occupational capacity (= suspected unnatural death; the competent insurance institution was notified of this suspicion for the purposes of further verification and, where necessary, to call for an autopsy) (= suspected unnatural death)

The above examples also serve to clearly illustrate that classifying the mode of death is in no way linked to the question of guilt. Death due to allergic shock occurred despite correct administration of medical treatment, but the adverse drug reaction (ADR) caused distinctly premature death. However, there are specific borderline cases involving therapeutically acceptable causation of premature death. This is true, for example, of cancer patients in whom morphine-based pain medication possibly leads to a premature death, as well as in cancer patients who die as a result of

the effects of medically indicated and correctly administered chemotherapy, although without this therapy their malignancy may have caused death at an earlier—or later—point in time.

Case Study
Diagnostic coronary angiography was performed on a 56-year-old man with an abnormal stress ECG. The patient suffered circulatory collapse and asystole 2 h following the hitherto complication-free intervention. Resuscitation attempts were unsuccessful. The man had felt perfectly well that morning as he walked by foot to the examination. Due to the short time interval between the intervention and death, the man's relatives expressed suspicion that an error in medical treatment had occurred. At autopsy, cardiac tamponade due to coronary wall rupture was identified, an extremely rare yet typical complication of coronary angiography that patients need to be informed of prior to the intervention, as was the case in this particular instance.

It is not intraoperative deaths in severely ill or polytraumatized patients who succumb to their underlying disease or injuries despite interventions performed according to correct medical practice that are seen as problematic, but rather those deaths involving either patients whose quoad vitam prognosis up to the time of death was not particularly alarming or indeed patients who were largely in good health.

The question of whether a death should be certified as unnatural in cases where the unavoidable risk of a medical intervention becomes a harsh reality is a controversial one. There are those who demand at least a remote indication of a breach of the recognized rules of medical diligence (treatment error) in order for death to be suspected as unnatural. However, with regard to the case study described above, one can assume—in contrast to advanced-stage cancer patients who die as a result of their malignancy—that coronary angiography was responsible for causing the patient's death, possibly many years prematurely.

Case Study
A 77-year-old woman was treated for a femoral neck fracture with a total endoprosthesis. The patient developed postoperative right ventricular heart failure in the recovery room and died. Autopsy confirmed the surgeons' suspected diagnosis of partially fracture-related and partially unavoidable surgery-related fatal bone marrow and fat embolism. It was not possible to establish beyond a doubt whether the woman would have lived longer had she not undergone the operation. Meanwhile, police inquiries revealed that the patient had suffered a fall while walking to the toilet at the nursing home for the elderly where she was a resident. In contravention of explicit instructions that the overweight woman should always have two caregivers to support her when walking, she had been accompanied on the occasion in question by only one caregiver, who was not strong enough to prevent the fall. The patient would not have died at that point in time had instructions been followed.

Bone marrow and fat embolism during (in particular the bone cement phase) and following endoprosthetic surgery for femoral head fractures is a known yet rarely fatal complication that the patient needs to be informed about. In the event of this type of embolism, death usually occurs as the result of a fall in the home (an accident) and is therefore classified as an unnatural death. Accidents, like the one described above, which could be avoided through due care and diligence can result in legal prosecution for negligent manslaughter.

Death on the Operating Table. The death of a patient directly during a medical intervention, notably on the operating table, is a particularly challenging situation. In such cases, classifying the death as "unexplained" and notifying the

relevant authorities, even if the treating physician temporarily makes himself or herself the subject of a criminal investigation, is advised. The death certificate should be filled out by an impartial physician in order to avoid any suspicion of concealment by the operating physician. This could be regulated either by law or, for example, by hospital regulations. Most particularly in cases of death on the operating table, the underlying disease and cause of death need to be established at autopsy before any comment can be made on the question of a possible treatment error. A review of medical malpractice claims reveals that autopsies almost always result in exoneration of the accused physician.

Important: **Classifying death as "natural" is not recommended in cases of death on the operating table since**, **at any subsequent official inquiry**, **the treating physician may be suspected of attempting to conceal a medical error**. **The death certificate should be filled out by an impartial physician uninvolved in either the diagnosis or treatment of the patient**.

The death certificate must be filled out once the external postmortem examination has been completed and all available information has been collected. In a departure from specifications in the WHO's international death certificate (Fig. 2.2), some death certificates require more differentiated information, such as the US Standard Certificate of Death (Fig. 2.8), which includes a special section requiring information on the cause of death (Fig. 2.9) and the approximate time between disease onset and death.

2.6 Second External Postmortem Examination at the Crematorium

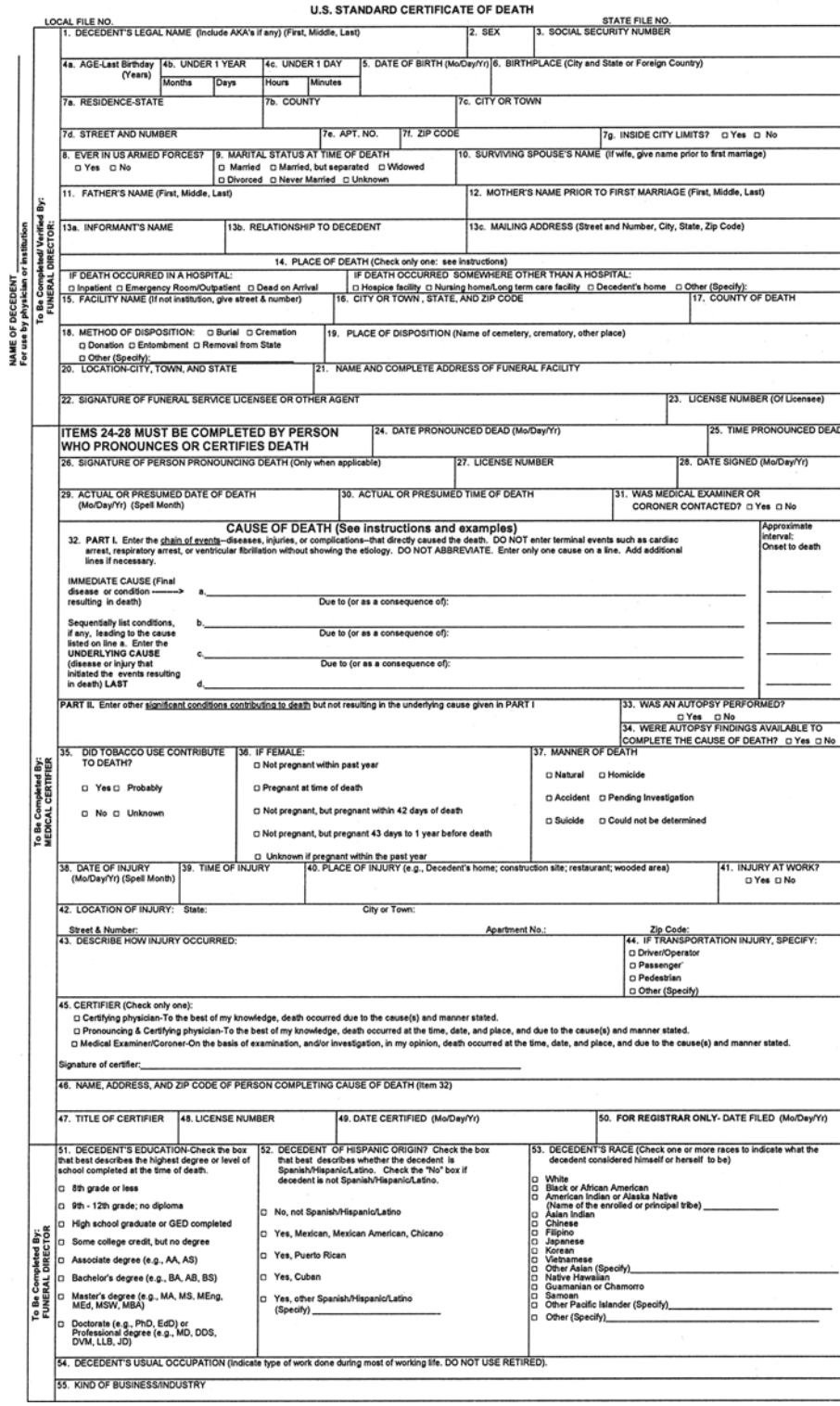
In countries where inhumation is predominantly practiced, a second external postmortem examination is sometimes required by law if cremation or burial at sea is planned. Since the body is irretrievably disposed of in both these cases, a second examination is designed to carefully reexamine the body and compare any findings with information given in the death certificate. Further information may be requested from the first certifying physician, as well as from other physicians involved in the previous treatment of the deceased, in order to clarify any conflicting information. If doubts persist as to whether death was natural, the body is not released for cremation. Any indication of an unnatural death should prompt the second certifying physician to notify the authorities. The public prosecutor's office will then decide whether a forensic autopsy is required. However, depending of the results of police inquiries, the public prosecutor's office can also release a body for cremation without calling for an autopsy.

Previously overlooked indications of homicide (small gunshot wounds or ligature marks missed by the first certifying physician), as well as reasonable suspicion of a treatment error or error in care, can give rise to an autopsy following the (second) external postmortem examination. Furthermore, a second external examination is intended to identify and further clarify deaths with long intervals since the harmful event (road traffic accident, occupational accident) that have been incorrectly classified as natural, as well as cases of suspected fatal occupational disease, prior to cremation.

2.7 The External Postmortem Examination and Recording Causes of Death/Fatal Injury

External postmortem examinations and death certification form the basis of statistical information on death due to natural causes or fatal external trauma. According to WHO World Health Statistics (WHO 2008), the following represent the most frequent causes of death:

1. Ischemic heart disease
2. Cerebrovascular disease
3. Lower respiratory infections
4. Chronic obstructive pulmonary disease
5. Diarrheal diseases
6. HIV/AIDS
7. Tuberculosis
8. Trachea, bronchus, and lung cancers
9. Road traffic accidents



U.S. STANDARD CERTIFICATE OF DEATH

LOCAL FILE NO. STATE FILE NO.

NAME OF DECEDENT
For use by physician or institution
To Be Completed/Verified By: FUNERAL DIRECTOR:

1. DECEDENT'S LEGAL NAME (Include AKA's if any) (First, Middle, Last) | 2. SEX | 3. SOCIAL SECURITY NUMBER

4a. AGE-Last Birthday (Years) | 4b. UNDER 1 YEAR Months Days | 4c. UNDER 1 DAY Hours Minutes | 5. DATE OF BIRTH (Mo/Day/Yr) | 6. BIRTHPLACE (City and State or Foreign Country)

7a. RESIDENCE-STATE | 7b. COUNTY | 7c. CITY OR TOWN

7d. STREET AND NUMBER | 7e. APT. NO. | 7f. ZIP CODE | 7g. INSIDE CITY LIMITS? ☐ Yes ☐ No

8. EVER IN US ARMED FORCES? ☐ Yes ☐ No | 9. MARITAL STATUS AT TIME OF DEATH ☐ Married ☐ Married, but separated ☐ Widowed ☐ Divorced ☐ Never Married ☐ Unknown | 10. SURVIVING SPOUSE'S NAME (If wife, give name prior to first marriage)

11. FATHER'S NAME (First, Middle, Last) | 12. MOTHER'S NAME PRIOR TO FIRST MARRIAGE (First, Middle, Last)

13a. INFORMANT'S NAME | 13b. RELATIONSHIP TO DECEDENT | 13c. MAILING ADDRESS (Street and Number, City, State, Zip Code)

14. PLACE OF DEATH (Check only one: see instructions)

IF DEATH OCCURRED IN A HOSPITAL: ☐ Inpatient ☐ Emergency Room/Outpatient ☐ Dead on Arrival

IF DEATH OCCURRED SOMEWHERE OTHER THAN A HOSPITAL: ☐ Hospice facility ☐ Nursing home/Long term care facility ☐ Decedent's home ☐ Other (Specify):

15. FACILITY NAME (If not institution, give street & number) | 16. CITY OR TOWN, STATE, AND ZIP CODE | 17. COUNTY OF DEATH

18. METHOD OF DISPOSITION: ☐ Burial ☐ Cremation ☐ Donation ☐ Entombment ☐ Removal from State ☐ Other (Specify): | 19. PLACE OF DISPOSITION (Name of cemetery, crematory, other place)

20. LOCATION-CITY, TOWN, AND STATE | 21. NAME AND COMPLETE ADDRESS OF FUNERAL FACILITY

22. SIGNATURE OF FUNERAL SERVICE LICENSEE OR OTHER AGENT | 23. LICENSE NUMBER (Of Licensee)

To Be Completed By: MEDICAL CERTIFIER

ITEMS 24-28 MUST BE COMPLETED BY PERSON WHO PRONOUNCES OR CERTIFIES DEATH

24. DATE PRONOUNCED DEAD (Mo/Day/Yr) | 25. TIME PRONOUNCED DEAD

26. SIGNATURE OF PERSON PRONOUNCING DEATH (Only when applicable) | 27. LICENSE NUMBER | 28. DATE SIGNED (Mo/Day/Yr)

29. ACTUAL OR PRESUMED DATE OF DEATH (Mo/Day/Yr) (Spell Month) | 30. ACTUAL OR PRESUMED TIME OF DEATH | 31. WAS MEDICAL EXAMINER OR CORONER CONTACTED? ☐ Yes ☐ No

CAUSE OF DEATH (See instructions and examples)

Approximate interval: Onset to death

32. **PART I.** Enter the chain of events--diseases, injuries, or complications--that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary.

IMMEDIATE CAUSE (Final disease or condition ------> resulting in death) a. _____ Due to (or as a consequence of):

Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST
b. _____ Due to (or as a consequence of):
c. _____ Due to (or as a consequence of):
d. _____

PART II. Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I

33. WAS AN AUTOPSY PERFORMED? ☐ Yes ☐ No

34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? ☐ Yes ☐ No

35. DID TOBACCO USE CONTRIBUTE TO DEATH? ☐ Yes ☐ Probably ☐ No ☐ Unknown

36. IF FEMALE: ☐ Not pregnant within past year ☐ Pregnant at time of death ☐ Not pregnant, but pregnant within 42 days of death ☐ Not pregnant, but pregnant 43 days to 1 year before death ☐ Unknown if pregnant within the past year

37. MANNER OF DEATH ☐ Natural ☐ Homicide ☐ Accident ☐ Pending Investigation ☐ Suicide ☐ Could not be determined

38. DATE OF INJURY (Mo/Day/Yr) (Spell Month) | 39. TIME OF INJURY | 40. PLACE OF INJURY (e.g., Decedent's home; construction site; restaurant; wooded area) | 41. INJURY AT WORK? ☐ Yes ☐ No

42. LOCATION OF INJURY: State: City or Town: Street & Number: Apartment No.: Zip Code:

43. DESCRIBE HOW INJURY OCCURRED:

44. IF TRANSPORTATION INJURY, SPECIFY: ☐ Driver/Operator ☐ Passenger ☐ Pedestrian ☐ Other (Specify)

45. CERTIFIER (Check only one):
☐ Certifying physician-To the best of my knowledge, death occurred due to the cause(s) and manner stated.
☐ Pronouncing & Certifying physician-To the best of my knowledge, death occurred at the time, date, and place, and due to the cause(s) and manner stated.
☐ Medical Examiner/Coroner-On the basis of examination, and/or investigation, in my opinion, death occurred at the time, date, and place, and due to the cause(s) and manner stated.

Signature of certifier: _____

46. NAME, ADDRESS, AND ZIP CODE OF PERSON COMPLETING CAUSE OF DEATH (Item 32)

47. TITLE OF CERTIFIER | 48. LICENSE NUMBER | 49. DATE CERTIFIED (Mo/Day/Yr) | 50. **FOR REGISTRAR ONLY**- DATE FILED (Mo/Day/Yr)

To Be Completed By: FUNERAL DIRECTOR

51. DECEDENT'S EDUCATION-Check the box that best describes the highest degree or level of school completed at the time of death.
☐ 8th grade or less
☐ 9th - 12th grade; no diploma
☐ High school graduate or GED completed
☐ Some college credit, but no degree
☐ Associate degree (e.g., AA, AS)
☐ Bachelor's degree (e.g., BA, AB, BS)
☐ Master's degree (e.g., MA, MS, MEng, MEd, MSW, MBA)
☐ Doctorate (e.g., PhD, EdD) or Professional degree (e.g., MD, DDS, DVM, LLB, JD)

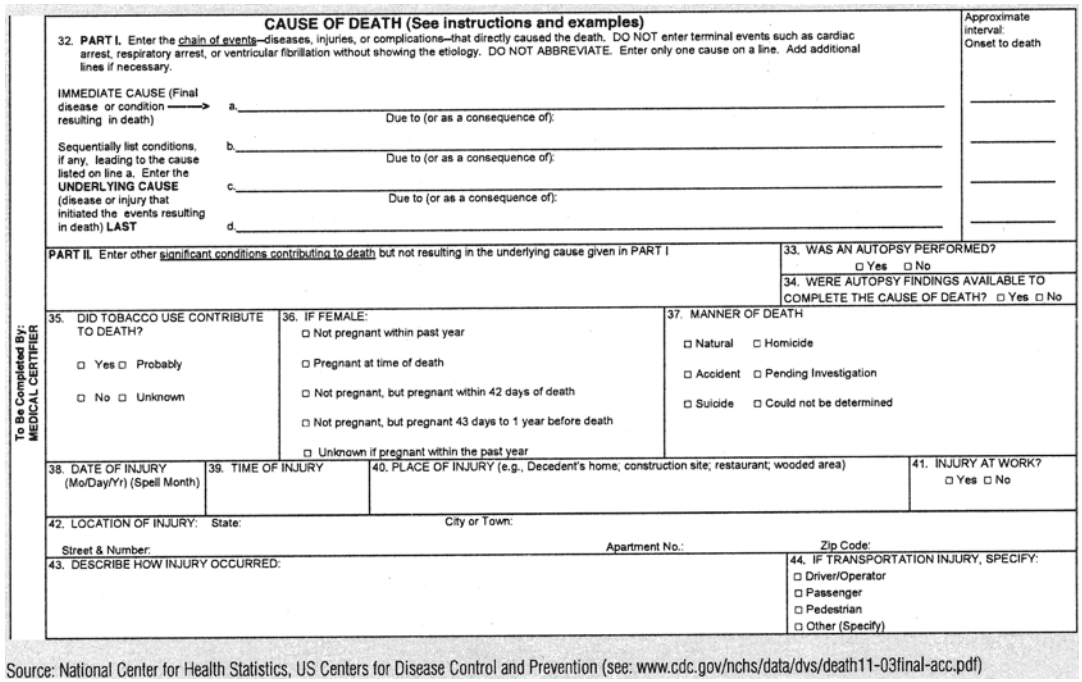
52. DECEDENT OF HISPANIC ORIGIN? Check the box that best describes whether the decedent is Spanish/Hispanic/Latino. Check the "No" box if decedent is not Spanish/Hispanic/Latino.
☐ No, not Spanish/Hispanic/Latino
☐ Yes, Mexican, Mexican American, Chicano
☐ Yes, Puerto Rican
☐ Yes, Cuban
☐ Yes, other Spanish/Hispanic/Latino (Specify) _____

53. DECEDENT'S RACE (Check one or more races to indicate what the decedent considered himself or herself to be)
☐ White
☐ Black or African American
☐ American Indian or Alaska Native (Name of the enrolled or principal tribe) _____
☐ Asian Indian
☐ Chinese
☐ Filipino
☐ Japanese
☐ Korean
☐ Vietnamese
☐ Other Asian (Specify) _____
☐ Native Hawaiian
☐ Guamanian or Chamorro
☐ Samoan
☐ Other Pacific Islander (Specify) _____
☐ Other (Specify) _____

54. DECEDENT'S USUAL OCCUPATION (Indicate type of work done during most of working life. DO NOT USE RETIRED).

55. KIND OF BUSINESS/INDUSTRY

Fig. 2.8 The US Standard Certificate of Death



CAUSE OF DEATH (See instructions and examples)

32. PART I. Enter the chain of events—diseases, injuries, or complications—that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary.

Approximate interval: Onset to death

IMMEDIATE CAUSE (Final disease or condition → resulting in death) a. Due to (or as a consequence of):

Sequentially list conditions, if any, leading to the cause listed on line a. Enter the UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST
b. Due to (or as a consequence of):
c. Due to (or as a consequence of):
d.

PART II. Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I

33. WAS AN AUTOPSY PERFORMED? ☐ Yes ☐ No

34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? ☐ Yes ☐ No

To Be Completed By: MEDICAL CERTIFIER

35. DID TOBACCO USE CONTRIBUTE TO DEATH? ☐ Yes ☐ Probably ☐ No ☐ Unknown

36. IF FEMALE: ☐ Not pregnant within past year ☐ Pregnant at time of death ☐ Not pregnant, but pregnant within 42 days of death ☐ Not pregnant, but pregnant 43 days to 1 year before death ☐ Unknown if pregnant within the past year

37. MANNER OF DEATH ☐ Natural ☐ Homicide ☐ Accident ☐ Pending Investigation ☐ Suicide ☐ Could not be determined

38. DATE OF INJURY (Mo/Day/Yr) (Spell Month)

39. TIME OF INJURY

40. PLACE OF INJURY (e.g., Decedent's home; construction site; restaurant; wooded area)

41. INJURY AT WORK? ☐ Yes ☐ No

42. LOCATION OF INJURY: State: City or Town: Street & Number: Apartment No.: Zip Code:

43. DESCRIBE HOW INJURY OCCURRED:

44. IF TRANSPORTATION INJURY, SPECIFY: ☐ Driver/Operator ☐ Passenger ☐ Pedestrian ☐ Other (Specify)

Source: National Center for Health Statistics, US Centers for Disease Control and Prevention (see: www.cdc.gov/nchs/data/dvs/death11-03final-acc.pdf)

Fig. 2.9 Extract from the US Standard Certificate of Death: cause of death

10. Prematurity and low birth weight
11. Neonatal infections[1]
12. Diabetes mellitus
13. Malaria
14. Hypertensive heart disease
15. Birth asphyxia and birth trauma
16. Suicide
17. Stomach cancer
18. Cirrhosis of the liver
19. Nephritis and nephrosis
20. Colon and rectum cancer

"Homicide" comes 22nd on the list. However, in most countries around the world, there is no reliable or structured system for recording fatal trauma. The WHO proposed a system for recording deaths which could be used as a supplement to current death certificates and which classifies fatal trauma according to type: road traffic incident, other transport incident, blunt force (struck/hit by a person, animal, or object), fall, stab/cut, animal bite, drowning/submersion, burn (smoke/fire/flames), burn (contact with heat/scald), poisoning, suffocation, choking, hanging, electrocution, firearm discharge/gunshot, explosive blast, and envenomation, among others. In 2012, both a short version and a long version of a fatal injury surveillance data collection form were proposed with the aim of improving the worldwide lack of data. Naturally, there are significant discrepancies in the rates of death due to trauma between different areas of the world, with South Africa, for example, demonstrating particularly high rates. However, cases of fatal trauma can also be recorded to a certain extent by applying the coding system used in the International Classification of Diseases (ICD 10; see Table 2.6).

2.8 Duty to Bury

The duty to organize and pay for the burial of a deceased person usually falls to relatives. Many countries specify a fixed hierarchy of relatives upon whom the duty to bury falls (e.g., spouse, biological or adopted children, parents, siblings, grandparents). If the wishes of the deceased are unknown, the type of burial (inhumation,

[1]Comprises severe neonatal infections and other noninfectious causes arising during the perinatal period

Table 2.6 External causes of injury and their corresponding ICD codes (Bartolomeos et al. 2012)

External cause of injury	ICD-10 Code
All injuries	Vo1-Y98
Unintentional injuries	V01-X59, Y40-Y86, Y88, Y89
1. Road traffic injuries	V01-V89, V99, Y850
2. Poisoning	X40-X49
3. Falls	W00-W19
4. Fires	X00-X09
5. Drowning	W65-W74
6. Other unintentional injuries	V90-V98, W20-W64, W75-W99, X10-X39, X50-X59, Y40-Y86, Y88, Y89
Intentional injuries	
1. Self-inflicted	X60-Y84, Y870
2. Interpersonal violence	X85-Y09, Y871
3. War	Y36
4. Other intentional injuries	Y35

cremation, or burial at sea) is also chosen by the relatives within the boundaries of national law. In many countries, regulations determine the point at which a duty to bury becomes relevant in the case of stillbirths or spontaneous abortions (e.g., from a body weight of 500 g) and/or whether relatives are entitled to bury low-body-weight embryos and fetuses. Time limits within which burial should take place, starting from the time of determination of death, are often specified (e.g., 5 days) unless there are legally recognized grounds to delay burial. In addition, there are regulations and laws limiting the period of time a body can remain buried in a cemetery (e.g., to 30 years), while others permit urn burials only in designated places and either permit or forbid urns to be taken home. Finally, there are also regulations relating to the scattering of a person's ashes at sea, in a specially designated woodland area, or on private land.

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Thanatology

3

Case Study

The body of a 42-year-old man was found in the sealed apartment where he lived alone; there were no signs of a break-in and physical conflict or that visitors had been in the apartment. The partially clothed body was discovered in the living room more or less seated in a left lateral position with its feet on the floor. Reddish-brown fluid exited from the mouth and nose. The tips of the second and third fingers on the right hand clearly demonstrated nicotine discoloration, and the skin on the left elbow showed unusual purplish punctiform discoloration and discrete linear scars, which were only visible in very bright light. Extensive green skin discoloration and marbling could be seen on the posterior side of the body facing the heating device; in addition, a number of fly eggs were found in the corners of the eyes. The low rectal temperature measured corresponded to room temperature with closed doors and windows. According to the police, the deceased was known to be a drug user. Although no narcotic paraphernalia (syringes, powder, etc.) was found in the apartment, numerous empty bottles of various alcoholic beverages, including high-proof spirits, were found. At autopsy, an aromatic odor emanated from the body and the urinary bladder was found to be filled to bursting with cloudy urine. It was not possible to establish a cause of death macroscopically at autopsy. In addition to the cause and mode of death, the police were keen to establish the postmortem interval.

Contrary to popular belief, forensic medical activities are by no means restricted to death resulting from violent trauma (homicide and manslaughter). All factors relating to death, i.e., previous history, circumstances of death, external examination and autopsy, further examinations to establish the postmortem interval, identity, and cause and mode of death (natural, unnatural), among others, form a central field of work in forensic medicine, brought together under the term "thanatology."

Thanatology (Greek *Thanatos* = death) is the scientific study of the causes and circumstances of death.

Differentiating between "cause of death" and "mode of death" has a crucial impact on how one proceeds following the discovery of a body.

Cause of death: The cause of death from a medical perspective, e.g., lung emboli, sepsis, myocardial infarct, hypertensive intracerebral hemorrhage, ruptured aneurysm, and malignancy.

Mode of death: The circumstances surrounding death and their legal relevance, such that the mode of death can be classified as "natural" or "unnatural." If it is not possible to establish the mode of death on the basis of an external

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examination and previous history alone, an autopsy examination should be considered (see Chap. 2).

Entries need to be made in the death certificate for both the cause and the mode of death, on the one hand, to enable a legal–criminal evaluation and, on the other, to permit any further necessary measures to be taken by the authorities, such as issuing a death certificate.

3.1 Death

The death of the organism is preceded by an agonal phase, leading to a loss of the integrating and coordinating functions of vital organs or organ systems. Principal among these is the breakdown of the cardiac circulatory system, respiration, and the central nervous system (CNS). Thus, the process of dying can have agonal phases of varying duration, ranging from seconds to hours (Table 3.1).

Depending on the cause of death, vital body reactions or a reaction in vital organ systems can (still) be expected. Thus, in the case of slow exsanguination, circulatory centralization with reduced blood pressure and increased heart rate (hypertension, tachycardia, shock symptoms) is seen. Dyspnea can be added to these symptoms in the case of asphyxial death. Central nervous reactions include, for example, tonic–clonic seizures (agonal phase or "death throes" in the real sense). In the case of death due to preexisting internal disease with an extended agonal phase, the imminent onset of hypoxic damage to the CNS results in the agonal phase not being consciously perceived by the dying person.

The agonal phase ends with clinical death (cardiac arrest). That an individual is dead is established on the basis of changes that are seen only following irreversible cardiac or respiratory arrest. Conclusive signs of death include livor mortis, rigor mortis, putrefaction, injury incompatible with life, and evidence of brain death, the latter being subject to national legal standards.

Types of Death. It is not always possible to assign a single, concrete, and nameable cause of death originating in an organ or organ system. Thus, disease in an organ can have fatal effects beyond the function of the organ itself. Indeed, there are diseases of a variety of organs or organ systems that only become fatal when they occur concurrently. The presence of parallel diseases in organs or organ systems is also seen, whereby each disease on its own could explain death. Against this background, it is possible to differentiate between various types of death on the basis of patient history, clinical symptoms, examination findings, and diseases diagnosed at autopsy (Table 3.2).

The types of death shown in Table 3.2 highlight how, when establishing the cause of death, the disease responsible for causing organ (system) failure needs to be embedded in a plausible chain of causality. Where this is the case, a justifiable cause of death is established. In the

Table 3.1 The agonal phase

	Ultrashort or absent agonal phase	Short agonal phase	Long agonal phase
Duration	Seconds	Minutes	Hours
Examples	"Krönlein" gunshot wound with instant evisceration of the brain, fragmentation of the organism by a rail vehicle, explosions causing rupture of the organism	Functional disruption that can only be survived for a short time, such as extensive hypertensive hemorrhage, cardiac tamponade, internal bleeding following ruptured aortic aneurysm	Chronic disease, such as developing sepsis or malignancy with impending death in the final stage: pallor, sharp nose, sunken eyes and cheeks, grayish or pale skin, cold sweats ("hippocratic face")

absence of any indication in the patient history of disease that could have caused death, and where the circumstances of death offer no explanation for acute death in particular, this should be noted in the death certificate. An increased incidence of cases of this kind at a particular location may give rise to the suspicion of serial homicide, e.g., the surreptitious administration of lethal medication in a care home or intensive care unit.

Important: **Any information relating to the cause of death established at autopsy and entered on the death certificate should have a pathophysiological basis**. **The diseases indicated should be of a nature that could cause morbid events leading directly to death**, **such that death at the time and under the circumstances stated is plausible**.

Table 3.2 Types of death

Linear type	Divergent type	Convergent type	Complex type
Disease and cause of death are found in one organ or organ system	Although disease is organ-specific, cause of death is non-organ-specific	Diseases in various organs or organ systems converge in a final phase to cause death	Diseases in various organs or organ systems could each in isolation represent an organ-specific cause of death

3.2 Brainstem Death and Brainstem Death Diagnosis

In practice, diagnosing brainstem death is relevant only in those patients in whom the other two "portals of death," i.e., the cardiovascular system and respiration, are substituted and thereby maintained at a functional level. Once intensive care measures of this kind have been ceased, organs can be explanted. The criteria for diagnosing brain death (complete brain death or partial brain death) are subject to the relevant national legislation. Table 3.3 lists the main criteria for the diagnosis of brainstem death diagnosis in Germany.

3.3 State of Apparent Death

Before issuing a death certificate, it is necessary to establish an individual's death medically. For this, conclusive evidence of death, i.e., rigor mortis, livor mortis, putrefaction, or injuries incompatible with life, is required. Incontrovertible evidence of death of this kind is absent in the case of apparent death.

State of Apparent Death: **States in which the normal signs of life** (**respiration**, **preserved cardiovascular function**) **are reduced due to dysregulation of the organism to a minimum extent**, **with the result that insufficient examination will fail to detect any residual function**.

Table 3.3 Brainstem death diagnosis in Germany

Criteria	Clinical symptoms	Long observation time	Short observation time
Massive acute primary or secondary CNS damage. To be excluded: Intoxication Neuromuscular blockade Hypothermia Circulatory shock Metabolic or endocrine coma	Unconsciousness, pupils unresponsive and no reaction to mydriatic eye drops, no brainstem reflexes: Corneal reflex Oculocephalic reflex Pain response to trigeminal stimulus Gag reflex Apnea	12–72 h: can vary depending on age and type of brain damage (primary, secondary)	Supplementary findings: Flat EEG (mandatory in infratentorial brain damage and children aged up to 2 years old) Absent evoked potentials (only in supratentorial and secondary brain damage) Evidence of cerebral circulatory arrest

Modified according to Deutsches Ärzteblatt (1997)

Where there is no clear clinical evidence of life (respiration, cardiovascular function) but at the same time no conclusive signs of death, the phenomenon referred to as "a state of apparent death" should be considered. The main goal here should be to perform an ECG examination. The organism is able to tolerate significantly longer periods of cardiac and respiratory arrest in the presence of hypothermia. One well-known case is that of a 6-year-old boy who fell through ice into water and could only be rescued after being submerged for 20 min. No brain damage could be seen following successful resuscitation. Thus, where a state of apparent death cannot be excluded and given appropriate resuscitation attempts, medical treatment should be ceased only after a 30-min flatline ECG. Even discrete ECG findings do not constitute a flatline ECG and should not be confused with cardiac arrest. In hypothermic patients with possible intoxication and following near drowning, longer reanimation times are required before rewarming or detoxication and/or the onset of conclusive signs of death are seen.

Inconclusive signs of death include:

- Absent reflexes
- Absent respiration
- Absent cardiac activity
- Dilated unresponsive pupils
- Reduced body temperature

Death should never be certified on the basis of these inconclusive signs of death. In the case of hypothermia, cold rigor should not be confused with rigor mortis.

Important: **No conclusive signs of death, no death certificate**!

The causes leading to an apparent state of death are summed up in the AEIOU rule according to Prokop:

- A = alcohol, anemia, anoxemia
- E = electricity, including lightning strikes
- I = injury (craniocerebral trauma)
- O = opium, narcotics, drugs with a central effect
- U = uremia (metabolic comas), hypothermia

Important: **Erroneously issuing a death certificate following insufficient examination of a patient who is in fact still living is always a contravention of the generally recognized codes of medical practice for pronouncing death**.

3.4 Supravital Reactions: Early Postmortem Changes

Definition: **Supravital reactions in the early postmortem phase refer to postmortem metabolic processes which, even following death and during the supravital phase, generally lead to tissue reactions triggered in a temperature-dependent manner** (**see Chap**. 7).

Alongside livor mortis and rigor mortis, the following supravital reactions are of particular importance in terms of the time of death or postmortem interval:

- Mechanical excitability of skeletal muscles
- Electrical excitability of skeletal muscles, particularly the facial mimic muscles
- Pharmacological excitability of the iris smooth muscles

3.4.1 Mechanical Excitability of the Skeletal Muscles

Mechanical stimulation of the skeletal muscles can be triggered by a vigorous blow to the biceps brachii muscle, for example, producing propagated excitation and contraction over the entire muscle (Zsako's phenomenon) for up to 1.5–2.5 h post-mortem (hpm). A significant reversible idiomuscular contraction may develop at the point of stimulation in response to the mechanical excitation for up to 4–5 hpm. A weaker idiomuscular contraction can still be triggered at up to 8–12 hpm, while a mild idiomuscular contraction may persist for up to 24 hpm. These Zsako reflexes (idiomuscular reactions) are sometimes easier to feel than they are to see and may be found on other points of the body in the early postmortem phase (1.2–2.5 hpm) by:

- Sharply tapping the area between the shoulder blades can cause these to approximate.
- Tapping between the metacarpal bones on the back of the hand with a reflex hammer causes the fingers to approximate.
- By tapping the muscles 8–12 cm immediately above it, an upward reflex movement of the patella can be elicited.

3.4.2 Electrical Excitability of Skeletal Muscles

Skeletal muscle excitation may be elicited postmortem primarily by electrical stimuli, i.e., appropriately affixed electrodes can induce muscle contraction. Evidence of electrical excitability and the propagation of muscle contraction permits inferences about the time of death to be made even at the site where the body was discovered. Thus, checking supravital electrical excitability of the skeletal muscles is an important part of the forensic estimation of postmortem intervals.

Transportable electrical excitation devices are used, some with flat surface electrodes particularly suited to testing the excitability of facial mimic muscles (Fig. 3.1). Electrical stimulation can also be performed on the thenar and hypothenar muscles.

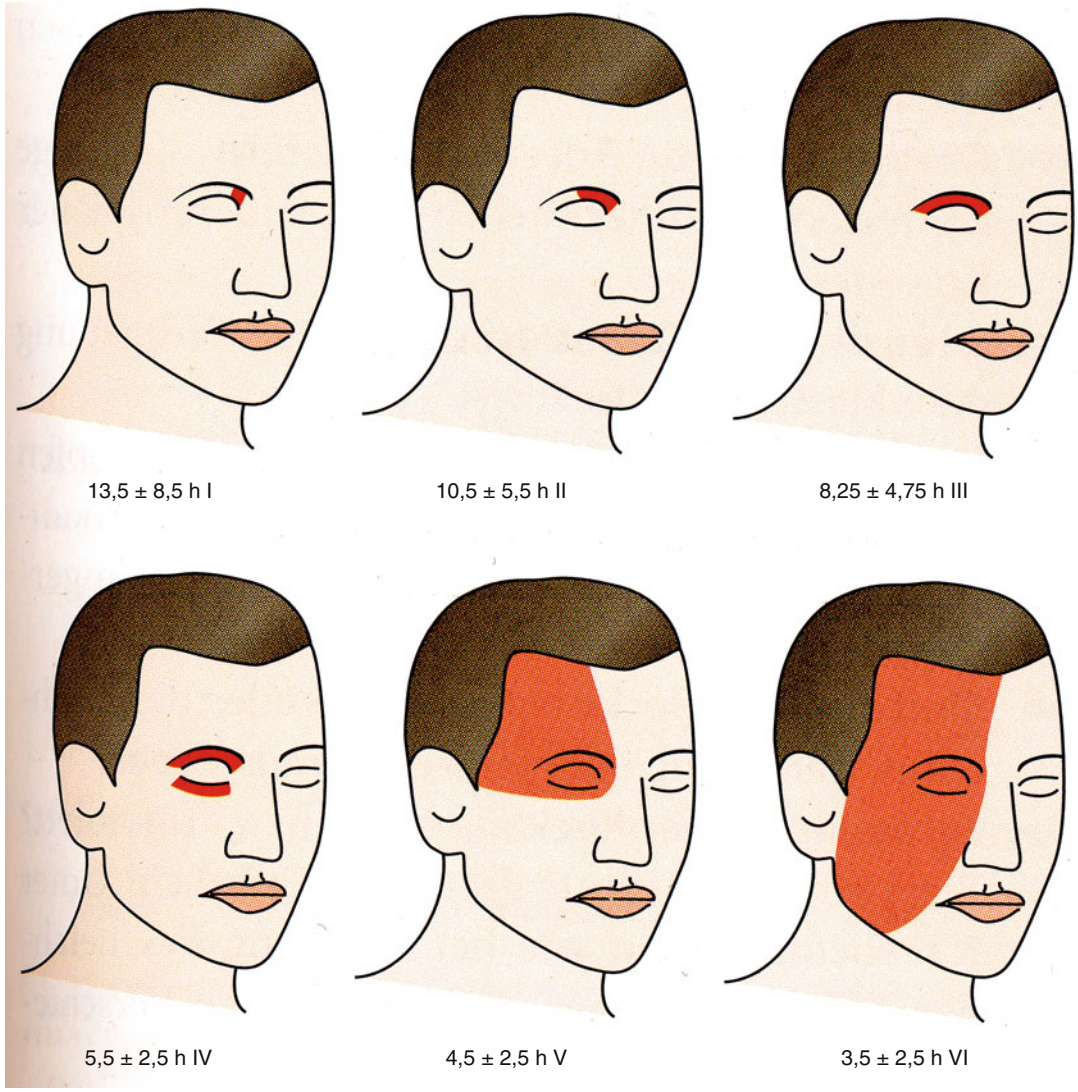

Fig. 3.1 Testing for electrical excitability of the facial mimic muscles. Contraction of the entire ipsilateral facial musculature can be seen in the early postmortem phase. As the postmortem interval increases, muscle excitation is limited to the point of stimulation directly proximal to the electrode (orbicular muscle of the eye) (According to Henssge et al. (2002))

A propagation of muscle excitation to areas distal to the electrodes is observed, e.g., following the application of puncture electrodes to the orbicular muscle of the eye (medial part of the eyelid) or to the orbicular muscle of the mouth (at both corners of the mouth). The observed reaction can then be graded:

- Early postmortem reaction in the entire ipsilateral side of the face
- Reaction restricted to the area of stimulation
- Reaction in only the eyelid
- Reaction in only part of the eyelid
- Reaction only directly at the point of electrode puncture

The same principle is applied when grading the stimulation of the orbicular muscle of the mouth. Facial mimic muscle excitability is given as up to 20 hpm, the orbicular muscle of the mouth 11 hpm, and the thenar and hypothenar muscles 10–12 hpm. Electrical stimulation of the pupil musculature using puncture electrodes applied to the conjunctiva at the corneal margin can trigger a reaction up to 20 hpm but rarely longer.

3.4.3 Pharmacological Excitability of the Iris Musculature

The use of pupillomotor pharmacological agents helps in the assessment of the initially preserved postmortem responsiveness of the iris muscles, whereby the striated iris muscles show longer pharmacological excitability than smooth iris muscles. Subconjunctival injection of mydriatics (adrenaline/noradrenaline, tropicamide, and atropine) or miotics (acetylcholine) can demonstrate preserved responsiveness—evidence of mydriasis or miosis—for up to 20 hpm, rarely up to 40 hpm. Other pharmacological agents show supravital reactions of shorter duration.

3.4.4 Livor Mortis (Postmortem Lividity/Hypostasis)

Increasing heart failure can cause visible and prognostically unfavorable local blood stasis as early on as during a protracted agonal phase. Actual livor mortis, however, appears only after irreversible circulatory arrest as a result of blood settling in dependent areas of the body, i.e., the lowest parts of the body, due to hydrostatic pressure or gravity, and represents the earliest sign of death. Initially, hypostasis causes blood vessels in the subepidermal corium to fill up, producing small bright-red spots. These spots then expand and become confluent, turning purple in color due to residual oxygen consumption (Fig. 3.2).

Linear sparing can often be seen within areas of livor mortis due to external pressure, in particular from tightly fitting clothes, working against the pressure of hypostasis, perhaps as the result of folds in clothes (Fig. 3.3).

Wherever the skin comes into direct contact with the underlying surface, i.e., the areas bearing the body's weight, the pressure exerted by the surface exceeds the hydrostatic pressure, thus causing sparing from livor mortis in those particular areas. Typical areas of sparing in a supine body include the shoulder blades (butterfly-shaped sparing), buttocks, and heels. If the supporting surface has a particular pattern, this

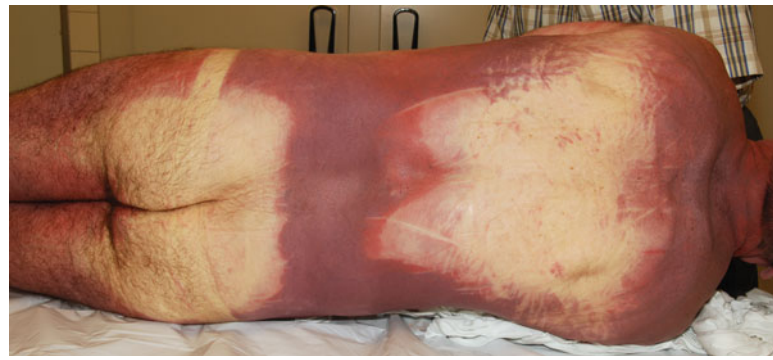

Fig. 3.2 Livor mortis. Butterfly-shaped area of sparing from *bluish-violet* livor mortis due to the supine position of the body. An additional area of sparing is seen in the belt area. Areas of lighter livor mortis are a sign of early postmortem reoxygenation of hemoglobin due to cold storage

pattern may be reflected as a pattern of sparing from livor mortis (Fig. 3.4). Similarly, sparing may be seen in skin folds or as a result of the extremities being in a particular position, e.g., rhombus-shaped sparing in the elbows if the arm is bent.

Intense livor mortis may be observed in cases of sudden death and persistent fluidity of postmortem blood. Any evaluation of livor mortis needs to take several factors into consideration (Table 3.4).

The first manifestation of livor mortis can be expected 20–30 min following irreversible cardiovascular arrest, initially as bright-red spots that subsequently become confluent and turn bluish-violet

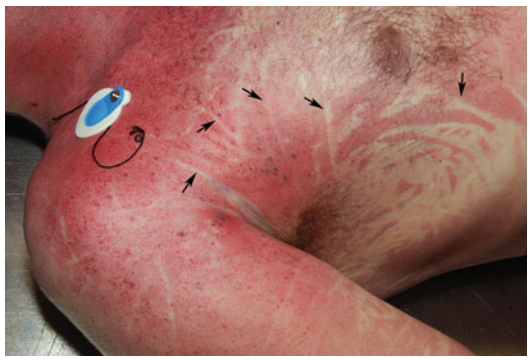

Fig. 3.3 Livor mortis. Linear sparing (*arrows*) within livor mortis due to folds in clothes

Table 3.4 Evaluating livor mortis

Criterion	Principal findings requiring clarification
Extent	Patchy or already confluent? Only on the back or both sides of the back up to the anterior axillary line?
Localization	Is it consistent with the position in which the body was discovered? Does it match a supine position? Is there a sock- or glove-shaped distribution in the upper and lower extremities in a body found in an upright position (hanging)?
Sparing	Is it consistent with the supporting surfaces? Are there other distinctive areas of sparing?
Pattern of livor mortis	Due to the contours of supporting surfaces or other forms of local compression?
Intensity	Is discoloration livid or pale?
Blanching on the application of pressure	Does livor mortis blanche when pressure is applied? Does blanching occur with blunt pressure (thumb) or only sharp-edged pressure (pincette or fingernail)? Does livor mortis remain fixed after turning the body or does it shift?
Color	Purplish to reddish violet? Pale reddish? Bright red? Brownish red? Greenish? Cold conditions, such as keeping a body in cold storage, cause a shift in the oxygen-binding curve and reoxygenation of hemoglobin, hence bright-red livor mortis; the same phenomenon is seen in hypothermic death

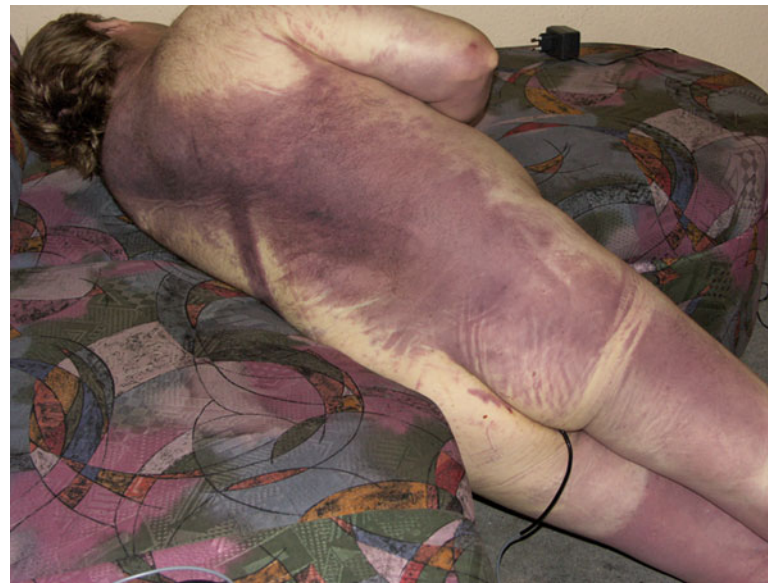

Fig. 3.4 The distribution of livor mortis is consistent with the (former) position of the body on the sofa with linear sparing matching the folds in the sofa fabric

Table 3.5 Color and intensity of livor mortis

Color	Etiology
Bright red	(a) As a result of CO intoxication (carboxyhemoglobin formation): cherry-red livor mortis, but only at COHb values of >30 % (b) In the case of hypothermic death and/or postmortem cold storage: renewed bright-red livor mortis due to oxygen diffusion through the skin and hemoglobin reoxygenation
Purple	Following early postmortem interval due to oxygen consumption
Brownish red	Intoxication from methemoglobin-forming agents, e.g., nitrite, nitrate
Greenish	Intoxication from sulfur, sulfhemoglobin formation
Pale	Due to blood loss or anemia

Any assessment of livor mortis requires good lighting conditions!

in color, reaching a maximum extent at 12 h. However, the type, extent, and intensity of livor mortis are subject to significant variation. Livor mortis of only mild extent and intensity is often seen following blood loss, as in the case of internal or external exsanguination or anemia. The color of livor mortis can also vary (Table 3.5). Livor mortis blanches under blunt pressure from a finger for around 10–20 hpm, while sharp-edged pressure can produce blanching for longer periods of time.

A body is usually placed in a supine position for external examination or transportation. If the body was originally discovered in some other position, e.g., in a prone position, it is possible for the livor mortis that initially formed in anterior areas of the body to disappear either partially or completely and for new areas of livor mortis to form in posterior body areas.

Important: **If the position of a body is changed following its discovery**, **livor mortis may shift completely within the first 6 h post-mortem at average central European temperatures**. **A partial shift in livor mortis can be seen at between 6 and 12 h post-mortem**, **thus producing a "double" livor mortis process**.

Depending on the ambient temperature, a gradual formation of fine spots of accumulated blood (vibices) can be seen as a result of hypostatic settling of blood within an area of livor mortis (Fig. 3.5).

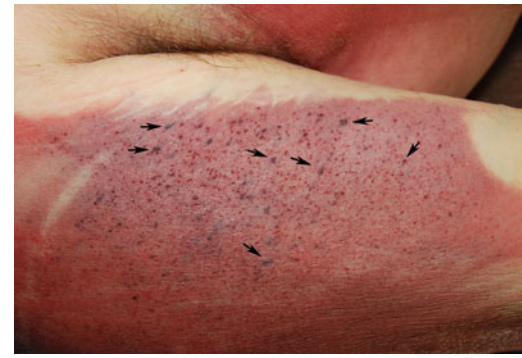

Fig. 3.5 Vibices. Abundant small purple vibices (*arrows*) within an area of livor mortis

Important: **If livor mortis seen on external examination is not consistent with the position in which the body was discovered**, **it can be assumed that the position was altered post-mortem**, **i.e.**, **the body was either turned or even transported**, **and hence the scene at which the body was discovered is possibly not the scene of death**.

Internal Livor Mortis. Hypostatic settling of blood in internal organs results in a finding of internal livor mortis at autopsy, e.g., particularly marked in the liver if the body is in a right lateral position, in the dorsal region of the lung in a supine position, or in the pelvic small bowel loops in an upright position (suspended position as in hanging).

3.4.5 Rigor Mortis (Postmortem Rigidity)

After livor mortis (the first conclusive sign of death), rigor mortis represents the second sign, the onset of which is seen at approximately 3–4 hpm (under normal central European conditions), depending on body weight, nutritional status, previous energy consumption, and ambient temperature. Nysten's rule is the accepted reference for the sequence in which rigor mortis affects the body (Table 3.6). In practice, however, variations from this sequence are observed.

Initially, the adenosine triphosphate (ATP) necessary for muscle contraction and decontraction can be resynthesized post-mortem

via the creatine kinase reaction and anaerobic glycolysis. This is no longer possible once the ATP level has fallen to below 85 % of its initial value, causing actin to bind irreversibly with myosin. This occurs primarily in those muscle groups where a significant fall in glycogen reserves has taken place, e.g., lower extremities while running a marathon. Rigor mortis affects both skeletal and smooth muscles, as well as the pupils and hair erector muscles, the latter causing so-called goose bumps (cutis anserina).

Assessing Rigor Mortis. In practice, the presence and intensity of rigor mortis are subjectively assessed by testing the mobility of joints and the resistance this produces. Fully established rigor mortis can generally only be "broken" manually by the examiner with the use of significant force. To assess the overall intensity of rigor mortis and any possible changes (increase in or resolution of rigor mortis), it is mandatory to examine multiple joints: elbows, knees, hips, jaw, and fingers.

Renewed-Onset Rigor Mortis. While on the one hand the onset of rigor mortis varies in time depending on the muscle group, glycogen level, and ambient temperature, on the other not all muscle fibers of a muscle belly are necessarily affected immediately. Thus, on testing, it is possible to identify rigor mortis that can be stretched or "broken" by force, only for rigor mortis to occur in other as yet unaffected muscle fibers.

Important: **The phenomenon of renewed-onset rigor mortis after prior stretching can generally be seen for up to 6–8 hpm**.

Rigor Mortis Resolution. As with its onset, the resolution of rigor mortis is to a great extent temperature-dependent. At normal central European ambient temperatures, rigor mortis can be expected to resolve after 2–3 days. For this to happen, proteolysis and the release of actin filament from the *Z* lines, biochemically accompanied by a rise in ammonia levels, need to take place. Table 3.6 provides an overview of the course of rigor mortis.

Table 3.6 Characteristics of rigor mortis

Onset	Usually after 2–4 h, earlier at high temperatures and possibly significantly later at low temperatures
Sequence (Nysten's rule)	Jaw, neck, upper extremities, trunk, lower extremities *Exception*: earlier onset possible following a drop in glycogen during the agonal phase
Testing	Subjective, by testing the mobility of several joints
Breaking	Possible in the early postmortem phase
Renewed onset	After breaking of rigor mortis in the early postmortem phase, renewed onset can be seen for up to 6–8 hpm
Resolution	Strongly temperature-dependent, usually starting after 1–3 days and completed after 2–5 days, possibly even after 2–3 weeks in low ambient temperatures
Extent	Also includes smooth muscles, e.g., pupils, as well as the hair erector muscles ("goose bumps" or cutis anserina)

3.4.6 Reduced Body Temperature

Besides livor mortis and rigor mortis, reduced body temperature is the most significant finding in terms of any forensic estimation of the postmortem interval. Body temperature does not usually drop instantly post-mortem; instead, a postmortem temperature plateau lasting 2–3 h is primarily seen. In the first instance, a radial temperature gradient between the core and the surface of the body needs to develop; only then can a continuous drop in core body temperature be assumed to take place at a steady ambient temperature. The cooling curve follows an exponential course according to Newton's law of cooling, whereby it levels off before ambient temperature and body temperature equalize, producing a largely sigmoid postmortem temperature curve.

The mechanism by which postmortem body temperature and ambient temperature equalize involves conduction and convection, as well as radiation and water evaporation to a lesser extent. These cooling characteristics prompted the development of a nomogram (according to Henssge), which enables an estimation of postmortem interval on the basis of a single deep rectal temperature and simultaneous measurement of the ambient temperature, assuming body weight is known and taking other parameters into consideration by applying a corrective factor (Figs. 3.6a, b and 3.7).

Using the rectal temperature time of death nomogram, the two simultaneously measured

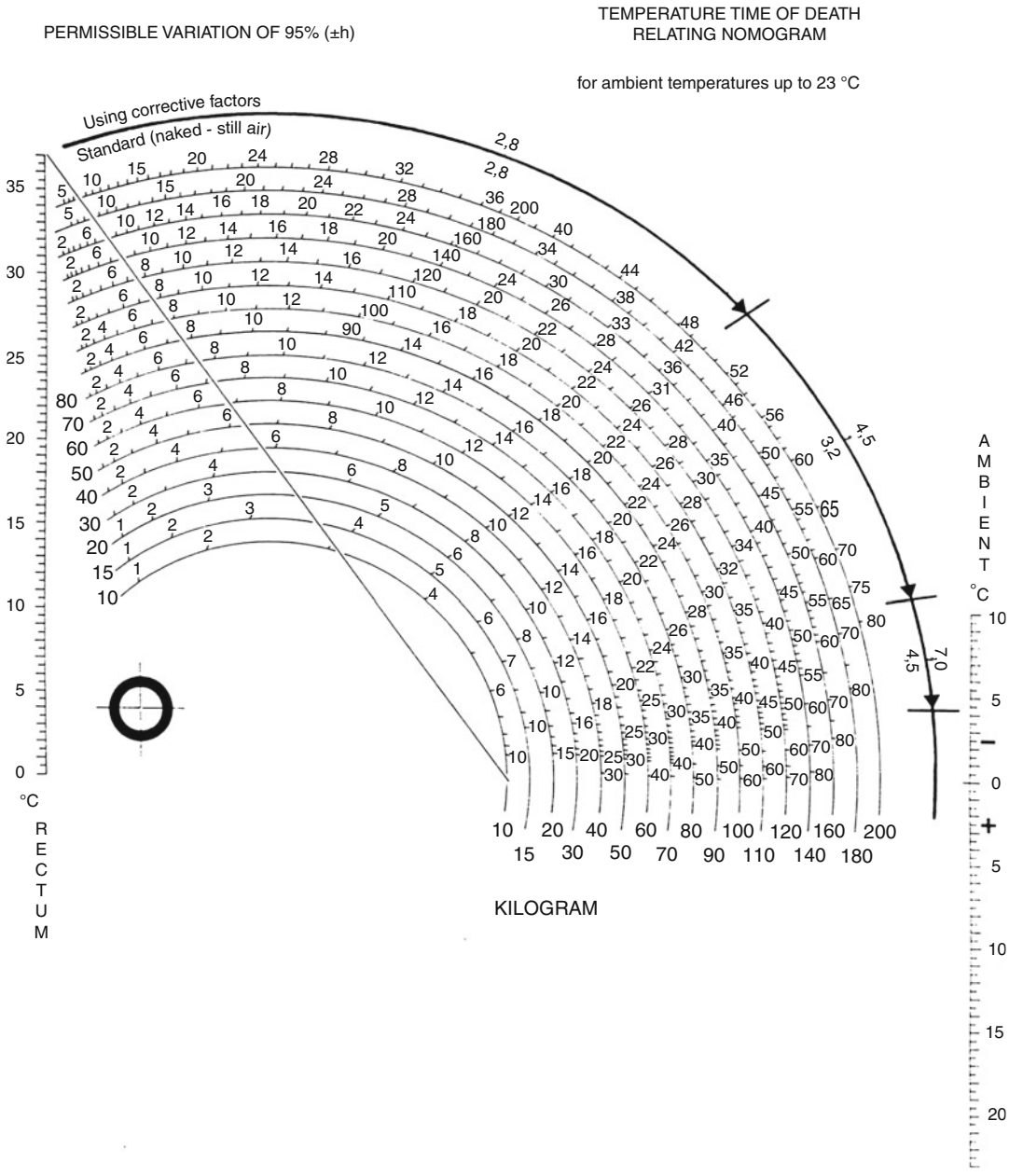

Fig. 3.6 (**a**) Henssge's nomogram method for estimating time since death from a single rectal temperature where the environmental temperature is below 23 °C (Henssge et al. 2002). (**b**) Henssge's nomogram method for estimating the time since death from a single rectal temperature where the environmental temperature is above 23 °C (Henssge et al. 2002). The nomogram is related to the chosen standard, that is, naked body extended lying in still air. Cooling conditions differing from the chosen standard should be adjusted by corrective factors of the real body weight, giving the corrected weight by which the death time is to read off. Factors below 1.0 may correct conditions accelerating the heat loss of a body, and factors above 1.0 may correct thermal isolation conditions (see Table 3.7)

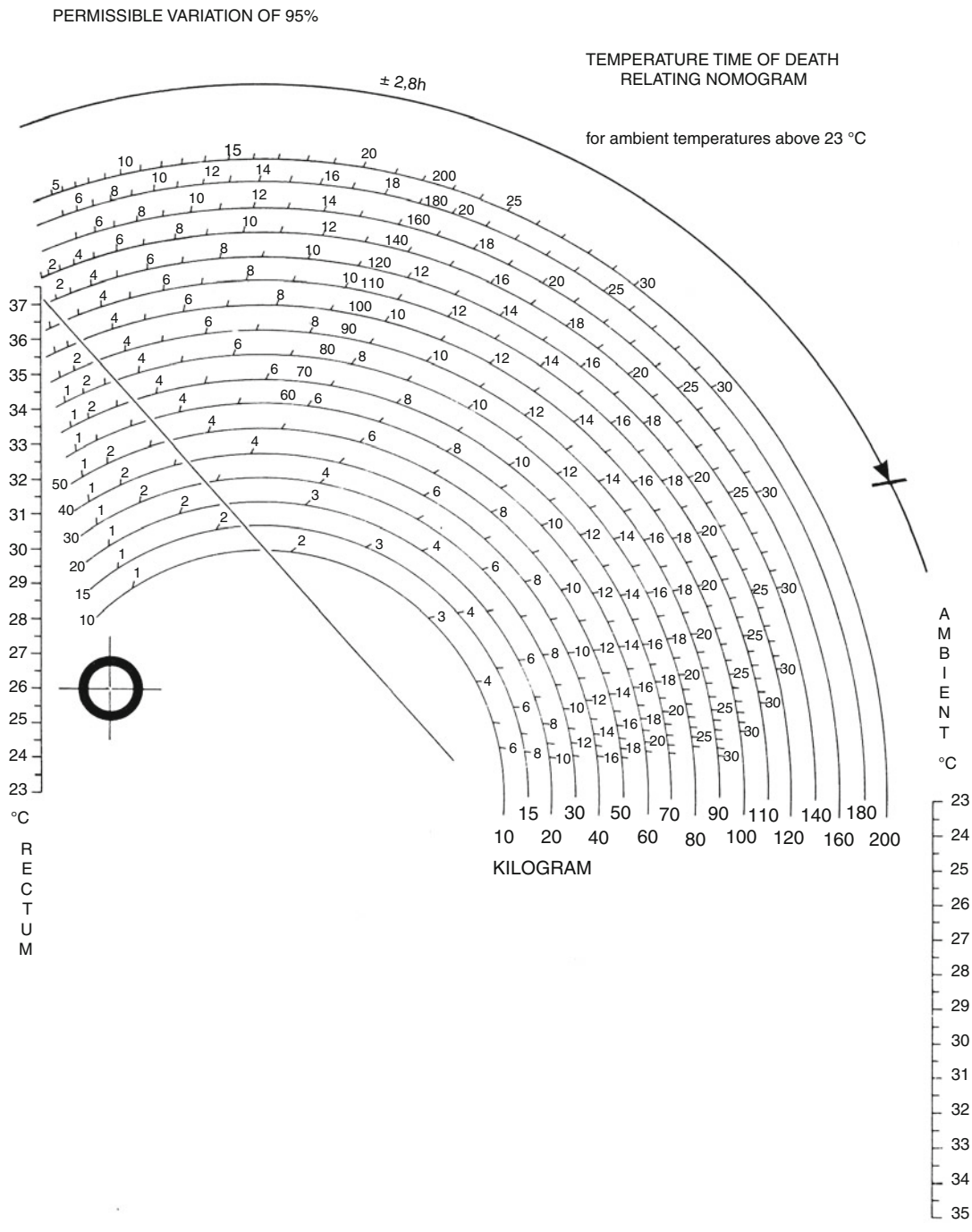

Fig. 3.6 (continued)

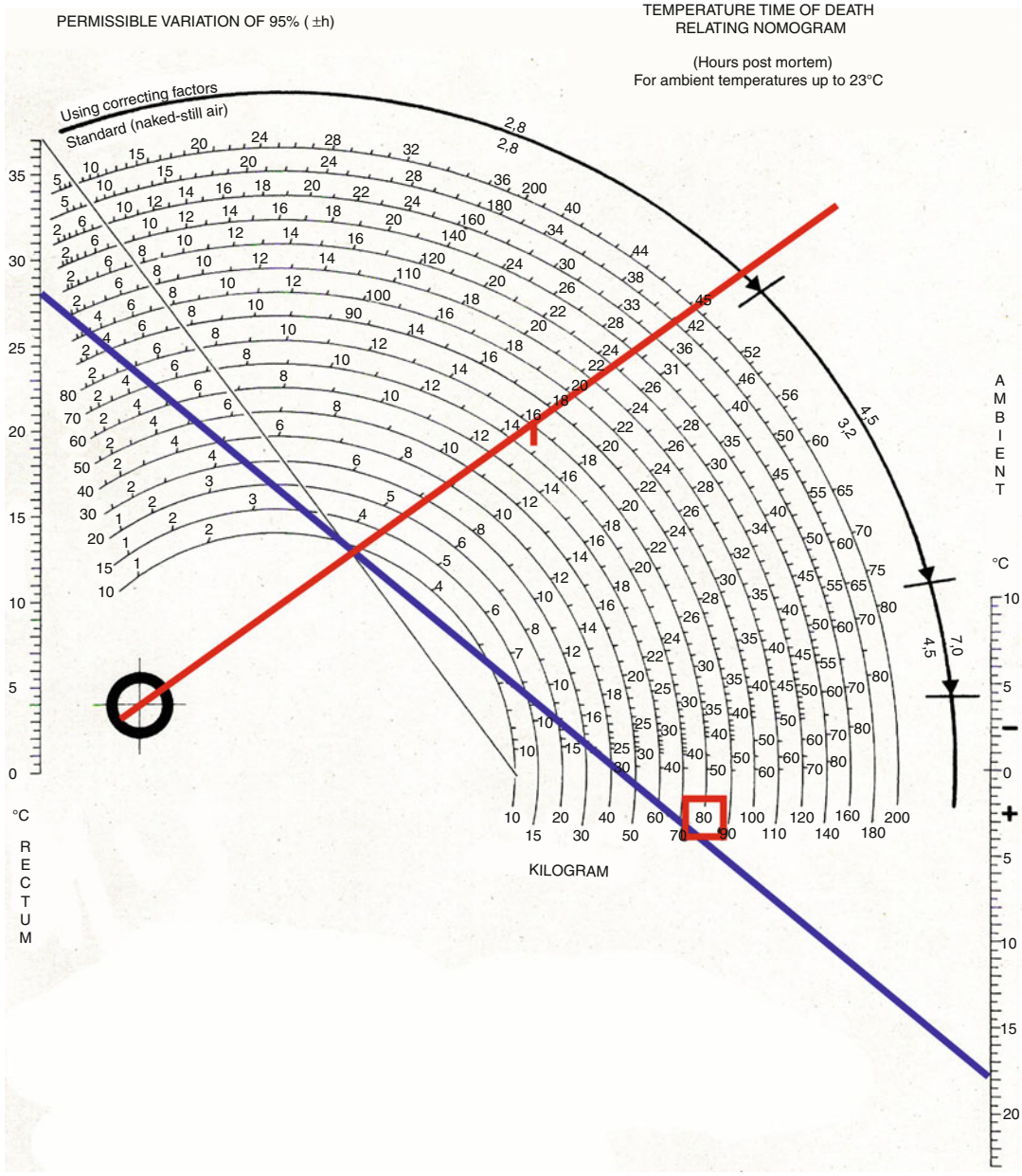

Fig. 3.7 Henssge's nomogram method for estimating time since death. A rectal temperature of 28 °C and an ambient temperature of 18 °C at a bodyweight of 80 kg yield an average time since death of approximately 16 h with 95 % tolerance levels of ±2.8 h (Modified from Reimann et al. (1990))

temperatures—ambient temperature and deep rectal temperature—are entered into the nomogram and connected by a straight line. This line intersects a fixed diagonal. A straight line is then drawn through the point of intersection of the diagonal and the first straight line, thus interconnecting the two temperature scales. The approximate time of death can be read from the semicircle for the relevant weight; the outer semicircle gives the 95 % confidence intervals.

Table 3.7 Empirical body weight corrective factors for bodies of average weight (reference, 70 kg)

Dry clothing/covering	In air	Corrective factor	Wet clothing/covering wet body surface	In air	In water
		0.35	Naked		Flowing
		0.5	Naked		Still
		0.7	Naked	Moving	
		0.7	1–2 thin layers	Moving	
Naked	Moving	0.75			
1–2 thin layers	Moving	0.9	≥2 thick layers	Moving	
Naked	Still	1.0			
1–2 thin layers	Still	1.1	2 thick layers	Still	
2–3 thin layers		1.2	More than 2 thick layers	Still	
1–2 thick layers	Moving or still	1.2			
3–4 thick layers	Still	1.3			
More thin/thick layers	No effect	1.4–1.8			
Thick bedspread		2.4			
+ clothing combined		2.8			

From Henssge et al. (2002)

Note: For the selection of a corrective factor (c.f.) for any case, only the clothing or covering of the lower trunk is relevant!

Insulating bases (e.g., thick foam upholstered bases) slow down the cooling process even in naked bodies up to a c.f. of 1.3; bases which accelerate the cooling process (e.g., concrete base of a cellar) require c.f. of around 0.75 for naked bodies or reduce c.f. for clothing by 0.1–0.2 units

However, conditions that may accelerate or slow down cooling should be taken into consideration by using empirically found body weight corrective factors (see Tables 3.7 and 3.8). In practice, two rectal temperature time of death nomograms are applied: one for ambient temperatures up to 23 °C and one for ambient temperatures from 23 °C.

Important: **The following rule of thumb applies**: **at average central European room temperatures with average clothing and physical condition and following a temperature plateau lasting 2–3 h**, **a postmortem drop in body temperature of 0.5–1.5 °C/h is seen**.

In addition to ambient temperature and deep rectal temperature, any factors that may influence cooling conditions are of relevance in the forensic estimation of time since death, including:

- Initial body temperature (preexisting increase in temperature or sepsis, previous sauna use or hot bath?).
- Physical condition/body proportions (body weight, density of subcutaneous fatty tissue).
- Body fat content (cachexia? obesity?).
- Condition of clothing: Are clothes dry or damp (one, two, or more layers)?
- Covering on the body.
- Body position or posture: Extended or curled up.
- Air and/or wind conditions.
- Located in fluid (water in particular; still or flowing fluid?).
- Proximity to a heat source (heater, spotlight, etc.).
- Heat-conducting or heat-insulating supporting surface (stone floor, carpet?).
- Large surface–volume ratio, e.g., in infants.
- Changes at the scene where the body is found, e.g., windows opened, heating or air conditioning switched off.

If it is clear that the postmortem interval needs to be established, ambient temperature and deep rectal temperature (possibly also the temperature of the surface supporting the body) should be measured as promptly as possible. If there is a significant difference between the temperature of the air and the surface temperature, use the mean. Deep rectal temperature (at least

Table 3.8 Dependence of corrective factors on body weight under strong thermal insulating conditions

Cooling conditions	Real body weight (kg)																	
	4	6	8	10	20	30	40	50	60	70	80	90	100	110	120	130	140	150
										1.3								
Clothing	1.6	1.6	1.6	1.6	1.5					1.4					1.3	1.2	1.2	1.2
Multiple layers	2.1	2.1	2.0	2.0	1.9	1.8				1.6				1.4	1.4	1.4	1.3	1.3
Bedcover	2.7	2.7	2.6	2.5	2.3	2.2	2.1	2.0		1.8			1.6	1.6	1.6	1.5	1.4	1.4
	3.5	3.4	3.3	3.2	2.8	2.6	2.4	2.3		2.0		1.8	1.8	1.7	1.6	1.6	1.5	1.5
	4.5	4.3	4.1	3.9	3.4	3.0	2.8	2.6	2.4	2.2	2.1	2.0	1.9	1.8	1.7	1.7	1.6	1.6
Clothing +	5.7	5.3	5.0	4.8	4.0	3.5	3.2	2.9	2.7	2.4	2.3	2.2	2.1	1.9	1.9	1.8	1.7	1.6
Bedcover	7.1	6.6	6.2	5.8	4.7	4.0	3.6	3.2	2.9	2.6	2.5	2.3	2.2	2.1	2.0	1.9	1.8	1.7
Duvet	8.8	8.1	7.5	7.0	5.5	4.6	3.9	3.5	3.2	2.8	2.7	2.5	2.3	2.2	2.0	1.9	1.8	1.7
	10.9	9.8	8.9	8.3	6.2	5.1	4.3	3.8	3.4	3.0	2.8	2.6	2.4	2.3	2.1	2.0	1.9	1.8

From Henssge et al. (2002)
Example: real body weight, 20 kg; chosen corrective factor (reference 70 kg), 1.6. Using a corrective factor of 1.9 results in a corrected body weight of 38 (40) kg

8 cm into the anal canal) should be measured using a special thermometer. Factors affecting cooling conditions need to be recorded (e.g., strong radiation—sun—cooling system, no high thermal conductivity of the surface beneath the body, no strong fever, or general hypothermia). The place of death must be the same place as where the body was discovered. Although body weight can be estimated at the scene where the body is found, a conclusive recording should be made when body weight is measured accurately prior to autopsy. The body weight corrective factor also needs to be estimated according to the information in Tables 3.7 and 3.8 before the relevant data can be entered in the nomogram. Personal experience is needed and for the selection of the corrective factor of any case, only the clothing or covering of the lower trunk is relevant. Known changes should be taken into account or should be evaluated, e.g., a change of the ambient temperature (the mean ambient temperature of the period in question, contact the weather station).

The nomogram cannot be used if a temperature-based estimation is impossible. This may be the case in the presence of sources of heat (radiation, underfloor heating) or cooling factors near the body or in rapidly alternating environmental temperatures.

3.5 Special Postmortem Changes

Drying of tissues in the postmortem absence of transudation and sweat secretion or moistening of the skin and mucous membranes is a particularly relevant postmortem change (Table 3.9). Drying is also caused by disruption to the moisture barrier following skin damage. Since the epidermis has no vascular supply, it is impossible to determine whether skin damage occurred ante- or post-mortem, hence the use of the neutral term "drying."

The position of the body (supporting surfaces, pressure points, etc.) or previous ante- or postmortem events should be able to provide an adequate explanation for the shape of skin drying artifacts.

Table 3.9 Common postmortem drying artifacts in the skin and exposed mucous membranes

Location	Cause, shape, and color
Lips	Linear drying along the border between the skin and mucous membranous part of the upper and lower lips
Tongue	Brownish drying if the tongue protrudes or if the mouth is open
Tip of the nose	Brownish drying and hardening
Scrotum	Brownish drying and hardening
Labia majora	Brownish drying and hardening
Cornea	Transverse linear drying if eyes are open post-mortem in a slit-like manner
Finger pulp/ extremities	Wrinkled, reddish-brown drying and hardening
Skin abrasions	Reddish-brown drying, e.g., extensor side of the knee following a fall. Also, skin abrasions incurred post-mortem, possibly during transportation of the body, same manner of drying
Shaped areas of drying	Showing the shape or contour of the affected area, e.g., defibrillator marks, as well as (ligature/manual) strangulation marks. Shaped areas of drying may reflect the contour of the implement used

3.6 Animal Scavenging

In addition to postmortem drying, very particular postmortem changes can be seen as a result of animal scavenging, even indoors; cats and dogs, as well as insects, can be responsible for these changes (see Fig. 3.8). Bird pecking may mimic stab wounds. Defects caused by animal scavenging show no underlying hemorrhage. The shape of defect margins may provide information about the kind of animal, e.g., rodent. In some cases, damage or defects caused by animal scavenging may initially arouse suspicion of homicide. Dogs can cause striated, reddish-brown, dried-out skin detachment as well as deep soft tissue defects (Fig. 3.8a, b).

Animal scavenging generally begins at unclothed and freely accessible points on the body (head, neck, and hands). Small vertebrates, in particular rodents like mice and rats, start scavenging at easily accessible areas such as the nose,

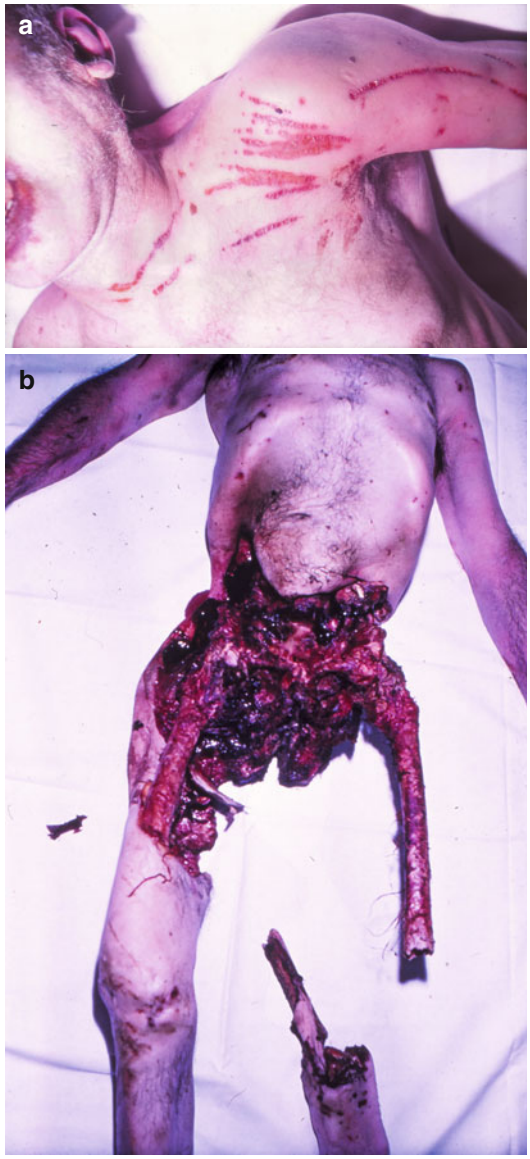

Fig. 3.8 (**a**) Striated and dried-out skin abrasions following dog predation (Alsatian). (**b**) Extensive traces of scavenging next to the body after it had been enclosed at home for an extended period of time

ears, lips, and fingertips. Rats tend to have a predilection for eyes, while fish scavenging defects are generally seen on bodies recovered from water. Typical evidence of gnawing and bites includes wounds with gently curved margins (Fig. 3.9a, b), occasionally also parallel wound margins. Lacerations and scratches are usually caused by canines, incisors, or claws. In the case

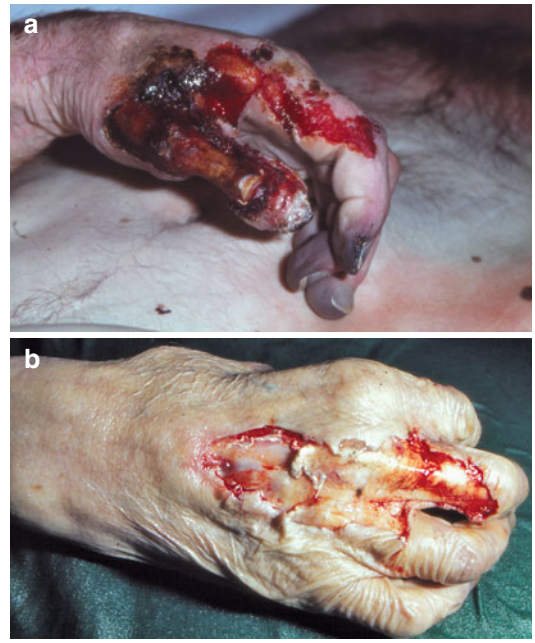

Fig. 3.9 (**a**, **b**) Rat scavenging defects on the hand with gently curved wound edges

of advanced animal scavenging, body parts may be carried away, for example, by foxes or wild boar. Birds tend to cause hole-like or small stab-like defects with the tips of their beaks.

3.7 Advanced Postmortem Changes

Following an early phase, decomposition advances at varying rates depending on temperature and moisture levels, among other factors, starting with autolysis and putrefaction and culminating in skeletonization.

Autolysis: The breakdown of organic structures by the body's own enzymes. Enzyme-rich internal organs (e.g., the pancreas) are the first to be affected, followed by other internal organs depending primarily on temperature. Autolytic processes include among others:

- Autodigestion of the gastric mucosa, possibly leading to perforation of the stomach wall and leakage of stomach contents into the abdominal cavity.

- Postmortem autodigestion of the pancreas.
- Progressive disappearance of membrane functions, associated with an extracellular rise in the potassium concentration and a drop in the sodium chloride concentration.
- A drop in pH value and a rise in lactate concentration.

Autolytic or postmortem biochemical processes vary greatly depending on the storage conditions of the body, on the temperature, on tissue moisture levels, etc. Thus, it is understandable that clinical–chemical laboratory diagnosis is sometimes barely possible and that laboratory values valid antemortem can generally only be used as references values to a limited extent. Case study investigations, however, have shown that values measured in the early postmortem phase following acute metabolic decompensation in diabetes (glucose, lactate, HbA1c) correlate well with levels measured antemortem and are of diagnostic value.

Putrefaction: The process of primarily bacterial destruction involving aerobic and anaerobic components and accelerated by warmth and moisture. The process of bacterial breakdown ends partly with H end products, that is to say, primarily hydrocarbons (CH_4), H_2S, and NH_3, and partly O_2 end products as a result of oxidative breakdown on the surface of the body.

The bacteria involved in the putrefaction process come from the skin surface, the airways, the gastrointestinal tract (physiological intestinal flora), the genital region, and the conjunctivae. Pathogenic germs, for example, in a deceased septic individual, can accelerate putrefaction. Common putrefaction agents include *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Bacillus subtilis*, as well as *Escherichia coli* bacteria and clostridia; fungi may also be involved.

The first sign of putrefaction is green discoloration of the skin, often starting in the right lower abdomen due to sulfhemoglobin formation in the presence of oxygen (Fig. 3.10).

Hemolysis in subcutaneous veins as well as the postmortem spread of bacteria via the vascular system causes "marbling," whereby the venous network becomes visible through the skin (Fig. 3.11).

Other simultaneous putrefaction-related changes to the body are seen, including:

- Swelling of the abdomen, eyelids, and mouth due to putrid gas formation.
- Tongue protrusion due to putrid gas pressure.

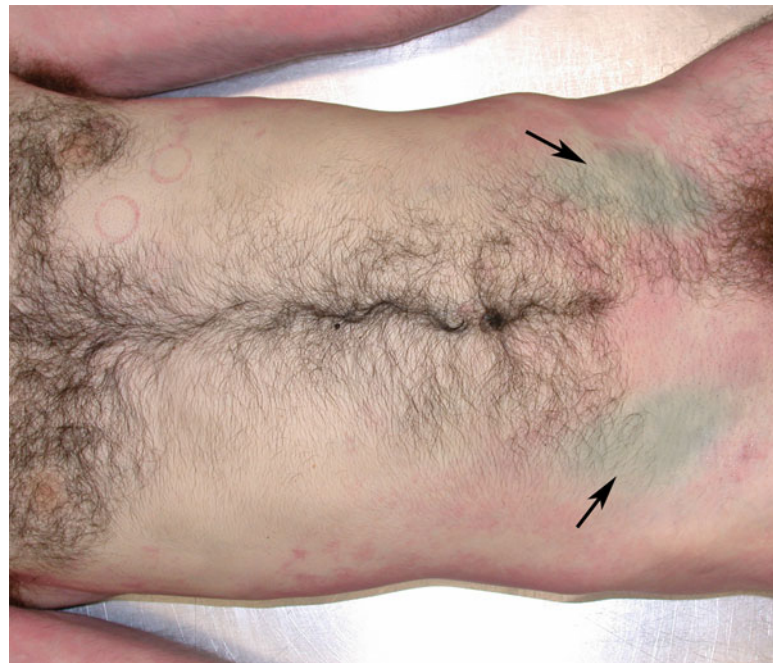

Fig. 3.10 Putrefaction. Early greenish putrefaction of the skin in the right and left lower abdomen (*arrows*)

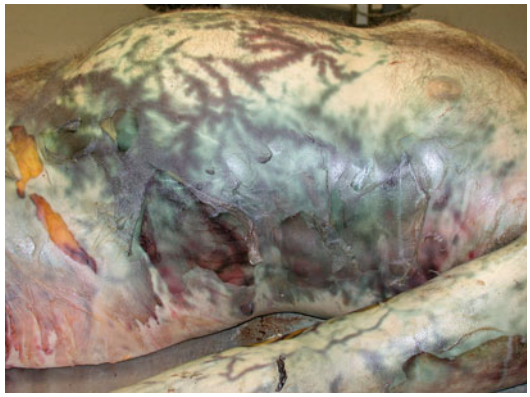

Fig. 3.11 "Marbling," whereby the venous network becomes visible through the skin. Postmortem putrefaction with abdominal distension due to gas formation, marbling, and the rupture of previously fluid-filled putrid blisters

- Escape of feces from the anus.
- In the case of pregnancy: Expulsion of the fetus from the uterus (postmortem fetal extrusion, "coffin birth").
- Generalized crepitus caused by in the tissue.
- Formation of fluid-filled putrid blisters between the epidermis and corium.
- Hair as well as finger- and toenails become increasingly easy to remove.
- Liquefaction or oily transformation of fatty tissue within the body.
- Proteolysis and formation of biogenic amines and alkaloids (ptomaines).
- Brain tissue becomes increasingly soft and liquid.
- Blisters penetrate to the internal organs, which take on the appearance of being permeated by foamy putrid blisters ("foamy organs") or honeycombed (e.g., "honeycomb liver").

The sometimes considerable abdominal protrusion that results from the buildup of putrid gases can cause postmortem fetal extrusion in pregnant women ("coffin birth") or rectal prolapse due to gas pressure (Fig. 2.4).

The degree of putrefaction does not permit any inferences to be made about time since death.

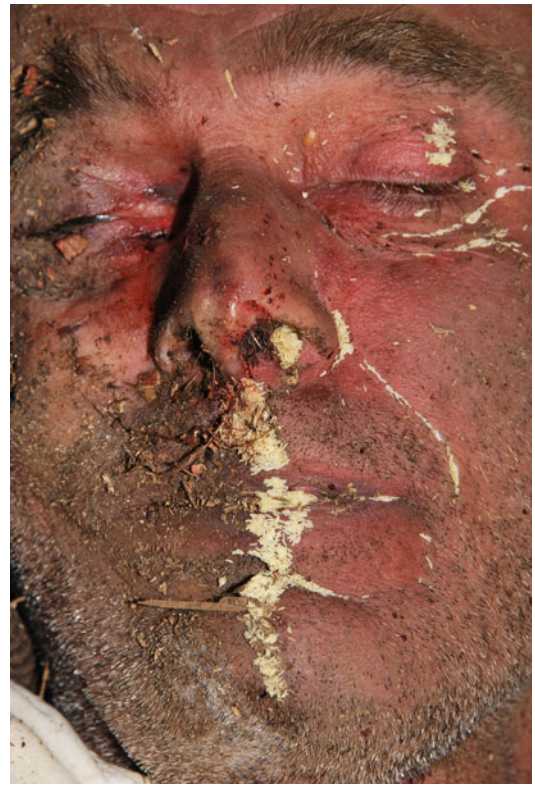

Fig. 3.12 Fly egg deposition. Abundant whitish-gray fly eggs deposited on the eyelids, nostrils, and mouth region post-mortem

Although in principle Casper's law is valid, it should be applied with caution.

Important: **Casper's law states that 1 week exposed to air = 2 weeks in water = 8 weeks buried in earth**.

Postmortem changes to a body may also be caused by fly egg deposition and maggot formation (Figs. 3.12 and 3.13).

The development cycle of flies or maggots becomes important in the estimation of postmortem intervals spanning long periods of time.

Whereas a body buried in soil can be expected to become largely skeletonized—depending on environmental conditions—after 20–30 years, certain environmental factors may be responsible for a body remaining in a relatively good state of preservation. Mummification is of particular relevance in this context.

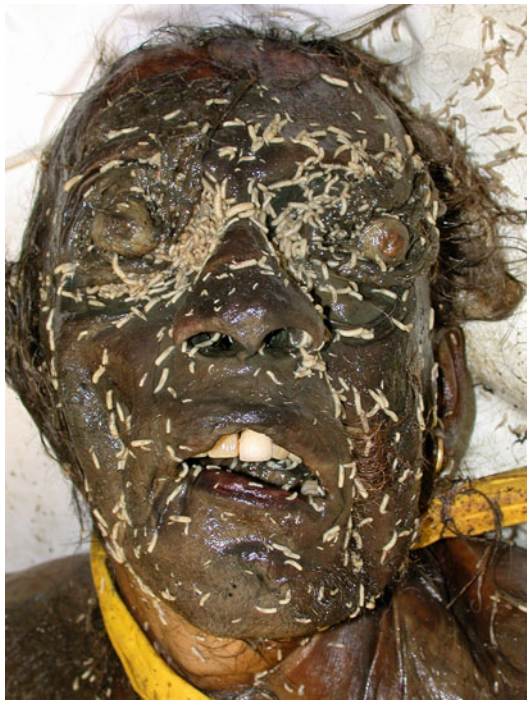

Fig. 3.13 Advanced putrefaction and extensive maggot colonization

Mummification: Preservation of the body due to rapid drying out, generally as a result of dry currents of hot or cold air (natural mummification). The process may begin as early as 1 week post-mortem, with partial mummification seen after several weeks and complete mummification after several months.

Mummification occurs when the moisture that promotes bacterial growth is absent; the skin dries out and becomes leathery. It is often possible to make numerous findings in a mummified body, including, for example, trauma-related injury or a natural cause of death, such as cardiac tamponade following ruptured myocardial infarct. In cases of incomplete mummification or where environmental factors are not wholly conducive to mummification, colonization of the skin surface with whitish-gray fungi is not uncommon (Fig. 3.14).

Adipocere may develop under anaerobic, moist conditions, whereby unsaturated fatty acids degenerate into saturated fatty acids.

Fig. 3.14 Mummification. Patches of whitish-gray fungal colonization of facial skin in a case of incomplete mummification

Subcutaneous fatty tissue may be affected within a matter of weeks, muscles within a matter of months. The process of a body's complete transformation to adipocere can take between months and years. While on the one hand the partial preservation of bodies is seen under anaerobic conditions, it may also be seen in bodies found in bogs ("bog bodies") as a result of the preserving effects of humic and tannic acids. Tissue freezing is responsible for the remarkable state of preservation seen in glacier mummies.

3.8 Forensic Entomology

The presence of insect colonization on a body may be helpful in the estimation of time since death, assuming the species of insect can be precisely identified and its development cycle is known, e.g., the *Calliphora erythrocephala* (blue bottle fly) or *Musca domestica* (house fly). Under certain circumstances, flies may be found to have colonized the entire upper respiratory tract (Fig. 3.15).

Flies may lay eggs as early on as in the agonal phase; post-mortem they show a predilection for skin lesions, the mouth, nostrils, and corners of the eyes, beneath the eyelids, as well as moist areas of the body. Depending on the species and the ambient temperature or conditions, entomological analysis is able to identify the sequence of generations, enabling inferences to be made

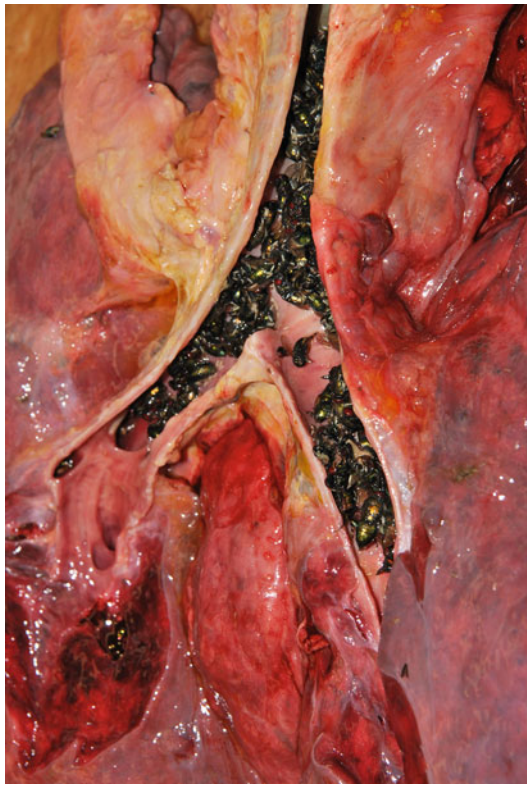

Fig. 3.15 Flies found at autopsy in the entire upper respiratory tract and extending to the main bronchi

Table 3.10 Stages of fly development

Stage	Duration
Egg deposition	24–48 h
Maggots	Over 10–12 days: firstly small then larger maggots showing typical sieve-like maggot scavenging defects to the skin, associated with urea production and proteolytic decomposition
Larvae	Larvae after 10–12 days
Pupae	Pupation following the larval stage for 10–14 days (Fig. 3.16)
New flies	New flies hatch at around 14 days following pupation; thus empty pupal cases indicate an minimum postmortem interval of 4 weeks

Note: These times may vary depending on the species and ambient conditions during the postmortem interval

about the postmortem interval. Thus, empty pupal cases indicate that at least one generation has reached maturity (Table 3.10).

Fig. 3.16 Fungal colonization and pupae

Important: **For the purposes of any forensic entomological estimation of time since death, several specimens of each maggot, pupal case, insect, beetle, etc., of varying appearance should be taken. A proportion of these are firstly killed by pouring boiling water over them and then stored in 70 % ethanol. The remaining specimens are stored alive at 4°C to enable breeding and subsequent species identification.**

3.9 Forensic Estimation of the Time of Death and Postmortem Interval

Any expert forensic estimation of time since death needs to take multiple parameters into consideration over and above livor mortis, rigor mortis, and postmortem determination of the ambient and deep rectal/core body temperature. In some cases, analyzing gastric contents may provide usual information against the background of normal digestive processes. Thus, findings that can only be made at autopsy may be highly relevant in terms of estimating the postmortem interval. Table 3.11 provides an overview of postmortem interval estimation.

A more accurate estimation of time since death may be possible with the help of information provided by the investigating authorities, relating, for example, to when the deceased was last seen alive, last emptied their letter box, or made their last telephone call.

Table 3.11 The main criteria for expert forensic estimations of time since death

Criterion	Approximate postmortem interval[a]
Livor mortis	Begins approximately 20–30 min post-mortem on the neck, becoming confluent within 30–120 min, culminating at 6–12 hpm, blanches under digital pressure for 10–12 hpm
Potential for livor mortis to redistribute	Up to 12–24 hpm, sometimes fixed at the original site after 6 hpm ("double" livor mortis)
Rigor mortis	Begins in the jaw at 2–4 hpm, culminating at 6–8 hpm, onset more rapid in hot and slower in cold conditions
Recurrence of rigor mortis	Renewed rigor mortis following breaking for up to 6–8 hpm
Resolution of rigor mortis	Highly temperature-dependent, usually beginning after 2–3 days, complete after 3–5 days, or after 2–3 weeks at low temperatures
Electrical excitability of mimic muscles	Complete ipsilateral contraction propagation 1–6 hpm, only proximal to electrodes up to 8 hpm (5–22 hpm)
Mechanical excitability of muscles	Zsako muscle phenomenon 1.5–2.5 hpm, idiomuscular contraction 4–12 hpm
Pharmacological stimulation of iris smooth muscle	Possible for up to approximately 20 hpm with mydriatics or miotics, rarely up to 40 hpm
Eye vitreous humor potassium level	Potassium levels rises continuously after death, enabling good time of death estimations in the first 2–3 days post-mortem
Degree of urinary bladder filling	Rule of thumb in the case of death during the night: Empty urinary bladder = death occurred in the first half of the night Full urinary bladder = death occurred in the second half of the night
Drop in body temperature	Postmortem temperature plateaus for 2–3 hpm, followed by a drop in temperature of 0.5–1.5 °C/h
Estimating postmortem interval using Henssge's nomogram	Approximate postmortem interval calculated in hpm following simultaneous measurement of core body temperature and ambient temperature, taking other parameters such as body weight into consideration and possibly also applying situation-specific corrective factors
Gastric emptying	Helpful if the time of last food intake is known. A light meal remains in the stomach for 90 min on average, a medium-sized meal for around 3 h, and a heavy meal for around 4 h. (important: identity of specific food components)
Green putrefaction of the skin, often starting in the right lower abdomen	From approximately 48–72 hpm
Marbling (venous network becomes visible through the skin)	From approximately 48 hpm
Green discoloration of the entire abdominal skin, sunken eyeballs	Approximately 1 week
Putrid blisters, generalized body distension	Approximately 2 weeks
Skin slippage, hair and nails easily removable, distinct facial swelling (individual is unidentifiable), putrid blisters in soft tissue (crepitation on palpation)	Approximately 3–4 weeks
Entomological postmortem interval estimation	Postmortem interval estimation possible after weeks depending on the species, stage of growth, and ambient conditions; up to 10 specimens of each fly, insect, pupa, maggot, etc., should be taken and stored in 70 % ethanol
Adipocere	As early as after 3–5 weeks in water or in heat under anaerobic conditions, after months up to a year when buried in earth
Skeletonization	Generally only after 20–30 years when buried in earth
Mummification	Possibly already mild after 1 week, partial after several weeks, and complete after months; long-term preservation, e.g., glacier mummies

[a]The information given here refers to average central European environmental conditions; significant variations are possible in individual cases!

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Autopsy (Syn. Postmortem Examination, Necroscopy)

4

Case Study

Following the death of a 48-year-old man of the Muslim faith, the deceased's relatives refused on religious grounds to grant permission to carry out a clinical autopsy. Since the manner of death was given as unexplained on the death certificate, the public prosecutor applied to the court for an order to perform a medicolegal autopsy. The judge was of the opinion that the interests of criminal prosecution in the case of a crime associated with the man's death took precedence over the individual interests of the deceased and his relatives in terms of postmortem physical integrity, particularly since the autopsy procedure would be dealt with in a respectful manner. The request of the relatives, who had been previously heard, that partial autopsy involving the opening of only the abdominal cavity would be adequate was rejected by the judge on the grounds that the German code of criminal procedure expressly stipulates the opening of all three body cavities: head, chest, and abdomen.

The corpse is accorded respect in all cultural spheres and, according to the prevailing beliefs in many countries, has personal protection rights that continue to be effective post-mortem. Thus, the dignity of an individual is to be respected even after death. The religious beliefs of the deceased and the bereaved require special consideration. Although relatives have a right to care for the dead, they do not have unlimited rights of disposal. Therefore, according to civil law, a body cannot be sold and may not become an object of commercial trade ("res extra commercium"). A body can be subject to confiscation for the purposes of criminal proceedings.

Legal uncertainties about ordering a clinical—as opposed to a forensic—autopsy have resulted in a comparatively low autopsy rate in Germany. The legal basis for autopsy lies partly in the laws of the individual German states and partly in federal law on forensic autopsy and autopsy for epidemic prevention, as well as in insurance law and hospital admission contracts.

4.1 Situations Calling for Autopsy and Types of Autopsy

Autopsies have served numerous purposes for decades, including:

- Determining underlying disease and cause of death in the individual case
- Expanding scientific knowledge (e.g., metastatic behavior of malignancies)
- Assessing disease response to therapy
- Comparing autopsy findings with diagnostic findings
- Gaining insight into the evolution of new diseases (HIV infection, AIDS, SIRS, etc.)

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- Revealing and explaining hitherto unknown aspects of forensic criminological and insurance medicine
- In the education, training, and further training of medical students, medical assistants, and physicians in specialist medical training or other forms of further training

Moreover, the importance of autopsy for the purposes of determining cause of death, defending medical malpractice claims, and consoling surviving relatives is not to be underestimated.

4.1.1 Clinical Autopsy in the Pathology Department

Clinical autopsies are carried out in the pathology departments of clinics and hospitals. In many countries, and depending on the legal framework, clinical autopsy is only authorized if the deceased gave their consent ante-mortem (explicit consent) or when the procedure does not go against the wishes of the deceased and consent is given by close relatives (presumed consent).

The legal framework whereby an autopsy can only be carried out if no objection was expressed during life (explicit dissent) is rare. Clinical autopsy is sometimes possible in cases where the bereaved, once fully informed, express no objection and allow a legally defined deadline to expire (presumed dissent). Any breach of legislation may be considered in the eyes of criminal law as "disturbing the peace of the dead," particularly in cases where an autopsy is carried out against the express wishes of the deceased.

4.1.2 Autopsies Ordered by an Authority

An autopsy performed for the sole purpose of determining the cause of death by order of an authority, such as a public health office or other legally empowered institution, falls under the category of administrative autopsy, much like autopsy for statutory accident insurance purposes. This type of legal framework for carrying out autopsies was active for instance in the former German Democratic Republic in the form of a catalogue of indications which, when present, would prompt either the state or other authorities to order an autopsy, e.g., in all cases of death during pregnancy or shortly after childbirth or medical treatment. In many countries, authorities have the legal framework to call for an autopsy in cases where an infectious disease is suspected and the risk to the population needs to be evaluated.

4.1.3 Special Medicolegal Autopsies

A court calls for a medicolegal autopsy on the order of the public prosecutor's office; in urgent cases, the public prosecutor's office can itself call directly for an autopsy to be carried out. It is highly unusual for a court not to carry out an order from the public prosecutor's office. The latter has considerable discretionary powers in terms of applying for an autopsy, such that estimates for Germany put the rate of medicolegal autopsies at only every 10th–20th unexplained death, i.e., by no means do all bodies retained by the police undergo medicolegal autopsy.

According to the German code of criminal procedure, a medicolegal autopsy must be carried out by two physicians. The code of criminal procedure also makes provision for the extent of autopsy (opening of all three body cavities) and specifically regulates autopsies on neonates. When performing an internal examination on a neonate, investigative efforts should be targeted at revealing whether the child was alive during or after birth and whether it had reached sufficient maturity—or was at least fit—to survive outside the womb.

Other regulations stipulate that an external examination and autopsy be carried out with the greatest possible speed, since even small delays may compromise the reliability of any medical findings on the cause of death. Thus, if a criminal offense cannot be excluded, or if autopsy findings are expected to be called into question at some later date, an autopsy is as a basic rule called for by the public prosecutor's office. A medicolegal or court-ordered autopsy should be carried out to

establish cause of death particularly in the case of deceased individuals "held in custody or official detention," e.g., forced detention in a psychiatric institution, prison inmates, and individuals in police custody.

4.2 Basic Principles of Autopsy Techniques at Medicolegal Autopsy

Over the course of many years, nationally and internationally comparable autopsy dissection techniques have evolved in forensic institutes and/or pathology departments. So-called partial autopsies only take place in exceptional cases where relatives would otherwise refuse to grant their consent-in all other instances, the head, chest, and abdominal cavities are opened. Dissection can be extended to include all body regions according to the line of inquiry. The variable approach to dissection at autopsy is discussed in detail in the relevant textbooks. All autopsies should include recording basic data and taking specimens for further investigations. Data include height and weight, as well as the weight of the brain, left and right lungs, heart, liver, spleen, left and right kidney, and possibly also the thymus. The specimens listed in Table 4.1 should be taken at autopsy for chemical–toxicological and histological or immunohistochemical analysis.

Depending on the line of inquiry, it is often necessary to take additional specimens to those mentioned in Table 4.1 for chemical–toxicological and histological, immunohistochemical, or cytological

Table 4.1 Representative specimens to be taken at forensic autopsy for chemical–toxicological and histological–immunohistochemical analysis

Specimen/investigation	Chemical–toxicological	Histological/immunohistochemical/cytological
Brain (multiple specimens)	+	+
Heart (multiple specimens)	–	+
Liver (left and right hepatic lobe at minimum)	+	+
Spleen	–	+
Kidneys (right and left)	+	+
Thyroid gland (right and left)	–	+
Lungs (one specimen per lobe at minimum)	+ (headspace vials to detect, e.g., volatile gases)	+
Gastric contents (milliliters or grams)	+	+ (histological analysis of gastric contents possible)
Pancreas (head and tail region)	–	+
Adrenal glands (right and left)	–	+
Fallopian tubes and ovaries, testes, epididymides, seminal vesicles (right and left), uterus	–	+
Cardiac blood (ml)	+	–
Femoral vein blood (ml)	+	–
Bile fluid (ml)	+	–
Vitreous humor (ml)	+	–
Cerebrospinal fluid (ml)	+	+ (rare)
Urine (ml)	+	–
Smear specimens (oral, anal, vaginal, penile)	–	+ (particularly in the case of a suspected sex offense)
Muscles	+	+ (rare)

analysis; examples worthy of mention here include:

- Removal of areas of skin along with subcutaneous soft tissue with a radius of approximately 5 cm at gunshot entry and exit sites to possibly determine firing range
- Removal of skin and subcutaneous soft tissue due to unexplained puncture sites
- Removal of additional tissue specimens for diatom detection in the case of suspected death by drowning (lung, kidney, brain, and medullary bone)
- Eyeball removal for the histological detection of retinal bleeding in the case of, for example, suspected fatal shaken baby syndrome
- Taking irrigation fluid from the respiratory tract in the case of, for example, suspected aspiration of textile fibers from a cushion due to asphyxia with a soft cover
- As a basic rule, it is essential to take sufficiently representative tissue specimens for histological and immunohistochemical analysis in order to ensure that no findings escape detection (e.g., a sufficient number of myocardial and lung specimens to enable detection or exclusion of myocarditis or pneumonia)
- Additionally, representative specimens need to be taken from macroscopically abnormal areas

Also as an additional measure, any abnormal contents of preexisting body cavities need to be quantitatively recorded (e.g., blood in milliliters, blood clots in grams), most notably in the case of effusion fluid or blood in the chest cavities, pericardium, and abdominal cavity. Where appropriate, volumes should be given for the contents of cysts, in either joint cavities or blood-filled cavities, for instance, following décollement. Coagulated blood, for example, in the form of epidural or subdural hematoma, can be used to determine blood alcohol concentrations or detect toxins in order to establish whether a victim suffered impairment at the time of an incident, even if death occurred at some later point in time.

As a basic principle, all injuries and pathological organ and tissue lesions should be described according to localization, shape, structure, color, size, and anatomical correlation. Moreover, all major blood vessels (arteries and veins, possibly also the extremities), the central and peripheral airways, the extrahepatic biliary tree, the efferent urinary tract including ureters and bladder, as well as the pancreatic duct require dissection. A number of basic guidelines on dissection procedures at autopsy and their forensic background are discussed below. For a more detailed discussion, the reader is referred to the relevant literature.

4.2.1 Back, Arms, and Legs

Particularly in the case of previous trauma, most notably involving falls and blows, the back, arms, and legs require careful dissection. The same applies to traffic accident victims. Following shoulder-to-shoulder incision above the level of the shoulder blades with the body in a prone position, an additional incision is made in the body midline over the vertebral bodies, becoming Y-shaped at the lower portion of the coccyx over the buttocks and continuing along the posterior side of both legs. Incisions over the posterior parts of the shoulder can be extended to the arms, and incisions in the extremities to the feet (heels) or hands. As the outermost stratum, the dermis is dissected first, followed by subcutaneous fatty tissue, muscle fascias, and finally muscles. Since blunt force trauma from an anterior direction with the victim in a supine position primarily causes hemorrhage in the form of abutment injuries, the scapular spines, anterior shoulder blades, outer and inner edges, and tip of the shoulder blades, as well as the spinous processes of the vertebra, need to be exposed, while hemorrhage in neighboring soft tissue needs to be excluded (Figs. 4.1 and 4.2). Further sites of hemorrhage may be found over the coccyx and in soft tissue in the gluteal region, as well as projected to the hip bones as a result of impact injuries. Dissection of the deep musculature permits among others visualization of rib fractures involving bleeding into the intercostal muscles and around the ischiadicus nerve, as well as iliosacral joint separation. In the neck area, skin and cutaneous soft tissue dissection can be extended down to the cervical spine. Particularly in the case of traffic accident victims with décollement, extensive

blood-filled cavities are sometimes found; these cavities can serve to provide blood specimens for blood alcohol determination.

4.2.2 The Head and Head Cavity

To investigate the head cavity, a mastoid-to-mastoid incision across the parietal region is made with the body in a supine position and the scalp detached from the cranial vault by folding the flap backwards and forwards (Fig. 4.3). If external injury to the scalp is present, e.g., following blunt or sharp local trauma, the resultant hemorrhage can be visualized by making an incision in the subcutaneous soft tissue. The fascia of the temporal muscles can then be inspected, and these muscles detached from the bony cranial vault. Punctate as well as small or large irregularly shaped sites of hemorrhage may be seen on the inner surface of the scalp as a result of stasis following compression trauma to the neck or due to hypostatic hemorrhage resulting from a head-down position.

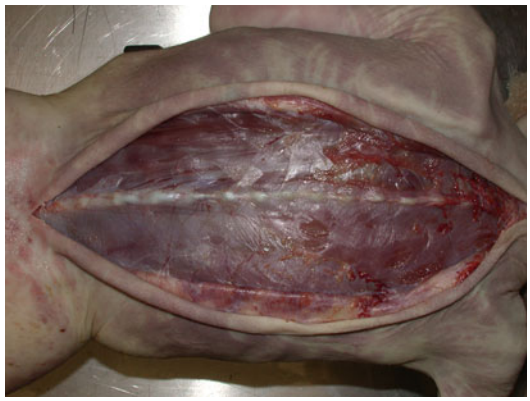

Fig. 4.1 Soft tissue dissection of the back to exclude hemorrhage extending to the spinous processes of the vertebra resulting from abutment injury

Before opening the cranium, the periosteum should be stripped from the skullcap in order to better identify any possible fissures, fractures, open sutures, hyperostosis, scarring, and necrosis.

The bony cranial vault is opened by making a cut using a circular saw. Electric oscillating saws or hand saws are used to this end. The use of undulating saws is contraindicated by the fact that, even when water and suction devices are used, aerosols and fine dust are produced and may be inhaled by those present at the autopsy. Since a significant portion of these dust particles are respirable, many pathologists and prosectors favor hacksaws, which produce only coarse non-respirable particles.

The saw cut should extend down to the dura mater, without, however, causing injury—which cannot always be entirely prevented. After removal of the bony cranial vault, fractures located above the occipital frontal circumference are visible, as are epidural hematomas. Either the

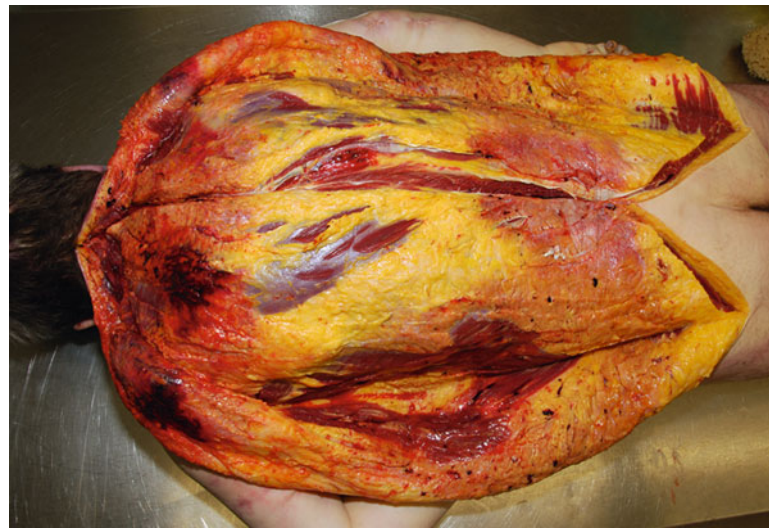

Fig. 4.2 Soft tissue dissection of the back revealing fresh hemorrhage extending to the left shoulder blade as a result of abutment injury

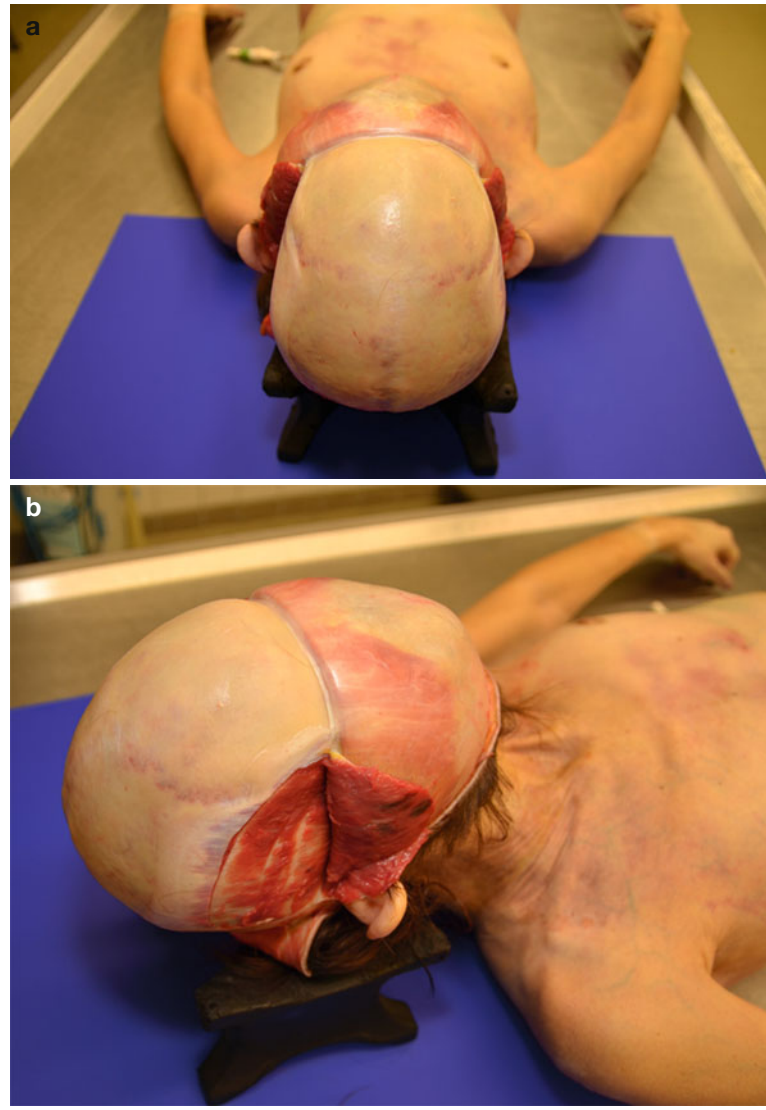

Fig. 4.3 (**a**) Detachment of the inner surface of the scalp and (**b**) the M. temporalis dexter

dura mater can then be cut open radially leaving the brain in toto intact or a horizontal flat cut extending from the level of the saw cut is made through the dura and brain, thereby permitting the upper portion of the brain and dura mater to be lifted in a cap-like manner, while at the same time affording a cross-sectional view of the cerebrum and both large brain ventricles. During dissection, particular attention should be paid to the bridging veins (superior cerebral veins) vulnerable to trauma, especially if the case history suggests acceleration trauma. Also of relevance is, for example, yellowish-brownish-ocher discoloration in the dura mater or brain tissue (here in the form of pseudocystic areas) as an indication of previous hemorrhage, with the subsequent detection of hemosiderin deposits using Prussian blue staining.

The lower portion of the brain, including the cerebellum, pons, and medulla oblongata, is removed together with the occipital lobes once both frontal and parietal lobes have been raised and the now visible tentorium cerebelli has been transected. If cerebrospinal fluid has not already been obtained by means of puncture, it can be taken at this point from the anterior basal cistern prior to opening the posterior cranial fossa. Cerebrospinal fluid gathers here once the

brainstem has been raised. Also prior to actual removal of the brain, specimens and smear samples can be taken for microbiological and viral analysis from the basal surface of the cerebrum and from the cerebellopontine angle, as well as from any sites demonstrating abnormally flat, usually grayish deposits (meningitis? lymphomatous meningitis? carcinomatous meningitis?).

Following removal of the brain, it is possible to visualize the circle of Willis at the brainstem and dissect its large branches. At the same time, the most frequent localizations of basilar artery aneurysms are visualized. If nontraumatic subarachnoid hemorrhage is present, a basilar artery aneurysm should be sought while still at autopsy by carefully rinsing blood away with water. Later, following fixation in formaldehyde, the blood will have hardened to such an extent that it can only be removed from the brainstem with great difficulty, with the added risk of producing artifacts.

Important: Horizontal sections of brain tissue are better suited to correlation with radiological, in particular computer tomographic findings than frontal sections.

The dura mater can be removed entirely either during or following removal of brain. Nondisplaced fractures of the skull base can then be identified, including, for example, fissures in the orbital roof.

"*Half-Moon*" *Incision*. This refers to a special dissection technique sometimes used in the case of neonates, infants, and young children. The cranial cavity is opened by means of two incisions running parallel to the sagittal plane, thereby exposing the cerebral falx and the cerebellar tentorium (Fig. 4.4). In particular, this enables visualization of tears in the cerebellar tentorium.

At forensic autopsy, the usually fresh brain is dissected by means of multiple sections in the frontal plane. The principle aim here is to detect hemorrhage, necrosis, and pseudocystic areas resulting from previous trauma. Pathology institutes sometimes fix the brain in formaldehyde for at least 1 up to 4 weeks prior to dissection and then undertake brain dissection separately—where available, at a neuropathological institute.

Important: Depending on the line of inquiry in the case of, for example, hypoxic brain injury survived for a prolonged period, autolysis and putrefaction, or in order to differentiate between fall-related brain hemorrhage and brain hemorrhage due to natural causes (e.g., hemorrhage following a fall vs. hypertensive brain hemorrhage), it is sometimes necessary to fix the brain in toto in a first step and perform its examination at some later point in a fixed state.

Optic Nerves and Eyeballs. Most notably in the case of increased intracranial pressure

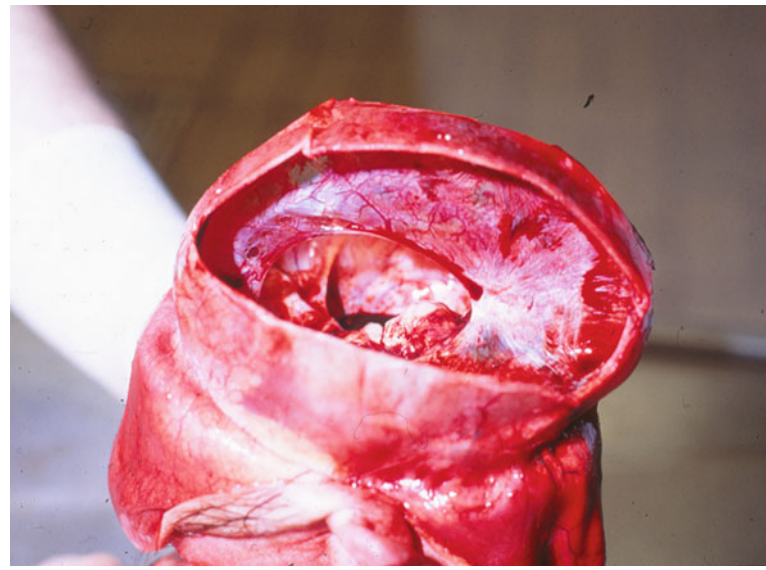

Fig. 4.4 "Half-moon" incision to visualize the cerebral falx

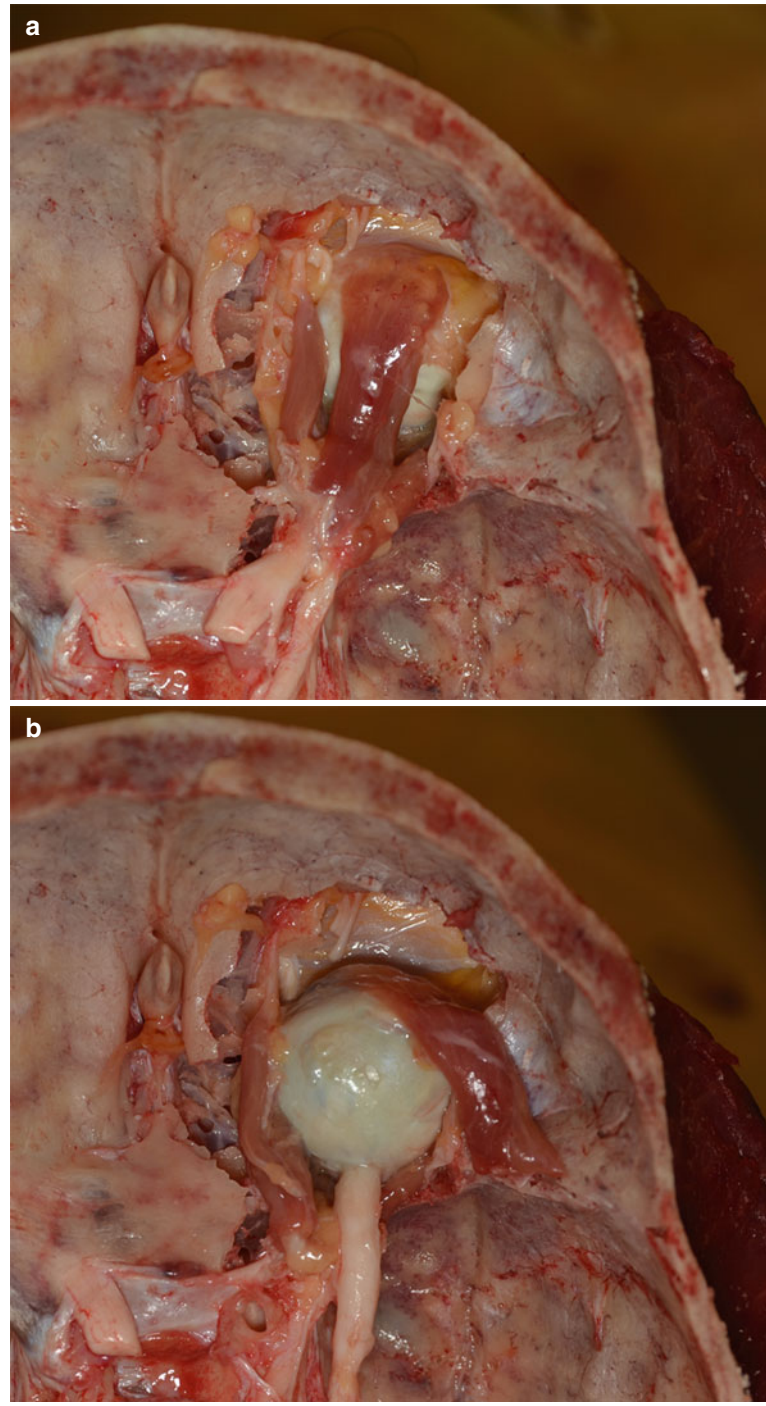

Fig. 4.5 (**a**) Dissection of the orbital cavity and visualization of the optic muscles and (**b**) the bulbus oculi and optic nerve

involving hemorrhage around the optic nerve sheaths (e.g., as seen in shaken baby syndrome), the optic nerves and eyeballs need to be dissected separately. To this end, a window is carefully placed in the bony orbital roof, which can then be extended dorsally through the optic canal. The retrobulbar fatty tissue and optic muscles can then be visualized (Fig. 4.5).

Three possibilities present themselves for the detection of retinal hemorrhage:

- In principle, ophthalmological findings can be made and documented photographically even post-mortem.
- Eyeballs can be removed completely and retinal hemorrhage detected using histological analysis (see Fig. 18.18).
- Only the posterior portion of the eyeball is removed at dissection for subsequent histological analysis. The anterior portion of the eyeball is stabilized by means of a tamponade in the posterior orbital cavity.

Ocular bulb luxation by means of transecting the four straight muscles of the eye is sufficient to visualize the orbital floor and lamina papyracea in the case of occipital trauma and blow-out fractures (e.g., in boxing injuries).

Further steps in the dissection process include removal of the hypophysis, opening of the sphenoid sinus by removing the sella turcica (watery fluid in the case of death by drowning?), opening the nose and paranasal sinuses from inside, as well as visualization of the tympanums, from which smear samples can be taken where appropriate for pathogen detection. During this process, the tympanic cavities, auditory ossicles, eardrums, as well as the internal carotid arteries all become visible. Opening the tympanum and paranasal sinuses is mandatory in the case of an appropriate indication, such as craniocerebral trauma, a diving accident, drowning, and sudden infant death.

Spinal Cord. Spinal cord removal is only rarely necessary, for example, following trauma or medical treatment that has given rise to medical malpractice claims or in the case of neurological disease. To this end, the entire length of the spinal canal should be opened, and the spinal cord and dural sheath removed completely (Fig. 4.6). Any particular features of cartilage or bone should then be documented. Further investigation of the spinal cord should take place following fixation. The spinal dura is examined internally and externally, the leptomeninx and its vessels inspected, and the spinal cord visualized slice by slice by means of parallel frontal sections.

Specimen Taking. The extent of specimen taking depends primarily on the line of inquiry. For example, analyzing cerebrospinal fluid and vitreous humor (native/frozen) for electrolytes, glucose, and lactate in cases of intoxication and impaired glucose metabolism (hypo- and hyperglycemia) is recommended; prompt analysis of native material obtained under sterile conditions is required for pathogen detection. For histological investigation, representative specimens should be taken from various areas of the brain and separated according to side and localization: frontal lobe including gyri and sulci, lentiform nucleus, thalamus, hippocampus, corpus callosum and caudate nucleus, cerebellum (cortex and medulla), pons, medulla oblongata, and cervical medulla. However, this level of specimen taking is not necessary in many cases of forensic autopsy. One possible alternative in the case of a special line of inquiry is to send the extracted brain in toto to a neuropathology institute. In such cases, however, the neuropathologist should be given precise questions to answer.

Important: In certain special cases, dissection to visualize the facial portion of the skull may be necessary; for identification purposes, it may also be necessary to completely remove the maxilla and mandible for subsequent radiological and/or morphological comparison with findings that may only become available at a later point.

4.2.3 Opening the Chest and Abdominal Cavities

The anterior trunk is opened by making a midline incision extending from the jugular fossa to the pubic bone. This incision is generally extended to a T-incision by making an additional cut from acromion to acromion along the clavicles. A midline incision running along the mid-neck to below the chin is seen predominantly in forensic medicine. Depending on requirements, the T-incision can either branch laterally along both arms or extend downwards to form a Y-incision over the groin area and both legs, assuming this has not

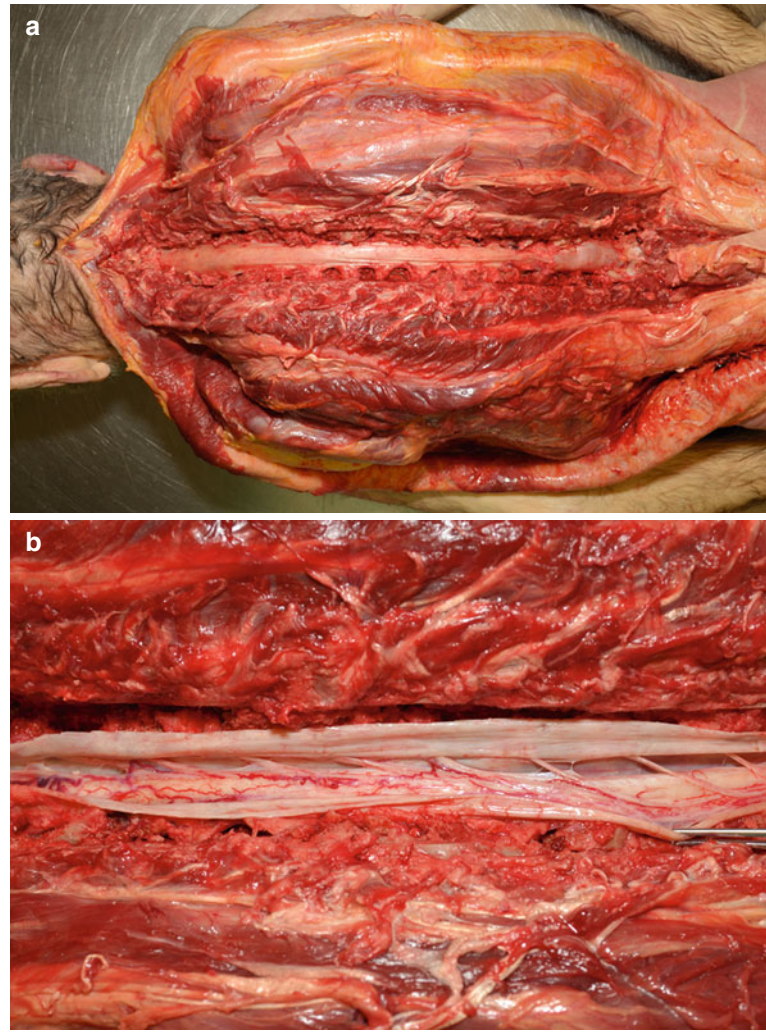

Fig. 4.6 (**a**) Spinal cord canal and surrounding dural sac exposed at autopsy. (**b**) Spinal cord and branching nerves exposed after opening the dural sac

already taken place as part of the back dissection (see above).

Chest Cavity. In a first step, a record is made of the status of the dome of the diaphragm at the level of the midclavicular line in relation to the ribs (e.g., at the level of the fourth rib or the level of the fifth intercostal space). Before opening the bony ribcage, an incision is made in the intercostal space to the parietal pleura. Attention should then be paid to whether the lungs are attached internally by the visceral pleura to the parietal pleura and whether, on opening, they sink back mildly, moderately, or distinctly ("small" pneumothorax test). The reliability of the pneumothorax test is limited on the one hand by preexisting postinflammatory adhesions between the visceral pleura and the parietal pleura and by extensive accumulation of effusion fluid in the pleural cavities on the other. If pneumothorax is already suspected at the outset of autopsy, a "large" pneumothorax test can be performed: a preformed pocket of soft tissue to the side of the chest is filled with water; the presence of pneumothorax is indicated if, on making an inci-

sion in the intercostal muscles and transecting the parietal pleura, air bubbles are seen escaping beneath the level of the water. In terms of differential diagnosis, advanced putrefaction involving the escape of putrefaction gases needs to be considered.

The bony ribcage is opened along the cartilage–bone border in the cartilage region and by making a transverse transection in the sternum. This process leaves the uppermost portion of the sternum between the sternoclavicular joints intact. Once the heart has been extracted and the cervical muscles visualized, dissection can then be continued as far as the base of the sternum and the clavicles by exposing the clavicular periosteum in order to exclude subperiosteal bleeding at the base of the clavicles (in the case of overextension trauma, death by hanging, or shaken baby syndrome, among others). If the sternoclavicular joints are to be opened, particular care must be taken during incision not to tear the blood vessels that lie beneath, most notably the brachiocephalic veins, since this would cause hemorrhage into the pleural cavity.

Prior to removing the chest organs, the area must be inspected for hemorrhage, inflammation, adhesions, effusions, etc.; moreover, the position of pacemaker probes, drainage tubes, and airways needs to be checked. The neck and chest organs to the level of the diaphragm are often removed together (as a so-called organ block) and then dissected. If esophageal variceal hemorrhage is suspected, it is advisable to ligate and remove the esophagus adjacent to the pharynx and leave the esophagus attached to the stomach for better visualization of submucosal esophageal varices (Fig. 4.7).

The Heart. The extent to which the pericardium is covered by the pulmonary lobes is inspected (senile emphysema, emphysema in the setting of COPD, emphysema aquosum, emphysema following aspiration), as are (postoperative?) adhesions, injuries, and effusions in the pleural cavity. The pericardium is opened using a lambdoid incision. The contents of the pericardium are recorded, and postinflammatory

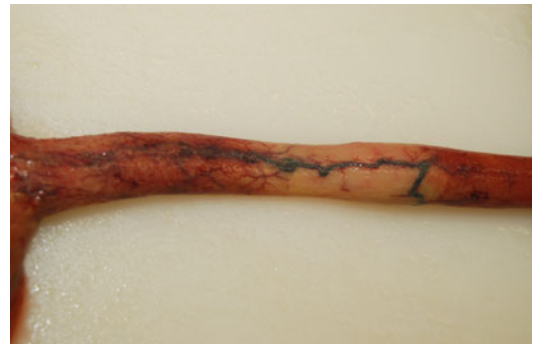

Fig. 4.7 Visualization of submucosal esophageal varices following ligation and eversion of the esophageal pipe

adhesions, e.g., due to bypass surgery, can generally be bluntly detached with ease. Where necessary, an air embolism test (according to Richter) can be performed: open and fill the pericardium with water, possibly record the buoyancy of the heart in water, pierce the right atrium, and collect rising gas or air bubbles (Fig. 4.8) in a headspace vial filled with water. Turn the vial upside down so that the water forms a seal at the bottom preventing the gas from escaping. Prior radiological diagnosis can be particularly helpful in the case of suspected air embolism. Gas and air bubbles in the coronary arteries should also be recorded.

Specimens of cardiac blood are taken during sectioning of the main afferent and efferent blood vessels of the heart. Larger and usually fulminant pulmonary thromboembolisms can be visualized in the main branches of the pulmonary arteries. When dissecting the heart, details are recorded of the width of the cardiac cavities, the foramen ovale (open, closed), left and right ventricular wall thickness, and size of the cardiac valves and their ability to swing open and shut (to exclude endocarditis), as well as details of the branch points and course of the coronary arteries.

The degree of coronary sclerosis in the coronary arteries needs to be recorded, including the localization of high-grade luminal narrowing and of lumen occlusion due to blood clots (coronary thrombosis). The localization and size of areas of cardiac muscle pallor and gray myocardial scar-

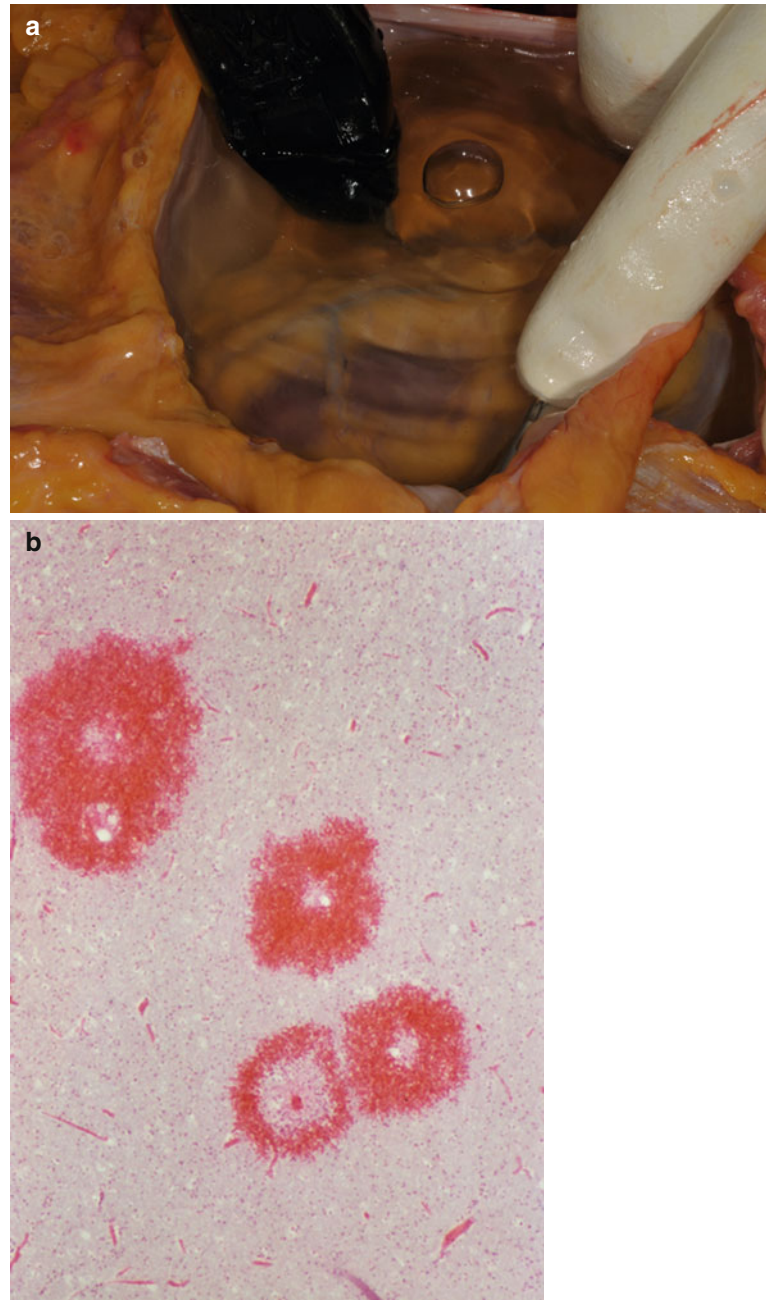

Fig. 4.8 (**a**) Air embolism test demonstrating rising air bubbles following incision in the wall of the right atrium beneath the water level. (**b**) Ring bleeding in the case of cerebral air embolism (He ×40) as a sign of vitality (see Chap. 7)

ring need to be recorded, along with fresh yellowish areas of necrosis or abnormal shadows or marbling, particularly those suggestive of a hemorrhagic wall.

Neck Organs. Only after removal of the brain and heart, and the associated drainage of blood, should soft tissue dissection of the neck be performed under artificial "bloodless" conditions; this is in order to prevent the misinterpretation of congestive hemorrhage or dissection-related artifacts, among others, as antemortem hemorrhage (Fig. 4.9).

Under artificial bloodless conditions, the neck muscles are dissected in situ layer by layer to the

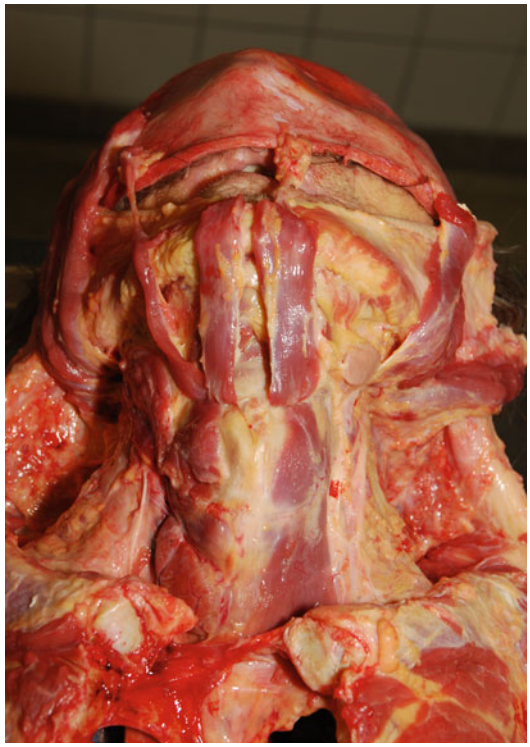

Fig. 4.9 Layer-by-layer dissection of the anterior soft tissue of the neck and neck muscles under artificial bloodless conditions

base of the sternum, clavicles, and thyroid lobes. This is followed by removal of the neck organs, including the tongue, tonsils, larynx, hyoid bone, esophagus, trachea, bronchial tubes, lungs, thyroid lobes, ascending aorta, aortic arch and its major branches (including the right and left common carotid artery bifurcation), descending aorta, and the thoracic aorta. If the case history suggests inhalation (e.g., soot particles, textile fibers) or aspiration (e.g., chyme), the respiratory tract should be dissected to at least the level of the lobar bronchi prior to detaching the lungs at their roots. In some cases, rinsing the upper respiratory tract and subsequent examination of the rinsing fluid or sediment (microscopically following centrifugation) can be helpful. Tongue sections should be inspected for bite wounds, e.g., resulting from physical confrontations or epileptic seizures.

Larynx and Hyoid Apparatus. Small fractures or fissures to the larynx and hyoid apparatus can sometimes be diagnosed radiologically. In addition to assessing the stability and elasticity of the larynx and hyoid apparatus, fractures and hemorrhage in immediately adjacent soft tissue need to be excluded. Normal variants should be recorded.

Lungs. The central and peripheral branches of the bronchial tree and the pulmonary artery branches should be dissected to detect or exclude aspiration, purulent bronchitis, pulmonary edema, and pulmonary thromboembolism. Lung parenchyma should be sectioned into slices and inspected for the brittleness of lung tissue, the extent of pulmonary emphysema should be assessed, and focal lesions in pulmonary tissue excluded. Whereas lobar pneumonia, for example, can be readily diagnosed macroscopically, early phases of (purulent) bronchopneumonia and viral pneumonia can often only be detected histologically. Focal scarring in the tip of the upper lobe could be an indication of previous pulmonary tuberculosis. Abnormal lymph nodes at the root of the lung should be removed.

Abdominal Cavity. Internal organs should be inspected for their normal location, in particular whether the appendix, gallbladder, spleen, and internal female genitals (fallopian tubes, ovaries, and uterus) are fully present and free from adhesions. Testes and epididymides are dissected separately. Preexisting (surgical) scars may indicate the partial or complete absence of internal organs or previous surgery and resultant postoperative adhesions. In addition to the thickness of subcutaneous fat in the chest and abdominal region, the level of the diaphragm in relation to the ribs or intercostal spaces should be noted (see above). The abdominal cavity including the lesser pelvis should be checked for effusion fluid, blood, and fibrinous deposits on the peritoneum. Acute hemorrhage in the retroperitoneal space, e.g., due to retroperitoneal hemorrhage from a ruptured abdominal aortic aneurysm, is visualized as shiny reddish-black hemorrhage.

In general, abdominal organs can be removed either in isolation or as part of an organ block:

- After applying two clamps at the level of transection of the small intestine at the duodenojejunal angle, the intestinal loops including the jejunum and ileum and extending to the cranial part of the sigmoid colon along with

attached mesenterium and pericolic fatty tissue are removed. In the case of inflammatory lesions, adhesions should always be detached bluntly with care under visual control. The small intestine should be inspected from the duodenojejunal angle to the cecum in a stepwise manner in order to detect perforations, invaginations, strangulation, segmental hemorrhagic infarctions, and areas suspicious for malignancy. The entire intestine should be opened and the contents carefully inspected.

- Isolated removal of the spleen, possibly also in an organ block with the liver, stomach, duodenum, and pancreas, most notably if the splenic artery and vein need to be visualized to the splenic pedicle.
- Removal of the adrenal glands from the upper renal pole.
- Dissection of the kidneys with adipose capsule, ureters, and urinary bladder (optional).
- Removal of the caudal part of the sigmoid colon, optionally with the urinary bladder, rectum, prostate, and seminal vesicles or fallopian tubes, ovaries, uterus, and vagina.
- Removal of the abdominal aorta and its branches to the iliac arteries.

Following removal, abdominal organs are dissected in a separate step, and specimens are taken for chemical–toxicological, histological, and possibly also microbiological or viral analysis. Taking blood from the femoral vein is particular important in terms of determining blood alcohol levels.

Liver, Stomach, Pancreas, Duodenum, and Spleen. The upper abdominal organ block can be removed in toto and dissected in a subsequent step. In addition to noting trauma-related hemorrhage, parenchymal tears, or, for example, incision or gunshot wounds, any variations from normal findings should be recorded as due to preexisting internal disease. Dissecting the splenic artery and vein separately enables arterial aneurysms or venous thrombosis to be excluded at those sites. In the case of liver cirrhosis, the esophagus can be ligated below the epiglottis, detached, and removed together with the upper abdominal organ block. For the purposes of visualizing esophageal varices, the esophagus is everted, starting at the level of the ligation site and moving towards the entrance to the stomach, thereby stopping blood escaping from the varices (see Fig. 4.7). The stomach is then opened retrogradely from the duodenum rather than along the large curvature.

Endocrine Organs. The thyroid lobes, testes, ovaries, pancreas, and pituitary gland are dissected or examined separately. With the exception of diabetes, endocrine diseases are rarely a direct cause of death. However, fatal thyroid dysfunction (e.g., thyroiditis, Hashimoto's lymphomatous goiter, hyperthyroiditis, and thyroid cardiomyopathy), as well as pheochromocytoma undiagnosed ante-mortem or functionally active adrenocortical adenoma, is occasionally encountered in forensic medical practice; pituitary dysfunction, such as Sheehan syndrome, is very rare.

Retroperitoneum. The retroperitoneal organs may demonstrate injury resulting from trauma or preexisting internal disease. Retroperitoneal hemorrhage is relatively common following ruptured dissecting or arteriosclerotic abdominal aortic aneurysms. Dissection extending to the ascending aortic branch usually reveals cardiac tamponade as the direct cause of death. Aortic dissection involving compression of the coronary artery branch points with no cardiac tamponade (bloodless aortic dissection) is seldom.

Skeletal System. As part of routine procedure at forensic autopsy, the cranial vault and skull base, the ventral aspect of the spine, internal aspect of the thorax skeleton, as well as the internal aspect of the pelvic skeleton are exposed. If certain structures need to be examined in greater detail, further dissection is required. This applies to the facial bones, larynx and hyoid apparatus, ribs, vertebrae, and pelvic bones, as well as all bones of the extremities. In the case of traffic accident victims, radiological diagnosis prior to dissection is often helpful. Clinically diagnosed and/or surgically treated antemortem fractures need to be visualized at autopsy. In this

context, it is important to note whether local surgical conditions are consistent with the expected findings; whether the surgical approach used—insofar as this can be evaluated—was according to standard medical practice, e.g., appropriate suturing; whether there was postoperative bleeding and infection; and whether prostheses were implanted correctly. Visualizing the skull and lower extremities (Messerer fracture?) is particularly important in traffic accident victims. In some traffic accident cases, it is necessary to simultaneously visualize vascular and nerve fibers.

Victims of Sex Offenses. In the case of a suspected sex offense, smear samples (oral, anal, and vaginal, including anterior and posterior vaginal vault and cervical canal, or penis) should be taken prior to dissection. Already at external examination, the genital and anal region should be carefully inspected, paying particular attention to injuries, hemorrhage, secretion deposits, possibly foreign pubic hair, as well as any other traces of foreign material. To ensure thoroughness of the examination, the external and internal genitalia are removed en bloc, i.e., together with the anus, rectum, and urinary bladder. To this end, the anogenital region requires a relatively wide incision margin, followed by detailed dissection.

Specimen Taking. Taking the specimens mentioned in Table 4.1 should form an integral part of any autopsy, even if case-specific variations are permissible, as is the case of brain specimens (see above). Hair specimens should be obtained dry as pencil-width bundles, placed in aluminum foil, and marked to indicate the end taken from proximal to the head. Skin and subcutaneous soft tissue should be included in any specimen taken of an injection site; taking material for comparative purposes can be helpful, for example, in terms of wound age determination. Particularly in cases of suspected drug abuse, taking a smear sample from the nasal mucous membrane should be considered, while taking specimens of putrefaction fluids and maggots may be helpful in the case of prolonged postmortem intervals. It may be helpful to remove the injured area in toto for the purposes of establishing a line of evidence and determining wound age; this also applies to gunshot entrance and exit wounds. Bones are sometimes retained for reconstructive purposes, the maxilla and mandible for identification purposes, and the larynx and hyoid apparatus to investigate neck trauma, this latter possibly as an organ block including the tongue. If DNA analysis is required to identify an unknown decedent but postmortem blood is not available, notably due to putrefaction, muscle specimens can be taken, for instance, from the greater psoas muscle.

4.3 The Autopsy Report

An autopsy report should be compiled both during and after a forensic autopsy. The report should in the first instance describe the principle findings at autopsy while documenting that the examination was conducted according to standard medical practice, i.e., in accordance with the requirements of a forensic autopsy. Therefore, the report should also include several short comments relating to negative findings, i.e., those instances where no local pathological findings could be made on the body. In this way, the autopsy report serves to document the fact that the forensic pathologist has examined all body regions and internal organs or, depending on the specific case, relevant anatomical structures and has recorded his or her findings. The autopsy report can additionally be supplemented by the pathologist's own photodocumentation (stored on CD-ROM or as color printouts of images taken at autopsy).

Writing an Autopsy Report. An autopsy report should firstly include the following information where available: name, surname, and address of the deceased; name of the forensic pathologist; name of the prosector; date, time, and place of autopsy; persons present (from the investigating authorities or the police, public prosecutor's office, or court); commissioning body or

reference of the commissioning authorities; and autopsy number given by forensic institute. Autopsy findings should be recorded in a structured manner, e.g., according to the following sample:

A. External examination (see Chap. 3)
B. Internal examination
   I. Cranium and cranial cavity
   II. Neck region
   III. Chest and abdominal cavities
   IV. Skeletal system
   V. Dimensions and weights (height, weight, organ weight, etc.)
C. Preliminary forensic assessment
   I. Reported case history at the time of autopsy
   II. Principal autopsy findings
   III. Cause of death
   IV. Interpretation (of findings in view of the reported case history)
   V. Specimens (according to the attached list and with recommendations where applicable for further analyses to establish cause and mode of death) and images attachment
   VI. Proviso (the forensic pathologist should reserve his final opinion insofar as information and investigation results are hitherto unavailable)
   VII. Signatures (where applicable, all pathologists or examining personnel should be named individually on the autopsy report and should each give their signature)

Naturally, autopsy reports can and sometimes need to be structured differently to the above example.

4.4 Dealing with Specimens Taken at Autopsy

Specimens taken at autopsy serve the primary purposes of autopsy insofar as they help in the first instance to establish, by means of the further investigations carried out on them, the cause and mode of death. Further investigations may include:

1. Further macroscopic investigation of, e.g., bones following maceration.
2. Macroscopic and radiological investigations of specimens taken from, e.g., the larynx and hyoid apparatus.
3. Histological and immunohistochemical investigations of organs and tissue samples (e.g., to detect early meningitis or diagnose myocarditis, myocardial infarct, ascending cholangitis, drug-reactive and/or drug-induced organ lesions, pneumonia, and vascular disease, etc.). Details on fixation, processing, and the use of special histological and immunohistochemical staining techniques can be found in the relevant literature on (forensic) histopathology.
4. Postmortem biochemical analyses (e.g., to determine glucose and lactate concentrations in the case of suspected glucose metabolism impairment, HbA1c, electrolyte levels in body fluids, and hormone levels in body fluids, e.g., catecholamines and thyroid hormones).
5. Postmortem chemical toxicological analyses to detect drugs and/or medications in organ specimens, tissue samples, and body fluids.
6. Investigations on special specimens (e.g., skin wounds to determine wound age or gunshot wounds to determine firing range).

The abovementioned principle investigations required to establish a reliable body of evidence in criminal proceedings should be carried out within a reasonable length of time. In order for the agent commissioning the autopsy to have time to order further investigations, the autopsy protocol should specify a period of specimen storage (e.g., 2 years); a separate commission should be requested from the commissioning agent if longer storage times are required.

Specimens removed at autopsy can be disposed of with the approval of the commissioning agent (e.g., police or public prosecutor's office) following expiry of the specimen storage deadline or earlier. Transferring specimens to relatives for secondary burial is also possible, particularly in cases where, in agreement with the agent commissioning the autopsy, whole organs (e.g., heart or brain) were retained for examination.

If organs and tissue specimens are to be taken at autopsy, be it clinical or forensic, for transplantation purposes (e.g., dura mater, tendon tissue, corneas, cartilage tissue), the regulations in the relevant country need to be observed. However, in order to avoid conflicts with the primary objective of autopsy, i.e., establishing the cause of death, careful documentation is paramount. This also applies to the use of organs, organ specimens, body fluids, and tissue specimens for scientific research, which is subject to monitoring by the relevant ethics commission.

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Exhumation

5

The removal of a body from a grave (exhumation) requires legal authority, often in the form of a court order. Next of kin may also have a prior right to be heard. Exhumation may be deemed necessary either in cases where a court order was not issued in a timely fashion around the time of death or if new evidence justifying exhumation has since come to light. Since it is often the case that particular questions require clarification, it is important to establish in advance whether exhumation is an appropriate approach to answering these questions.

The utility of exhumation varies greatly depending on the period of time a body has spent in an inground grave (burial time). It may be possible to detect poisoning many years after burial—depending on the preservability of the poison—particularly in the case of heavy metals. Hair analysis also offers the possibility of confirming regular use of medications such as ß-blockers.

Many relevant factors are unknown prior to exhumation, and in most cases, it is impossible to safely determine them retroactively. Exhumations are typically performed to clarify cause of death and determine identity in cases of intoxication, suspected homicide, medical malpractice, or accidents, including traffic accidents. The authorities concerned are often interested in those findings which can be reasonably expected from exhumation, depending on both the nature of the diagnostic question being asked and the specific length of time since burial.

5.1 Macroscopic Findings on Exhumation

The conditions prevailing in an earthen grave can differ significantly and may vary to a considerable degree according to the climate zone and time of year. Although groundwater level is avoided when the depth of a grave is determined, water may nevertheless accumulate in the case of prolonged rainfall, with the result that a body may need to be recovered from a water-filled casket (Fig. 5.1).

It is often possible to make numerous findings after exhumation despite factors such as autolysis, putrefaction, possible animal predation, fungal colonization (Fig. 5.2), mummification, adipocere, and artifacts (e.g., caused during body recovery). There is no linear correlation between postmortem interval and the detectability of findings, which depends far more on ambient conditions and the diagnostic question being posed. A body may have undergone decomposition to an extent that it is no longer possible to make a targeted assessment.

In the case of a naturally mummified body, putrefaction, autolysis, as well as possible animal predation and mummification processes take place simultaneously. When this is the case, mummification, that is to say, the drying out of tissue, inhibits other decomposition processes. Brain tissue often takes on a pulp-like consistency within days or weeks, making a differentiated assessment impossible (Fig. 5.3).

However, important findings such as extensive intracranial or intracerebral hemorrhage may be

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Fig. 5.1 Exhumation from a water-filled casket following prolonged rainfall

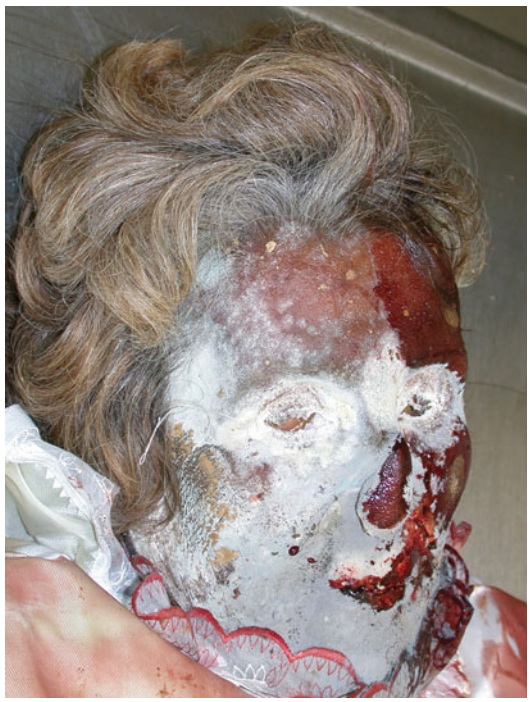


Fig. 5.2 Extensive whitish-gray fungal colonization following a burial time of 7 months

detectable for longer periods of time. Lack of water tends to slow down autolysis and putrefaction; this can mean that the internal organs of a body may undergo complete decomposition, while the outer layer, in particular the skin, remains preserved by the mummification process, especially in cool, dry ambient conditions. The surface of the body is dry and rigid and has a light brown-ocher color (Fig. 5.4).

In the case of mummification, skin injuries such as incision and stab wounds as well as gunshot wounds can still be detected after long burial times. Even larger wounds and hemorrhage are identifiable in mummified bodies if subcutaneous soft tissue can be well visualized (Fig. 5.5). Dental findings, including evidence of dental treatment, can be made after significantly longer burial times (Fig. 5.6).

Adipocere. This refers to the transformation of body fats into adipocere. Fatty tissue either undergoes an oily transformation or becomes a greasy waxlike mass that eventually dries out and hardens with time to the consistency of chalk, particularly

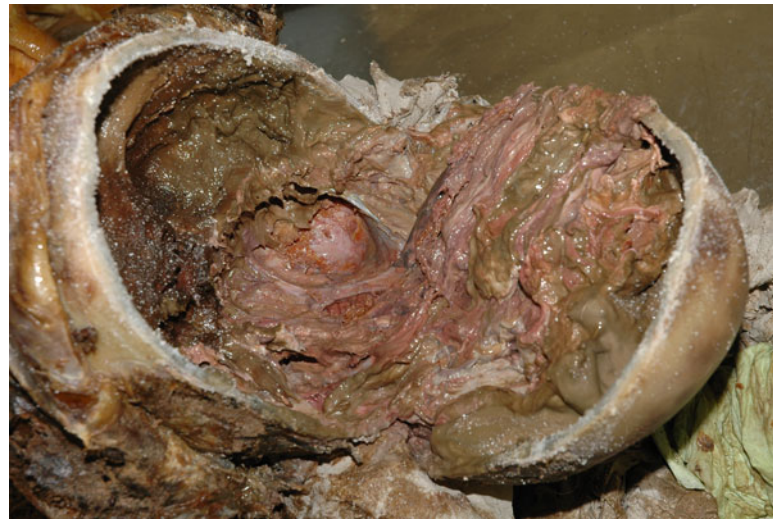
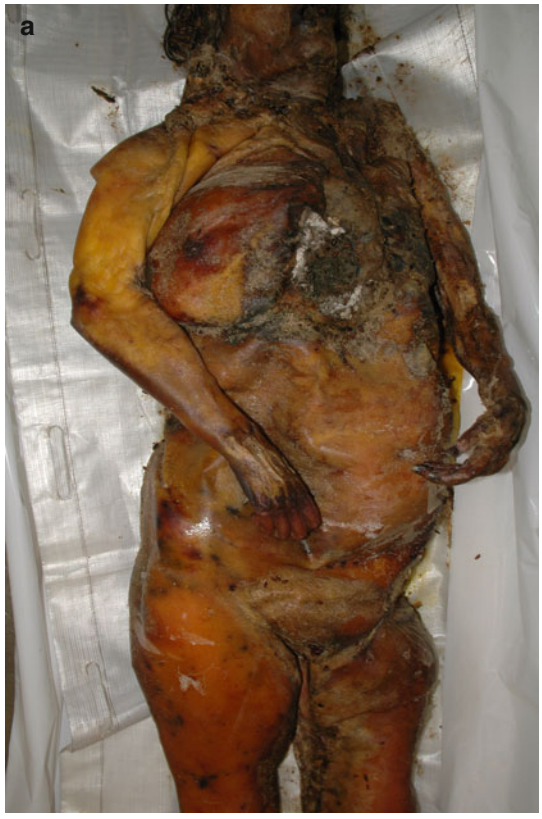


Fig. 5.3 Brain tissue that has undergone pulp-like transformation (burial time, 7 weeks)



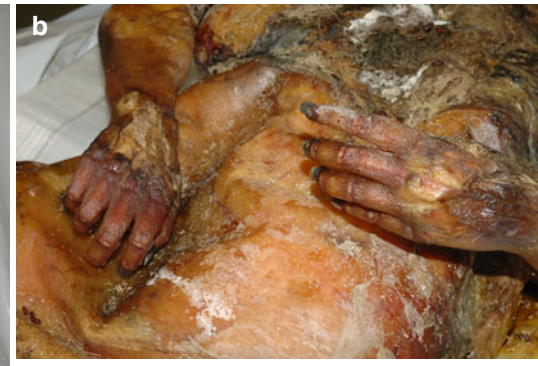


Fig. 5.4 Burial time, 6 years. (**a**) Mummification in dry, cool ambient conditions with brown-ocher-colored drying and stiffening of the body surface. (**b**) Partial involvement of the extremities, including hands and fingers, in the mummification process

under dry-air conditions. Once adipocere has formed, it may be difficult, if not impossible, to detect findings in internal organs; injuries, on the other hand, can remain well identifiable. Table 5.1 lists a selection of macroscopic findings that can still be made even after prolonged postmortem intervals.

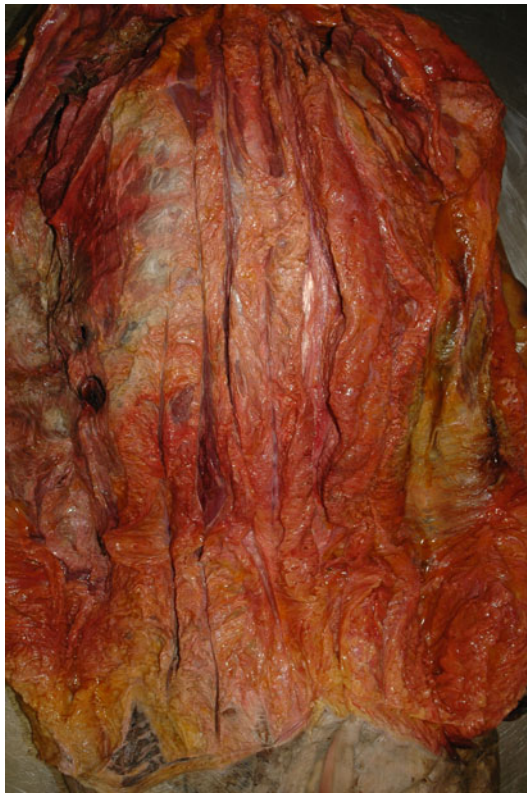


Fig. 5.5 Soft tissue dissection of the back in a partially mummified body (burial time, 1.5 years); anatomical structures can be well visualized

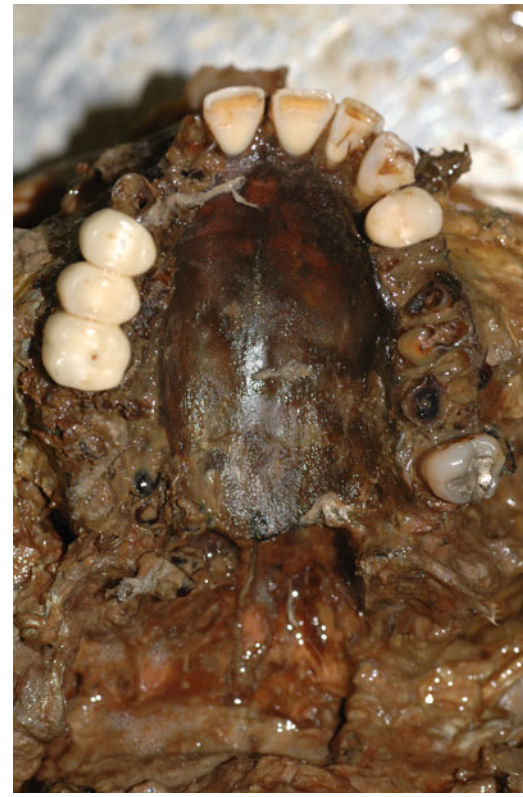


Fig. 5.6 Dental status on exhumation following a burial time of 1.5 years

Even after exhumation, it is possible to differentiate between ante- and postmortem injury and changes to a body. Postmortem artifacts include injuries caused by a primary event (e.g., fire-related artifacts such as heat fractures), postmortem transport, as well as the breaking open of a casket lid and other changes related to the recovery of a body from a grave. Depending on the deceased's prior history, injury may have been caused by previous attempts at resuscitation, e.g., serial rib fractures. Other types of postmortem damage develop according to the postmortem interval, such as autolysis of the pancreas, gastric wall rupture and escape of gastric contents, rectal prolapse due to gas buildup in the abdomen, and hypostatic accumulation of blood. Internal organs gradually lose their color to become dirty reddish brown, e.g., the vascular intima throughout the body. Depending on the position of the body, lesions such as extensive petechiae or ecchymoses may be seen, particularly in the head, neck, and shoulder region in the case of a head-down position.

Table 5.1 A selection of pathomorphological findings correlated with burial times

Macroscopic finding on exhumation	Burial time
Epidural/subdural hematoma	2.8 months
Cerebral contusion	2.1 months
Hematothorax	2.5 years
Blood aspiration	1.7 months
Cardiac tamponade	3 years
Intestinal bleeding	1–2 years
Strangulation marks	Months
Electrical burns	2 months
Conjunctival hemorrhage	2.6 months
Meningitis	3.5 months
Coronary sclerosis	7.5 years
Coronary thrombosis	10 months
Pulmonary thromboembolism	13 months
Pneumonia	2 years
Liver cirrhosis	1 year
Oligodendroglioma	39 days
Cerebral hemorrhage	66 days/73 days
Aortic aneurysm rupture	193 days
Deep vein thrombosis	75 days

Table 5.1 (continued)

Macroscopic finding on exhumation	Burial time
Fresh myocardial infarction accompanied by cardiac tamponade	97 days/64 days
Myocardial scars	304 days/1,581 days
Left ventricular hypertrophy	240 days/128 days
Cor pulmonale	267 days/128 days
Prostatic venous plexus thrombosis	202 days/27 days
Bronchial carcinoma	202 days/13 days
Caseous pulmonary tuberculosis	236 days/10 months
Lobar pneumonia	37 days
Pleural mesothelioma	157 days/210 days
Fresh pulmonary embolism	168 days/13 months
Status following hepatic rupture	49 days
Cavernous hemangioma of the liver	38 days
Cholecystolithiasis	478 days/7.5 years
Ascites	50 days
Perisplenitis cartilaginea	176 days/6 weeks
Gastric cancer	168 days
Pseudomelanosis coli	71 days
Esophageal varices	39 days
Ventricular ulcer	170 days/157 days
Inguinal hernia	112 days/127 days
Inguinal testis	60 days
Renal cysts	240 days
Full urinary bladder	236 days
Prostate hyperplasia	478 days/2.5 years
Decubital ulcer over the sacral bone	111 days
Dermal scarring	478 days/7.5 months
Tattoos	72 days
Parasternal rib fractures due to resuscitation	114 days
Crural ulcers	30 days/9 weeks

Modified from Karger (2004), Ulm (2008) and based on additional information in the literature

5.2 Histological Findings on Exhumation

Histological and histopathological findings following prolonged postmortem intervals have been the subject of several investigations in the past (Table 5.2). The detection of usable microscopic findings depending on the postmortem interval, especially in the context of exhumation, is by nature temporary and reliant on many factors. Thus, methods of forensic–histological diagnosis are limited in the presence of autolysis and putrefaction, being only of modest use, for example, in autopsies on bodies taken from mass graves. In the case of mummification, on the other hand, tissue and organoid structures, as well as potential pathological findings, can be seen microscopically for a significantly longer period of time as compared with in the presence of autolytic and putrefactive processes. However, microscopically, numerous problems of differentiation are encountered, for example, when distinguishing tubular necrosis in the kidney from purely autolytic changes. Detecting acute myocardial infarction can also be challenging and is only possible for a limited period of time. Finally, structures are prone to various autolytic processes in the postmortem period. Leukocytes and nuclei of granulocytes are seen as exceedingly resistant to autolysis and putrefaction. Evidence of bronchopneumonia could be shown following exhumation even after 392 days. Others have diagnosed confluent bronchopneumonia after a postmortem interval of 95 days. However, there is no specific sequence or timeline for changes to internal tissues and organs resulting from autolysis and putrefaction, nor can a fixed time or period of time be determined for single organs. In general, the uterus is considered to be an organ relatively resistant to putrefaction.

5.3 Chemical–Toxicological Analysis Following Exhumation

Experience with a number of poisons or types of intoxication has been gained in terms of potential toxin detection following prolonged burial times (Table 5.3). This process involves taking very particular specimens, such as the bottom boards of a casket, casket fittings, and soil samples from above, below, beside, and at some distance from the casket. For autopsy purposes, samples of the following, where available, should be taken for chemical–toxicological analysis:

- Blood (cardiac blood, femoral vein blood)
- Liver sections
- Kidney sections
- Lung sections
- Brain sections
- Bile fluid
- Gastric contents

Table 5.2 Detectability of selected histological findings subject to postmortem periods according to data in the literature

Finding	Postmortem period	Author(s)
Electrical burns	3 weeks	Walcher (1937)
Peritonitis, sepsis, septicopyemia	17 days/42 days	Althoff (1974)
Polynuclear alveolar cells	15 days	Althoff (1974)
Early cell infiltration	65 days	Althoff (1974)
Subserous hemorrhage	65 days	Althoff (1974)
Chronic meningitis	52 days	Althoff (1974)
Coronary thrombosis and myocardial fibrosis	90 days	Althoff (1974)
Chronic bronchitis	27 days	Althoff (1974)
Bronchopneumonia	133 days/95 days	Althoff (1974)
	392 days	Naeve and Bandmann (1981)
Immunohistochemical analysis using myeloperoxidase	19 months	Schulz et al. (1999)
Brain metastasis of lung cancer	44 days	Althoff (1974)
Liver metastasis of hemangiosarcoma	27 days	Althoff (1974)
Bronchial anthracofibrosis	80 days	Althoff (1974)
Stenosing coronary sclerosis	133 days	Althoff (1974)
Intimal sclerosis of the coronary arteries	19 months	Schulz et al. (1999)
Ganglion and glial cells	114 days	Walcher (1937)
	1,212 days	Althoff (1974)
Positive evidence of iron in the tissue	1–2 years	Walcher (1937)
Fat embolism	8–10 days (exp.)	Lubarsch (1900)
	4–8 weeks	Walcher (1925, 1928)
	4.5 months	Strassmann (1921–1931)
Coronary thrombosis	8 months	Banaschak et al. (1998)
	3.5 months	Grellner and Glenewinkel (1997)
	3.9 months	Althoff (1974)
	96 days	Stachetzki et al. (2001)
Acute myocardial infarction	6 weeks	Breitmeier et al. (2003)
Detected immunohistochemically using necrosis marker C5b-9(m) and	12 months	Karger et al. (2004)
NP57 (indicates neutrophilic leukocytes)	63 days, 487 days	Ortmann et al. (2000)
	128 days	Ortmann et al. (2000)
Detection of hemosiderin in the dura	8.5 months	Breitmeier et al. (2005)
Liver fibrosis	4.5 months	Breitmeier et al. (2005)
Shock liver	6 months	Breitmeier et al. (2005)
Glomerulonephritis	3.2 months	Breitmeier et al. (2005)
Cervical artery dissection	20 months	DeGiorgio et al. (2007)
Alzheimer's disease	2 months	Gelpi et al. (2007)
	20 months	Omalu et al. (2005)
Previous cerebral contusion	3.5 weeks	Grellner and Glenewinkel (1997)
Cerebral edema	13 weeks	Grellner and Glenewinkel (1997)
Thyroiditis	3.75 months	Grellner and Glenewinkel (1997)
Nodular goiter	7 weeks	Grellner and Glenewinkel (1997)
	13.5 weeks	Althoff (1974)
Thyroid adenoma	3 months	Grellner and Glenewinkel (1997)
Tracheitis	2 weeks	Grellner and Glenewinkel (1997)
Epicarditis	3.75 months	Grellner and Glenewinkel (1997)
Lipomatosis cordis	2.5 years	Grellner and Glenewinkel (1997)
Myocardial hypertrophy	3.5 months	Grellner and Glenewinkel (1997)
	4.25 months	Naeve and Bandmann (1981)

Table 5.2 (continued)

Finding	Postmortem period	Author(s)
Myocardial granulation tissue	3.5 months	Grellner and Glenewinkel (1997)
Myocardial fibrosis or scarring	2.5 years	Grellner and Glenewinkel (1997)
	2 years	Nordmann (1939)
Acute pulmonary trauma	5 weeks	Grellner and Glenewinkel (1997)
Pulmonary edema	3 months	Grellner and Glenewinkel (1997)
	2.5 years	Thomas (1979)
Chronic pulmonary congestion	2.1 months	Grellner and Glenewinkel (1997)
	3.5 months	Naeve and Bandmann (1981)
Pulmonary amyloid bodies	3.5 years	Grellner and Glenewinkel (1997)
Shock lung	2 weeks	Grellner and Glenewinkel (1997)
Pneumonia	3.75 months	Grellner and Glenewinkel (1997)
	1.1 years	Naeve and Bandmann (1981)
Immunohistochemical detection of neutrophilic granulocytes with NP57	24 months	Karger et al. (2004)
Lung emphysema	2.5 years	Grellner and Glenewinkel (1997)
	2 years	Nordmann (1939)
	26 days	Raestrop (1926)
Anthracosis	7.5 years	Grellner and Glenewinkel (1997)
Tuberculosis	1.5 months	Grellner and Glenewinkel (1997)
	10 months	Nordmann (1939)
Pulmonary artery sclerosis	2.5 years	Grellner and Glenewinkel (1997)
Pulmonary thromboembolism	1.5 months	Grellner and Glenewinkel (1997)
Pulmonary fat embolism	4.5 months	Strassmann (1921–1931)
	1–2 months	Walcher (1925, 1928)
	1.2 months	Naeve and Bandmann (1981)
Hepatic capsular fibrosis	2.5 years	Grellner and Glenewinkel (1997)
Hepatocellular necrosis	6 days	Grellner and Glenewinkel (1997)
Fatty degeneration of the liver	3 months	Grellner and Glenewinkel (1997)
	10 years	Siegel (1985)
Fatty liver hepatitis	2 weeks	Grellner and Glenewinkel (1997)
Periportal infiltration	16 days	Althoff (1974)
Splenic artery hyalinosis	3.75 months	Grellner and Glenewinkel (1997)
Septic spleen	8 days	Grellner and Glenewinkel (1997)
	16 days	Althoff (1974)
Scarring of the renal cortex	3 months	Grellner and Glenewinkel (1997)
	4.8 months	Naeve and Bandmann (1981)
Renal shrinkage	6 weeks	Grellner and Glenewinkel (1997)
	3 years	Walcher (1937)
(Chronic) pyelonephritis	6 months	Grellner and Glenewinkel (1997)
	5 weeks	Althoff (1974)
Prostatic hypertrophy	2.5 years	Grellner and Glenewinkel (1997)
	2.5 months	Riepert (1993)
Corticoadrenal hyperplasia	3.5 weeks	Grellner and Glenewinkel (1997)
Adipose tissue	2 years	Grellner and Glenewinkel (1997)
Alveolar structure and epithelium	4.8 years	Grellner and Glenewinkel (1997)
	1.25 years	Walcher (1937)
	2 weeks	Althoff (1974)
Amniotic fluid components	4.5 months	Strassmann (1921–1931)
Bone marrow	3 months	Grellner and Glenewinkel (1997)

(continued)

Table 5.2 (continued)

Finding	Postmortem period	Author(s)
Residual brain tissue in adipoceratous cadavers	73 years	Erman (1882)
Brain structures	3 months	Grellner and Glenewinkel (1997)
Myocardium	2.5 years	Grellner and Glenewinkel (1997)
Hepatic cells	2.5 years	Grellner and Glenewinkel (1997)
Neuronal and glial cells	17 years	Grellner and Glenewinkel (1997)
	3.3 years	Althoff (1974)
Pancreas	4.8 years	Grellner and Glenewinkel (1997)
	4 weeks	Walcher (1937)
Renal structures and cells	2.5 years	Grellner and Glenewinkel (1997)
	3 years	Walcher (1928)
Skeletal muscle, including transverse striation	4.8 years	Grellner and Glenewinkel (1997)
Thyroid structure	2.5 years	Grellner and Glenewinkel (1997)
Infectious arteritis of the hepatic artery	9 days	Dedouit et al. (2010)
Reticulum cell sarcoma	16 months	Sierra-Callejas and Pribilla (1978)
Expanded lung tissue in a newborn caused by breathing	4.5 months	Strassmann (1921)
Displaced textile fibers at the site of bullet entry	7 months	Strassmann (1921)
	1 year	Strassmann (1921)
Interstitial lung fibrosis	223 days	Stachetzki et al. (2001)
Keratinizing squamous cell lung cancer	43 days	Stachetzki et al. (2001)
Brain metastasis of small-cell bronchial cancer	73 days	Stachetzki et al. (2001)
Immunohistochemical detection of glucagon	Negative from 14 days post-mortem	Wehner et al. (2001)
Immunohistochemical detection of calcitonin	Negative from 13 days post-mortem	Wehner et al. (2001)

From Dettmeyer (2011)
Sometimes only the postmortem interval is stated in the literature, but not if mummification or autolysis and putrefaction can be assumed

Table 5.3 Toxin detection following exhumation

Toxin	Years prior to detection	Toxin	Days prior to detection
Arsenic	9	Phosphorous	152
Lead	9.5	Hydrogen cyanide	116
Thallium	8	Fluorine	53
Strychnine	6	Aconitine	48
Antimony, barium	5	Mercury	30
Atropine, scopolamine	3	Carbromal	28
Morphine derivate	1.1	Mineral acids	23
Colchicine	0.8	Barbiturates	21
Parathion (E 605)	17		
CO	0.6		
ß-Blockers (hair)	7		
Digitoxin	1.4		
Chlorprothixene	5.5		
Diazepam	2.7		

From Forster (1986), Karger (2004), and own results

- Intestinal contents, subdivided into small and large intestine contents
- Muscular system
- Fatty tissue (particularly in the case of volatile gases such as anesthetics)
- Hair
- Finger- and toenails
- Vitreous fluid
- Synovial fluid (knee joint)
- Cerebrospinal fluid

Gaining information on poison concentrations from chemical–toxicological analysis following exhumation is problematic, since although it is still possible to identify a poison as such, the postmortem metabolization of toxic substances needs to be taken into account. Thus, drawing conclusions about fatal intoxication should be done so with caution and only when all other facts have been taken into consideration.

5.4 Exhumation of Mass Graves

A distinction is made between mass graves intended as a final place of burial and those intended as a temporary measure. Mass graves are usually created when there are large numbers of deceased, and these numbers, combined with external circumstances or time considerations, prohibit normal burial.

Most notably in postwar periods (including civil war), and especially in the case of possible war crimes, there is a desire to remove the deceased expediently. Typically, bodies are randomly placed in large pits and possibly also strewn with quicklime to accelerate the process of decomposition. Mass graves of this kind are usually opened and mass exhumations undertaken as part of criminal proceedings at the International Criminal Court in the Netherlands. In addition to identifying victims, it is also important to establish the cause of death in each individual case. As the time interval between death and exhumation increases, it becomes ever more challenging to assign individual body parts or bones to the relevant individuals.

It is not uncommon in the case of terrorist attacks, serious accidents such as air crashes and ferry disasters, as well as natural disasters like earthquakes, floods, and tsunamis for the sheer number of bodies to vastly exceed normal cooling and storage capacities. In such cases, creating temporary mass graves may be the only option available. However, this type of mass grave is created in a well-ordered manner to facilitate, as far as possible, the later exhumation and examination of the deceased.

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Establishing Identity

6

Case Study

During the summer months, a group of youths noticed a sleeping bag that had apparently become caught in a hedgerow within the designated flood-risk area of a river. After opening the zip of the sleeping bag a few centimeters, they saw a human skull and other bones. The police were informed.

The sleeping bag was found to contain items of clothing and a whole human skeleton. In addition, valid identity and health insurance cards were also recovered. Both documents were issued to a 67-year-old man known to the homeless community. Following osteological analysis carried out as part of a forensic autopsy ordered by the court, it was concluded that the remains were those of a male of advanced years. A skull–photo comparison between the skull found at the scene and the photo on the identity card was not able to either conclusively establish or exclude the victim's identity. However, it should be noted here that the identity photo was very low resolution and the face was covered to a great extent by a beard.

Some weeks later, the police received information from the homeless community that the missing 67-year-old man had "reappeared." The police were able to ascertain the man's whereabouts and interview him. He recounted how the previous autumn he had lent his health insurance card together with his identity card to a "buddy" who had wished to see a doctor. He had not seen the buddy since that time.

Old biplanar head X-rays of the friend dating back 12 years were found. An X-ray of the recovered skull was made and compared with the 12-year-old anteroposterior image (comparative X-ray analysis): an alignment of the bony structures on both images could be seen. Good correspondence was seen between the frontal sinuses, structures subject to high individuality. Identity was subsequently established "with a probability bordering on certainty."

Identification in its wider sense refers to identifying an unknown person, for example, in a closed-circuit television video (CCTV), whereas identification in a narrower sense refers to establishing the identity of a deceased individual from their remains.

Identification is a two-step process:

- The first step involves gathering information about the unknown individual in order to investigate missing persons who could correspond to the unknown decedent.
- In a second step, antemortem data is compared with postmortem data.

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Results from these two steps enable *identity* to be either *excluded* or *established*. If identity is established, an indication of the *probability of identity* is additionally expected.

The following sections deal with the identification of deceased individuals primarily by means of external examination and autopsy but also by means of methods such as radiological investigations.

6.1 Visual Identification by Relatives

Identification by relatives or other individuals who knew the deceased is permissible by law in a number of countries. However, this can sometimes be challenging or barely possible due to the degree of decomposition of the body. There have been numerous cases whereby presumed relatives have been shown a badly decomposed body, were barely able to look at the body due to emotional stress, and confirmed its identity in response to preset expectations. As an alternative, many facilities prefer to use only photos of a body in order to reduce the emotional stress experienced by relatives.

Prior to visual identification, it is important to establish whether, given the condition of the body, this approach is viable. If the body is in a poor condition, the identifying person should be asked to name concrete distinguishing features that may help to identify the deceased.

6.2 Identification Using Personal Effects

Objects found with a body or human remains play an important role in the identification process. While their value as an indicator of identity is undisputed, relying on objects found with the deceased for the purposes of establishing identity can lead to serious errors. Even in the case of an identity card, the possibility of theft or—as illustrated in the case study above—loan during life should be taken into consideration.

Important: The greater the individual nature of an object found with a body, the more likely there is to be at least an association between the owner of the object and the deceased. However, this does not necessarily mean that the deceased is the owner of the object.

6.3 Body Modifications and the Results of Medical Treatment

Body modifications such as tattoos (Fig. 6.1) and piercings have become more common in recent years. At the same time, the increasing number of personal photos in the digital age means that good antemortem images are available for comparison purposes; these can be used for direct comparison with a body. Moreover, attempts can be made to photograph tattoos or piercing configurations on a body from angles and distances comparable to those on antemortem photos. Computer-assisted *superimposition techniques* can then be used to compare ante- with postmortem images (Fig. 6.2).

Aesthetic Surgery and Trauma Surgery. Breast augmentation is the most commonly seen

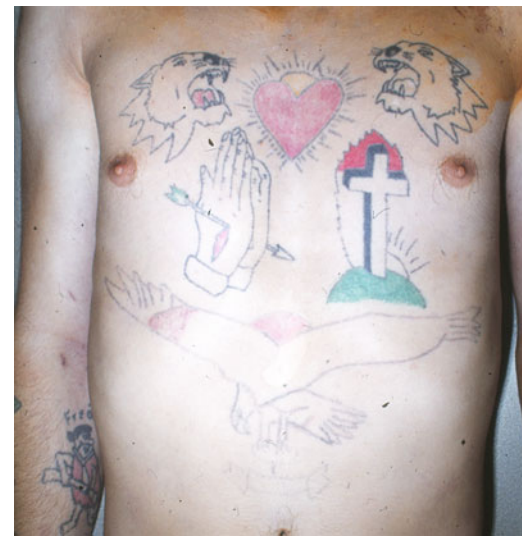

Fig. 6.1 Tattoos as body modification

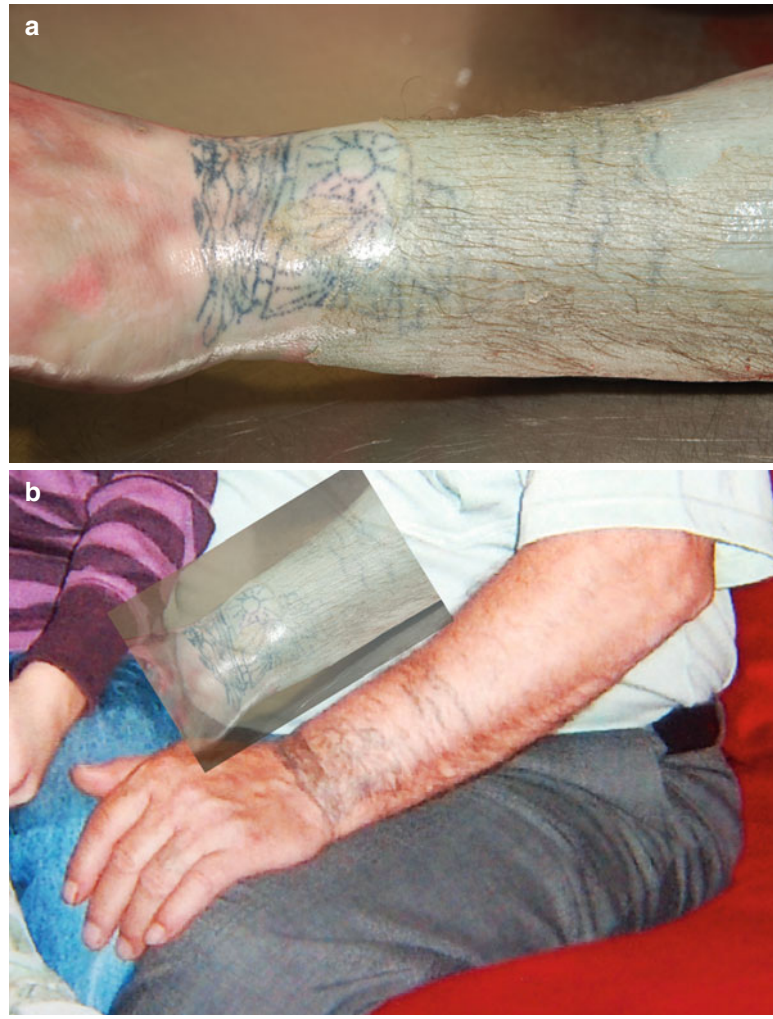

Fig. 6.2 Superimposition of ante- and postmortem photos of a tattoo. (**a**) A highly distinctive tattoo on the extensor side of the lower right forearm could be seen on the badly decomposed body. (**b**) A family photo clearly showing a tattoo on the individual in question was available. The tattoo on the body was photographed at a similar angle and placed over the tattoo on the family photo using superimposition technology (shown here slightly transposed)

form of aesthetic surgery in women, usually involving the use of silicone implants. Many manufacturers give their implants serial numbers, making it possible to trace the date of implantation and the name of the patient from these numbers. Metal plates, medullary nails, or total endoprostheses used in orthopedic and trauma surgery may also carry a serial number (Fig. 6.3). Although the above constitute "mere objects," their fixed attachment to the body makes them highly reliable indicators of identity.

Implanted metal plates are not the only devices to assist in identification—the serial number of a pacemaker may be equally as helpful.

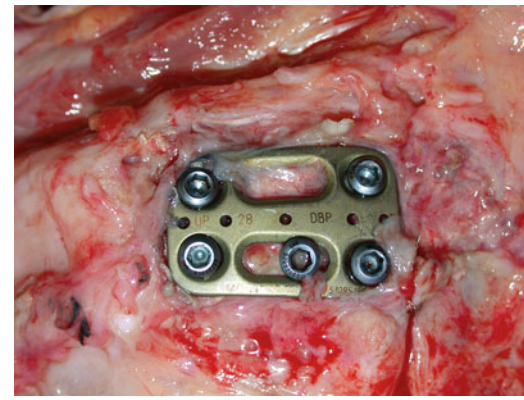

Fig. 6.3 Metal plate with a serial number, implanted during trauma surgery. It was possible to indentify the deceased conclusively with the aid of the serial number

Complex Identification. As discussed above, body modifications or surgical procedures can produce highly individual distinguishing characteristics or provide unique implant serial numbers that can be of great assistance in the identification process. On the other hand, antemortem material or information that might normally have been well suited to comparative investigation may be worthless. For instance, if lower leg X-rays of a missing person are available but the deceased lacks the limb in question as a result of amputation, for example, due to diabetes, comparative X-ray analysis for the purposes of establishing identity is no longer possible.

6.4 Forensic Odontostomatology

Teeth provide a considerable amount of information about a person. A specialized field of forensics known as "forensic odontostomatology" is concerned with forensically relevant information gained from dentition.

For the purposes of forensic identification, the dentition of the deceased to be identified is analyzed in terms of the presence or absence of teeth, particular dental alignment, and dental treatment (Fig. 6.4). Dental treatment to teeth that are present is specified, e.g., crown, filling, and bridge, and a record is made of which of the five surfaces of each tooth is affected. This postmortem dental record is documented using a purpose-made form (Fig. 6.5).

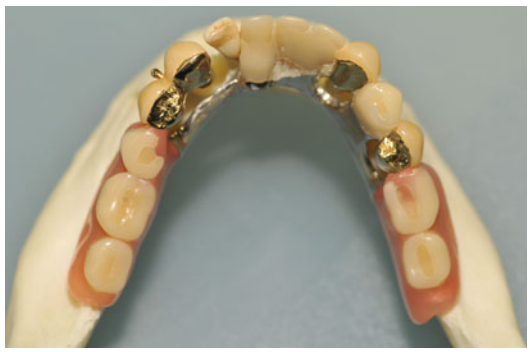

Fig. 6.4 Postmortem dental findings in a lower jaw. Complex dental treatment with partial prosthetic treatment (Image courtesy M.A. Verhoff, specimen courtesy of C. Grundmann, Duisburg)

A wide variety of dental formulas using many different coding systems for the individual teeth are currently in use internationally. Furthermore, in the past, different dental formulas were used at different times even within one country. Translating a foreign dental formula into one's own familiar formula can be challenging.

In Germany, a person's dental status is recorded on each visit to the dentist, such that current dental records are available for all regular dentist visits. Therefore, for the purposes of comparison, a treating dentist can compile or reconstruct the antemortem dental status of a missing person using dental records. Matches and variations are sought when comparing ante- and postmortem dental records. For example, if tooth 24 shows a filling post-mortem that is absent in the antemortem dental status, this is not considered an exclusion criterion since additional treatment by another dentist may have been performed. On the other hand, if tooth 24 is documented as having a filling ante-mortem but is found to be untreated post-mortem, this would exclude identity. However, possible errors should be borne in mind, in particular errors made during the compilation of antemortem dental records. Thus the above exclusion, for example, would carry all the more weight if teeth 23 and 25 were also untreated post-mortem, thereby ruling out the possibility of a simple "slipup" when the antemortem dental records were made.

Important: The greater the correspondence between ante- and postmortem dental records, the higher the likelihood that the records relate to the same person.

In contrast to forensic DNA analysis, there is no basis for calculating a probability of identity in forensic odontostomatology. Instead, an experienced dentist is required to estimate whether dental treatment is of a sporadic or of a more regular nature. In extreme cases, an isolated

			Filling		
X	Extracted	RF	Root canal filling	C	Crown, material unknown (mark pivot if pivot crown)
Xpm	Extracted / missing postmortem	SilF	Silicate filling	CG	Crown, gold
C	Caries	CO	Composite resin filling	CR	Crown, resin
AB	Abrasion	AM	Amalgam filling	CJ	Crown, jacket
TA	Tooth avulsed	IL	Inlay	CV	Crown, veneer
	Diastema in mm	...F	Other filling material described	CPa	Crown, Palliag

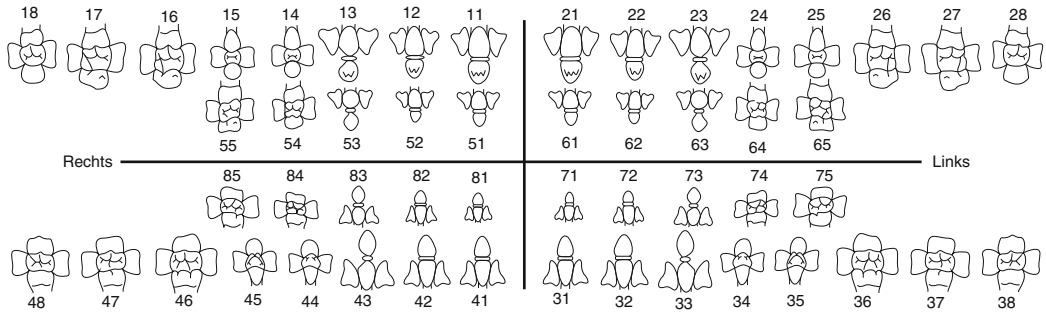

Fig. 6.5 A data entry form to record ante- or postmortem dental status. Using this form enables precise graphic documentation of dental status. The predefined abbreviations are used to record the various forms of dental treatment

instance of dental treatment recognized by the treating dentist can be sufficient to establish identity.

6.5 Comparative X-Ray Analysis

Given the large number of X-ray images produced on the basis of medical indications, the likelihood that antemortem X-rays of a missing person exist is relatively high. X-rays are then made of the body, using the same beam path as in antemortem images where possible. Any *correspondence in trabecular bone structure* or scar formation, etc., becomes apparent on direct comparison of ante- and postmortem X-rays. The *frontal and maxillary sinuses* show highly individual patterns on anteroposterior skull X-rays (Fig. 6.6)

As an accessory measure, ante- and postmortem X-rays can be compared using superimposition techniques: the two images are digitalized (if not already the case) and semitransparently superimposed using an appropriate image processing program. By shifting or scaling the images, a "match" is generally readily apparent.

In extreme cases, very small X-ray images, such as digital intraoral dental X-rays, are sufficient to establish identity (Fig. 6.7).

6.6 Skull–Photo Comparison

In cases where an intact skull—ideally including the lower jaw—is available, this can be used for comparison with portrait photos of a missing person. To this end, it is important to ensure that the skull is photographed at the same angle and from the same distance as the portrait photo. Additionally, spacers can be attached to defined points on the skull, which have been cut to correspond to soft tissue thickness at each individual point as previously investigated experimentally (Fig. 6.8). Using superimposition techniques, the portrait photo is superimposed over the skull and the size scaled (Fig. 6.9). For the purposes of identification, the facial proportions of the skull and photo need to correspond, and ideally the spacers should end at the facial borders shown on the portrait photo. Depending on how old the photo is, greater consideration may need to be given to possible age-related soft tissue variations or jaw atrophy due to tooth loss.

The most challenging aspect of skull–photo comparison is orienting the skull to match the angulation on the photo. Methods permitting superimposition already at the orientation stage are helpful here. This was first made possible by filming the portrait photo and the skull simultaneously

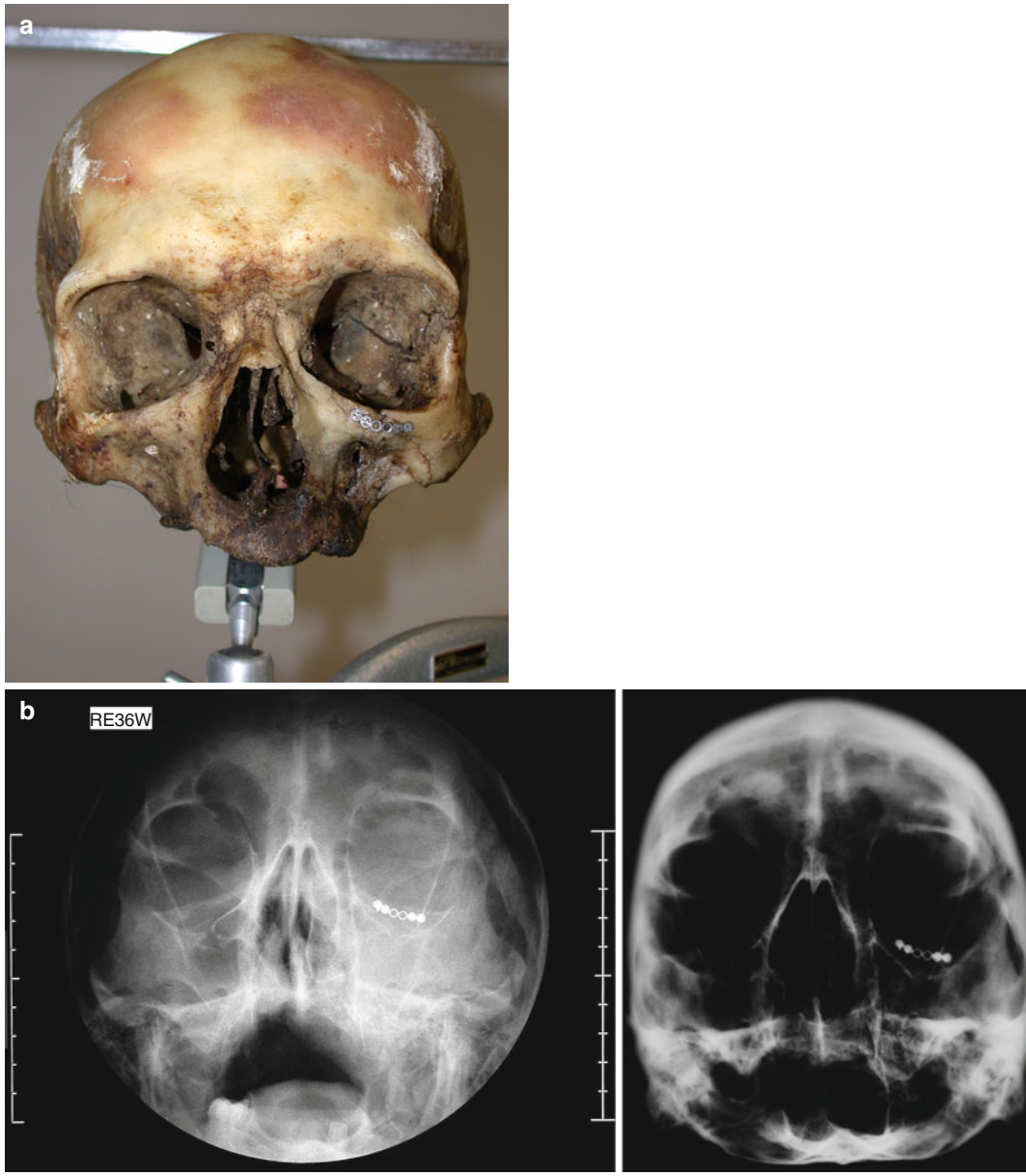

Fig. 6.6 Comparative X-ray analysis of a frontal skull X-ray. (**a**) A healed midfacial fracture that had been treated with a perforated plate could be seen on the skeletonized skull of this unknown deceased individual. The type of treatment suggested that it must have been performed at a center for maxillofacial surgery. Research was undertaken at the nearest center into a patient who had been treated with a plate of this kind 18 months previously. A postoperative follow-up X-ray was available (**b**, *left*). An X-ray of the skull using a comparable beam path was made (**b**, *right*). A highly individual correspondence could be seen, e.g., in the shape of the frontal sinus. Superimposition also produced a correspondence

with two different video cameras and relaying the videos to a video mixer for subsequent processing. This enabled the skull to be aligned in real time with the superimposed semitransparent portrait photo. Modern methods use the live-view function in digital cameras and a computer screen to display the video, onto which the digitalized portrait photo then can be mapped.

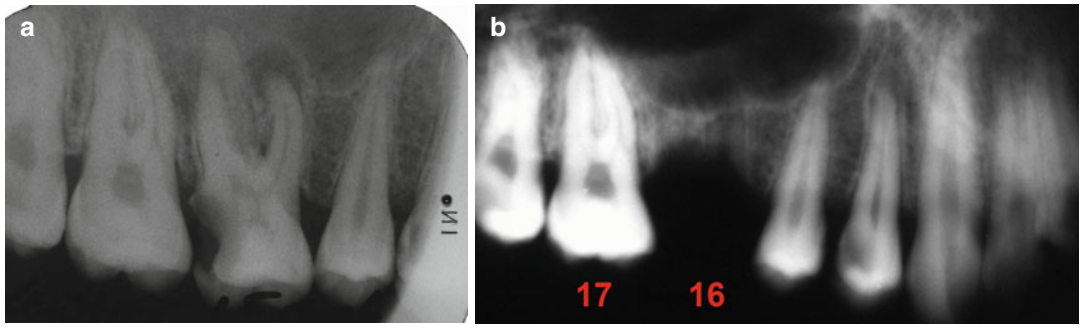

Fig. 6.7 Comparative X-ray analysis using a digital intraoral X-ray of teeth 15–18 (*top*), the only image available for a missing person. The indication for this X-ray is apparent: caries on tooth 16 and root canal infection. As a result, the tooth was extracted 3 months prior to death. The postmortem image (*bottom*) shows distinct correspondence between teeth 15, 17, and 18, as well as bony structures of the upper jaw. Tooth 15 has already moved somewhat distally towards the gap. Superimposition confirmed the correspondence

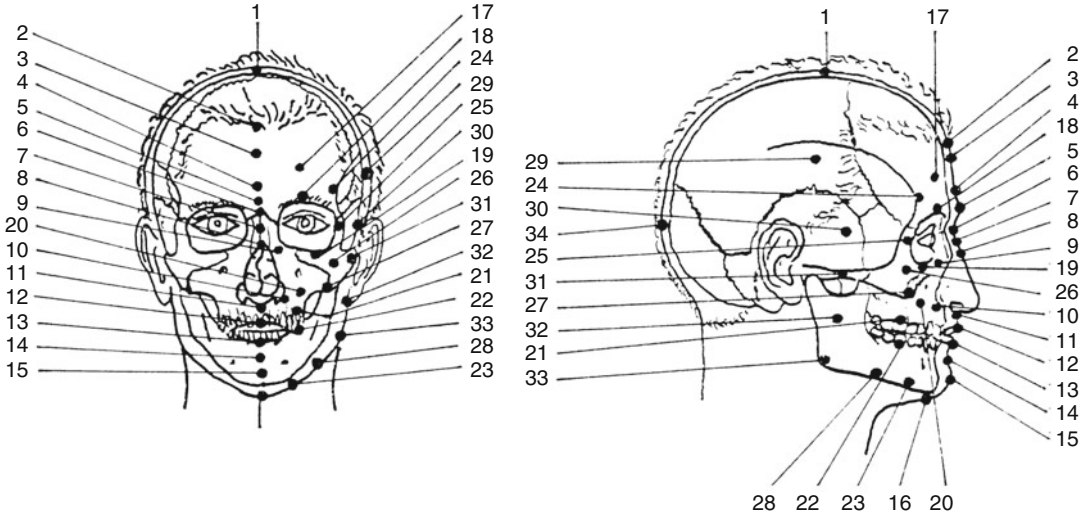

Fig. 6.8 Soft tissue landmarks on the skull. A total of 34 landmarks were defined on the skull and face in order to measure soft tissue thickness. In the case of skull–photo comparison or facial soft tissue reconstruction, these points on the skull are sought and marked with spacers that correspond to statistically investigated age-, gender-, and race-dependent average soft tissue thicknesses (landmarks) (From Helmer (1984))

6.7 Fingerprinting

Due to their high level of individuality, the analysis of fingerprints has been an established method of criminalistics for over 100 years (Fig. 6.10). Whereas early analysis depended on the direct comparison of fingerprints and their morphological description, digitalization opened up the way to the compilation of databases and automatic comparison.

Obtaining fingerprints is always indicated in the case of an unidentified decedent, the body's state of preservation permitting. Various techniques have been developed to obtain usable fingerprints from decomposed bodies: skin removal, "boiling," and electronic fingerprint scanning.

Fingerprints obtained from a body can be compared with national criminal investigation databases; however, a match will only be found if

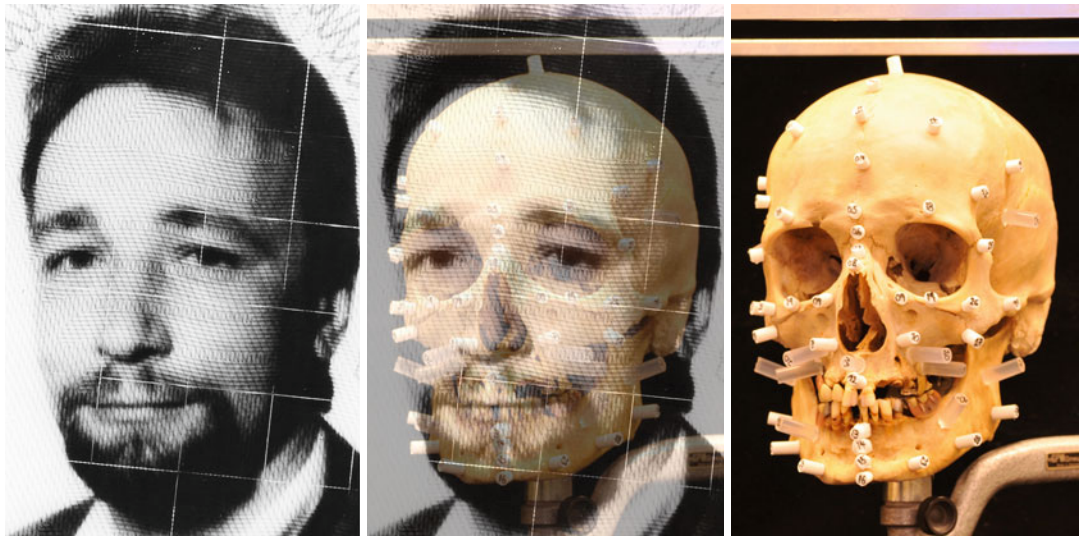

Fig. 6.9 Establishing identity by means of skull–photo comparison. An identity card photo taken more than 25 years prior to death was the only photographic material available for comparative purposes (*left*). The macerated skull was landmarked using spacers for central European men in the 50–59 years age group, placed at an identical angle to that on the portrait photo, and photographed (*right*). Similarities between the forehead, eye sockets, and nose were already apparent on morphological comparison. Once superimposed (*center*), the bony dimensions of the upper face and midface fit in the portrait photo. The boundaries set by the spacers in these areas line up with the skin surface. The spacers on the right lower jaw apparently protrude too far. However, taking the individual's dental status into consideration, one can assume that the lower jaw underwent significant modification in the 25–30 years prior to death; hence, this is not interpreted as an exclusion criterion

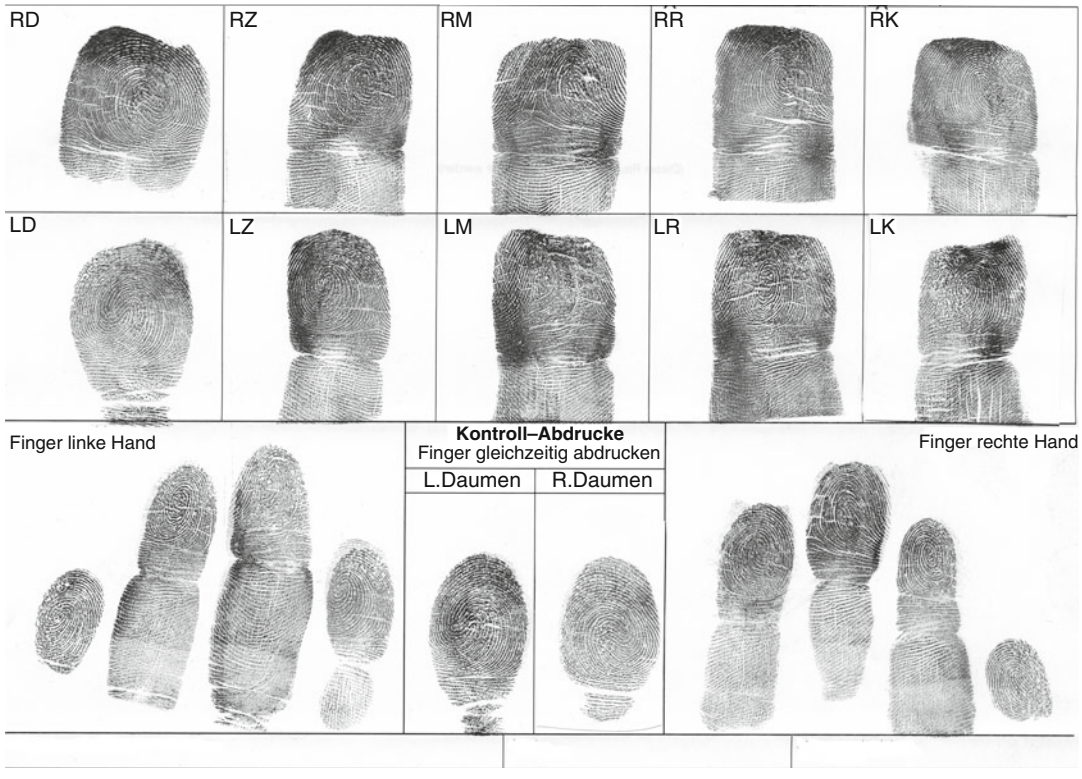

Fig. 6.10 Fingerprints for comparison purposes

an individual was fingerprinted during life. Where other investigative approaches lead to a missing person, fingerprints can be obtained from the individual's personal effects for comparison purposes. However, there will always be uncertainty here as to whether another individual has also touched the object.

6.8 Large-Scale Disasters: Disaster-Victim Identification (DVI)

A "large-scale disaster" is by definition one involving more than 100 victims. One particular aspect of large-scale disasters is the multitude of victims on the one hand and the often limited scenario on the other. In the case of a plane crash, for example, the passenger list provides the names of victims. However, a passenger may have travelled under a false identity. The strict procedure followed in such cases—usually led by the relevant identification commission of the respective office of criminal investigation—involves collecting all available antemortem data relating to the individuals suspected of being victims and comparing these with postmortem data. Investigations of this kind are usually carried out by interdisciplinary ante- and postmortem teams comprising criminal investigators, dentists, and forensic pathologists.

Methods used to establish identity in situations such as these include forensic odontostomatology, forensic DNA analysis, and fingerprinting. Having said that, all the methods discussed in this chapter are equally suitable. Method selection depends on the state of preservation of the body or remains, on the one hand, and on the availability of antemortem data, on the other. In Germany, dental records play the most important role in forensic identification following a large-scale disaster, since antemortem data is usually available for the majority of victims. Once this line of investigation has been exhausted, forensic DNA analysis and comparative X-ray analysis are then performed.

Most large-scale disasters are international incidents, given that victims often originate from different countries and the incident may have taken place in a yet another country. Establishing the cause of death and identity of victims is, in the first instance, the task of the national authorities of the country where the incident took place. National law applies. International collaboration in the identification process is only possible if the country in question authorizes—or requests—such collaboration. If this is the case, Interpol is placed in charge and coordinates the existing national DVI teams from its headquarters. The potential involvement of teams depends on the expected nationality of victims. Ensuring that standard forms are used, e.g., for collecting antemortem data of the missing person and postmortem data of the victim, is part of Interpol's coordination task. To this end, easy-to-use forms have been developed to replace the multitude of different documentation systems used worldwide. Moreover, the forms have been designed in such as way as to simplify comprehensive electronic data collection. The electronic collection of ante- and postmortem data, as well as automated comparison, forms the basis of modern large-scale disaster management. Since errors can occur in both the collection and the transmission of ante- and postmortem data, concepts in automated error-tolerant comparison have had to be developed.

6.9 Photographic Identification

Photographic identification is rooted in a long tradition of meticulously documenting characteristic facial features in a standardized form. Meanwhile, a separate field based on modern photographic methods has become established and plays a particularly relevant role in judicial routine. CCTV cameras are seen in many areas of public life. As a result, images of varying quality and taken under a variety of conditions are produced, which for identification purposes need to match a person with the highest possible level of probability of identity.

Important: By committing their face to a two-dimensional image, every human being creates a "landmap" comprising an individual

pattern of personal characteristics arranged in a specific constellation.

In order to *directly compare photos of two potentially identically persons*, images showing similar head posture and line of vision and taken using similar photographic techniques are required. Depending on image quality, *more than 100 characteristic features can be distinguished* and evaluated according to shape, structure, and degree of prominence. Problems may arise as a result of age differences at the points in time at which the images being compared were taken. For example, identity card photos of the individual under investigation may have been taken at an unknown point in time, possibly long before the document was actually issued.

In order for photographic identification to be successful, the established methods need to be consistently applied and documented. Subject to this condition, direct photographic comparisons can assist in the identification process, for example, in bank raids, computer fraud, the falsification of documents, traffic offenses, as well as many other types of criminal offense.

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Vital Reactions

7

All cases of fatality require that findings such as blunt force injury be analyzed to establish whether injuries are of ante- or postmortem origin, the latter possibly including "injury" to the body during transportation, as a result of dismemberment, or due to postmortem animal predation. If an injury was incurred ante-mortem, the age of the wound—among other factors—and whether there is a causal link between the injury and death are of particular interest. Likewise, findings at the scene where the body was discovered, such as traces of blood, may provide insight into the incident, the victim's position at the time of the incident, as well as the victim's capacity to act in the posttrauma period. Numerous so-called vital signs are encountered in forensic practice, the most relevant of which will be discussed here. Some findings are almost certainly of antemortem origin, while others are only *likely* to have been caused before death. Notable examples include:

Defense wounds: These wounds, either active or passive, are considered evidence of a physical confrontation or preserved capacity to act and are always caused ante-mortem.

Contact traces: Blood, saliva, or other fluids such as vomited gastric contents may be found either on the body or at the scene where the body was discovered. Although contact traces of this kind often occur ante-mortem, this is not always the case. According to the course a contact trace follows, it may be possible to estimate the victim's position, i.e., standing, sitting, lying in a supine or prone position, lying in a left or right lateral position, bending, or kneeling. Contact traces cannot always be unequivocally interpreted as a vital sign. Caution is advised in the case of long postmortem intervals and the escape of putrefaction fluids.

Also of interest is how long the victim survived an instance of blunt force trauma (survival time), as well as the sequence in which injuries were incurred. In addition to injuries from blunt force trauma, there are a number of vital reactions and processes considered as evidence that the victim was alive at the time of the incident.

Vital Reactions and Processes: Sometimes local findings, on other occasions verifiable and more complex physiological events providing evidence that an extraneous force or noxa (trauma, intoxication, etc.) came into contact with a living organism and that the organism was able to respond either locally or systemically.

Macroscopically visible vital reactions, e.g., injuries with hematomas, may be found on either external or internal examination, while other vital signs can only be detected using microscopy, chemical–toxicological analysis, or postmortem biochemical analysis. Vital findings that can be detected at the scene where a body is found or on external examination are given in Table 7.1.

Fat embolism, COHb levels in blood, and "agonochemical" stress reactions accompanied, for example, by increased adrenalin and noradrenalin blood levels, all represent vital signs detectable by microscopic, chemical–toxicological, or biochemical analysis. Tissue

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Table 7.1 A selection[a] of general findings indicating vitality made at the scene where a body is found or on external examination

Sign of vitality	Practical example/comments
Hematomas	Hand marks on the upper arm (age can be roughly estimated from the color and margins of the hematomas)
Defense wounds (active/passive)	To the extensor side of the lower arms or palms of the hands from attempting to resist a knife attack
Bloody shoe prints made by the victim at the crime scene	Shoe prints provide evidence that the victim was able to move
Traces of blood spattering from opened arteries	Blood spattering only occurs if the circulatory system is intact to provide the necessary blood pressure
Livor mortis of limited extent and intensity	Can be a sign of internal or external blood loss (caution: individuals with preexisting anemia), possibly requires investigation by autopsy
Bright red livor mortis, also in the nail-bed region	Carbon monoxide poisoning following active smoke inhalation; death due to hypothermia
Contact traces	Contact traces of blood originating from an injury consistent with the position of the body
Petechiae (somewhat more numerous)	Occur only if the circulatory system is intact (caution: hypostasis-related blood extravasation post-mortem, e.g., into the conjunctivae in a head-down position, does not represent vital petechiae)
Secretion of saliva and mucus	Rivulets of saliva from the mouth in the case of death by hanging
Subcutaneous emphysema	Palpable crepitus caused by gas in tissues in divers (caisson disease), gas inhalation, injury-related dispersion of air to soft tissues (Fig. 7.1) due to pneumothorax Caution: gas buildup due to putrefaction
Crow's feet-like areas of sparing from heat injury to the skin	From shutting eyes tightly due to extreme heat (see Fig. 12.8)
Wound healing response	Reddened wound margins due to reactive hyperemia and/or early wound organization

[a]See Tables 7.5 and 7.6 for other signs of vitality in the respiratory and gastrointestinal tracts

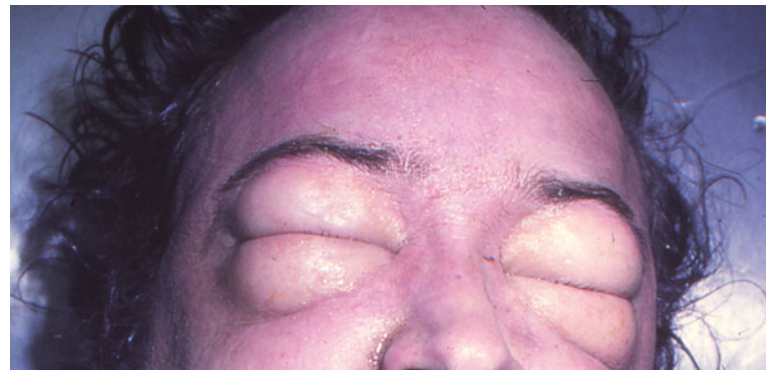

Fig. 7.1 Injury-related dispersion of air to soft tissues

reactions in the form of tissue injury organization, as in myocardial infarction or skin injury, are vital reactions.

Metabolic processes, such as increased blood glucose levels, constitute vital reactions that can be detected post-mortem, particularly by measuring glucose and lactate levels in cerebrospinal fluid or vitreous humor. If the combined values of glucose and lactate in liquor add up to more than 362 mg/dl, a lethal diabetic coma is considered probable, at values above 415 mg/dl highly probable, assuming all other

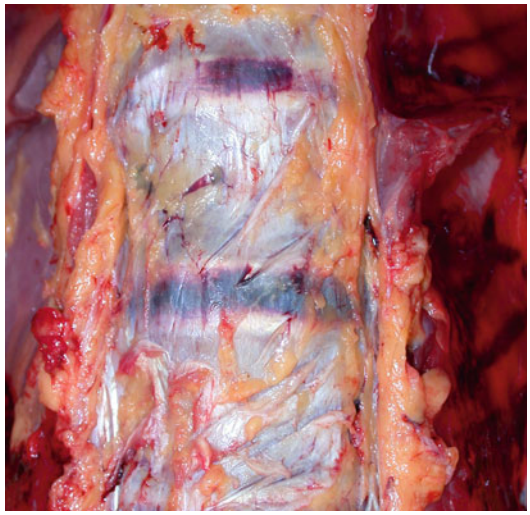

Fig. 7.2 "Simon's hemorrhage" into the intervertebral discs is a sign of vitality in death by hanging

explanations have been ruled out. Combined values (glucose + lactate) of 500 mg/dl and higher are often found in the case of death in diabetic coma. In cases such as these, determining the HbA1c value can be helpful. This combined glucose and lactate value according to Traub (1969) is believed to remain stable for up to 200 h post-mortem. Glycogenic nephrosis could represent a further indication of antemortem hyperglycemia.

When interpreting vital reactions, patterns of evidence at the scene of the incident should also be taken into consideration, since these can sometimes permit inferences to be made about the victim's capacity to act (e.g., the victim's shoe- or footprints in areas of blood in cases where traces of blood are found on the soles of shoes or socks) or the sequence of injuries. Arterial spurting patterns need to be differentiated from projected blood patterns caused either by striking a bleeding wound or a pool of blood or by spatters of blood originating from the striking instrument. Hemorrhage frequently provides evidence that the organism has been exposed to an effect during life but can often only be detected at autopsy, e.g., seizure-related hemorrhage into the tongue musculature in death due to epileptic seizure or into the intervertebral discs in death by hanging (Fig. 7.2).

7.1 Vital Reactions: Hemorrhage

Injury to arteries, veins, and capillaries generally leads to hemorrhage per rhexis at the site of trauma, e.g., wound margins with hematomas. However, if the difference in pressure is sufficiently great, postmortem blood loss is also seen. This type of blood loss needs to be differentiated from hemorrhage per diapedesis or hemorrhage due to clotting disorders (e.g., in hemorrhagic–hypovolemic and septic shock or during heparin or phenprocoumon-therapy). Capillary bleeding manifesting as skin petechiae or mucosal ecchymosis is the result of an increase in intracapillary pressure. The following applies to congestive hemorrhage caused in this way:

Important: The higher the intracapillary congestive pressure, the shorter the time to onset of petechiae or ecchymosis.

Petechiae and ecchymosis may also appear post-mortem, particularly in the facial skin, conjunctivae, and buccal mucosa in the case of a head-down position. Therefore, with increasing postmortem intervals, congestive hemorrhage of this kind should only be interpreted as a vital reaction in body areas not subject to hypostasis.

Hemorrhage often causes hematomas which, by virtue of their type (size, shape), localization, and color, are of particular relevance in expert forensic reconstructions. Table 7.2 provides an overview of macroscopically detectable hemorrhage as a sign of vital reactions.

Death by exsanguination occurs in adults from a blood loss of around 1.5 l or after acute blood loss of approximately 40 % of blood volume. Hemorrhagic–hypovolemic shock can develop despite immediate substitution (blood transfusion, volume substitution). Both the macroscopic and microscopic detection of shock organs represents a reliable sign of vitality (e.g., myocardial necrosis, shock lung with hyaline membranes, megakaryocyte embolism, hemorrhage and edema, shock liver with centrilobular necrosis, shock kidneys, acute ulcers of the mucosa of the stomach and/or small intestine as signs of shock).

Hematoma Age. The age of hematomas appearing posttrauma can only be approximated according to their borders and color and depends

Table 7.2 Hemorrhage as a vital reaction

Type of trauma	Vital reaction of the organism
Injury to large arterial or venous blood vessels	Hemorrhage into surrounding soft tissue (e.g., into intercostal tissue in rib fractures, femoral musculature in femoral fractures, and retroperitoneal soft tissue) or in preformed body cavities (hematothorax, cardiac tamponade, intra-articular hemorrhage, bloody ascites)
Injury to large vessels and significant blood loss (exsanguination)	Sign of hemorrhage: livor mortis of mild extent and intensity (DD: anemia!), the color of internal organs becomes more prominent, wrinkling of the splenic capsule, striated subendocardial hemorrhage, anemia of the skin and mucosa
Compressive force accompanied by an increase in intracapillary pressure (e.g., atypical hanging, ligature strangulation, manual strangulation)	Bleeding in the form of skin petechiae (e.g., in the neck and facial skin above the level of compression in ligature strangulation, above and below the site of trauma in Perthes pressure congestion)
Traumatic basilar skull fracture	Bleeding into the oral cavity accompanied by deep blood aspiration (*caution*: passive postmortem passage of blood into the respiratory tract is possible)
Hematomas of the skin and mucosa	A vital reaction if well demarcated, distant from areas subject to hypostasis, and already showing changes in color or histological signs of hematoma organization; enzyme and immunohistochemical analyses are sometimes helpful in determining vitality and establishing wound age

on several factors, most notably the localization of hemorrhage, its extent, and its depth in tissue.

Important: When examining surviving victims of violent trauma, it should be borne in mind that hematomas often reach their most intense manifestation 12–24 h following an incident.

Hematomas of limited extent and intensity are resorbed more rapidly than large hematomas. The relatively sharp borders of the early grayish-blue hematoma are replaced by more poorly defined borders with increasing age. In cross section—orthogonal to the skin—fresh hematomas are remarkable in that they have a shiny blackish-red color and well-demarcated borders, while older hematomas take on an increasingly matt brownish-red color and lose their well-defined borders. Most notably large hematomas that extend deep into tissue as far as hemorrhagic cavities can be striking in terms of their "fresh" appearance at the center, while border areas already show yellowish-green discoloration.

Any assessment of hematomas, in particular age estimations, should be made with caution. Sometimes it is only possible to clarify whether hematomas occurred at the same or at different time points. Although subject to considerable variation, the color changes seen in hematomas in relation to the passage of time are largely assumed to correspond to the times shown in Table 7.3. Older hematomas always demonstrate signs of vitality, most notably color changes and histological signs of hematoma organization.

Table 7.3 Changes commonly seen in hematomas

Color and borders	Hematoma age
Grayish blue: well-defined borders and shiny blackish red in cross section	Fresh
Bluish violet: no longer quite fresh and with somewhat poorly defined borders	Several days at most
Greenish: no longer fresh and with undefined borders	At least 4–5 days, usually 6–8 days
Yellowish: clearly older and with notably ill-defined borders	Approximately 8 days
Brownish red	No assessment possible

Important: A description of each hematoma in terms of its localization, shape, orientation, borders in relation to the surrounding area, and color with the aid of a color scale is recommended, alongside photodocumentation using a scale of reference. Patterned hematomas showing the imprint of an object are of particular interest.

7.2 Vital Reactions: Embolisms

Any type of embolism is considered a vital reaction, since a functioning circulatory system is a prerequisite for the embolic spread of substances, cells, foreign bodies, etc. Autologous as well as

Table 7.4 Types of embolism relevant in forensic medicine

Type of embolism	Possible causes or relevance in an expert appraisal (examples)
Acute or recurrent pulmonary arterial thromboembolism	For example, patients confined to bed following trauma (recent or older traffic accident?). Spontaneous thrombosis and thromboembolism? Proper anticoagulation? Infected thrombus?
Fat embolism (in lung arterioles and septal capillaries, in renal glomeruli, intracerebral)	Posttraumatic (detachment?) Death due to hypothermia? Status post liposuction? Preexisting disease, such as fatty liver?
Amniotic fluid embolism	Death during pregnancy? During birth?
Megakaryocyte embolism	Shock: terminal shock due to various causes
Foreign body embolism following intravenous drug abuse	"Junkie pneumopathy"
Gas embolism: air embolism, nitrogen embolism	Particularly following cut-throat injuries with opening of larger veins, suicidal venous injection of air, nitrogen embolism in the case of caisson disease (decompression sickness)
Bone marrow embolism	Posttraumatic in the case of fractures to large long bones (e.g., traffic accidents), shock, intraoperative, primarily in femoral head endoprosthesis surgery
Tissue embolism	Embolic spread of specific organic tissue, in rare cases as a tumor tissue embolism (parenchymal embolism)
Arterial embolism	Generally thromboembolisms, originating from (occasionally infected) parietal thrombi in the left heart (atrium thrombus), the heart valves, the endocardium, or after traumatic damage to the vascular intima Atrial fibrillation? Endocarditis? Thrombosed myocardial aneurysm?
Cholesterol crystal embolism	Rare, arterial-embolic spread of cholesterol crystals from atherosclerotic plaques
Parasitic embolism	Rare, embolic spread of parasites or parasite components
Bacterial embolism	Bacterial spread in the presence of sepsis, such as focal nephritis in the case of bacterial endocarditis lenta; septic or infected (thrombo)embolus
Iatrogenic embolism	e.g., TURP syndrome with intraoperative embolic spread of rinsing fluid via the open veins of the prostatic venous plexus, embolism following puncture, lime cement embolism in the case of total endoprosthesis, silicone embolism syndrome
Tumor embolism	Rare, embolic spread of tumor cells
Other foreign body embolisms	e.g., embolically spread projectile after a gunshot wound
Traumatic embolism	Embolism caused directly by trauma, e.g., cerebral embolism following trauma to the carotid artery, dissection and thrombosis of the carotid or vertebral artery following chiropractic therapy

foreign substances may be spread (fat, cells, tissue, air, oils, or projectiles) (Table 7.4). A number of embolisms can only be detected microscopically.

A paradoxical embolism is considered a rare entity:

Paradoxical Embolism: The embolus originates in a vein of the systemic circulation and is spread via the foramen ovale or arteriovenous anastomosis into the arterial circulation.

Histological analysis may help to:

- Localize the origin of the thrombosis or thromboembolism.
- Classify the intensity of a pulmonary fat embolism in particular (Table 7.5).
- Attribute a megakaryocyte and bone marrow embolism to either a trauma, a surgical intervention, or a shock event.
- Determine survival time following embolism or estimate the age of thromboses and thromboembolisms.
- Determine the age of other embolisms.
- Detect remnants of an embolism that has taken place (e.g., hemosiderin deposits at fibrous tissue junctions in the case of a macroscopically detectable rope-ladder pattern in pulmonary artery branches-due to an old, survived pulmonary embolism organized by connective tissue).

Forensic pathology investigations are largely concerned with those thrombo- and fat embolisms which, once detected, are able to establish a causal link between an incurred trauma and death (Figs. 7.3, 7.4, 7.5, and 7.6).

Lethal fat embolism requires approximately 20–30 g fat, while air embolism needs air volumes of at least 70 ml once veins adjacent to the heart have been opened (see Fig. 4.8). At autopsy, samples of the air or gas should be taken and analyzed using gas chromatography in order to either detect or exclude typical putrefaction gas components such as carbon dioxide, hydrogen, methane, and hydrogen sulfide.

In the case of embolism, obstruction of the pulmonary circulation causes a partially acute/partially gradual rise in pressure, resulting in right heart failure. Moreover, an initially survived pulmonary fat embolism should prompt investigation of a possible cerebral fat embolism, which can cause death after hours or days.

Table 7.5 Classification of pulmonary fat embolism

Extent of fat embolism	Form of fat embolism	Localization of fat embolism
I = mild fat embolism	Teardrop-like	Scattered, but in every field of vision at 25× magnification
II = distinct fat embolism	Lake- or sausage-shaped	Multiple fat emboli, disseminated in every field of vision
III = massive fat embolism	Fat emboli with antler-like configuration	Visible in huge numbers in all regions, no field of vision without fat emboli
0 = no fat embolism	Punctiform when present	Possibly visible in isolation, never in all fields of vision

According to Falzi et al. (1964), Modified from Janssen (1977), From Dettmeyer (2011); evaluation at 100× magnification

7.3 Vital Reactions: Findings in the Respiratory and Gastrointestinal Tracts (Acute Pulmonary Emphysema, Aspiration, Inhalation, Erosions of the Gastric Mucosa)

In addition to hemorrhage and embolism, numerous other findings provide evidence of vitality at the time of trauma or injury. Aspiration and inhalation of fluids, gases, and other substances into the tracheobronchial system provide postmortem evidence of a reaction of the organism. Generally, aspiration is interpreted as a vital sign when blood, soot or soot particles, and gastric contents have been aspirated; victims that have been buried alive may aspirate the substance in which they have been buried, such as sand,

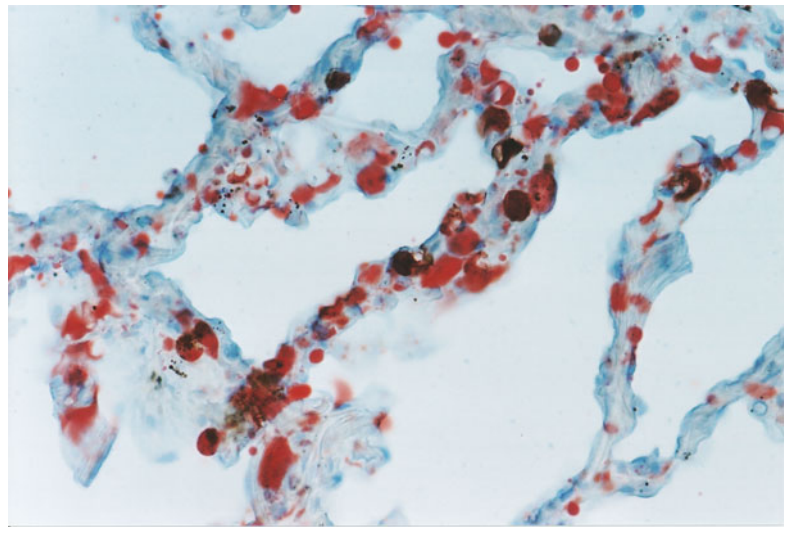

Fig. 7.3 Pulmonary fat embolism with fat droplets in the capillaries of the alveolar septa: a polytrauma patient after a traffic accident and a survival time of approximately 9 h (Sudan III ×400)

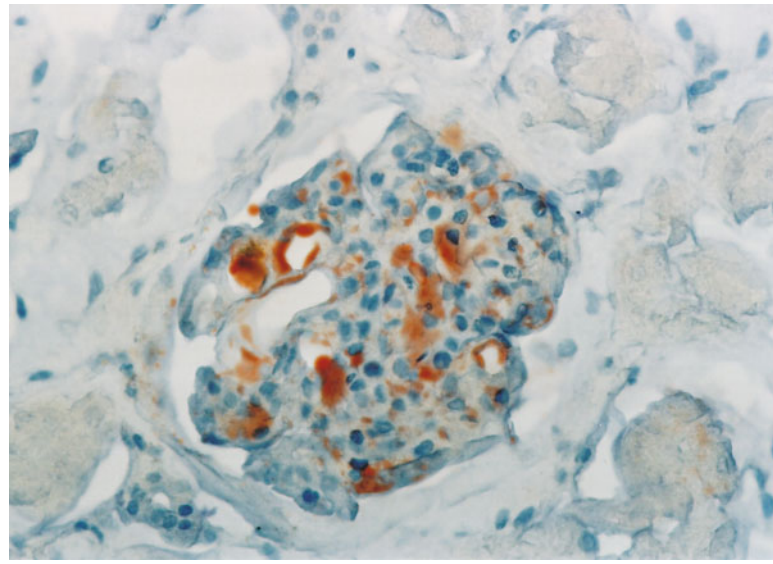

Fig. 7.4 Renal glomerular fat embolism in a polytrauma patient: a traffic accident victim with a survival time of approximately 6 h (Sudan III ×400)

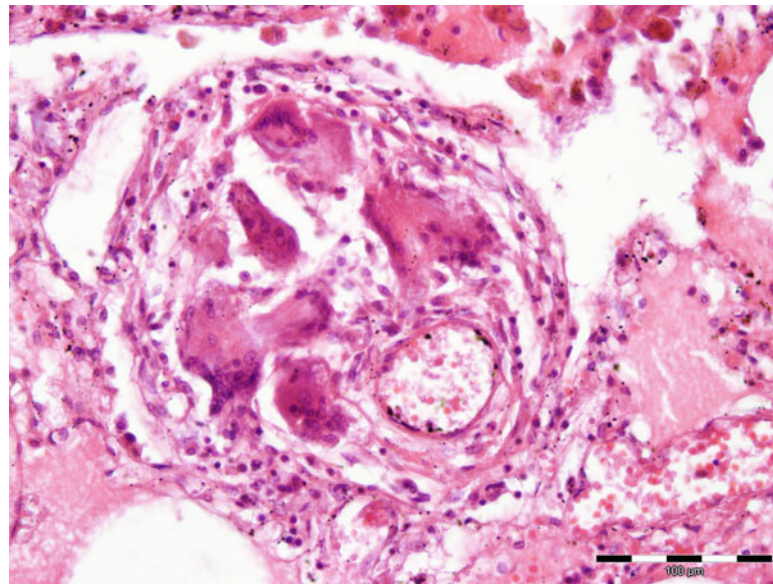

Fig. 7.5 Pulmonary granuloma in a case of junkie pneumopathy: embolically spread foreign material following intravenous injection and a foreign body reaction (H&E ×400)

whereas drowning victims aspirate the drowning medium (fresh-/seawater or other fluids) in the case of death by drowning.

Important: Evidence of deeply aspirated material reaching as far as the peripheral branches of the bronchial tree (in the bronchioles) is required, since fluids can reach the main bronchi in a passive manner, e.g., during transportation of the body.

Findings of varying severity in the respiratory tract caused by trauma-related overinflation of pulmonary tissue (acute pulmonary emphysema) and following aspiration or inhalation can provide evidence of vitality at the time of trauma (Table 7.6, Fig. 7.7); other findings verifying a vital reaction can be found in the gastrointestinal tract (Table 7.7).

Although the detection of drowning medium in the stomach is considered a sign of vitality, it is not necessarily evidence of death by drowning. White foam emanating from the mouth and nostrils (a "foam cone") is an antemortem event;

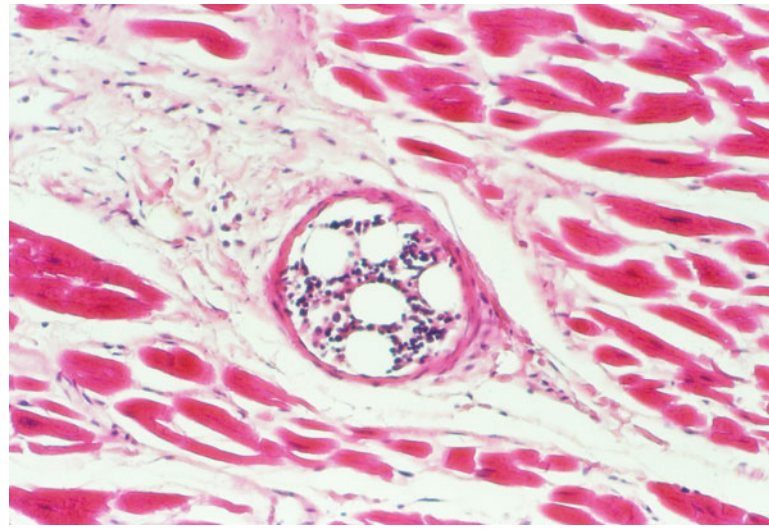

Fig. 7.6 Pulmonary bone marrow embolism: death on the operating table during implantation of a femoral head endoprosthesis following femoral neck fracture (H&E×100)

Table 7.6 Important vital reactions: respiratory tract findings following inhalation/aspiration

Finding	Cause and evidence of a vital reaction
Acute pulmonary emphysema	Final vigorous respiratory excursion [asphyxia, neck compression, respiratory tract obstruction—with the exception of bolus death—particularly peripheral overinflation of the lungs and ruptured alveolar walls (histology!)]; pulmonary lobes meet in the midline, e.g., in death by drowning, emphysema aquosum *Caution*: resuscitation with ventilation, putrefaction
Deep soot inhalation[a]	Smoldering fires: detection of soot in the peripheral branches of the bronchial tree often accompanied by toxic COHb levels in blood
Heat inhalation injury	Hot air inhalation: histologically detectable thermal injury to the respiratory epithelium with basal vacuoles and elongated cell nuclei, often associated with soot particle deposition
Blood aspiration[a]	Frequent finding in craniocerebral trauma with basilar skull fracture, chessboard-like or leopard skin-like arrangement of subpleural hemorrhages following deep blood aspiration
Amniotic fluid aspiration	Peripartum aspiration of amniotic fluid, histological detection of amniotic fluid components, particularly anucleated keratin lamellae in the pulmonary alveoli (Fig. 7.8)
Brain tissue aspiration	Evidence of brief preservation of respiratory activity in severe craniocerebral trauma
Fluid aspiration	Aspirated blood or other fluids mix with oxygen in the respiratory tract to produce bloody foam or foamy fluid ("foam cone" at the mouth and nostrils possible)
Positive hydrostatic test	In neonates to establish vitality outside the uterus

[a]*Caution:* A sign of vitality if extending to the peripheral branches of the bronchial tree—a passive passage of the "aspirate" into the peripheral bronchi is in principle possible; chyme aspiration during the agonal phase is relatively common! Intubation- and ventilation-related spread is also possible!

however, its relevance in terms of cause of death needs to be considered against the background of all other available information.

Important: A (blood-tinged) foam cone emanating from the mouth and nostrils may be due to the aspiration of blood or drowning medium or a sign of severe hemorrhagic pulmonary edema with foamy fluid rising retrogradely as far as the mouth and nostrils, e.g., narcotics fatalities.

In the case of bodies found in water following death by drowning, a foam cone may only appear once the mouth and nostrils have been placed above water level.

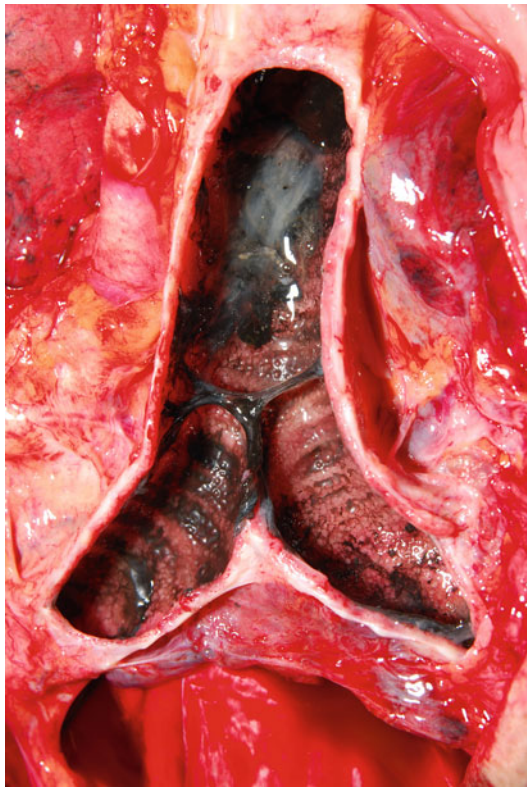

Fig. 7.7 Deep aspiration of soot particles as a sign of vitality at the time a fire broke out

Table 7.7 Important vital reactions: selected findings in the gastrointestinal tract

Finding	Cause and evidence of a vital reaction
Swallowing of soot	Detection of soot particles in the stomach following active antemortem swallowing in fire deaths
Erosions of the gastric mucosa	In the context of a shock event or in the form of Wischnewsky spots in hypothermic deaths (see Chap. 12)
Swallowing of other substances/fluids	Involuntary swallowing or peristaltic transport of blood, foreign bodies, tooth or denture fragments, or drowning medium (when in isolation, this is not considered evidence of drowning!)
Radial tears in the gastric mucosa	Drowning may lead to distension of the gastric wall, causing tears in the gastric mucosa
Positive hydrostatic test on stomach and intestines	Used in combination with the hydrostatic test on lungs in neonates to establish vitality outside the uterus

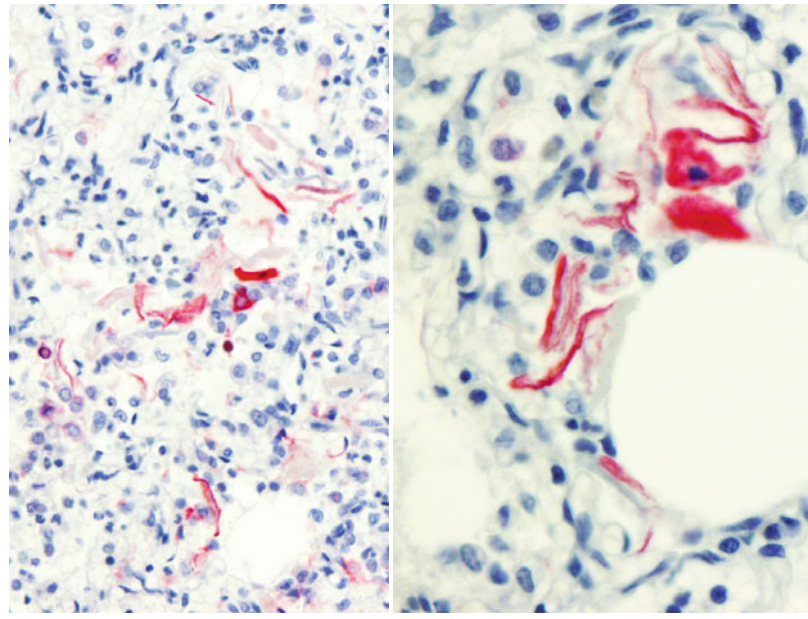

Fig. 7.8 Neonaticide: positive hydrostatic test (see Figs. 20.2 and 20.3). Lung tissue following amniotic fluid aspiration: intra-alveolar, partially anucleated keratin lamellae exfoliated from the fetal epidermis can be seen using immunohistochemistry (anti-cytokeratin ×250; ×400)

7.4 Other Vital Reactions: Skin Reactions, Wound Healing, Fat and Muscle Tissue, and Biochemical Vital Reactions

In the same way that small hemorrhages in soft tissue due to blunt force trauma can be inflicted post-mortem, (dermal) injuries or electrical burns with discrete hyperemic margins need not necessarily be of antemortem origin. Tissue transection during life triggers a retraction response of varying intensity, e.g., in muscle tissue, arterial and venous vascular stumps, skin, and connective tissue. Tissue responses of this kind are not a reliable vital response, particularly not in the supravital phase when tissue can retract post-mortem. Hemorrhage can be seen in the case of blunt force trauma and destruction of structures outside the immediate area of trauma, e.g., subperiosteal bleeding at the base of muscles due to tearing forces, in which case it is considered to have occurred ante-mortem. The same applies to subperiosteal bleeding at the base of the clavicle/neck muscles due to tearing forces in the case of hanging.

When estimating the age of a vital reaction histologically (forensic time estimation), enzyme- and immunohistochemical staining can be helpful in the early posttraumatic phase.

Histologically and/or *immunohistochemically detectable reactions*, particularly at wound margins, sometimes represent a vital reaction:

- Immunohistochemically detectable expression as an indication of a wound reaction (e.g., fibronectin, TGF-α)
- Granulocyte, monocyte, macrophage, and fibroblast invasion
- Subsequently branched capillary blood vessels

The principle wound healing processes can be identified using conventional histological staining techniques. The time periods given in Table 7.8 can be used as a guide when determining the approximate age of injuries: an estimation

Table 7.8 Chronology of wound healing

Time following injury	Histological findings and enzyme histochemical reactions
<20 min–1 h	Hemorrhage with destroyed tissue and cells, but with no cellular reaction, in particular no signs of granulocytic invasion
<1 h	Neutrophil granulocytes, partly marginated to the inner vascular wall, partly amoeboid migration into the tissue
1 h	Fresh hemorrhage, tissue edema, local acidosis, single polymorphonuclear leukocytes, evidence of ATPase, unspecific esterase, aminopeptidase, increased histamine, serotonin, α-esterases
2 h	Mast cell degranulation, infiltration of polymorphonuclear leukocytes, fiber necrosis, ground substance segregation, extracellular activation of fermentation: glucosidase, monoamine oxidase
2–4 h	Monocytic-cell invasion, phagocytic reactions
4–6 h	Peripherally increasing reactive hyperemia, fibrin deposition, peripheral formation of a leukocyte wall, also involving granulocytes
6–8 h	Necrobiosis of cells and tissue, distinct inflammatory demarcation, increasing phagocytosis
8–12 h	Increase in and further activation of mononuclear cells and histiocytes, invasion of single macrophages, evidence of alkaline phosphatase, cytochrome oxidase, and phosphorylases
12–16 h	Mononuclear cells gradually predominate, leukocyte degradation
16–32 h	Mobilized histiocytic cell elements, formation of collagen fibers with fibroblasts and fibrocytes, angioneogenesis with first branched capillary blood vessels
32–72 h	Formation of granulation tissue with collagen fiber tissue and capillary blood vessels, embedded macrophages (siderophages, lipophages)
3–4 days	Ground substance formation, denser collagen fiber tissue, potential decrease in the number of macrophages, new formation of mast cells, possibly polynuclear foreign body giant cells
4–10 days	Decrease of histochemical reactions in collagen fiber tissue, densification of scar tissue, decrease in the number of leukocytes and macrophages, possible persistence of siderophages
>10 days	Denser scar tissue with fewer cells, decreasing vascularization, potential persistence of hemosiderin deposits; after a significant length of time, basophilic calcium salt deposits are also possible

From Dettmeyer (2011), Modified according to Janssen (1977)
Considerable variations or differences are possible

Table 7.9 Terminologically possible statements on vitality as well as guideline time periods for the estimation of wound age based on conventional histological staining of correctly obtained tissue samples (Dettmeyer 2011)

Statement	Histological findings
Wound incurred shortly before or after death	Wound shows no signs of an active immune reaction or active wound healing: no conclusion as to vitality or wound age possible
Vital wound, i.e., incurred ante-mortem	Signs of an active immune reaction, in particular invasion of neutrophil granulocytes, invading macrophages, and fibroblasts Hemorrhage, fibrin deposition, and thrombocyte aggregates alone are not sufficient to assume a vital injury; this also applies to detectable peripheral hyperemia (supposedly reactive) at the wound margin
Fresh vital injury (hours to a few days)	Clear signs of a body reaction with invasion of neutrophil granulocytes and signs of an early wound repair process: macrophages, fibroblasts, branched capillary blood vessels, hemosiderin deposits, polynuclear foreign body giant cells. Fibrin deposition and thrombocyte aggregates alone do not permit a reliable statement on wound age
Vital wound, no longer fresh (a few days to weeks, in the single-digit range)	Signs of resorption and wound repair extending from the wound margin to the deep recesses of the wound, clear collagen fiber tissue (fibroblasts, fibrocytes), invading macrophages and lymphocytes, hemosiderin pigment-laden macrophages, polynuclear foreign body giant cells, granulation tissue with capillary blood vessels coated by endothelium, areas of scarring with scant cells
Vital injury, not yet old (weeks to months)	Repaired wound with scar tissue, partly vascularized containing loosely spread lymphocytes and macrophages
Vital, old healed injury (many months to years)	Dense collagen scar tissue without leukocytes, no or few embedded blood vessels, residual hemosiderin pigment deposits; basophilic calcium salt deposits can occur in old and dense bradytrophic scar tissue

Considerable intra- and interindividual variations possible

of the time interval between the time of injury and the time of death. Nevertheless, a certain degree of caution is recommended when determining either. The formulations given in Table 7.9 may be helpful in the determination of wound age.

In specific cases, biochemical processes may be seen as vital reactions, in particular the so-called agonochemical stress reaction, whereby catecholamine levels measured post-mortem are believed to correlate with the duration of the agonal phase. This type of analysis can be helpful in, for example, the differentiation between homicide by strangulation and death due to vasovagal inhibition.

7.5 Postmortem Manipulation

Postmortem manipulation or artificially produced effects can sometimes lead to findings that would appear to be of antemortem origin. Active insufflation of gases, fluids, substances, etc., can produce the clinical picture of emphysema (resuscitation and ventilation!), i.e., postmortem infiltration of fluids due to high hydrostatic pressure depending on water depth. Postmortem insufflation of hot air can cause mucosal injury similar to that seen in antemortem heat inhalation injury. Extensive charring of the trachea and lungs can simulate soot aspiration. No solid components are able reach the stomach without postmortem manipulation, nor can peristaltic transport of gastric contents to the duodenum take place.

7.6 Signs of Vitality: Indications of Preserved Capacity to Act Posttrauma

Not all instances of trauma cause a victim to immediately lose their capacity to act. Even following stab, incision, or gunshot wounds, an individual's capacity to act may be partially or entirely preserved, either temporarily or for an unlimited period of time. The capacity to act depends on the localization and severity of injury. Alongside conscious actions, unconscious automatisms, reflexes, and seizures are also possible.

A preserved capacity to act should be considered in the following example cases:

- Self-inflicted injuries, which only in the sum of their effects subsequently led to an incapacity to act or the last of which caused the individual's loss of capacity to act.
- Conflicting statements, relating in particular to the chronology of an event.
- The scene at which the body was discovered is not the same as the crime scene and the possibility that the unconscious individual or body was moved by a third party can be ruled out.

In forensic practice, a preserved capacity to act is seen even in cases of gunshot wounds to the head, particularly when only the frontal lobe is injured (generally in the case of small-caliber projectiles). However, brainstem injury, as well as injury to the midbrain and diencephalon, generally causes an immediate incapacity to act.

In the case of stab or incised wounds, the time to loss of capacity to act depends largely on the speed of blood loss. Injury to the cardiac cavities, aorta, and pulmonary artery can cause rapid massive hemorrhage; acute cardiac tamponade (death from around 250–300 ml blood in the pericardium) or massive blood aspiration, on the other hand, may have a limiting effect. Less rapid blood loss usually results in a preserved capacity to act until hemorrhagic shock occurs.

Compression trauma to the neck, most notably hanging, causes extremely rapid loss of capacity to act and consciousness, whereas in the case of skull fractures and cerebral injury, an individual's capacity to act may be preserved depending on injury intensity and localization. Injury to the cervical medulla, while not impairing an individual's consciousness, can cause immobility, which in turn renders an individual unable to act. Abdominal stab wounds may be survived for a significant period of time if no large blood vessels are injured. Purulent peritonitis is a common cause of death following long survival times.

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Blunt Force Trauma

8

Case Study

The body of an approximately 40-year-old man was found at the base of a stone staircase, where the head lay in a pool of blood, the legs on the bottom step. Of the victim's otherwise undisturbed clothing, only the shirt showed extensive traces of blood on the front and somewhat more medially on the back side. Tears could be seen to two upper button holes, and the shirt seams on the left-hand side were torn at the armpit. Hematomas and abrasions to the right side of the forehead, tip of the nose, and chin were apparent, as well as a longitudinal laceration to the oral mucosa, a left-sided monocle hematoma, and a striated laceration to the back of the head above the occipital frontal circumference. In addition, two hematomas each measuring up to 2.5-cm diameter could be seen in the medial third of the flexor side of the right forearm, as well as two smaller hematomas on the inner and outer side of the left upper arm, respectively. A nasal bone fracture was palpable. A distinct odor of alcohol was detected at autopsy. The cause of death was found to be a basal skull fracture and deep blood aspiration at a blood alcohol concentration of 2.6‰.

8.1 Injuries: General Forms, Descriptions, and Causes of Death

A central task of forensic medicine is to assess injuries in order to establish their origin. How and when they originated play an important role here. Often, the objective of an assessment is to support or refute specific allegations or to establish which of varying reports on the sequence of events is more probable, e.g., on the cause of uni- or bilateral periorbital hematomas (monocle hematoma or "raccoon eyes," Fig. 8.1).

Making a precise description of an injury, including its exact location, extent, color, and the tissue types or layers involved, is paramount in forensic practice. This description is documented in writing and findings are photographed (with a reference scale). Using this information, the injury can then be assigned to an injury type in a first step.

The sequelae of traumatic injury need to be precisely identified before a comparison can be made with the alleged incident. Injury findings often form the only basis on which to reconstruct an incident, added to which the information provided by affected individuals is not always accurate, particularly if those persons may face legal consequences.

In addition to differentiating between primary and secondary causes of death following trauma, vital reactions are of great importance, i.e., findings indicating that the victim was alive at the time of the incident or blunt force trauma (see Chap. 7). Time

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estimations are also important in the reconstruction process, e.g., the age of a wound, hemorrhage, thromboembolism, or other types of embolism.

Alongside blunt trauma, further distinctions are made between the various types of trauma:

- Sharp and semi-sharp force (Chap. 9)
- Mechanical trauma, including gunshot wounds (Chap. 10)
- Exposure to heat and cold (Chap. 12)
- Electrical energy, including lightning (Chap. 13)
- Various forms of asphyxia and abnormal pressure conditions (Chap. 14)
- Death by starvation or dehydration (Chap. 16)
- Various forms of intoxication (Chaps. 29 and 30)

In cases where trauma has lethal sequelae, primary trauma-related or immediately fatal courses need to be distinguished from secondary sequelae of trauma (Table 8.1).

Any adequate expert assessment requires a precise description of all injuries resulting from blunt force trauma (Table 8.2). Assessments aimed at reconstructing an incident become all the more challenging when only inaccurate and generalized information is provided.

Injury types (Table 8.3) can show overlaps, intermediary forms, and combinations, sometimes making an exact classification difficult if not impossible. Numerous textbooks omit to mention "semi-sharp force," for example, as a distinct type of trauma.

Patterned wounds caused by kicks (Fig. 8.2), hammer (Fig. 8.3), manhole cover (Fig. 8.4), blackjack (Fig. 8.5), and a special hammer (Fig. 8.6).

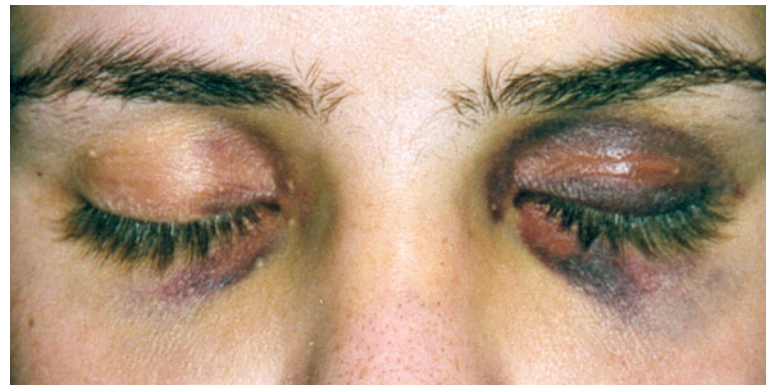

Fig. 8.1 Older bilateral periorbital hematomas due to blows from a fist

Table 8.1 Immediately fatal trauma (primary cause of death) and late trauma effects (secondary cause of death or trauma-related complications)

Primary causes of death	Secondary causes of death or trauma-related complications (examples)
Internal or external bleeding, depending in particular on preexisting cardiac disease and rate of bleeding at a blood loss from around 1.5 l (in adults)	Protracted hemorrhagic/hypovolemic shock (cold, moist, pale skin, thirst, nausea, possibly also disorientation, increased shock index: pulse/blood pressure)
Embolism, e.g., fat embolism in the case of soft tissue trauma, air embolism if large veins near the heart are opened, pulmonary thromboembolism	Pulmonary thromboembolism if delayed rather than acute in onset, e.g., after trauma-related immobility and deep vein thrombosis; posttraumatic fat embolism due to blood fat emulsification (one third to three quarters of all pulmonary capillaries shift, constant rise in pressure in the lesser blood circulation and increasing right ventricular load until the heart's ability to compensate is exceeded)

Table 8.1 (continued)

Primary causes of death	Secondary causes of death or trauma-related complications (examples)
(Partial) Destruction of vital organs (in particular the heart, lungs, brain, liver, spinal cord), in either isolation or combination following a traffic accident, a fall from a significant height, being struck or crushed by a railroad vehicle, or an explosion	Depending on posttrauma survival time: infections, ranging from secondary wound infections to systemic inflammatory response syndrome (SIRS), where two of the following five conditions are met: Neutrophil granulocytes >10 % Heart rate >90/min Respiratory rate >20/min Leukocytes >12,000/µl or <4,000/µl Body temperature >38 or <36 °C Sepsis or septicopyemia due to the spread of microorganisms (bacteria, fungi) and toxic circulatory failure in response to, e.g., bacterial endo- and exotoxins
Asphyxia, e.g., due to compression trauma to the neck or chest (e.g., Perthes pressure congestion in crush asphyxia)	Hypoxic organ damage, cerebral damage in particular, with subsequent trauma-related confinement to bed and complications such as thrombosis and thromboembolism and pneumonia
Trauma involving mechanical impairment to the function of internal organs (e.g., epi-/subdural hematoma, bilateral pneumothorax, traumatic cardiac tamponade)	Massively increased cerebral pressure with vasovagal cardiac arrest; respiratory insufficiency, reduced oxygen saturation, and hypoxic circulatory arrest; right ventricular decompensation
Burns, smoke inhalation	Burn disease involving the release of toxic substances from necrotic tissue; in the case of heat inhalation injury, involving heat-induced damage to the respiratory epithelium and secondary bacterial tracheitis, bronchitis, and purulent pneumonia
Sudden death due to vagal inhibition, controversial and only diagnosed once other causes of death have been excluded, e.g., bolus death, carotid sinus reflex death, stimulation of vagal fibers in trauma to the neck or solar plexus (blow to the abdomen)	
Anaphylactic/allergic shock: as an immune reaction to either immunogens (proteins, foreign serum, insect bites) or haptens (medications such as analgesics, antibiotics like penicillin, X-ray contrast medium) with specific or cross-reacting antibodies	Allergy-related deaths following exposure to relevant allergens, macroscopic edema (e.g., glottic edema) or skin blisters (e.g., Lyell syndrome), microscopic detection of increased mast cells, eosinophil granulocytes, serum antibodies
Cardiac arrhythmia, e.g., myocardial contusion, which can be assumed if other causes of death are excluded and local skin injuries, subcutaneous hemorrhage, and microhemorrhages in the myocardium are present	
Central paralysis, i.e., due to central regulatory mechanism failure following direct traumatic injury to the CNS or indirect hypoxic injury (asystole, neck compression, intoxication such as CO poisoning)	Primarily nonlethal trauma with sub- or epidural hematoma and/or intoxication, possibly protracted over hours or days, leading to increased intracranial pressure and compression of the brainstem in the foramen magnum

Table 8.2 Examples of accurate and inaccurate descriptions of injuries

Inaccurate description	Accurate description	Assessment
Hematomas to the forearm	Clearly demarcated bluish hematomas, 2–3 cm in diameter, to the mid-third of the flexor side of the forearm	Hematomas incurred as defense wounds
Hematomas to the skull	Unclearly demarcated hematoma, approximately 3 cm in diameter, in the body midline above the occipital frontal circumference	Injury from a blow if a fall down stairs is excluded
Bleeding skin wound to the chest	Longitudinal, smooth-edged wound measuring 2.5 cm without tissue bridging at the pointed ends of the wound and unilateral beveling on the lower wound edge, 3 cm below the left nipple	Stab/incised wound, probably from a knife with a double-edged blade; injury localization allows inferences to be made about the perpetrator's intentions
Gunshot wound to abdominal skin left and right	Gunshot wound with roundish-oval central defect, 0.8 cm in diameter, with wound edges that cannot be approximated and an abrasion ring in abdominal skin on the right side, 2 cm below the costal arch at the level of the midaxillary line; stellate gunshot wound, 1.5 cm in diameter, with wound edges that can be approximated and at the same level on the left side	Through-and-through gunshot wound to the upper abdomen, entry wound on the right side, exit wound on the right
Hematoma of the oral mucosa	Longitudinal hematoma, 1.3 cm in length, to the lower lip mucosa 2 cm to the right of the body midline aligned with a loosened canine	Tooth-mark hematoma, most likely an injury from a blow
Skin reddening over the knees and elbows	Flat, dry, reddish-brown skin crusts on the extensor side of the knee at the level of the lower margin of the kneecap and on the outer side of the elbows	Older skin abrasions at a location typical of falls

Table 8.3 Types of trauma

Trauma type	Subtype	Mechanism	Weapon, instrument, or object (examples)	Findings
Sharp force trauma	Incision	Incision with a sharp blade	Knife, shard, glass, razor, paper	Smooth wound edges
	Stab wound	Stabbing with a sharp blade	Knife, shard, scissors	Smooth wound edges, wound corners
Semi-sharp force trauma		Blow: combination of sharp and semi-sharp force trauma; biting	Axe, sword, teeth	Partially smooth, partially ragged wound edges; crushing or tearing of soft tissue, bone fractures also possible
		Sawing	Saw	Ragged/grooved skin injury, in part regularly spaced; typical notch formation in bone
Blunt force trauma	Non-patterned wounds	Blows, kicks, or pushes involving large surfaces of impact, falls onto flat surfaces	Hand, fist, (shod) foot	Hematomas, abrasions, lacerations, possibly also bone fractures
	Patterned wounds (Figs. 8.2, 8.3, 8.4, 8.5, and 8.6)	Blows with a sharp-edged object, kicks (shoe soles), falls onto prominent objects	Hammer, stone, stick, beam, door frame, table edge, shod foot	Hematomas, abrasions, lacerations showing recognizable edges or shapes of the surface of impact; possible bone fractures; typical parallel marks in the case of blows from a stick; shoe profile marks
Pointed-instrument injury		Gunshot, impalement	Firearm, bolt gun, beak, arrow (bow, crossbow, harpoon)	Roundish wound with a central defect; possibly also with edges that can be approximated, usually skin tears in a stellate configuration

Table 8.3 (continued)

Trauma type	Subtype	Mechanism	Weapon, instrument, or object (examples)	Findings
Thermal injury	Heat	Burns, scalding	Open flames, heated objects; hot liquids, gases, and steam; ambient temperature	Local: first- to third-degree burns, scalding Systemic: hyperthermia
	Cold	Heat loss	Low ambient temperature; rarely, cold objects	Local: frostbite Systemic: hypothermia
Electrical energy	Electricity, lightning	Electrical current flow, electric shock	Electrical conductor	Local skin reddening, electrical burns, coagulation, burns

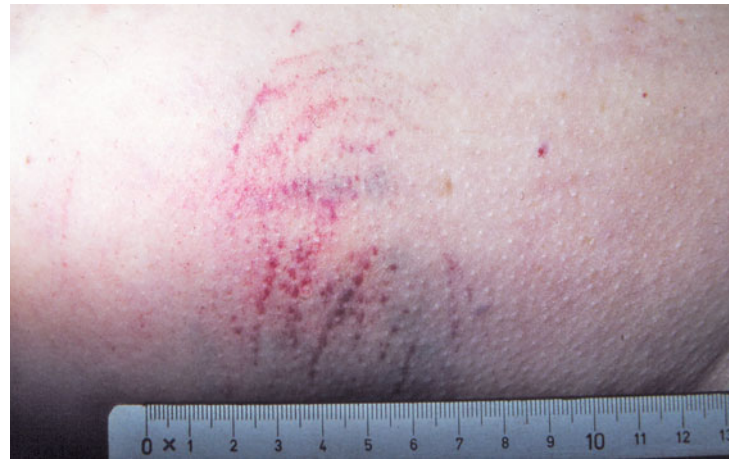

Fig. 8.2 Patterned wound caused by kick with signs of shoe sole

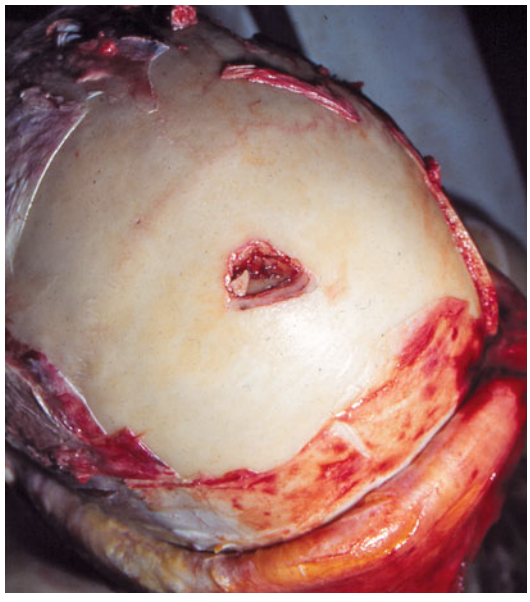

Fig. 8.3 Wound caused by a hammer

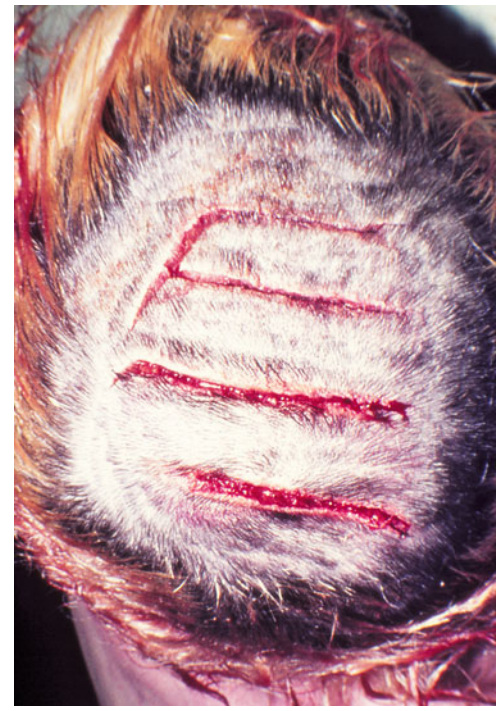

Fig. 8.4 Patterned wound caused by beating with a manhole cover

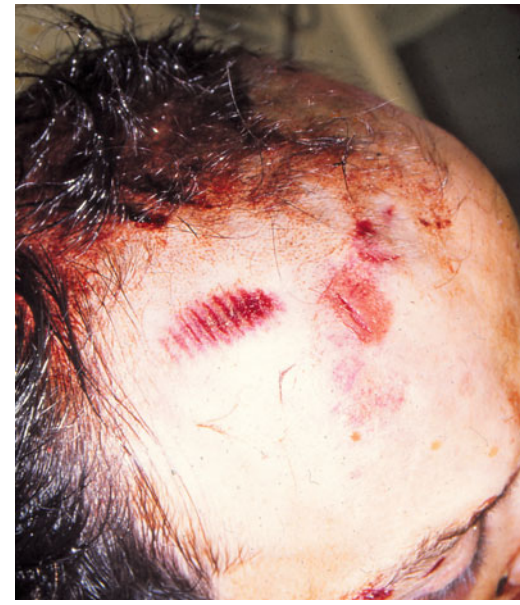

Fig. 8.5 Wound caused by blackjack localized above the hat brim line

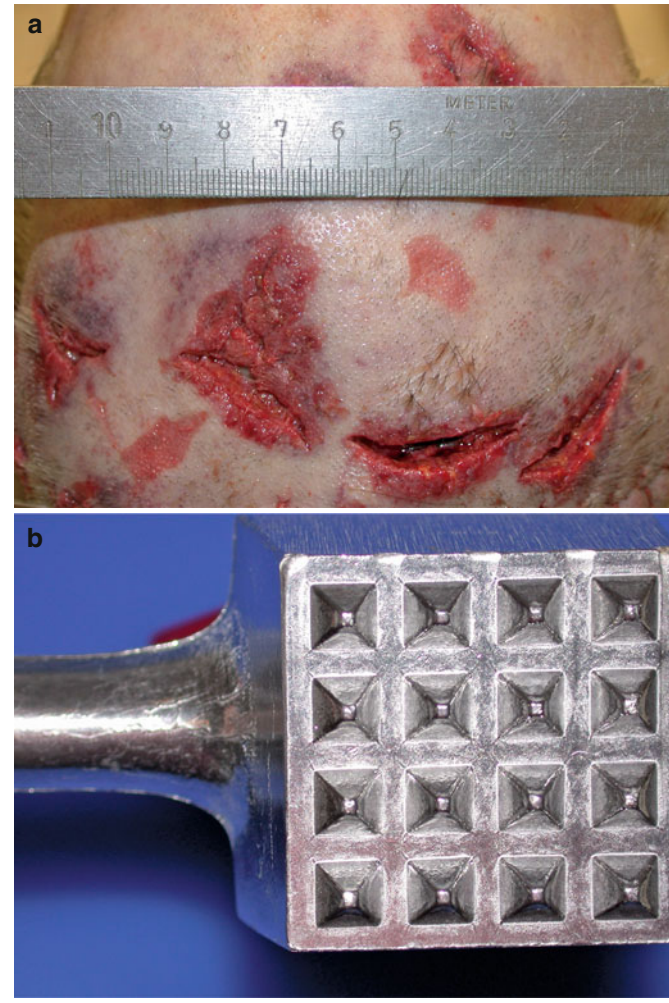

Fig. 8.6 (**a**) Patterned wound caused by (**b**) a special hammer

8.2 Types of Blunt Force Trauma

Case Study

A 57-year-old patient with a laceration to the forehead and multiple hematomas on the trunk and extremities was brought to the emergency department in the early hours of the morning. The patient vomited repeatedly and stated that he had no recollection of how he incurred the wounds. A blood alcohol level of 0.54‰ was measured. The person accompanying the patient claimed that the injured man had been assaulted by a man following an argument in a bar. The man was admitted as an inpatient due to suspected traumatic brain injury. A CT scan excluded intracranial hemorrhage. Parallel to this and with the patient's consent, the treating physicians informed the police. The accompanying person provided the police officers with the exact addresses of the victim and other witnesses.

A judicial hearing followed approximately 15 months later. The first-treating physician was called as an expert witness. He claimed to have no recollection of the case, since he had treated over 100 similar cases in the meantime. He was shown his handwritten findings: "Amnesia, nausea, vomiting, a laceration to the head, multiple hematomas." The physician stated that he was unable to provide more precise information for the purposes of the hearing. In particular, he was unable to recall the color of the hematomas. Furthermore, when questioned, he stated that no photodocumentation had been made of the injuries, nor was this his responsibility. He had treated the wounds and excluded life-threatening injury. In his opinion, he had taken all necessary steps for the patient's treatment.

The defendant had previously alleged that the 57-year-old had hit his head on a bar stool. Shortly after the incident, the named witnesses stated that they had not seen anything. The person who had taken the victim to the emergency department had meanwhile deceased. In his summation, the defense lawyer emphasized that all the indications pointed to a fall and that however many hematomas there may have been, all or at least most could have been incurred prior to the incident in question. The court accepted this line of argument and acquitted the defendant.

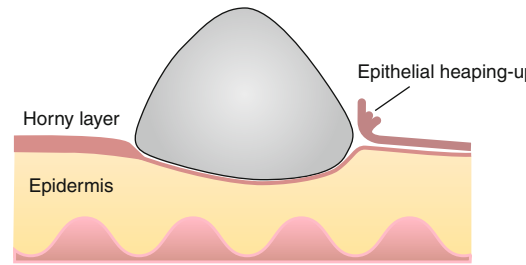

Fig. 8.7 Raised portion of the epidermis (skin tags) that can be folded back unilaterally following tangential blunt force to the skin ("heaping up" of the epithelium)

Blunt force trauma is characterized by the body colliding with an object or surface. This may involve an object being brought into contact with the body, as in a blow or a push, or the converse in the case of a fall, whereby the body moves towards the object. Traffic accidents often produce a combination of these two movements.

Depending on the impacting surface and the site affected on the body, an abrasion is produced, either as a patterned or as a non-patterned injury. Abrasions occur when blunt force impacts the skin at a tangential angle, causing portions of the epidermis to become raised ("heaping up" of the epithelium; Fig. 8.7).

For example, if the impact surface (25 × 25 cm) of a mallet strikes the mid portion of the skull, the borders of the resulting wound are determined by the anatomy of the skull, i.e., its convexity. The injury produced shows no characteristic pattern of the instrument and the injury is referred to as non-patterned. If the same area of the skull impacts a flat surface such as a wooden floor in a

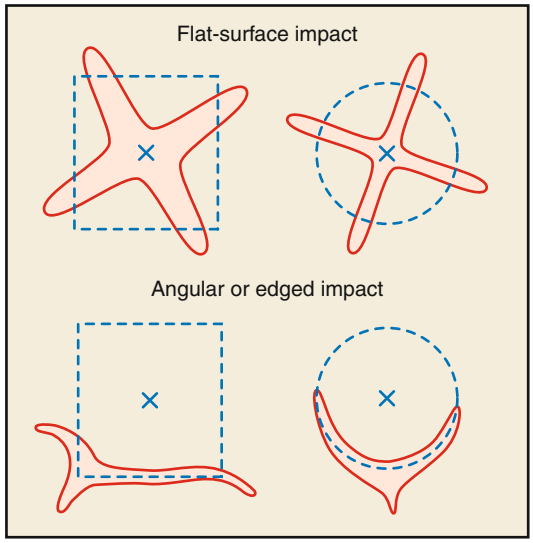

Fig. 8.8 Wound morphology depending on whether the flat (**a**, **b**) or angular (**c**, **d**) aspect of the impacting object striking the skin

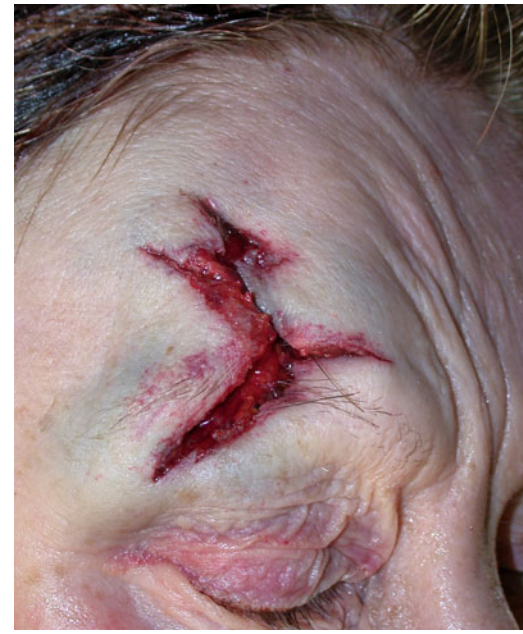

Fig. 8.9 A laceration from an angular impacting object

fall, a non-patterned injury that is indistinguishable from the injury mentioned above may be produced. This second blunt force injury would also be classified as a non-patterned injury. In contrast, if the mallet mentioned above was used to strike the middle of the back, the injury borders would be determined by the edges of the impact surface. Ideally, the entire impact surface would be reflected in the skin injury on the back, thus producing a patterned injury. If the edge of a mallet's impact surface rather than the center strikes a skull, the wound will show borders at least on one side and can be classified as a patterned injury. Thus, wound morphology depends in particular on whether the flat or angular aspect of the impacting object strikes the skin (Figs. 8.8 and 8.9).

In addition to the impacting object, injury sequelae from blunt force trauma depend on intensity, direction of movement (force vector), and postexposure interval. If the skin is broken, the tissue bridges at the base of the wound and at the wound corners represent an important diagnostic criterion (Fig. 8.10).

The angular aspect of an object striking the skull often does so at an oblique angle, producing a relatively smooth-edged wound that at first glance lacks tissue bridging. This type of wound, however, generally shows beveling in the direction of the impacting force, extending from the surface to deeper within the wound, and shows undermining on the contralateral side. Tissue bridging can be seen in areas of undermining. Typical blunt force injuries are given in Table 8.4.

The surface(s) or edge(s) of the impacting object may be visible in all skin injuries and some bony injuries (patterned injuries), enabling inferences to be made about the instrument or weapon used. A typical example of patterned blunt force trauma is "double" hematomas. These are produced, for example, by blows from a stick, where blood is forced to both long sides of the site of skin impact and subcutaneous capillaries rupture, forming anemic impact marks (Fig. 8.11). Double hematomas of this kind are also referred to as "parallel contusions" (Fig. 8.12).

If strongly convex areas of the body's surface are struck, parallel contusions tend to meet at their ends in an archlike pattern, producing a generally elongated outline. Baseball

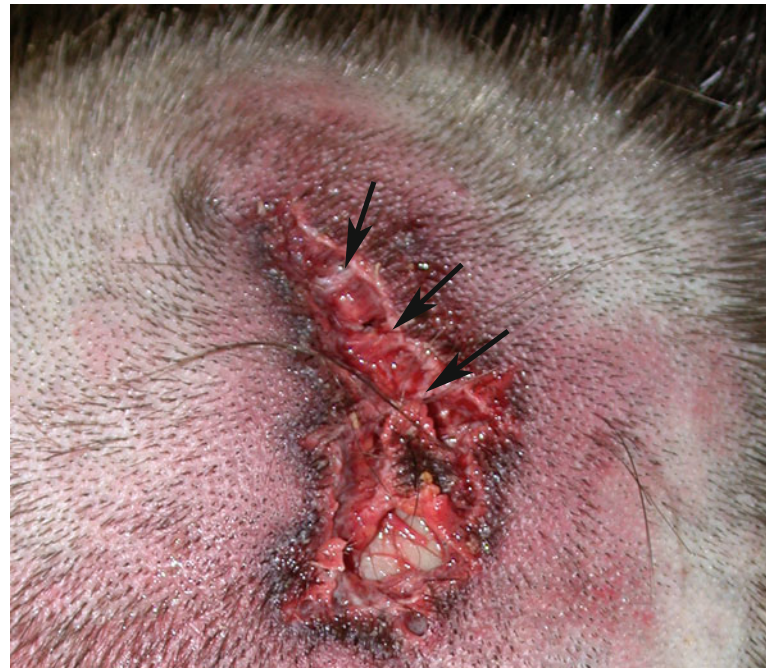

Fig. 8.10 A laceration with tissue bridging (*arrows*) at the base and corners of the wound following blunt force trauma

Table 8.4 Typical blunt force injuries

Injury sequelae from blunt force trauma	Features/conditions
Signs of anemia or hypemia	No dermal destruction; earliest phase; massive blunt force trauma and flexible impact surface, e.g., water as impact surface, can also be of longer standing
Erythema (hyperemia)	Second phase following blunt force trauma; always temporary, possibly visible at margins if skin is damaged
Intracutaneous hematomas	Stretching and tearing of blood vessels
Subcutaneous hematomas	Moderate blunt force often in an orthogonal direction to the skin; stretching and tearing of blood vessels; depends on tissue fragility, e.g., produced more easily in women and children
Hematoma in deeper tissue	Stronger blunt force, usually in an orthogonal direction to the skin; stretching and tearing of blood vessels; depends on tissue fragility; may expand subcutaneously
Abrasions	Blunt force must have tangential component; primarily rough surface and direct skin impact, at most thin clothing; often as concomitant injury at wound edges if skin continuity is broken
Stretch lacerations	Stretching or tearing of the skin; primarily over bony prominences, possibly at some distance from the site of trauma; affects the epidermis, extending to the dermis
Lacerations	Blunt trauma of significant intensity, primarily to the skull and bony prominences of the face or extremities (where bones serve as a point of abutment); simultaneous crushing and displacement of skin to the point of tearing; wound shape and configuration determined by the impact surface and direction of trauma
Avulsion	Blunt force with a significant tangential component; displacement of intact skin against the subcutaneous fatty tissue accompanied by tearing and cavity formation, secondary massive hemorrhage (possibly large hemorrhage cavities)
Organ tearing or rupture	Massive blunt force trauma, e.g., a fall from a great height or a traffic accident, or local blunt force trauma from kicks
Bone fractures	Massive blunt force trauma; bending and burst fractures
Traumatic brain injury	Very particular fracture pattern in the neurocranium due to its egg-like structure; problems in intracranial pressure increase in the case of posttraumatic hemorrhage or cerebral edema

bats have been known to produce oval or even round outlines. On the other hand, a whip or belt striking the same site will produce two parallel lines, since the material adapts to the skin surface.

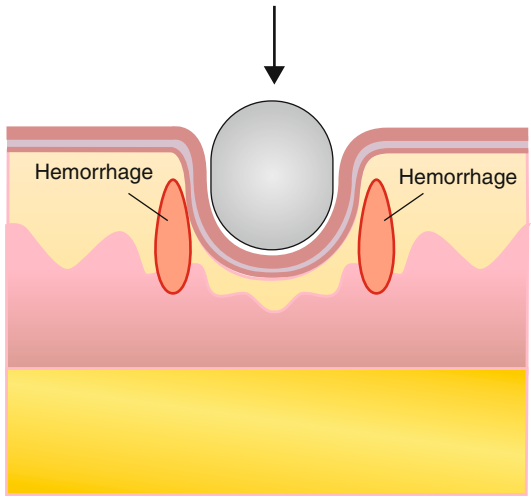

Fig. 8.11 Mechanism of injury for "parallel contusions"

Expert assessments often need to distinguish between injuries from blows and injuries from falls, as well as the sequence in which the injuries occurred. To this end, injury patterns need to be considered as a whole:

- Number of injuries
- Localization(s)
- Shape
- Injury age

Conflicting statements need to be checked for plausibility. The victim usually alleges to have been struck, while the suspect describes a fall (see Case Study). Falls tend to produce unilateral injury patterns. Therefore, it should be possible to reconstruct the distribution and number of injuries with a one-sided sequence of events. If injuries corresponding to patterned blunt force trauma are seen, a fall would have needed to involve a prominent object such as a table edge, a projection from a wall, or a stone lying on the ground.

The hat brim line rule is applied in the case of head injuries: typically, injuries incurred from a fall are located below an imaginary line

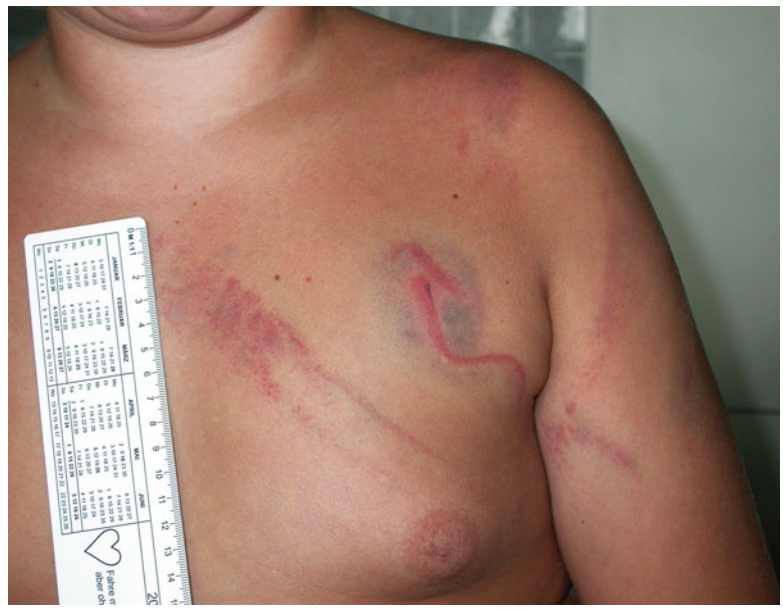

Fig. 8.12 "Parallel contusions." Double linear striated hematomas (*left*) from a blow with a belt and belt-buckle imprint (*right*); additional hematomas on the shoulder and upper arm

marked at the place where the brim of a hat would sit. Injuries incurred from blows, however, tend to be localized above the hat brim line (Fig. 8.13).

However, the hat brim line rule is only meant as a guide; it does not apply in the case of falls down stairs.

Fist blows generally strike below the hat brim line. In addition to a monocle hematoma resulting from a fist blow (also involving blow-out fractures in the case of boxing punches), teeth may act as an abutment to a blow from a fist in the mouth area, producing injuries to the oral mucosa in the shape of tooth marks (Figs. 8.14 and 8.15).

Falls headfirst also tend to represent exceptions to the hat brim line rule. Therefore, the circumstances and alleged facts of a case need to be considered when applying the rule.

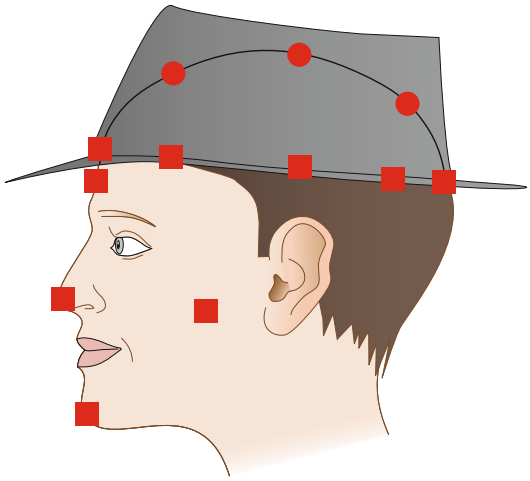

Fig. 8.13 The hat brim line rule, used to differentiate between injuries from falls and injuries from blows

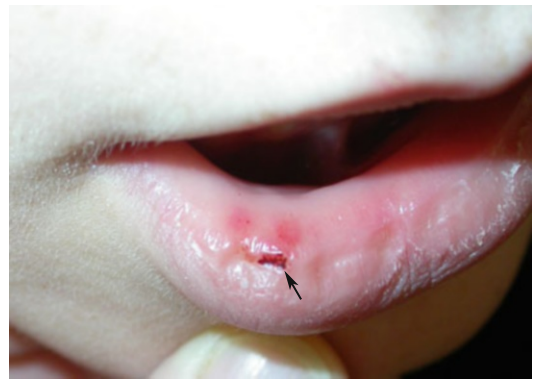

Fig. 8.15 Small tooth mark (*arrow*) from a blow to the lip; central pallor, parallel hemorrhage on the margin, and a clearly defined skin laceration corresponding to the tip of a tooth are visible

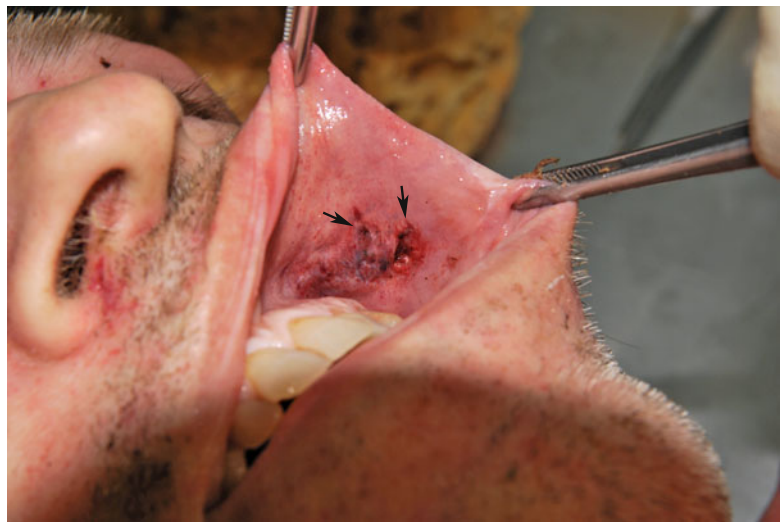

Fig. 8.14 Two closely spaced tooth marks (*arrows*) in the oral mucosa produced by a fist blow to the mouth

Case Study

Following a domestic fight between a 53-year-old woman and her partner, the woman called the police, who issued a restraining order against the man. On their arrival, the police had observed that the woman had a heavily bleeding head wound; she alleged that her partner had struck her with a chair. The man, on the other hand, reported that the woman had fallen against a door frame while inebriated. After putting up significant resistance to the restraining order, the man, on whom the police detected a strong smell of alcohol, was taken to the police station for a blood test. Forensic blood alcohol testing yielded a value of 1.98‰.

The police carried out a breath alcohol pretest on the woman, obtaining a result of 0.00‰. No order to collect evidence was given. The woman was taken by ambulance to the nearest hospital for her injury to be treated. Once the wound had been cleaned, the treating physician documented a straight, 4-cm longitudinal wound in the body midline to the highest point of the skull involving tissue bridging at the wound base; the injury was treated using four interrupted sutures.

At the judicial hearing, the man stood by his claim that the woman had fallen against a door frame. The forensic medical expert had only the medical report and witness statement on which to base his assessment, in which he maintained that the injury resulted from blunt force trauma involving impact from an angular object. In view of the localization of the injury, a fall was unlikely, although a fall that involved striking the edge of a door frame could not be conclusively ruled out. Taking the overall picture into consideration, the court found the man guilty and charged him with dangerous bodily harm.

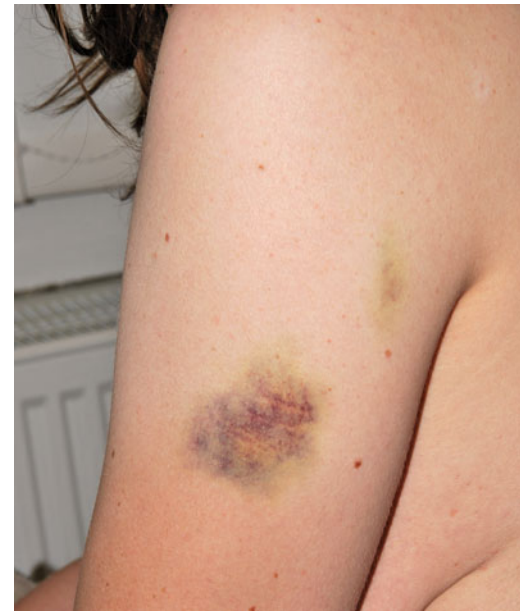

Fig. 8.16 A somewhat older hand mark to the upper arm caused when the perpetrator grabbed the victim vigorously in the course of a sexual crime

In addition to blow- and fall-related injuries resulting from physical conflict, attention should be paid to other findings that may provide insight into an incident. These include in particular hand marks on the upper arms (Fig. 8.16), defense wounds (parrying injuries) to the extensor side of the lower arms, and hematomas on the inner side of the thighs, the latter primarily in sexual offenses.

8.3 Injuries to Internal Organs

A distinction needs to be made between injuries to internal organs within the chest and abdomen and injuries due to craniocerebral trauma.

Lung Injuries. Lung contusions are usually the result of chest compression, generally occurring in combination with serial rib fractures (SRF). Rib fractures can in turn lead to skewering injuries to the lung. Deceleration trauma is a further mechanism of lung injury, causing hemorrhagic infiltration into tears in the pulmonary hila. Lung

contusion injury always requires differentiation from foci of blood aspiration; often, this can only be achieved with a good level of certainty once the extent of tissue injury has been established histologically.

Cardiac Wall or Aortic Arch Rupture. Injury of this kind can occur as a direct result of compression between the sternum and thoracic spine or due to deceleration. Aortic rupture may initially be incomplete and thus remain undetected.

Injury to Abdominal Organs. The liver and spleen are the most important organs in terms of abdominal organ injury. The rupture of either organ can follow a two-stage course, i.e., a tear in the parenchyma can occur in the presence of an intact capsule. A symptom-free interval lasting until the capsule ruptures due to hemorrhage into the organ is followed by massive blood loss.

Two-stage splenic rupture is often seen in children following a fall over bicycle handlebars, and failure to consider a two-phase course may lead to medical malpractice claims. Intestinal rupture due to blunt force trauma is seldom seen, occurring rarely after a fall from a great height or in the case of preexisting disease such as chronic inflammatory bowel diseases. Hemorrhage into or even rupture of the greater omentum or the mesentery is seen following kicks to the abdomen, particularly if the victim is already lying on the ground.

Fractures. Various types of fracture are seen depending on the intensity (impulse = mass × acceleration) of force and its localization (Fig. 8.17).

The fracture patterns found on a body can provide insight into the direction and intensity of blunt force trauma. The best known example of this is the butterfly fracture, which occurs when significant blunt force is exerted by a circumscribed surface area to a load-bearing long bone. This constellation is seen, for example, when a pedestrian is struck by a car bumper. The bone bends and ultimately cracks as a bone wedge is formed. The base of the wedge points in the direction of the impacting force and is located either at car-bumper level or somewhat lower in the case of a prior braking maneuver (see Chap. 21).

Secondary complications of bone fractures can include:

- Hemorrhage from the blood vessels of the bone
- Fat and bone marrow embolism after opening of the medullary cavity and transport of fat droplets and blood-forming bone marrow through open veins
- Organ penetration causing organ damage and hemorrhage
- Skin puncture (open fracture): Infections
- Lack of stability, e.g., in the chest following SRF: Impaired respiration
- Traumatic brain injury (TBI)

Traumatic brain injury is caused by blunt force trauma to the head, possibly in combination with sharp or semi-sharp force trauma.

Important: Injuries may develop outside the cranial cavity, on the cranial bone, and within the cranial cavity.

In the case of mild blunt force to the head, hemorrhage beneath the epicranium or lacerations are restricted to the site of impact. Depending on the localization and direction of force, the skull or brain is affected only once stronger force is exerted. Thus, it should be borne in mind that skull fractures without injury to the brain are seen as frequently as injury to the brain without fractures.

Cases of victims remaining conscious despite extensive skull fractures are not uncommon. Indeed, the patients are able to stand up and walk around. One proposed explanation for this phenomenon is that the impacting force is absorbed by the cranial bone, thus preventing its propagation to the brain.

Brain injuries that lead to loss of consciousness and death may also occur in the absence of skull fractures. Cerebral edema, intracranial bleeding, or diffuse axonal injury (DAI) can play an important role here, as in "shaken baby" syndrome (see Chap. 18). A fracture to the neurocranium in the setting of an intracranial pressure increase may serve as a "natural" pressure release mechanism.

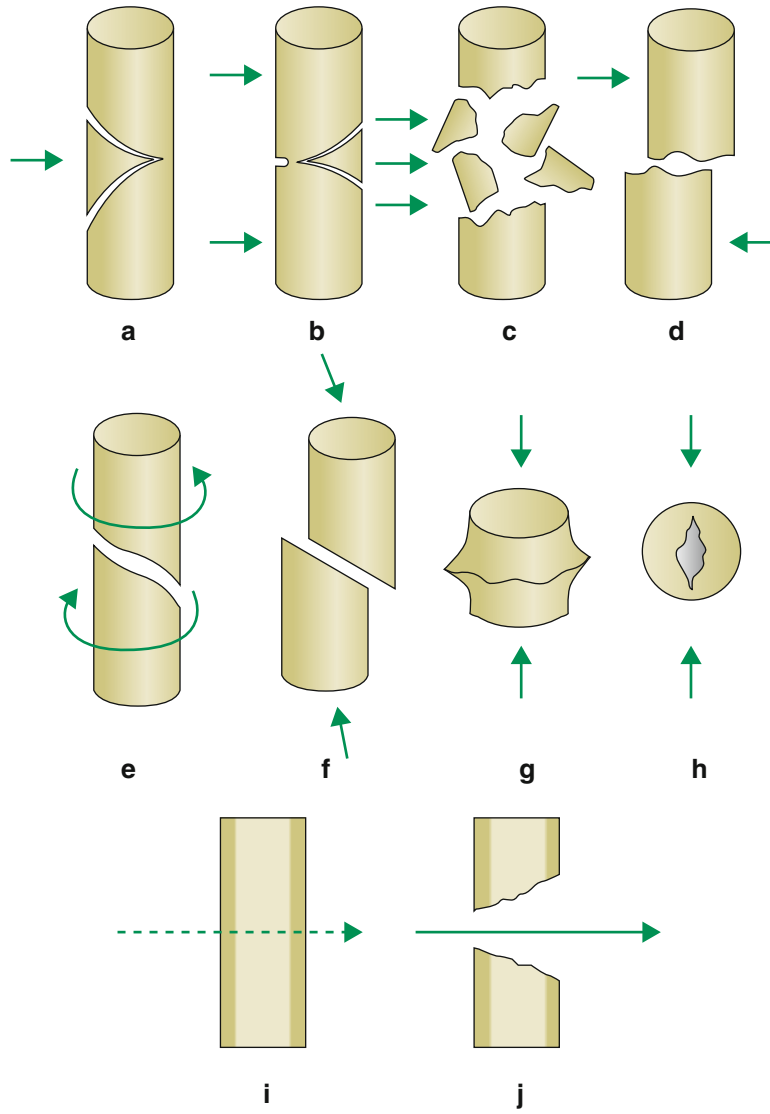

Fig. 8.17 Basic fracture types. *Arrows* indicate the direction of force. (**a**) Butterfly fracture. (**b**) Flexion wedge fracture. (**c**) Comminuted fracture. (**d**) Transverse fracture. (**e**) Spiral fracture. (**f**) Oblique fracture. (**g**) Compression fracture. (**h**) Rupture of the internal structures of long bone shafts or the skull.(**i**, **j**) Outward beveling to the skull in the direction of fire (Herrmann et al. 1990)

Whether TBI is open or closed is a further criterion by which to gauge injury severity. In the case of open TBI, the brain is exposed, i.e., there is a continuous local defect in the epicranium, neurocranium, and dura. Depending on injury severity, brain substance defects or even loss may have occurred. Moreover, open TBI always carries the risk of life-threatening infection.

Extremely severe injury causing disintegration of the neurocranium and destruction of the brain, as seen in cases involving victims struck or driven over by rail vehicles, cause immediate death.

Degree of TBI Severity. The degree of TBI severity is clinically assessed according to the Glasgow Coma Scale (Table 8.5). A maximum of four, five, or six points can be achieved for the three criteria "eye opening," "verbal response," and "motor response," respectively. These points are then added together, yielding a potential maximum score of 15 points (fully conscious). The minimum score of 3 is associated with deep coma or death.

Injury to the Epicranial Aponeurosis. Subcutaneous hemorrhage, hemorrhage in the epicranial aponeurosis or temporalis muscles, as well as lacerations to the epicranium tend to occur directly at the site of the impacting force. Isolated injuries to the well-vascularized scalp can cause fatal exsanguination. Subaponeurotic

hemorrhage, on the other hand, builds up beneath the epicranial aponeurosis and is almost always seen in a skull-fracture setting, without necessarily being associated with the site of direct blunt force impact (Fig. 8.18).

Skull Fractures. The skull encloses the cranial cavity and is comparable to a double-walled eggshell in an intact egg in terms of function. The cranial vault is composed of the typical structures comprising the diploe sandwiched between the outer and inner tables. Due to this particular anatomical structure, special fracture types unseen elsewhere on the skeleton are encountered on the neurocranium:

- Bending fractures
- Burst fractures

Table 8.5 Glasgow Coma Scale (GCS)

Points	Eye opening	Verbal response	Motor response
6	–	–	Obeys commands
5	–	Able to converse, oriented	Localizes pain stimuli
4	Spontaneous	Able to converse, disoriented	Withdraws from pain stimuli
3	In response to speech	Inappropriate words	Flexion to pain stimuli
2	In response to pain	Incomprehensible sounds	Extension to pain stimuli
1	No response	No response	No response

Severity: 14–15 points, mild; 9–13 points, moderate; 3–8 points, severe

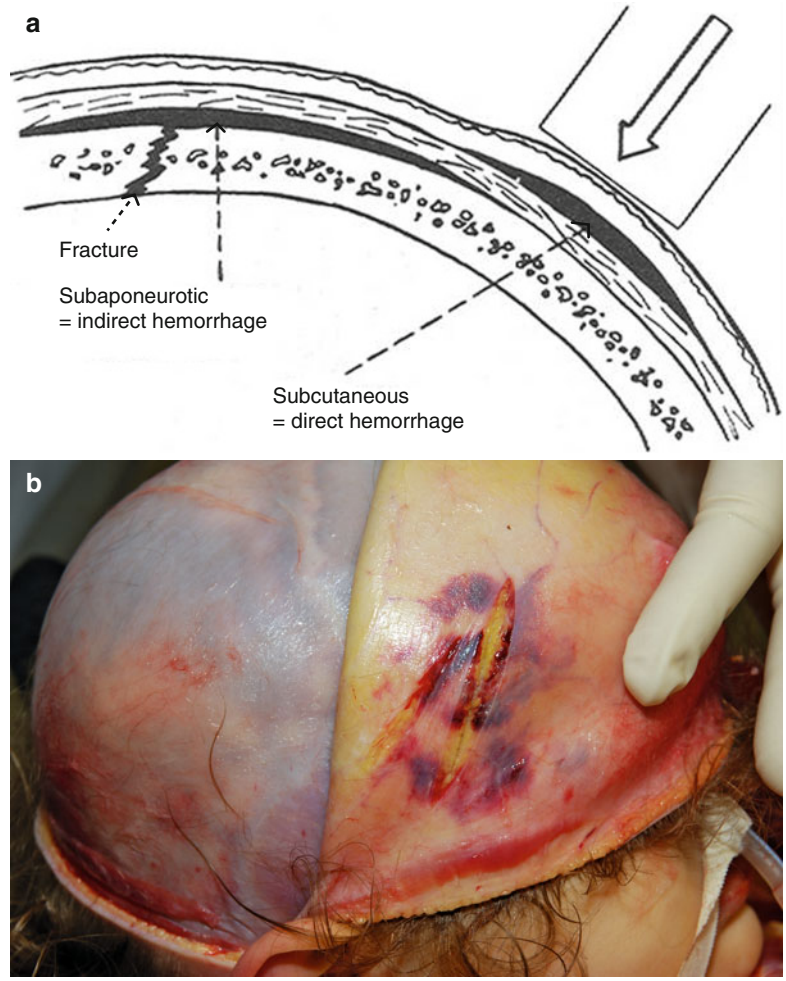

Fig. 8.18 (**a**) Subcutaneous hemorrhage due to direct external trauma to the skull as well as subaponeurotic hemorrhage due to a skull fracture. (**b**) Subcutaneous hemorrhage due to direct external trauma to the skull

- Depressed fractures
- Buttonhole fractures
- Terrace fractures
- Basilar skull ring fractures
- Basilar skull hinge fractures
- Radial fractures
- Cranial suture separation

Firstly, a basic distinction is made between bending and bursting fractures.

Bending Fractures. This type of fracture is caused by local deformation to the cranial vault directly at the site of blunt force impact, thereby producing greater tensile strain on the inner table than the outer table. Thus, the former fractures first, followed by the latter. The radial fracture is the typical bending fracture seen on the cranial vault. It is characterized by a combination of fracture lines extending from the center of trauma impact (meridional) and fracture lines following a circular peripheral course (equatorial), producing what is known as a "spider's web" fracture pattern (Fig. 8.19).

Basilar Skull Ring Fractures. Ring fractures to the base of the skull around the major foramen are considered a special form of bending fracture. Ring fractures are caused by either compression or traction to the base of the skull against the spinal column (Fig. 8.20).

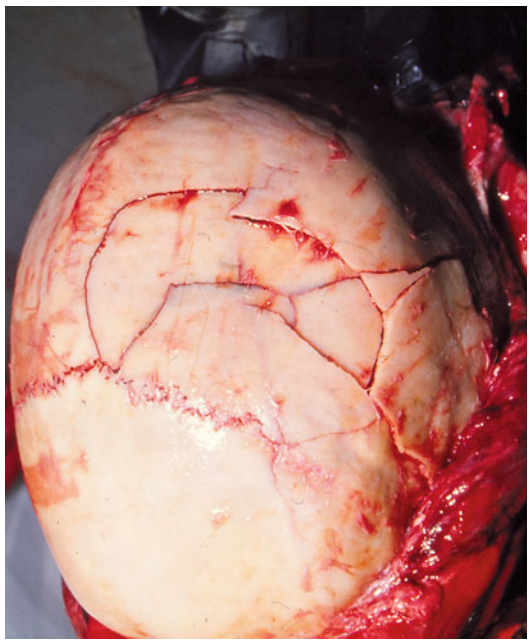

Fig. 8.19 Radial fracture following blunt force trauma to the skull

Buttonhole and Terrace Fractures. Patterned blunt force trauma to the cranial vault can also cause bending fractures. If force delivered in an orthogonal direction makes an imprint on the outer and inner tables, a buttonhole (or perforating) fracture may be produced. Patterned blunt force trauma impacting the skull at an angle produces a terrace fracture (Fig. 8.21). In the case of both fracture types, the fracture pattern on the outer table may correspond to the size and shape of the impacting object, permitting conclusions to be drawn about the weapon or instrument used (Fig. 8.22).

Burst Fractures. This type of fracture is caused by general deformation of the skull and occurs not only at the site of direct blunt force trauma. Bilateral pressure to the neurocranium (transverse pressure) reduces its transverse diameter. As the longitudinal diameter increases, the greatest tensile strain can be measured in the region of the transverse diameter. Thus, an indirect fracture forms along the transverse diameter. Frontal and occipital pressure to the skull would produce a longitudinal fracture by the same mechanism.

Important. The following applies to cranial burst fractures: transverse pressure produces transverse fractures and longitudinal pressure produces longitudinal fractures (Fig. 8.23)**.**

In addition, massive frontal or occipital blunt force trauma can result in longitudinal skull fractures, while massive lateral trauma can result in transverse fractures. This type of fracture is seen following falls from a great height as well as in traffic accident victims.

Indirect fractures can occur on the cranial vault as well as at the base of the skull. Typical basilar skull fractures are shown in Table 8.6.

Falls primarily on the back of the head can lead to so-called contrecoup orbital wall injury, particularly to the orbital roof (so-called orbital sign). Victims may develop unilateral (monocle) or bilateral (raccoon eyes) periorbital hematomas even in the absence of direct blunt force trauma to the orbital cavity. Thus, the orbital sign can be of diagnostic relevance when differentiating between blows and falls, particularly in the presence of a fall-related injury to the back of the head accompanied by a typically fine fracture to

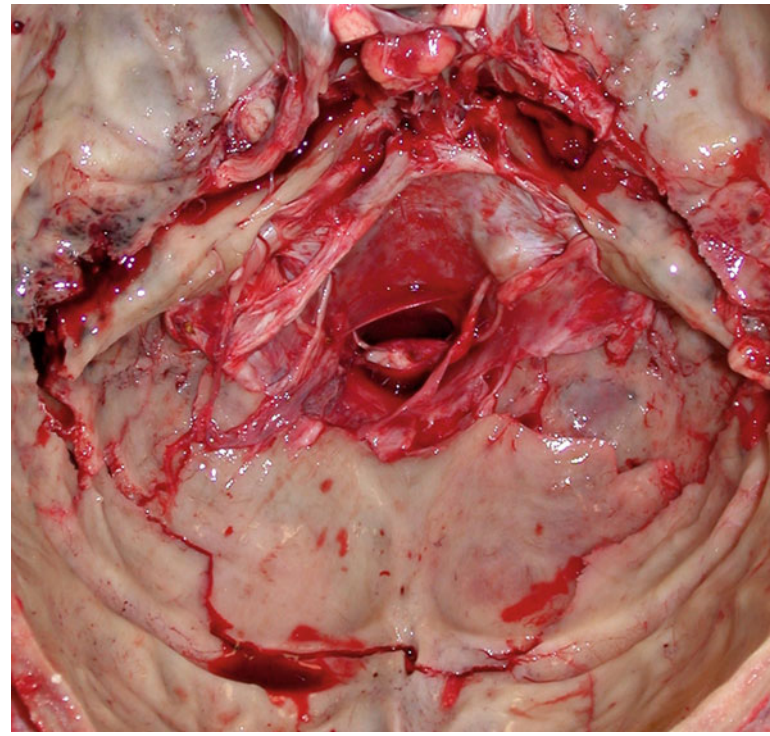

Fig. 8.20 Extensive basilar skull ring fracture following a fall from a significant height

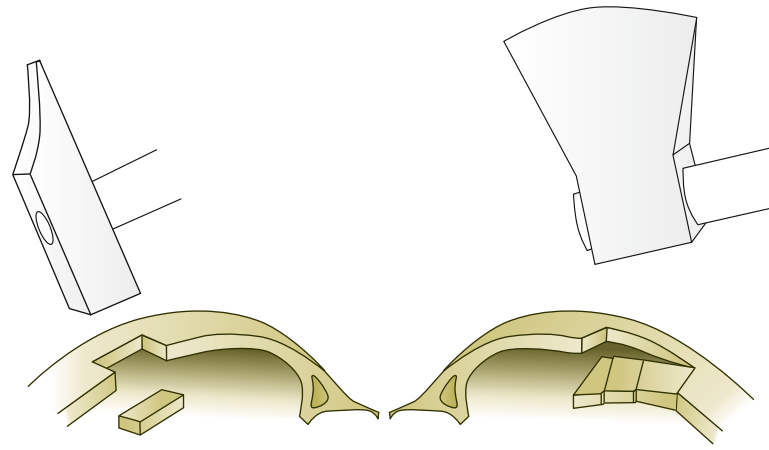

Fig. 8.21 Mechanisms of injury for buttonhole and terrace fractures

the orbital cavity without any indication of trauma or a blow to the eye region from the anterior direction.

In the case of multiple skull fractures, Puppe's rule can be helpful in identifying the sequence in which fractures occurred.

Puppe's Rule: Fracture lines extending out from a fracture zone always end at the point where they intersect with preexisting fracture lines.

Exceptions to Puppe's rule are seen in the case of two high-speed blunt force injuries in rapid succession or virtually simultaneously, e.g., a through-and-through gunshot wound to the head: as the fracture lines originating from the gunshot entry wound form, fractures caused by the gunshot exit wound are already developing. Therefore, fracture lines originating from the gunshot entry wound may end when they intersect fracture lines caused by the gunshot exit

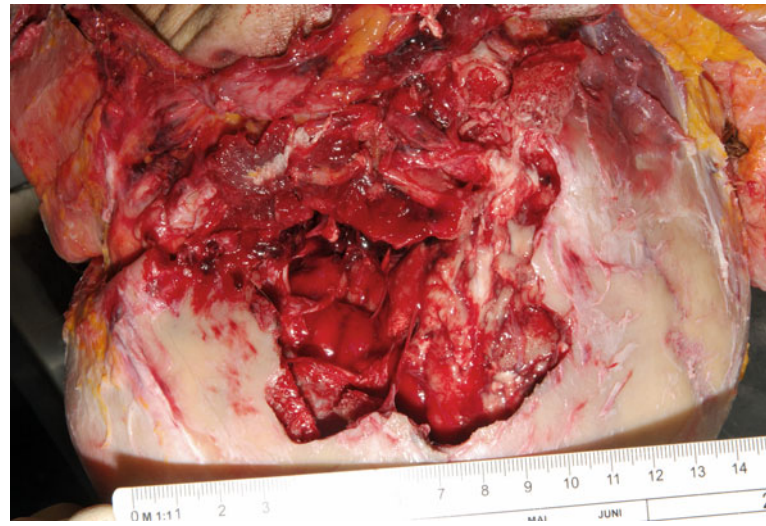

Fig. 8.22 Buttonhole fractures following blows from a hammer. The angular shape of the fractures matches the shape of the impacting surface of the instrument used

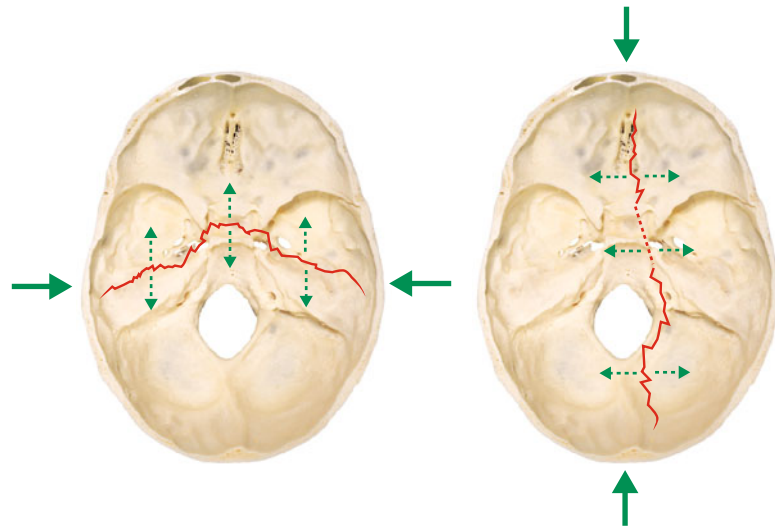

Fig. 8.23 Transverse pressure produces transverse fractures and longitudinal pressure produces longitudinal fractures

Table 8.6 Typical basilar skull fractures

Basilar skull fracture	Mechanism	Example
Hinge (transverse) fracture to the base of the skull (Fig. 8.24)	Massive side-to-side compression	The victim's head, lying in a lateral position on the road, is driven over by a vehicle
Longitudinal fracture to the base of the skull	Massive frontal and occipital blunt force trauma	Impact to the forehead or back of the head due to a fall
Ring fracture to the base of the skull	Compression of the skull base against the cervical spine	Falls involving landing on the feet or parietal region
	Traction of the skull base away from the cervical spine	Traffic accident involving a body being pulled towards the feet while the head is held in a fixed position

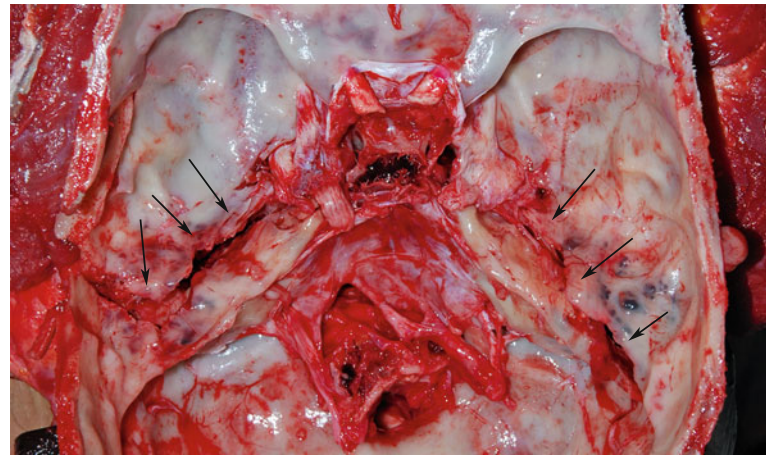

Fig. 8.24 Basilar skull hinge (transverse) fracture

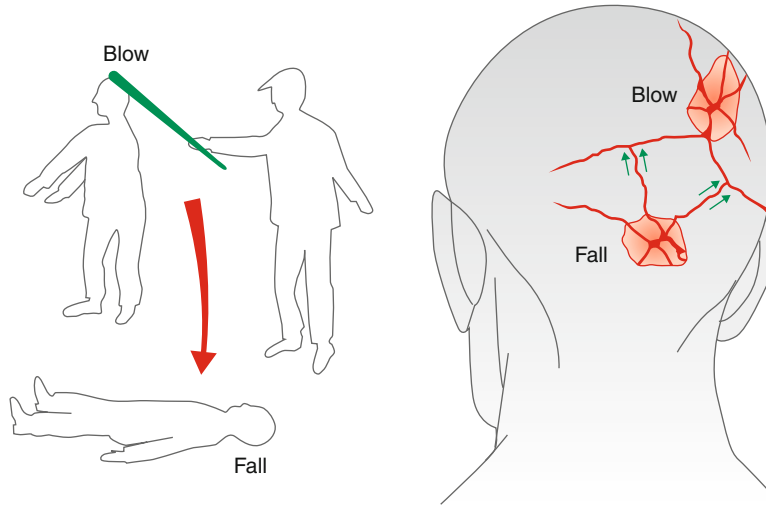

Fig. 8.25 Sequence of injuries from blows and falls according to Puppe's rule

wound. However, the sequence of injuries can be determined according to Puppe's rule in the case of a double fracture system due to injuries from blows or falls (Fig. 8.25).

Facial Bone Injury. Blunt force trauma to the face can cause isolated fractures to the nasal bone, zygomatic arch, or lower jaw. Blunt trauma to the eye, particularly in the form of a blow from a fist or impact from a tennis ball, can cause excessive orbital pressure. In addition to injury to the eyeball, this may cause the orbital wall to burst (blow-out fracture). Massive blunt force trauma to facial bones often produces typical fracture patterns that can be classified according to Le Fort (Table 8.7).

Table 8.7 Classification of midface fractures according to Le Fort

Le Fort type	Finding
Le Fort I	Separation of the hard palate from the maxilla extending through the piriform aperture, canine fossa, maxillary sinus, and greater wings of the sphenoid processes
Le Fort II	Pyramid-shaped separation of the midface involving oblique fractures through the bridge of the nose and extending diagonally through the orbital cavity and zygomatic process of the maxilla
Le Fort III	Separation of the midfacial skeleton from the craniofacial skeleton

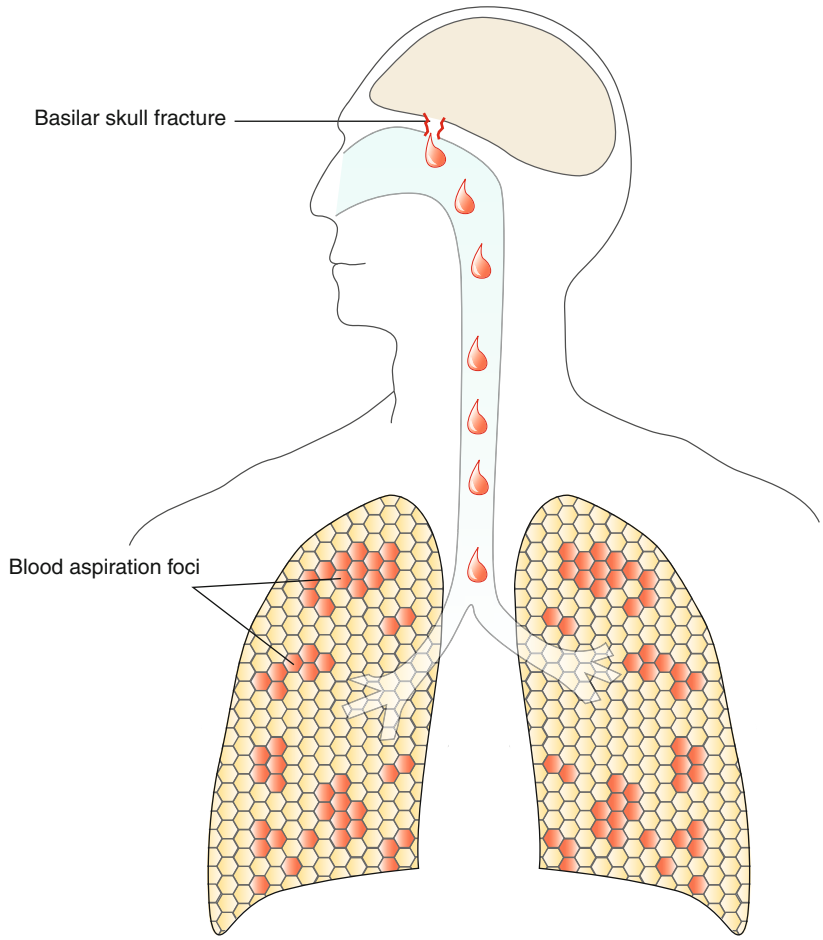

Fig. 8.26 Basilar skull fracture with fatal blood aspiration

Alongside damage to the central nervous system, the indirect effects of blunt force trauma can also be life-threatening, as in the case of basilar skull fractures accompanied by hemorrhage into the oral cavity and subsequent blood aspiration in an unconscious victim following traumatic brain injury (subpleural hemorrhage!) (Fig. 8.26), or fractures accompanied by significant blood loss, causing death due to exsanguination (Fig. 8.27).

8.4 Forensic Neurotraumatology: Brain Injury

The brain is relatively mobile within the cranial cavity. Depending on the type of blunt force trauma, the brain is subjected to either an axial (i.e., propagated in a straight line) or a rotational force. The brain is propelled in one direction and then slowed down; when combined, these effects can cause the various layers of the brain to move against one another.

Coup–Contrecoup Injury: Due to mass inertia, brain tissue is forced against a point of impact (coup), and then rebounds to strike the contralateral side of the dura (contrecoup), thereby creating negative pressure (tissue-damaging suction). The brain follows the motion of the head, coming to rest only after the latter has stopped moving. This causes secondary tissue compression at the point of contrecoup and possibly also, although to a lesser extent, negative pressure at the coup site.

Rotational Injury: The brain lags behind the rotational movement of the cranium, producing shearing forces that affect the brain, meninges,

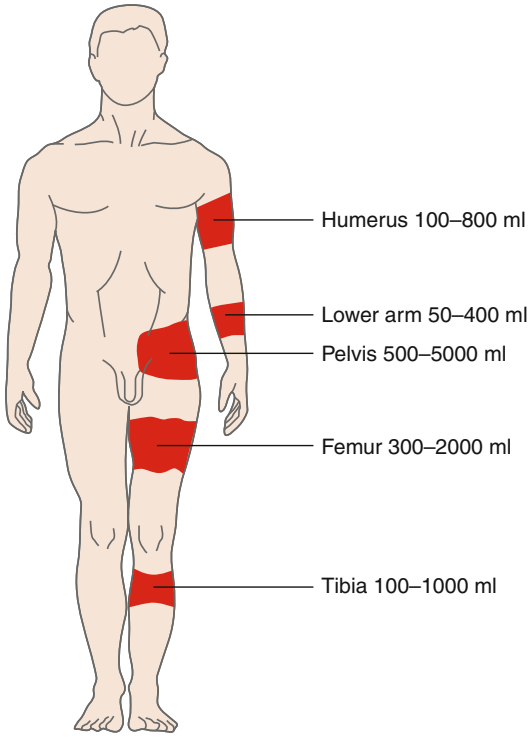

Fig. 8.27 Fractures accompanied by subsequent blood loss leading to death by exsanguination

and vessels. This delayed braking of the brain may produce secondary shearing forces in the opposite direction.

Brain injury is classified into three grades according to severity:

Grade 1 = commotio cerebri (concussion): There is no morphological correlation of this diagnosis, which is made purely on the basis of clinical information. Symptoms may include transient loss of consciousness, antero- and retrograde amnesia, nausea, and vomiting. Although rare, a posttraumatic semiconscious state is sometimes observed.

Grade 2 = contusio cerebri (cerebral contusion): Contusions of the cerebral cortex represent an important morphological criterion. These occur at the site of impact (coup), as well as at the contralateral site (contrecoup), where they are often more intense. Morphologically, cortical contusions are punctate or spotted areas of hemorrhage, with coup lesions occurring mainly on the cortical crests, contrecoup lesions predominantly in depressed areas.

Grade 3 = compressio cerebri (cerebral compression): Intracranial hemorrhage or cerebral edema causes increased intracranial pressure, leading to secondary cerebral damage. Brainstem compression at the tentorial gap and medulla oblongata in the foramen magnum can cause death due to impaired central regulation.

Diffuse Axonal Injury (DAI). DAI may be detected microscopically even in the absence of skull fractures or other macromorphologically visible lesions. Thus, for example, a focal axonal accumulation of the β-amyloid precursor protein can be seen below the cortex, either beneath the point of contusion or in the corpus callosum, approximately 3–4 h posttrauma.

Intracranial Hemorrhage. An overview of the types of intracranial hemorrhage, their causes, and the mechanism by which they form is given in Table 8.8.

An interval free from neurological symptoms between the time of injury and the onset of intracranial pressure symptoms is often observed in the case of epidural hematomas, less frequently with subdural hematomas.

In practice, it is important to differentiate early signs of traumatic injury from the symptoms of alcohol intoxication. Problems potentially arise if neurological deficits or symptoms (e.g., somnolence or impaired articulation) in a heavily intoxicated patient whose case history points to traumatic brain injury or who shows relevant signs of external injury are attributed solely to alcohol consumption. Failure to carry out close clinical observation or perform imaging studies for diagnostic purposes can lead to charges of negligent homicide in cases where unsupervised patient's die as a result of their injuries.

Acute Subdural Hematoma. The crescentic shape of acute subdural hematomas caused by TBI is remarkable on CT and at autopsy. Blood from hematomas can be used to determine blood alcohol levels at the time of injury. Acute subdural hematoma is usually caused by bleeding from cortical contusions at the contrecoup site.

Hemorrhage from sites of cortical contusion into white matter is not uncommon and should not be mistaken for intracerebral hemorrhage due to natural causes.

Table 8.8 Types of intracranial hemorrhage

Type	Localization	Origin	Mechanism/cause
Epidural hematoma (Fig. 8.28)	Between the inner table and dura	Usually the middle meningeal artery, less frequently the anterior or posterior meningeal artery	Generally bending fractures to the adjacent cranial vault. Possible vessel laceration due to tearing forces in the sulcus, even in the absence of fractures
		Uni- or bilateral sinus rupture	
Subdural hematoma (Fig. 8.29)	Between the dura and the arachnoid	Cortical arteries in crest areas	Coup–contrecoup injury, contrecoup region
		Bridging veins, arteries at the brain surface	Rotational injury (in adults) or skull fracture (e.g., from a blow from a fist) subjecting vessels to shearing forces
		Bridging veins	Shaken baby syndrome
Subarachnoid hemorrhage	In the arachnoid meshes	Basal artery, vessel lacerations below bone fractures	Rotational or coup–contrecoup injury
		Anterior communicating artery, middle cerebral artery, internal carotid artery, vertebral artery	Rupture of preexisting aneurysms
Intracerebral bleeding	Cerebral white matter	Site of cortical contusion	Coup–contrecoup injury
			Massive rotational injury, "central brain rupture"
	Hypertensive intracerebral hemorrhage		Spontaneous rupture of atherosclerotic arteries (common: hypertensive intracerebral hemorrhage)

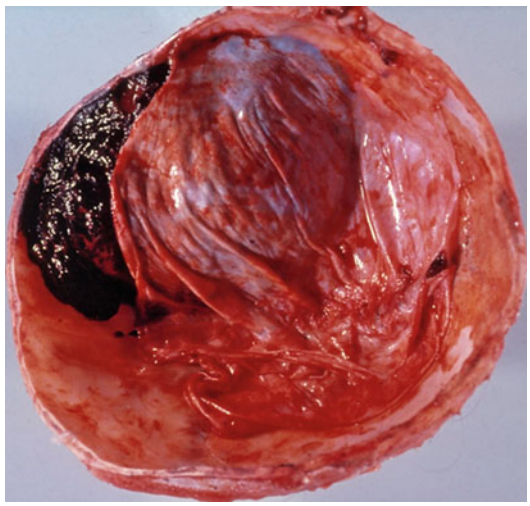

Fig. 8.28 Posttraumatic epidural hematoma between the cranial vault and the dura following injury to the middle meningeal artery

Hygroma. A hygroma is a fluid-filled exudation cyst; its interpretation as posttraumatic is controversial.

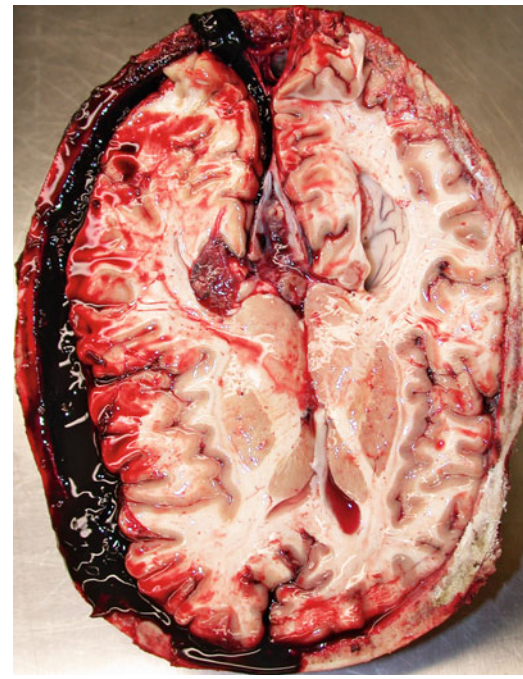

Fig. 8.29 Extensive subdural hematoma with midline displacement

Epidural Heat Hematoma. Prolonged exposure to heat causes blood within the cranium to be forced inwards and accumulate, together with fat, between the dura and inner table of the cranial vault. Heat hematomas are brick red in color and do not represent a sign of vitality (see Fig. 12.14).

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Pointed, Sharp, and Semi-sharp Force Trauma

9

Cutting implements, particularly knives, cause injuries classified as sharp force trauma, i.e., stab and incised (cut) wounds. Semi-sharp trauma comprises chop wounds, e.g., from saws, and bites wounds. Pointed force trauma includes injuries caused by firearms and pointed instruments, comparable to puncture wounds.

Instruments that cause sharp force injury differ from those that cause semi-sharp and pointed force injury (see Table 9.1).

Sharp and semi-sharp force cause varying wound morphologies and can be differentiated from blunt force injury by careful consideration of the wound edges, wound base, and wound angle (Fig. 9.2). In the case of injury caused by glass or glass fragments, tiny splinters of glass can sometimes be identified in the wound radiologically.

A distinction is made between stab and incised wounds. A stab perforates tissue by means of a pointed instrument in a primarily perpendicular force vector in relation to the surface of the body. In the case of a cut wound, a sharp edge (blade) moves across the body at a tangential angle. Depending on the instrument used and the course of movements, a *combination of stab and incised wounds* may be seen. Knives often produce this type of combined wound.

Important: The general morphological criteria of sharp force trauma include tissue perforation (of varying depth), smooth wound edges, as well as smooth perforation to the wound base and at the wound corners; contusion, drying, abrasion, and tissue bridges are absent.

However, if a knife penetrates to the point where the handguard comes in contact with the skin, dried and abraded wound margins may be seen in rare cases, as well as occasional contusions.

The general principle applied in the differentiation between stab and incision wounds is as follows:

Stab wound = depth exceeds length

Incised wound = length exceeds depth

Table 9.1 Examples of instruments and objects that cause sharp and semi-sharp force trauma

Sharp force	Semi-sharp force	Pointed force
Knives (dagger)	Axes	Knife point
Glass shards (broken bottle, Fig. 9.1)	Hatchets	Skewer (e.g., barbecue skewer)
Scissors	Chain saws	Forks
Porcelain shards	Grinders	Needles
Sharp-edged plates and sheets of metal	Swords	Ice picks
Carpet cutters	Machetes	Screwdrivers[a]
Scalpels	Saws	
Razor blades	Propellers	
Scythes	Lawn mower blades	
	Lances and spears[a]	
	Hooks	
	Drills	

It is not always possible to attribute individual instruments to a specific pattern of injury conclusively, since the instruments mentioned here can have varying forms

[a]Puncture wounds are also possible here, comparable to piercing wounds with a knife point

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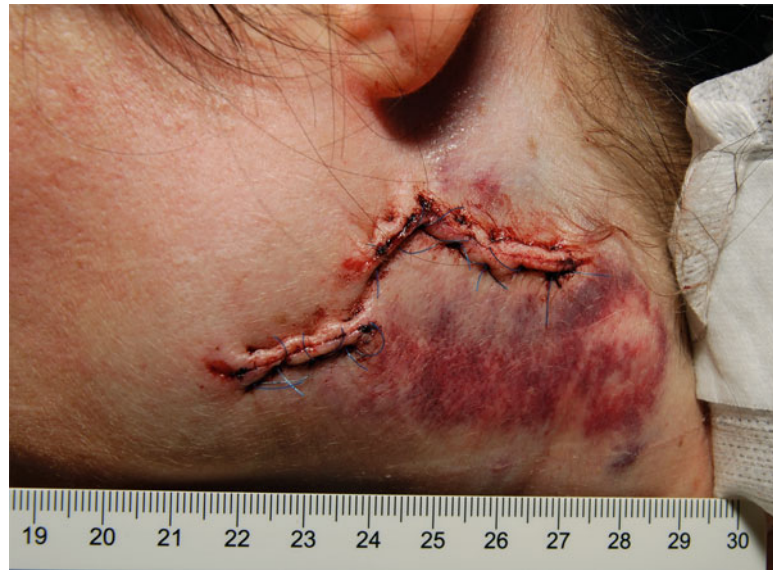


Fig. 9.1 Broken bottle injury

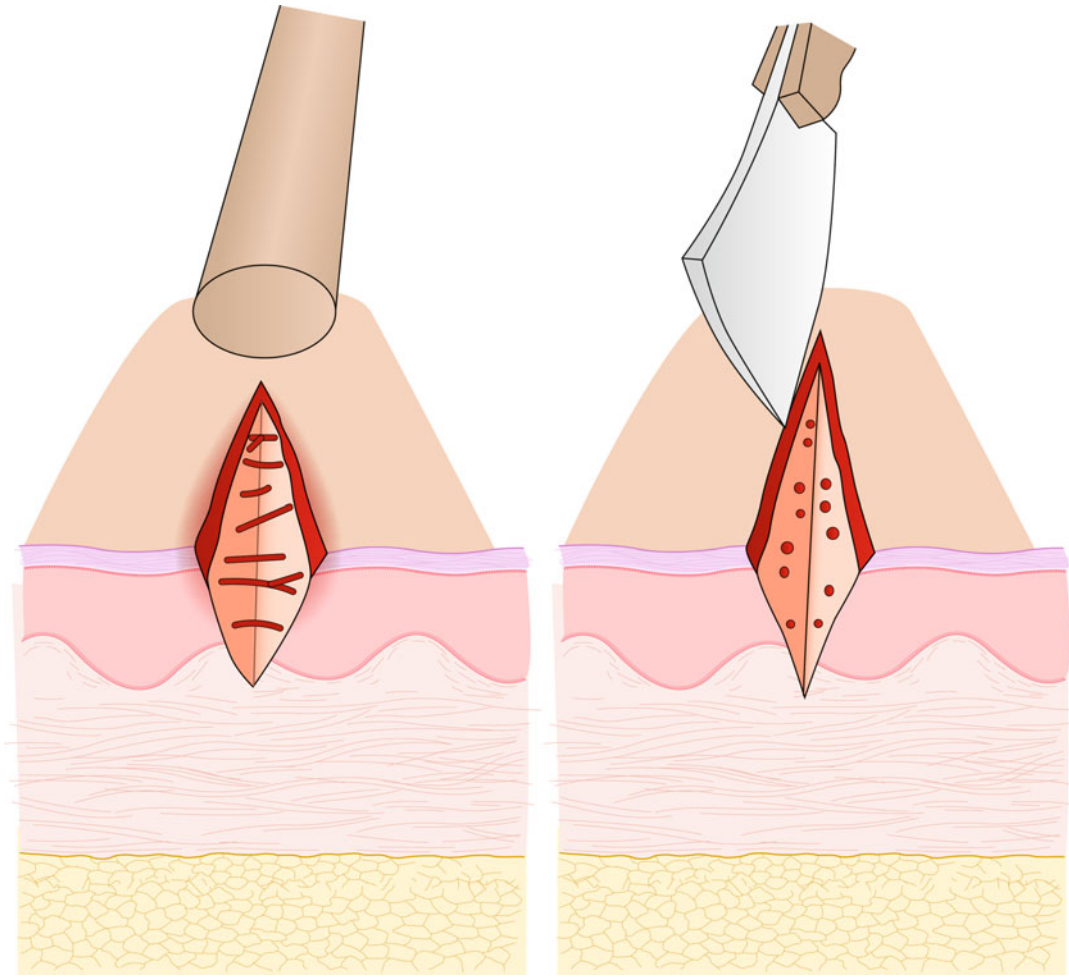


Fig. 9.2 Wound morphology in sharp and in blunt trauma: tissue bridges are absent in sharp trauma

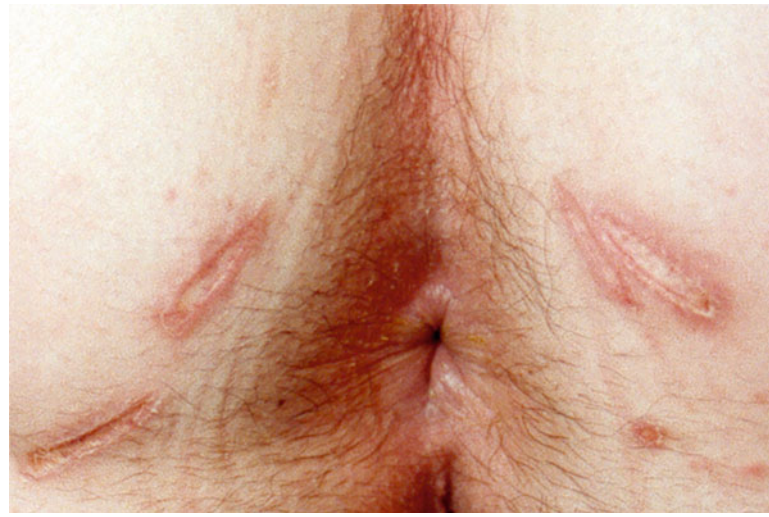


Fig. 9.3 Healed injuries inflicted to the perianal region with a broken bottle

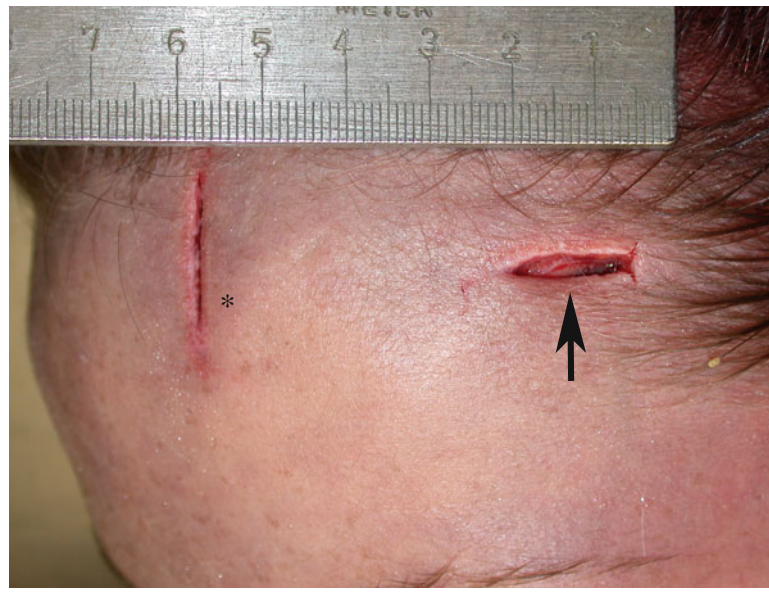


Fig. 9.4 Stab wound (depth greater than length) showing a knife-back contour and a small dovetail defect at one end (*arrow*), as well as a neighboring incised wound (length greater than depth; *asterisk*)

Depending on the mass of the instrument (weapon) and the speed of movement at the time it hits the body, semi-sharp force may involve a blunt force component in addition to the stab/incised wound. *Injuries resulting from a combination of sharp and blunt force* are referred to as *semi-sharp force* injuries and, if left untreated, can leave broad scars (Fig. 9.3).

9.1 Stab Wounds

Stab wounds are most commonly inflicted with a knife. At first glance, stab wounds are almond-shaped or even elliptical. The gaping wound edges are smooth. In the case of double-edged blades, e.g., a stiletto, both ends of the wound are pointed, while a single-edged blade produces a wound that is pointed at one end and squared off at the other (Fig. 9.4.) Depending on the width of the knife back and the extent to which the stab wound gapes, the squared-off end may be striking in that it appears as a v-shaped notch with two right angles or as a small dovetail defect. Approximating the two wound edges permits a better appraisal of the stab wound.

The length and width of a knife blade can only be determined from the wound if the knife entered and exited at a perpendicular angle, i.e., a pure stab wound to non-taut skin. If a wound is a combined

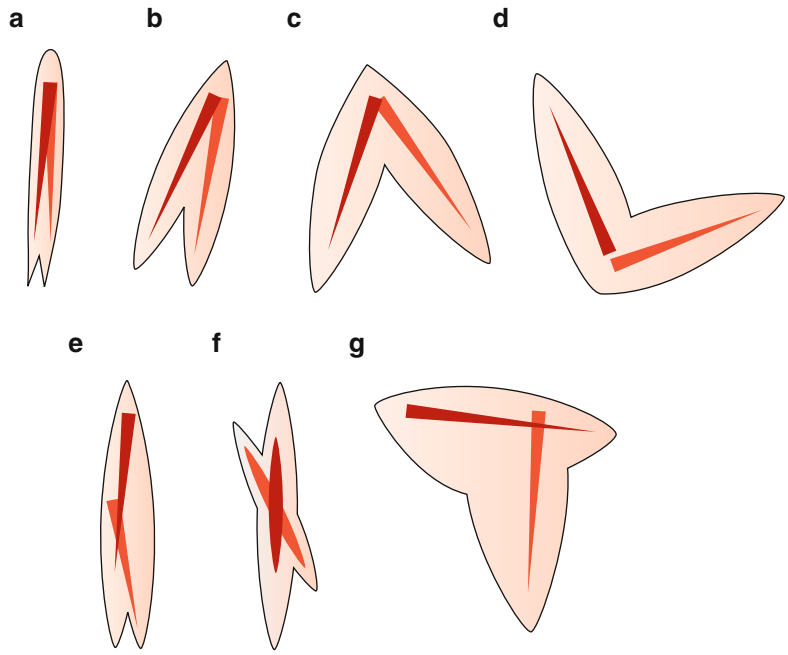


Fig. 9.5 Mechanism of formation and various forms of large dovetail defects: **a**–**d** = the knife is rotated or the victim attempts to evade assault; **e**–**g** = simultaneous coarse incising motion; **f** = wound configuration with two dovetail defects (only possible in double-edged knife wounds) Modified according to Höhn (1985), Saukko and Knight (2004), Hochmeister et al. (2007), Dettmeyer and Verhoff (2011)

stab/incised wound, it is no longer possible to determine the width of the knife blade. However, it is possible to measure the width of the stab wound in a great number of cases, and if multiple stab wounds of the same width are present, it is possible to state the width of the knife blade with relative accuracy.

In order to draw conclusions on the direction of the stab wound, the course of the wound track should be documented as precisely as possible at autopsy. Together with entrance stab wound analysis, it is possible to surmise the position of the perpetrator and victim and/or the sequence of events surrounding the stab wound and the position of the knife in the perpetrator's hand.

Although it may seem evident that blade length and width could also be determined from the stab wound, any determination becomes problematic if the blade has not penetrated fully. Thus, the length of the stab wound may not correspond to the maximum blade width, and wound depth may not equate to the actual length of the blade. On the other hand, the stab wound track may be longer than the blade if the blade was thrust in to its full length and soft tissue yields under pressure. The stab wound may be wider than the blade if, as is often the case, a cutting motion has additionally been used as the knife was thrust in (stab/incised wound).

Important: A stab wound from a knife may be wider or narrower than the width of the blade and its depth greater or lesser than the length of the knife.

If a knife is turned on its long axis as it is thrust in or particularly as it is withdrawn, or if the victim moves at the moment the knife penetrates, the stab wound may demonstrate a large dovetail defect; defects of this kind can vary in degree (Fig. 9.5).

Atypical blades, such as those with serrated spines or which are double-edged at the tip of the blade and widen into a broader knife back, can produce characteristic stab wounds (Fig. 9.6). The wound angle produced by the blade of a serrated knife, such as a large bread knife, shows a particular wavelike striation at the wound edges. Not infrequently, a fine skin abrasion extending from the pointed end of the wound in the cutting direction can be seen in stab wounds when the angle at which the blade is withdrawn from the wound is not completely vertical.

A variety of pointed instruments are capable of inflicting stab wounds (see Table 9.1). In cases where less sharp objects are used, discrete abrasions around the point of skin penetration may be seen. More rarely, a number of sharp weapons or

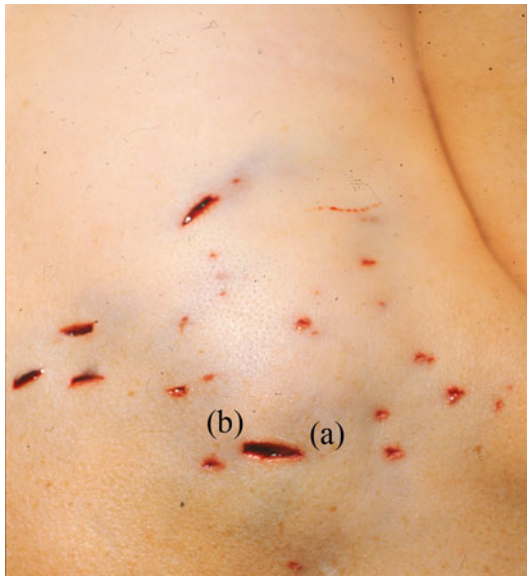


Fig. 9.6 Piercing wounds to the skin as well as deeper stab wounds with a pointed wound end (**a**) and a squared-off wound end (**b**) produced by a single-edged knife with a knife back

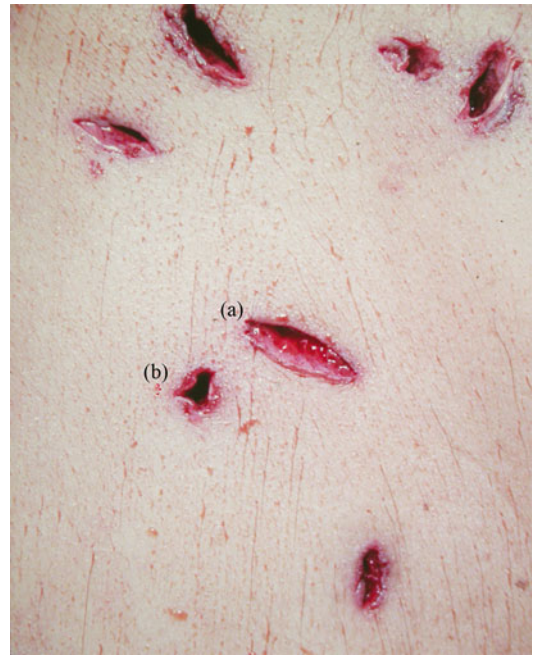
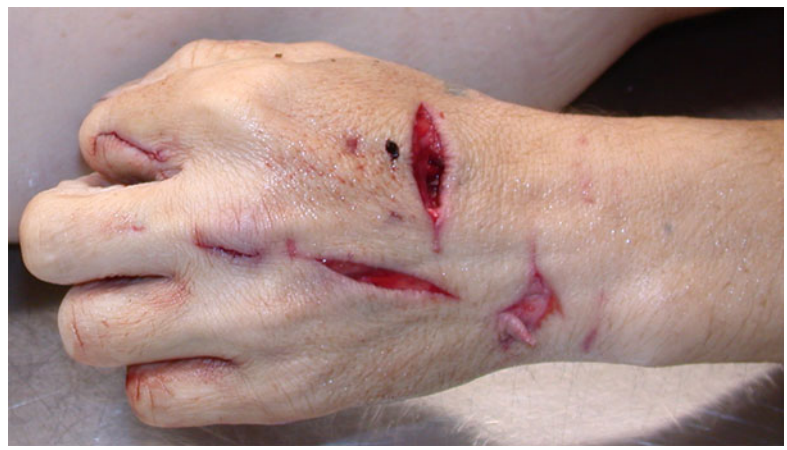


Fig. 9.7 Combined assault with a knife and scissors. Typical knife stab wounds, one with a small dovetail defect (**a**) and multiple triangular injuries caused by stabbing with scissors (**b**)

Fig. 9.8 Passive defense injury to the back of the hand following an assault with a knife



instruments may be used, either simultaneously or in rapid sequence, such as a knife and a pair of scissors (Fig. 9.7).

An assault with a knife can produce passive defense wounds (backs of hands, extensor side of the forearms, etc.; Fig. 9.8) as well as active defense wounds (e.g., palms of the hands, the area between the thumb and index finger; Figs. 9.9 and 9.10).

Passive defense wounds: These refer to the injuries incurred when a victim has held his/her hands or arms raised in front of the face for protection, resulting in stab and cut wounds from the assault weapon to the outer sides of the upper arms, to the extensor side or little finger side of the forearm, and the extensor side of the hands (backs of hands).

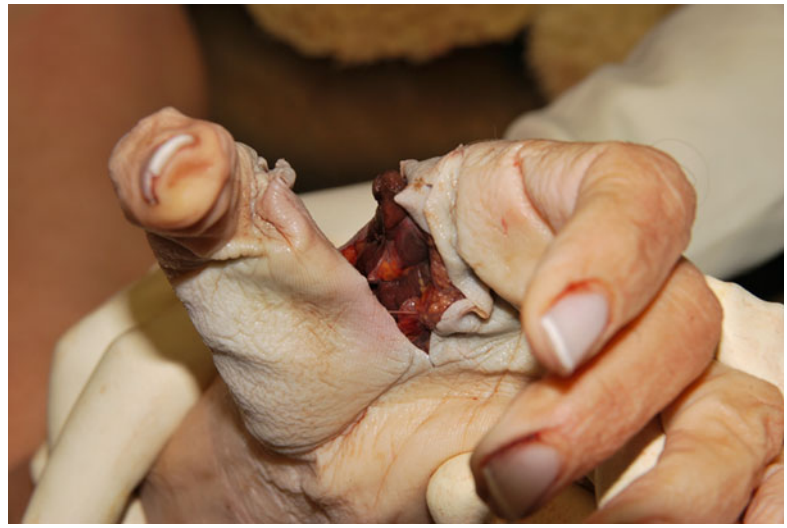


Fig. 9.9 Active defense wounds involving cuts to the palms of the hand and flexor sides of the fingers incurred in a knife assault

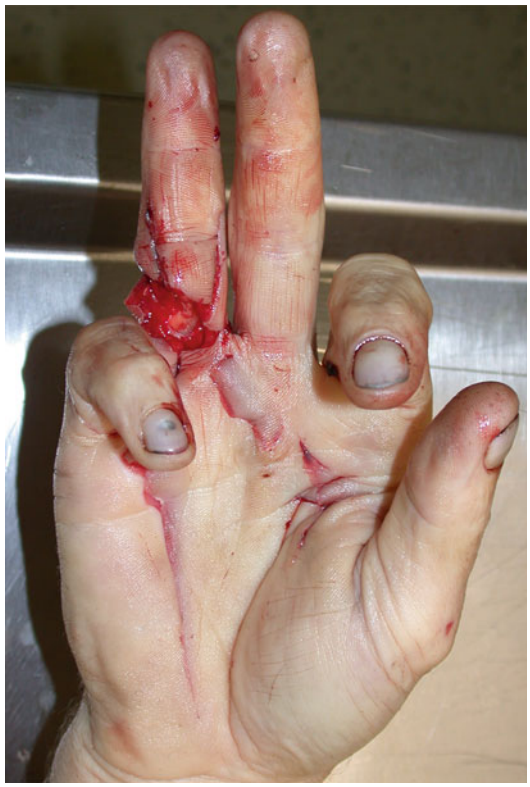


Fig. 9.10 Active defense injury between the thumb and index finger following a knife assault

Active defense wounds: These refer to the stab and incised wounds to the flexor side of the fingers and palms of the hands incurred when a victim reaches for the knife blade; the area between the thumb and index finger is typically affected.

Fatal stab wounds to the heart are occasionally overlooked at autopsy; moreover, wounds of this kind do not always produce significant external blood loss. The background history to such cases often includes reports that the victim complained of only mild and uncharacteristic symptoms following a single stab wound.

Since intercostal skin, intercostal soft tissue, and cardiac muscles have good shape stability, the shape of the weapon used (blade width, knife back) to inflict a stab wound to the heart can often be clearly identified from the wound track through skin, subcutaneous soft tissue, the pericardium, the epicardium, and the cardiac muscle.

It is not uncommon for the wound track to end in a ventricular lumen. In most cases, cardiac tamponade is the cause of death. On dissection, attention should be paid along the course of the wound track to whether an intercostal artery was injured, resulting in hemorrhage into the pleural cavity, as well as to whether a rib has been notched or a coronary artery pierced. When a knife is virtually rotated on its side, a horizontal wound track through the soft tissue between the ribs without notching of neighboring ribs is possible. Stab wounds to the fontanelles in the context of neonaticide and infanticide constitute a special type of stab wound which causes

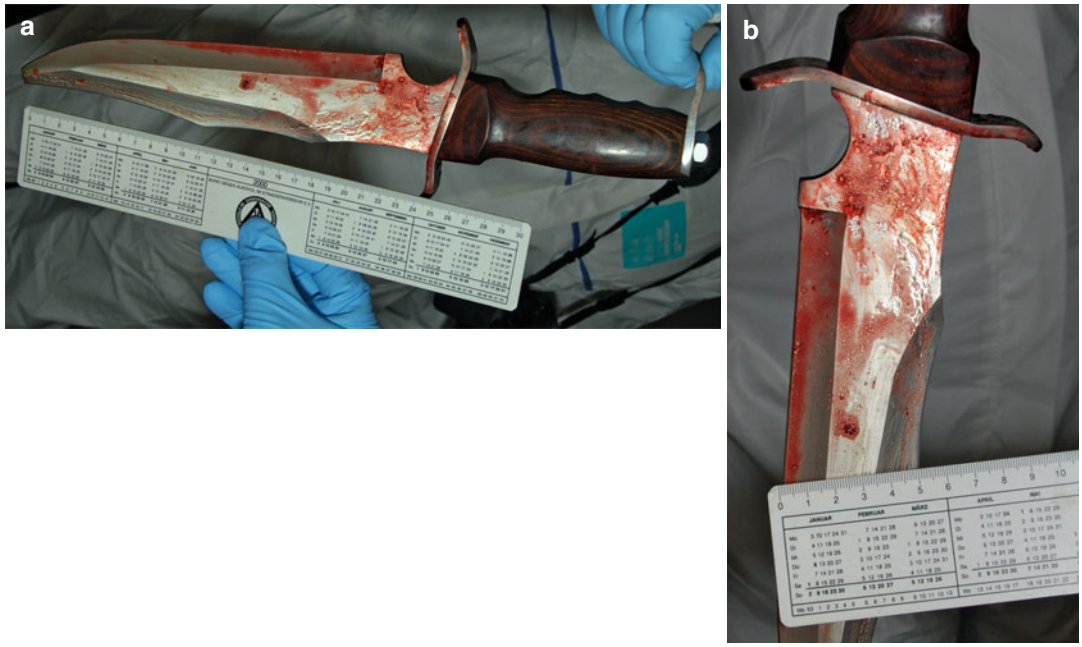


Fig. 9.11 (**a**, **b**) Determining the length, width, and type of blade (single-edged, double-edged, knife back, pointed, smooth blade, blade shaft) in a knife used to inflict injury

intracranial hemorrhage and which may be evident at autopsy only in the form of a discrete piercing of the hairy scalp over the anterior fontanelle, particularly if a pointed instrument like a needle has been used.

In all cases of stab wounds, the pattern of injury, i.e., length of the wound track, length of the stab wound, possible blade marks, sharpness of the blade, and serrated edges, needs to be compared where possible with a crime instrument, particularly when this is a knife (Fig. 9.11a, b).

9.2 Incised Wounds

An incised wound is produced when an object with a sharp edge—a blade from a functional perspective—comes in contact with the skin and underlying tissue. If the blade contacts the skin at a slanted angle (i.e., the longitudinal axis of the blade in relation to the skin surface), one wound edge will be beveled, while the opposing edge will be undermined. There is no tissue bridging under any circumstances. As with stab wounds, it is important to establish whether structures deeper in the wound have been injured, thus causing life-threatening injury, major vessels being particular noteworthy here. Both venous and arterial exsanguination (internal or external) is possible. *Air embolism* should also be considered if *major veins have been opened*; a gas volume of between 70 and 150 ml would be sufficient for this to occur. In the case of deeper stab and incised wounds to the neck, the trachea may have been opened, causing massive blood aspiration and leading to asphyxiation. In such cases, pulmonary tissue typically has a reddish pattern, occasionally referred to as a "chessboard-like pattern," as a result of blood aspiration.

9.3 Fatal Stab/Incised Wounds: Differentiating Between Suicide and Homicide

Tentative stab or incised wounds are characteristic of stab/incised wounds with suicidal intent, seen occasionally to the neck (Fig. 9.12), but more often to the flexor side of the wrists.

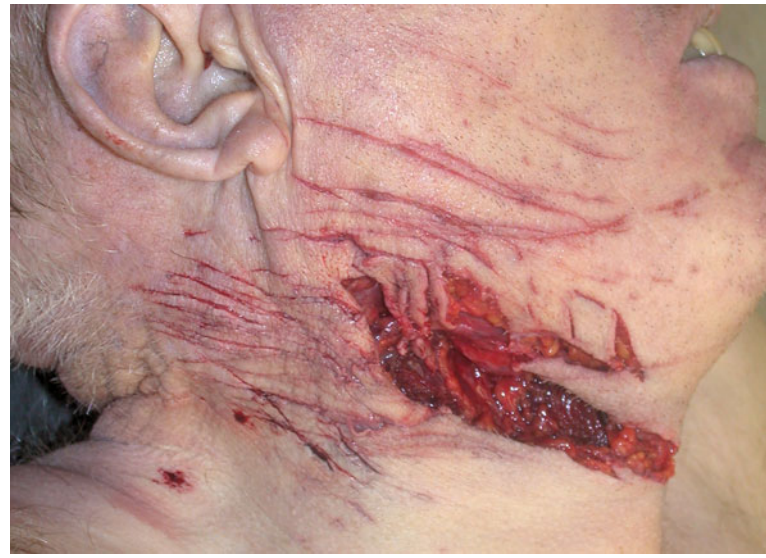


Fig. 9.12 Tentative cuts inflicted with suicidal intent accompanied by deep stab/incised wounds to the soft tissue of the neck

Tentative wounds are superficial, usually parallel incisions in the epidermis, at most the dermis, found adjacent to individual deeper skin incisions causing vessel injury. Injury is localized to an area easily accessible to the individual's dominant hand, predominantly to the flexor side of the left wrist or left side of the neck (in the case of right-handed individuals). These wounds are attributed to the onset of pain experienced when the incising instrument comes into contact with the skin and prevents the incision being made with the initially intended force. When the instrument comes in contact with the skin a second time, renewed pain again restricts the incision to a superficial one. This process is apparently repeated until a certain pain threshold is exceeded and deeper, ultimately more effective incisions can be made. Scarification to chest skin localized around a suicidal stab wound to the heart has a similar etiology. When an instrument with an extremely sharp blade such as a razor blade has been used, attention should be paid in good light to very fine, linear skin incisions in the hands of the victim (in the case of suicide) or the suspected individual (in the case of homicide).

At autopsy, multiple parallel scars generally running in a transverse direction across the flexor side of the wrists may be an indication of previous suicide attempts. Superficial scars or incised wounds running in either a transverse or longitudinal direction on the forearms and resulting from self-harm ("cutting") need to be differentiated from tentative wounds. Autoaggressive components in the context of psychological disorders, predominantly borderline-type personality disorders, form the basis of this behavior. Morphologically, the fact that none of the incisions or scratches pierces the dermis completely is striking. Comparable, and generally recent, self-inflicted incised injuries to various areas of the body are occasionally seen on examination, accompanied by inaccurate reports about an accident. Here again, it is necessary to establish whether the pattern of injury corresponds to self-inflicted injury or injury inflicted by others (see Chap. 17).

If major neck veins have been opened as a result of deep stab or incised wounds, in particular the internal jugular vein, sudden death due to air embolism is possible and may preclude fatal exsanguination (Fig. 9.13; see Chap. 11, Fig. 11.1).

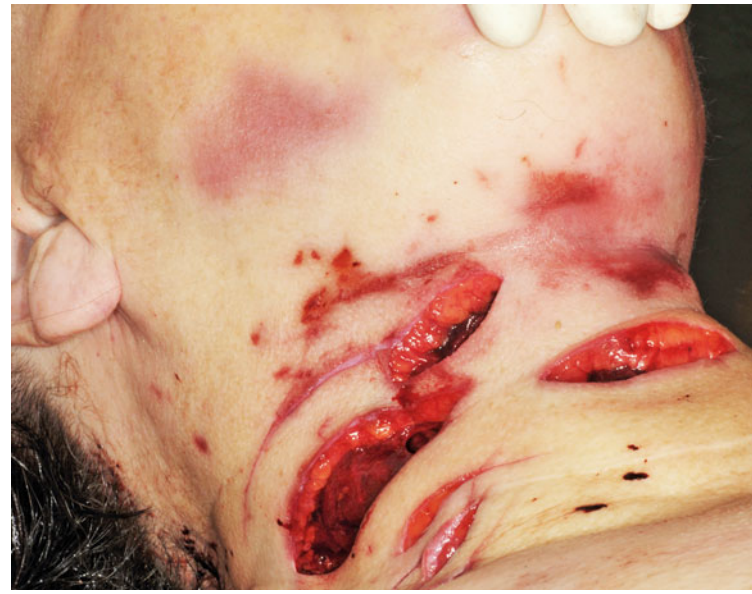


Fig. 9.13 Stab and incised wounds to the neck with opening of the deeper internal jugular vein (homicide rather than a tentative wound, see Fig. 9.11)

9.4 Semi-sharp Force Trauma

The term "semi-sharp force trauma" is not uncontroversial; it is used to describe wounds which, although caused by instruments, cannot be conclusively classified as either sharp force or blunt force trauma or which are a combination of both.

9.4.1 Chop Wounds

Characteristic features of chop wounds include *smooth-edged transection of tissue with crushing and abrasion of the surrounding skin* or tissue. Depending on the weight of the instrument used, the degree of blunt trauma resulting from this weight, and the site of trauma, bone fractures or combined forms of bone incision and fracture are possible. Chop wounds or injuries due to semi-sharp force are typically caused by swords, machetes, sabers, axes, or hatchets; however, airplane and boat propellers, as well as tools such as grinders (see Table 9.1), can also produce chop wounds.

9.4.2 Saws

Although the extent of blunt force trauma appears to be negligible in the case of saw injuries, the criteria for sharp force trauma are not always fulfilled. The teeth of a saw can cause microtraumas in rapid succession that could be classified as blunt trauma, while the action of sawing, on the other hand, corresponds to a cutting action ("saw cut"). These factors support the classification of sawing as semi-sharp force.

In criminal investigation terms, saw marks play a role virtually exclusively as postmortem injuries in the context of dismemberment for the purposes of disposing of a body. Bodily harm or homicide using a chain saw constitutes rare exceptions. From the perspective of the insurance medical examiner, questions regarding whether a saw injury is actually present, whether it resulted from an occupational accident, or whether it is the result of self-mutilation for the purposes of making an insurance claim occasionally require clarification.

Saws produce *ragged soft tissue lacerations* arranged in a linear pattern. Marks of a more characteristic nature can be seen on bones that have come in contact with or been severed by a saw. *Bone defects* produced in this manner permit *inferences to be made about the configuration of the saw blade* (size of teeth, mono- or bidirectional saw). By means of experimental comparisons, it may be possible to attribute a bone injury to a particular saw.

9.4.3 Chain Saws and Grinders

Injuries produced by chain saws or grinders, for example, can show a mixed morphological picture: smooth wound edges on the one hand, somewhat ragged on the other with abrasions around the wound margins or skin detachment and skin tags, as well as varying degrees of ragged soft tissue beneath the skin depending on the depth of penetration. Strong pulling action may produce small epidermal cuts transverse to the cutting direction, which appear strikingly like fine scratches (Fig. 9.14).

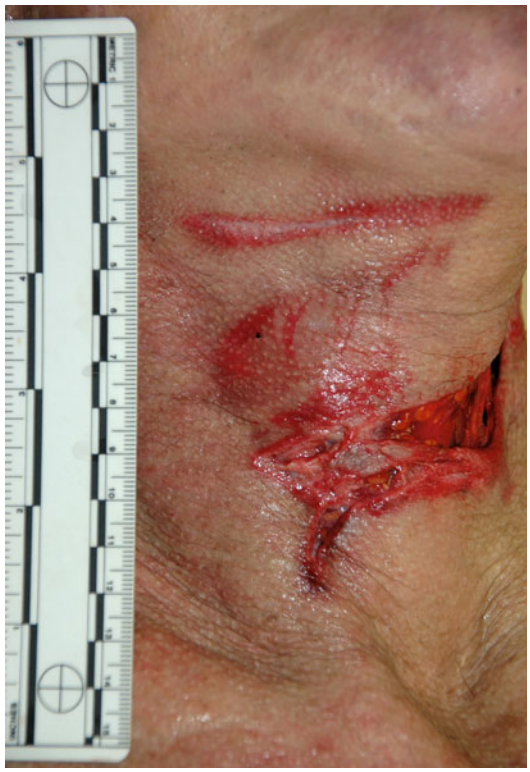


Fig. 9.14 A combination of smooth and ragged wound edges following injury to the skin and subcutaneous soft tissue of the neck with a grinder (suicide)

9.4.4 Bite Wounds

Bite wounds inflicted by humans are seen in the context of physical conflict, sexual offenses, and very rarely in the form of cannibalism. Bite wounds from animals or as a result of animal depredation are found primarily in the case of long postmortem intervals in homes or outdoors.

Human Bite Wounds. Bite wounds inflicted by humans are occasionally associated with sexual offenses. A *round to ovoid bite mark* produced by the teeth of the upper and lower jaws is characteristic (Fig. 9.15). Bites can produce indentations, abrasions, bruising, and even perforations in the skin of the victim. Skin perforations caused by bites from both humans and animals carry the risk of severe wound infection.

Important: Photographed at the correct angle (perpendicular to the surface) and using a scale, bite wounds can sometimes be attributed to a perpetrator retrospectively. Modern methods are able to work with 3D surface scans of a bite wound.

The classic method of producing *three-dimensional documentation* of a bite wound with teeth marks involves making a cast using a flexible polymer commonly found in dentistry.

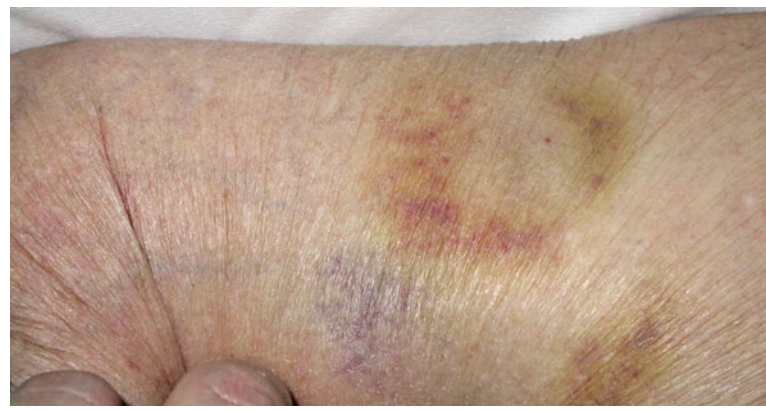


Fig. 9.15 Somewhat older bite wound of characteristic shape

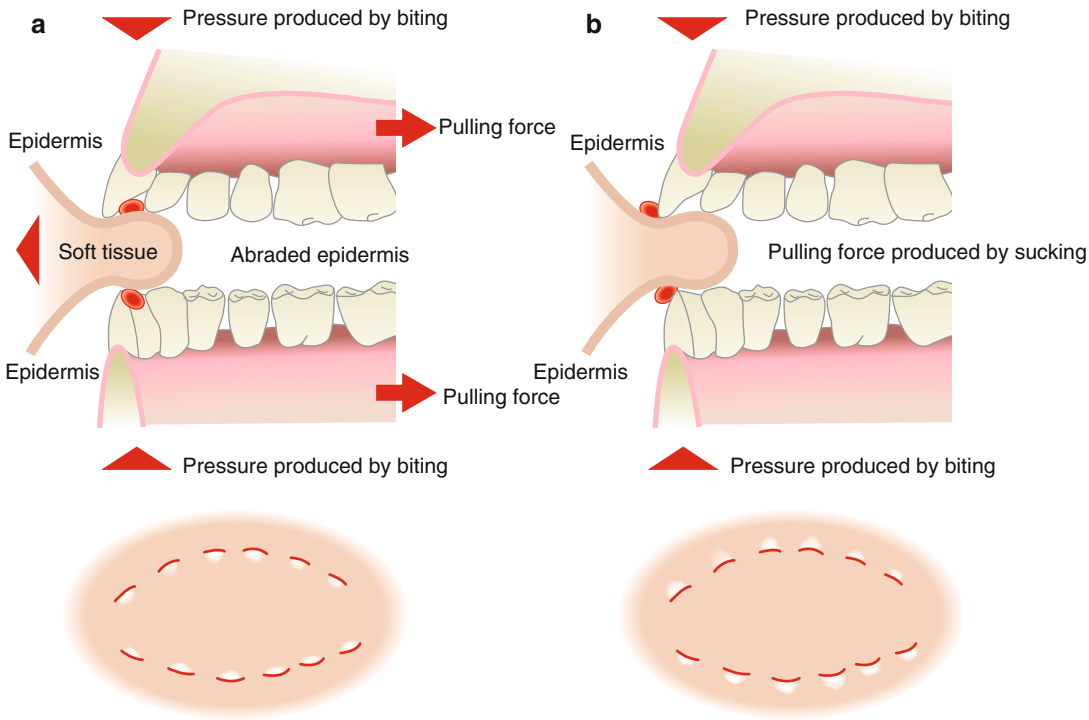


Fig. 9.16 A normal bite and a suction bite. Varying sites of epidermal abrasion in biting (**a**) and sucking (**b**)

However, it should be borne in mind that marks can only be identified in the early phase following a bite, i.e., within a few hours. Shortly after a bite wound has been produced, it is possible to make a distinction on the basis of wound morphology between a *defense bite* and a *suction bite* (Fig. 9.16).

Defense Bites: Defense bites typically involve the epithelium being pinched between the inner side of the dental arch (teeth and skin move in opposing directions on pulling away).

Suction Bites: In the case of suction bites, on the other hand, the epithelium is forced to the outer side of the dental arch (skin fold is sucked into the mouth).

Taking smears for forensic DNA analysis within 24 h of a bite wound can be helpful in the identification of a perpetrator. In addition to the post-expositional time interval, cleaning measures used following injury are of relevance.

Bite wounds should be documented as early as possible following injury. At an early stage, the number of teeth and their position in relation to one another are often easily identifiable and can be used for the purposes of identifying the perpetrator (Fig. 9.17).

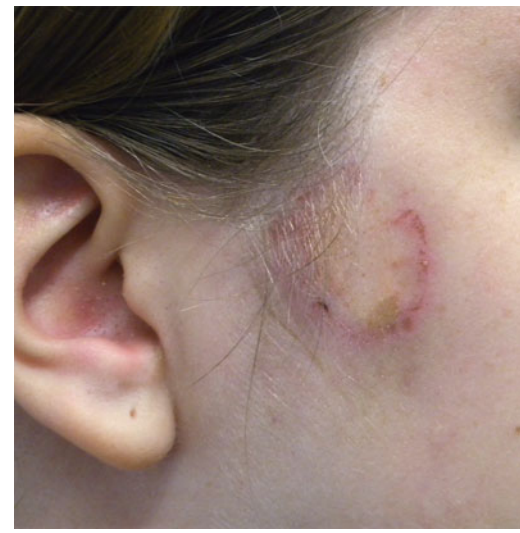


Fig. 9.17 An approximately 10-h-old bite wound to the skin due to domestic violence: parallel wounds run in a crescent-shaped course with mild bluish discoloration and individually demarcated teeth marks

As early as 2 days following injury from (human) bites, contusions lose their definition and teeth marks can no longer be identified. All that remains are two arc- or crescent-shaped and unclearly defined areas of reddish-brown skin discoloration located at bite distance from one another. It may not even occur to the inexperienced observer that these are bite wounds. Nevertheless, appropriate photodocumentation is advisable, since at least the dimensions of discoloration can be recorded and thus also the size of the jaw estimated and possibly compared with that of a suspect.

Animal Bites and Animal Depredation in Humans. Animal attacks on humans are occasionally seen, primarily by dogs in central Europe and only rarely by cats. Eyeteeth frequently cause skin perforation, while the remaining teeth produce crushing and abrasion wounds that often permit a reconstruction of the teeth (Fig. 9.18a–c).

As with human bites, *correlating* a bite wound to a particular animal is possible using special *DNA analysis*, such as canine forensic STR systems. Occasionally, animal bite and depredation wounds are seen at autopsy if animals (usually dogs or cats) are enclosed in a home with a body for an extended period of time. Postmortem animal depredation may produce an ambiguous pattern of injury initially suggestive of homicide.

9.5 Pointed, Sharp, and Semi-sharp Force Trauma to the Bone

Deep stab or incised wounds can cause injury to bony structures. Depending on the force with which an instrument, such as a knife, comes into contact with the body, fractures to the ribs, or at least peripheral v-shaped notches, along the course of the wound track are possible; these can be easily felt at autopsy. Depending on the force of the stabbing action and the stability of the instrument used, fractures to the face and skull, piercing stab wounds to the skullcap (Fig. 9.19a) and impression fractures (Fig. 9.19b), or even punctures involving fractures to the skullcap are possible.

In the case of deep incised wounds to the neck, incisions running transverse to the body may be seen, e.g., to the anterior side of the larynx, combined with transection of major neck vessels including the jugular vein and common carotid artery (Fig. 9.20).

Small particles of bone are sometimes carried along the wound track. Although characteristic traces of a particular instrument may be found on the bone tissue, conclusions above and beyond the fact that sharp or semi-sharp force trauma has taken place are usually not possible.

Coarser bone injuries are seen particularly in the context of semi-sharp force trauma involving the use of instruments such as hatchets or axes that have served as striking tools. Here, unilateral deeper indentations and splintered bone particles are seen, as well as fractures starting at the point of contact with the instrument. Saw teeth, depending on their size and configuration, may leave a characteristic pattern, while defective saws can produce a corresponding pattern of injury to the bone.

9.6 Causes of Death in Pointed, Sharp, and Semi-sharp Force Trauma

The cause of death in the case of sharp and semi-sharp force trauma depends not only on the intensity of trauma but also to a very great extent on the localization of injury. If both chest cavities are opened, hematothorax, pneumothorax, or a combination of both may be the cause of death. A deep incision to the throat whereby major neck veins are opened can cause fatal air embolism, while a single stab wound to the pericardium and cardiac muscles may trigger fatal cardiac tamponade. In the case of acute cardiac tamponade, 200–300 ml of blood within the pericardium causes death, while larger volumes of blood may be tolerated in milder protracted bleeding due to slow pressure-related pericardial expansion (chronic cardiac tamponade). However, stab wounds to the heart can often cause pericardial blood to be forced into both mediastinal soft tissue and the pleural cavities as a result of the stab wound to the pericardial sac.

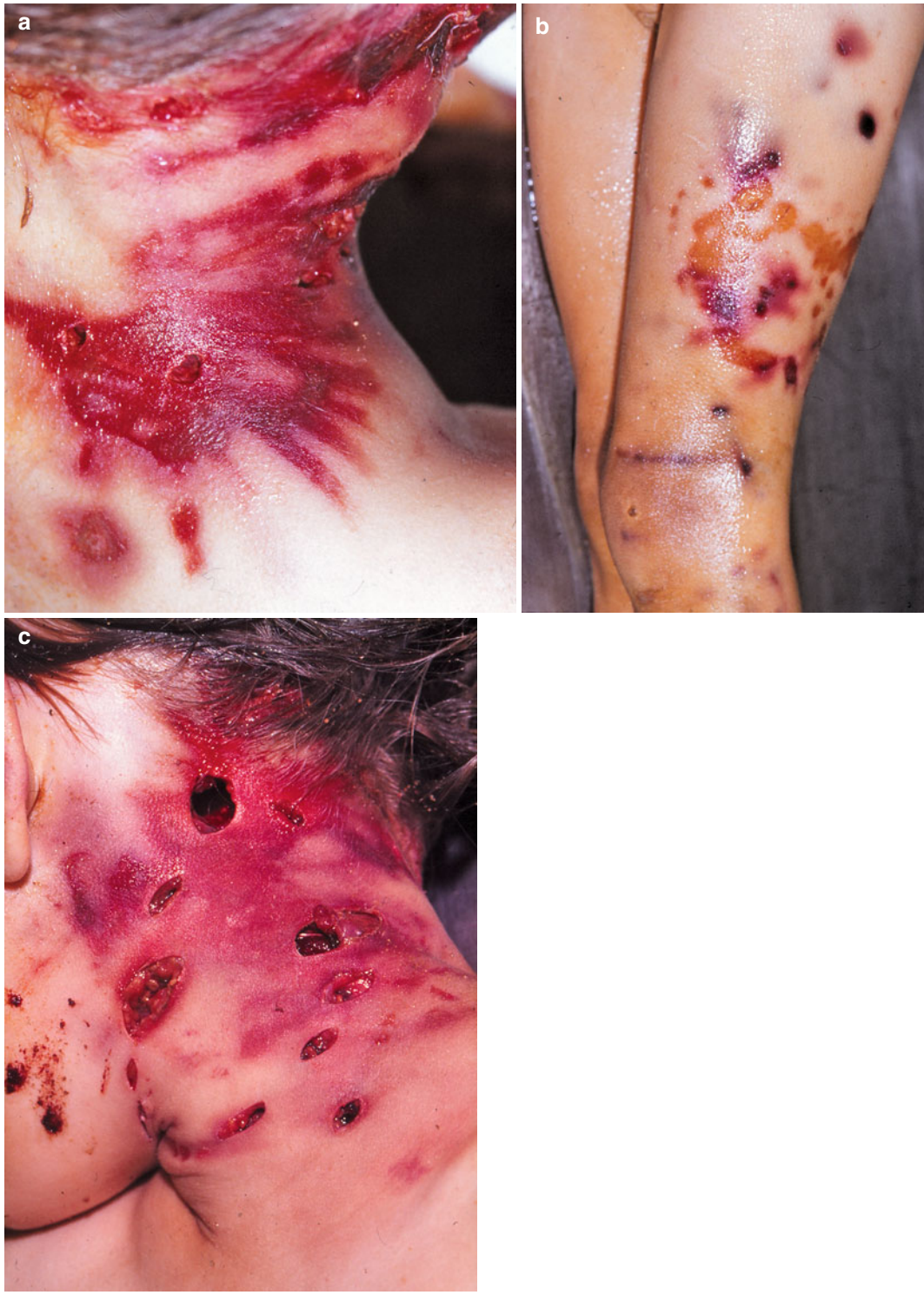


Fig. 9.18 (**a**) A 5-year-old boy who was attacked by an Alsatian dog: (**a**) linear bite-related skin abrasion, (**b**) both fresh and postmortem teeth marks on the extremities, and (**c**) deep bite wounds to the neck; cervical spine fracture was detected at autopsy

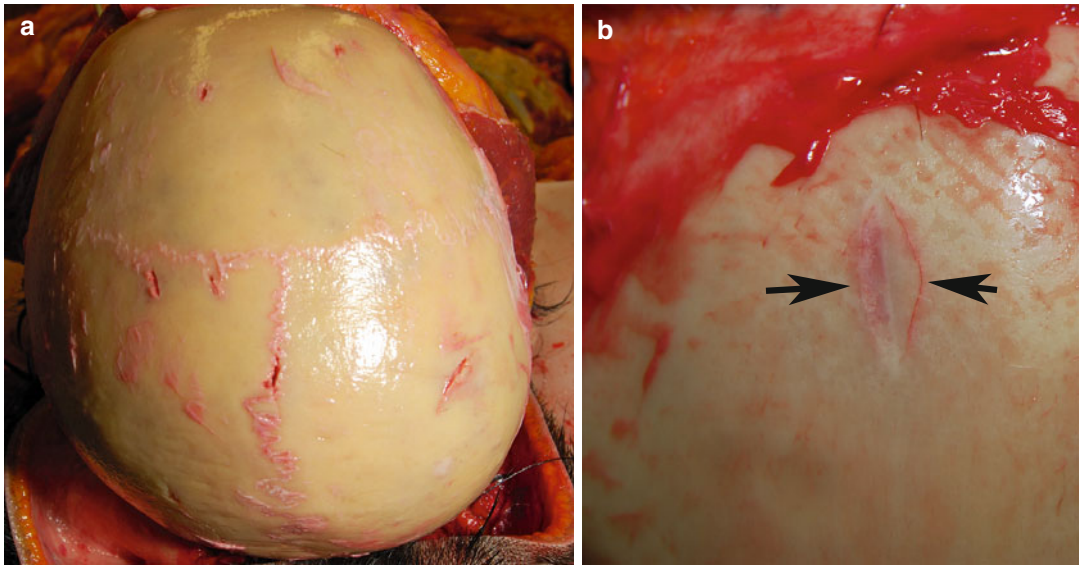


Fig. 9.19 (**a**) V-shaped notches to the bony skullcap caused by stab wounds. (**b**) Semi-sharp force: impression fractures (*arrows*) to the bony skullcap following a blow from the semi-sharp edge of a cooking pot

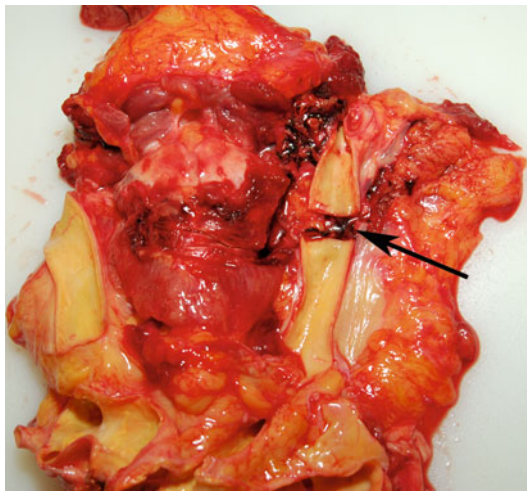


Fig. 9.20 Deep incision to the anterior neck (*arrow*) with incision-related v-shaped notches to the anterior larynx and transection of the jugular vein and common carotid artery

If a stab wound causes injury to an intercostal artery, a pelvic artery, or the aorta, relatively rapid internal exsanguination ensues, and blood is seen in the chest or abdominal cavity at autopsy, where blood volumes of up to 2.5 l may be found. Blood loss of around 30 % of total blood volume is considered life-threatening, while blood loss of more than 60 % of total blood volume is generally fatal. In the case of fatal internal bleeding, between 1.5 and 2 l of blood is found in the body cavities (pleural and abdominal cavities) of individuals with healthy cardiac parameters. Only an approximate estimate of blood loss or volume can be made if bleeding into soft tissue, in particular retroperitoneal soft tissue, has taken place.

It is not inconceivable that blood volumes found in body cavities at autopsy were increased in the course of resuscitation. However, the speed of blood loss plays an important role, as well as any possible volume substitution measures. In the presence of preexisting cardiac disease, such as stenosing coronary sclerosis, cardiomegaly, or valvular heart disease, smaller volumes of blood loss can lead to death.

Stab wounds that cause hemorrhage into the soft tissue of the neck and subsequent asphyxia due to retropharyngeal hematoma are comparatively rare. If, however, blood enters the airways following a stab wound, fatal blood aspiration may precede fatal exsanguination; where this is the case, lung sections show a "chessboard-like" pattern resulting from the aspirated blood (Fig. 9.21a–c).

Coarse semi-sharp force trauma to the skull can cause fatal craniocerebral trauma involving skull

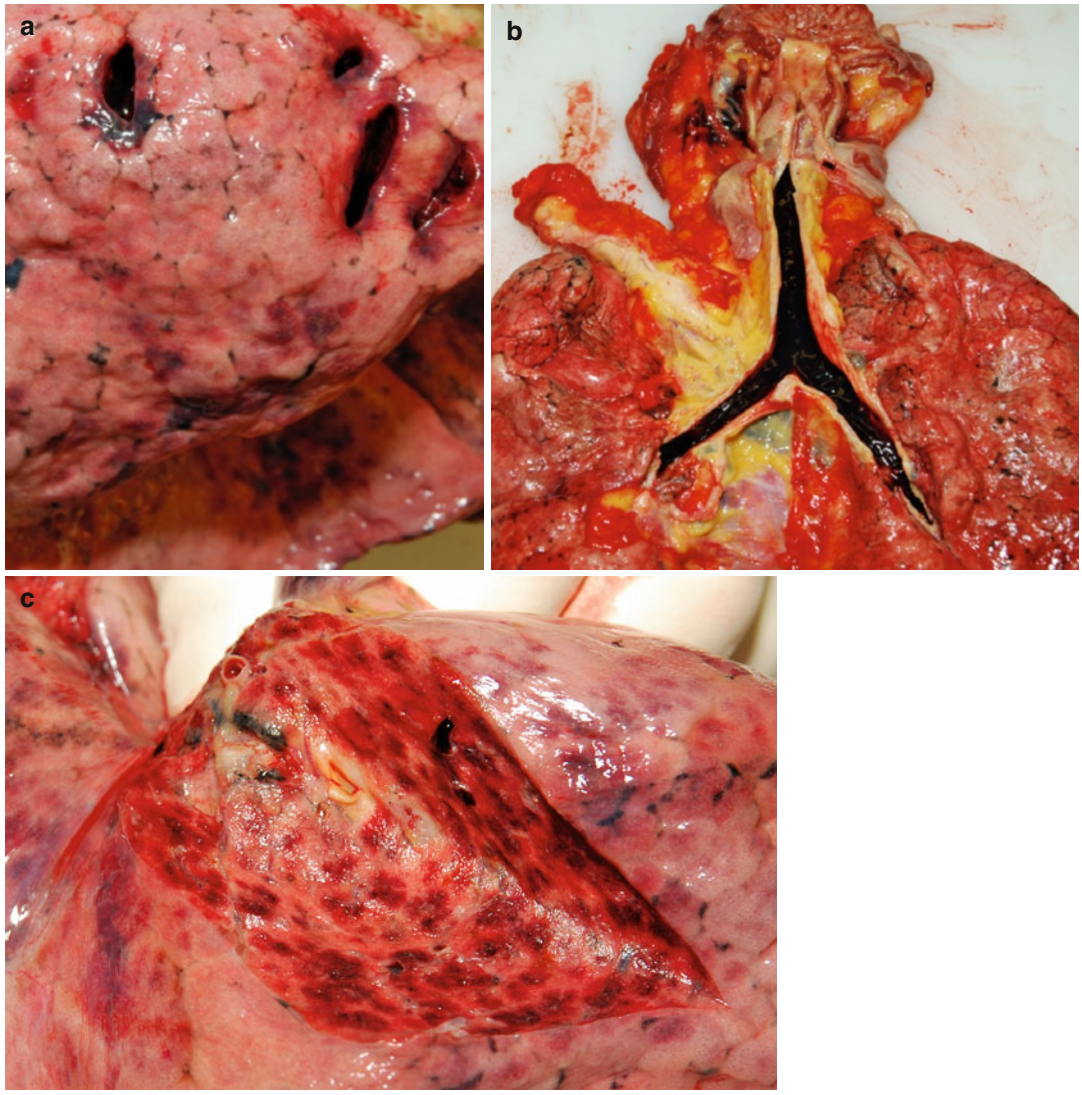


Fig. 9.21 (**a**) A deep stab wound to pulmonary tissue. (**b**) Copious blood is also seen in the upper airways. (**c**) Blood aspiration to the lung periphery with a chessboard-like pattern

fractures, injury to the meninges and brain, as well as intracranial hemorrhage. In the case of fatal (protracted) blood loss, typical signs of exsanguination can be seen on the body or at autopsy:

- Livor mortis of limited extent and intensity.
- Puckered splenic capsule.
- The intrinsic color of internal organs, in particular the thyroid gland, kidneys, and liver, becomes prominent.
- A single strip of subendocardial hemorrhage at the level of the left ventricular outflow tract.

At the same time, in the case of external bleeding, a comparison should always be made with blood detected at the scene where the body was discovered.

Chop wounds often affect bony structures, where indentations, ragged chipping defects, as well as bending and impression fractures are seen. Chop wounds to the skull may produce scalp laceration, severe destruction of the bony skull, cerebral tissue injury, and intracranial hemorrhage.

9.7 Medicolegal Aspects of Death Due to Pointed, Sharp, and Semi-sharp Force Trauma

Four questions in particular frequently require clarification by the forensic medical expert in the case of sharp and semi-sharp force trauma:

- Do the injuries permit conclusions to be drawn about the particular instrument used?
- Can a distinction be made between self-inflicted injury and injury inflicted by others?
- Following injury, was the victim able to act and if so, for how long?
- Can a distinction be made between antemortem injuries caused by sharp and semi-sharp force trauma and postmortem injuries?

9.7.1 Inferences About a Particular Weapon or Instrument

Although conclusions in this regard can often only be drawn with caution, it is generally possible to state what type of weapon or instrument is more likely to have been used and whether there was any third-party involvement. The pattern of injury sometimes makes it possible to conclude that more than one weapon or instrument was used. In some cases where a knife has been used, conclusions can be drawn about blade width and length as well as whether the knife was single- or double-edged (knife-back contour?). A serrated blade can leave a correspondingly serrated wound edge. Conclusive statements about the instrument or weapon used in the case of semi-sharp force trauma are more challenging.

9.7.2 Sharp and Semi-sharp Force Trauma: Differentiating Between Self-Inflicted Injuries and Injuries Inflicted by Others

Although the vast majority of stab wounds are caused by a third party, the possibility of self-inflicted injury with suicidal intent needs to be evaluated in the case of doubt; to this end, a series of criteria need to be taken into consideration (Table 9.2).

Stab wounds with suicidal intent are generally to the left anterior chest over the heart. Clothing is often not involved. If several stab wounds have been inflicted, these are generally close together and run in the same direction (often horizontal). In addition, tentative wounds are seen; stab and incised wounds to other areas of the body may also be encountered, particularly to the flexor side of the wrists, to the elbows, and possibly also in the groin area. All injury localizations need to be checked for the ease with which they could be accessed by the victim himself or herself when holding the instrument in his or her own hand. Incised wounds with suicidal intent are typically not found on the side of the body of the dominant hand, i.e., not ipsilaterally but rather contralaterally. In particular, incised wounds to wrists and elbows run more frequently in a transverse direction than in the rarer longitudinal direction. Suicidal incised wounds to the neck by right-handed individuals are usually to the left side, running from superior left to inferior right. Nevertheless, a comparison always needs to be made with the pattern of bloodstains found at the scene of death, on the body itself, and on clothing.

Homicidal incised wounds to the neck, if inflicted from behind, often involve the entire anterior side of the neck and are relatively deep. The wound usually commences at a relatively high point, sloping slightly downwards in its further course and ending at a lower point on the neck.

In the case of death caused by stab wounds, diffusely distributed stab wounds are found particularly when resistance has been shown, often to body regions inaccessible to the victim's own hands, such as the back, and combined with defense wounds. Multiple stab wounds, in some cases exceeding 100, permit inferences to be made about the psychological status of the perpetrator; thus, the presence of psychiatric disease in a suspect needs to be considered in any evaluation of a case (Fig. 9.22).

Table 9.2 Differentiation between suicide and homicide in sharp and semi-sharp force trauma

Suicide	Homicide
Clothing rarely involved, stab wound site often exposed	Clothing possibly penetrated or torn
Localized stab wounds over the left anterior side or heart region (rarely neck, head, or solar plexus)	Irregularly distributed stab wounds with no predilection for side
Limited number of deep stab wounds (usually less than ten, but multiple superficial stab wounds possible)	Multiple stab wounds more frequent
Wound track possibly runs downwards, horizontally, and upwards	Wound track generally runs slightly or distinctly upwards
No concomitant injuries	Active and passive defense wounds common in sharp and semi-sharp force trauma (hands, forearms)
Tentative wounds, primarily to the flexor side of the wrists, but possibly also to the neck	No tentative wounds
Stab/incised wounds only to body regions accessible with own hands	Stab/incised wounds also to body regions not easily accessible with the victim's own hands
Injuries generally not to the body side of the dominant hand but rather to the contralateral body side	Injuries also to the body side of the dominant hand
Regular distribution of bloodstain pattern on body	Diffuse, irregular, and poorly defined bloodstain pattern on body
Intensity of injury is overall uniform, crescent-shaped and horizontal incisions absent	Injuries of varying intensity, including deep incised wounds to the neck generally following a horizontal/crescentic course
Sparing of body regions particularly sensitive to pain, e.g., nipples	Body regions particularly sensitive to pain may also be involved
Stab/incised wounds to the neck show no injury to deeper structures (thyroid gland, trachea, larynx)	Deep stab/incised wounds to the neck involving the thyroid gland, trachea, and larynx
No signs of physical conflict at the scene where the body is found or at the scene of death	Signs of physical conflict at the scene where the body is found or at the scene of death
In some cases, further suicidal actions (hairdryer in the bath, ingestion of tablets, etc.)	No actions suggestive of suicide
Motive for suicide, possible suicide note	No motive for suicide, no suicide note
Instrument used generally found with the body	Instrument used may be absent or located at some distance from the body

If semi-sharp force trauma has been used for the purposes of self-mutilation (see Chap. 17), wound edges, such as those of an amputated body part, should undergo careful examination. Moreover, taking tissue samples is recommended to demonstrate whether, for example, a local anesthetic was used.

9.7.3 Ability to Act Following Pointed, Sharp, and Semi-sharp Force Trauma

In this context, "ability to act" describes those actions consciously performed by the victim following injury due to sharp and/or semi-sharp force trauma; these do not include reflex actions and semiautomatic behavior such as self-defense movements.

The phase during which a victim is capable of acting prior to death can be short or relatively long depending on the site and type of injury. Before any conclusions can be drawn, all findings must be considered as a whole, particularly autopsy results and the extent of blood loss. Thus, in the case of a stab wound to the heart involving transection of a coronary artery or of major neck vessels on both sides, one must assume a short period of time in which the victim was able to act. However, the victim's ability to act in the case of intercostal artery injury with hemorrhage into the chest cavity could be longer, while ability to act may be maintained for a considerable length of time in the presence of an abdominal

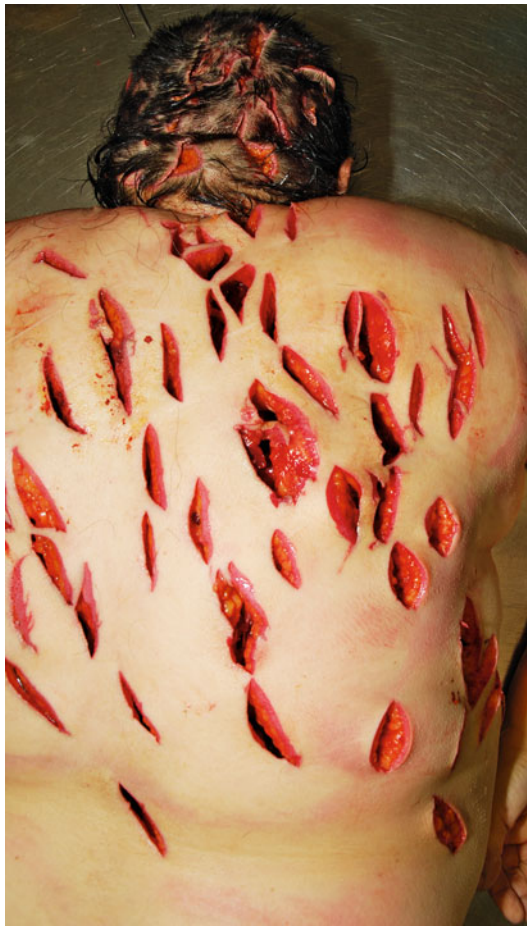


Fig. 9.22 Multiple irregularly distributed deep stab wounds to the head and back: homicide by a perpetrator suffering from paranoid psychosis

stab wound causing no injury to major blood vessels. It is quite plausible, therefore, for a suicide victim to dispose of or conceal a used knife and to perform other purposeful actions.

9.7.4 Antemortem vs. Postmortem Injuries in Pointed, Sharp, and Semi-sharp Force Trauma

Wounds with hematomas and injury that has clearly led to internal or external blood loss can be classified unequivocally as antemortem injuries. In the case of multiple stab/incised wounds, it is not always possible to state conclusively for some injuries whether or not the victim was still alive at the time the injuries were inflicted. One should consider the possibility that the victim died of exsanguination during the course of injury infliction and that the final stab and incised wounds were inflicted on a dead body. The absence of hematoma at wound margins, as well as the absence of any histological criteria for antemortem injury in particular, suggests only a very short survival time or postmortem injury to the body, e.g., during dismemberment for the purposes of transportation or to conceal body parts. Rarely, dismemberment is carried out in the context of some form of perverse libido development or is accompanied by feelings of hate and revenge. More commonly, a perpetrator attempts to make the identification of a body impossible by dismembering it and dispersing the individual body parts.

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Gunshot and Blast Wounds

10

Case Study

A police officer was found on his bed in a half-sitting position. His duty pistol was also found on the bed between his legs. A gaping, plate-sized wound could be seen in the right parietal region, a smaller wound approximately 2 cm in diameter in the left. A large volume of cerebral matter had exited the cranium. To the right of the body, fragments of cerebral tissue and small fragments of the cranium were on the ground and partially on the wall up to a distance of around 4 m. A case of suicide involving a single shot entering the left side of the head and exiting on the right side appeared the most likely scenario. However, a police investigation of the domicile was unable to locate and retrieve the projectile.

A forensic autopsy was ordered at which it became apparent that the smaller gunshot wound in the left parietal region could be approximated and showed no central defect or other signs of gunshot entrance. After shaving the hair in the right temporal region at the anterior margin of the larger wound, a muzzle imprint from the gun could be seen in the skin. On dissection, cone-shaped beveling in the bony defect from the inner to the outer table of the left temporal bone was interpreted as a sign of gunshot exit. In the light of autopsy results, the firing direction was amended: entrance gunshot wound to the right temporal region, exit gunshot wound to the left parietal region. With this information, a renewed search of the domicile was undertaken, and the projectile could be located to the left of the body. The projectile was from a cartridge used in police deployment (quick-defense bullet), which was typically deformed.

The particular wound morphology observed in this case was attributed to the use—and misuse with suicidal intent—of the cartridge intended for police deployment: the considerable level of destruction at the gunshot entry site caused the rapid buildup of intracranial pressure to be released via the gunshot entry wound, resulting in extreme "backspatter."

Gunshot wounds are generally treated as an injury entity in their own right; however, it would not be unreasonable to classify them as a special form of blunt trauma. In the present work, the authors propose an alternative classification as "puncture trauma": a bullet or similar object having a minimal surface area but travelling at great speed comes into contact with a body. A similar mechanism to that of a projectile is seen with bird beaks, arrows, or spears. Thus, injuries due to arrows (bow or crossbow), spears, and bird beaks can be classified as "puncture trauma." Bird-beak injuries most commonly affect the scalp and cranial vault in hikers or joggers who inadvertently approach the nests of birds of prey, such as buzzards.

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The effect of a gunshot depends on the energy output and the radial acceleration of tissue. Non-projectile-related tissue injury is additionally caused by blast waves, various particles from the propelling charge, and displaced tissue, particularly bony fragments. Thus, it is reasonable to assume that alarm guns can cause life-threatening injury.

10.1 Firearm Laws

In most countries, the purchase and possession of firearms are subject to obtaining a permit. In Germany, the Weapons Act (*Waffengesetz*, WaffG) regulates the acquisition, storage, handling, maintenance, and trade of weapons. Permission to own a weapon is granted with a weapon owner's license, while permission to carry a weapon is granted in the form of a weapons license in accordance with Art. 10, Para. 4 of the Weapons Act. "Carrying a weapon" in the sense of the Weapons Act refers to having a weapon ready for use, which is permitted only in exceptional cases and certain places. Applicants aged under 25 years need to support their application for a weapon owner's license with a medico-psychological expert appraisal; marksmen who practice sport shooting may possess large-caliber weapons and pistols only from the age of 21 years. Special transport regulations apply to hunting weapons.

However, not only firearms are considered as weapons: Art. 1, Paras. 2 and 3 of the German Weapons Act states the following on the concept of weapons:

> (2) Weapons shall mean
> 1. Guns or equivalent objects and
> 2. Portable objects
> a) which are by nature intended to remove or reduce a human's ability to attack or defend, in particular cutting weapons and thrust weapons;
> b) which, due to their properties, method of operation or how they work, are able to remove or reduce a human's ability to attack or defend, even if not intended for that purpose, and are referred to in this Act
> (3) Handling a weapon or ammunition shall refer to anyone who acquires, possesses, hands over to others, carries, transfers, shoots, manufactures, works on, repairs or trades in weapons or ammunition

According to the Firearms Protocol of the United Nations, which has led the way for weapons laws in numerous countries, it is not permitted to carry cutting and thrusting weapons, knives with fixed blades that can be opened with one hand (folding or pocket knives), or knives with a blade exceeding a length of 12 cm. Carrying a dummy gun in public in Germany is, with exceptions, forbidden, as are Taser guns (long-range electroshock devices). In the wake of several shooting attacks in Germany, the German authorities are now empowered to carry out unannounced inspections on the private premises of weapons owners.

10.2 Types of Firearms and Ammunition

In forensic practice, gunshot wounds are seen in cases of suicide, homicide, and accidents associated with the handling of firearms; injuries are occasionally also seen in the context of examining survivors of gunshot injury. The type of gunshot wound or pattern of evidence is determined by the type of weapon used. Table 10.1 provides a systematic overview of firearm types.

Rifled Barrel. Only shotguns are smooth-barreled. All other firearms produced today have a rifled barrel. Rifling describes spiral, parallel groves along the entire length of the barrel; this rifling imparts a spin to the bullet, thereby stabilizing its trajectory. The surfaces protruding between the grooves are referred to as "fields." The fact that the projectile takes on a highly individual notch pattern, making it possible to attribute it to a particular weapon, is of forensic relevance.

Cartridge Composition. Modern cartridges comprise:

- A case
- The propellant (powder charge)
- A primer
- A bullet
- A cartridge designation

A cartridge's name is made up of its caliber in millimeters (mm) or inches and its case length, perhaps also some additional designation.

Table 10.1 A classification of selected firearms

Types	Subtypes	Particular features
Handgun	Revolver	Essentially single-action weapons: the hammer strikes the firing pin when the trigger is pulled; the hammer needs to be cocked manually before each shot
		Single-action revolver: hammer can only be cocked manually
		Double-action revolver: hammer cocking can also occur when the trigger is pulled; the cylinder turns when the hammer is cocked, either to the left or to the right, depending on the type of weapon; can fire 5–9 shots; empty cartridges remain in the cylinder after discharge
	Pistol	Today nearly all multishot self-loaders: once discharged, recoil of the slide causes ejection of empty cartridges and loading of new cartridges. Manual pullback of slide to load the first cartridge before first shot. Cartridges are contained in a magazine in the butt. Lockable slides are required for pistols with greater muzzle impulses. Single-action pistols are used as sports guns
	Submachine gun	Automatic weapon uses a variety of pistol ammunition. Simple construction (open-bolt blowback operation). Often not adapted to single-shot action. Most famous model: Uzi. Capacity of 30–50 shots depending on the magazine, drum magazine gives up to 100 shots
Long gun (rifle)	Sporting gun	For firing individual bullets, rifled barrel. Hunting rifles are primarily single-shot.
		Military rifles are usually automatic weapons (assault rifles) with gas-operated reloading. Magazine holds 20–30 shots. Single-action, bolt action, and often adjustable to have a burst of fire limited to three shots. Best known assault rifle worldwide: the Russian AK-47 (Kalashnikov)
		Machine guns: designed for rapid-succession firing of rounds, with ammunition usually fed from a belt, up to 250 shots per belt. Heavy, stable mount needed
	Shotgun	Single-shot smoothbore firearm for firing shot. Almost exclusively used as a hunting gun
		Double shotgun: double bore. Combination with a rifle barrel is possible
		Rifle and shotgun barrels: side-by-side = cape guns; over-and-under = combination guns
		Three-barreled guns are referred to as drillings, with common combinations being: two shotgun barrels side by side above one rifle barrel
Blank firing pistols		Replicas of real revolvers or pistols with the same principle of functioning. The barrel mock-up, which must be firmly attached to the frame, is fitted with carbide metal baffles. Shoot blank shells

Numerous special weapons and historical firearms, although relevant in individual cases, are not listed here

Caliber. Caliber is not measured as the outer diameter of a bullet, but as the diameter of a weapon's barrel. The inner diameter of the barrel is determined by its fields. Table 10.2 shows a selection of commonly used cartridges and their dimensions.

Blank Cartridges. Often referred to as "blanks," blank cartridges are essentially built much like normal cartridges, only without a bullet. Instead, the end of the case is either sealed with a plastic plug or crimped.

Blank cartridges are used primarily in blank firing pistols but can in principle be used in any weapon with an appropriate caliber. In addition, cartridges with large propellant charges (mostly 9-mm caliber) are used in nail guns. Blank cartridges are also used in military rifles and machine guns in the context of military maneuvers.

While real blanks are loaded with only propellant, there are also cartridges that contain irritants in powder form; typical examples of such irritants would be CS gas (2-chlorobenzalmalononitrile) or capsaicin (from red chilli peppers and the active component in pepper sprays).

Types of Bullet. A distinction is made between bullets made entirely of the same material, such as lead, and jacketed bullets, which have a uniform core and a case made of a thin layer of another material, e.g., a lead core with a steel or copper jacket. Partially jacketed bullets, which

Table 10.2 The weight, velocity, and kinetic energy of selected projectiles

Cartridge	Mass (g)	Muzzle velocity (m/s)	Kinetic energy (J)
Handguns			
.32 ACP	4.6	276	175
7.62×25	5.6	424	496
9×18 Makarov	6.2	323	321
9-mm Parabellum	8	393	630
.40 S & W	10	347	606
.357 Magnum	10.2	376	725
.47 Magnum	15.6	411	1.320
Small arms			
5.45×39	3.4	910	1.430
.243	6.5	902	2.640
7.62 × 39	8	701	1.970
.270	8.4	933	3.660
7.62×51 (.308)	9.7	838	3.420

Table 10.3 A classification of bullets according to how they behave in the body

Bullet type	Behavior in the body
Nondeforming	Retain shape after penetrating the body, lower energy output, through-and-through wounds more common
Deforming	On penetrating the body, the surface area transferring energy is increased; no loss of material
Fragmenting	Fragment on penetrating the target, high energy output, extensive tissue destruction, so-called dumdum bullets

break up on entering the body and cause particularly extensive injuries, are commonly used for hunting. Bullets can be classified according to how they behave in the body (Table 10.3).

The various types of ammunition were, and still are, developed for widely varying requirements:

Hunting ammunition should kill as quickly as possible, while the primary objective of combat ammunition is to injure, necessitating that one or two other soldiers tend the injured soldier, temporarily making them also unable to fight. Sniper ammunition needs to kill fast and effectively, ideally without causing injury to nearby individuals. Police authorities increasingly use quick-defense ammunition, or "manstoppers," in duty weapons: the actual tip of the full-jacketed hollow-point bullet is made out of a plastic. The bullet expands when it penetrates tissue, resulting in less penetration depth and greater energy output, thereby stopping the subject while ideally causing as little injury as possible.

Shot Shells. Shot shell cases are generally plastic; only the tip containing the primer is made of metal. Then comes the propellant, which is separated from the shot pellets by a wad in order to prevent these elements from mixing. In modern shot shells, this wad has been replaced by a plastic "cup" containing the shot pellets and whose high, hollow base ensures the appropriate distance from the propellant. Shot pellet diameters vary according to the game being hunted. The shot pellets begin to "scatter" after leaving the barrel of the shotgun.

The Primer. The formerly widely used Sinoxid (main components, lead tricinate and barium nitrate) have largely been replaced today by lead-free primers.

The Propellant. The propellant imparts energy for the shot. The oldest known explosive to be used as a propellant is gunpowder (75 % potassium nitrate, 15 % charcoal, 10 % sulfur; produced in powder mills). Nitrocellulose (guncotton) has become prevalent in modern ammunition; this propellant is produced by nitrating cellulose with a nitrating agent (a mixture of concentrated salpetric acid and concentrated sulfuric acid). In di- or polybasic powders, an additional propellant in the form of nitroglycerine is added; burning produces CO_2, CO, H_2O, H_2, and N_2. An equivalent amount of nitrocellulose powder compared to gunpowder produces an approximately threefold greater volume of gas and explosive power.

10.3 Entrance Gunshot Wounds

When a projectile strikes the skin, tissue is carried in the direction of fire. In addition, radial acceleration is caused by the spin of the bullet. A limit velocity of approximately 50 m/s is needed to penetrate the skin.

Prerequisite signs of a gunshot entrance wound include a central tissue defect (wound edges cannot be approximated) and an abrasion ring. In addition, a bullet wipe mark between the central defect and the abrasion ring, as well as a contusion ring around the abrasion ring, may form (Fig. 10.1).

Abrasion Ring. It is assumed that the abrasion ring is produced by temporary depression of the skin and abrasion in the direction of fire. A bullet striking the skin at an orthogonal angle produces an abrasion ring of about 1–2 mm in width.

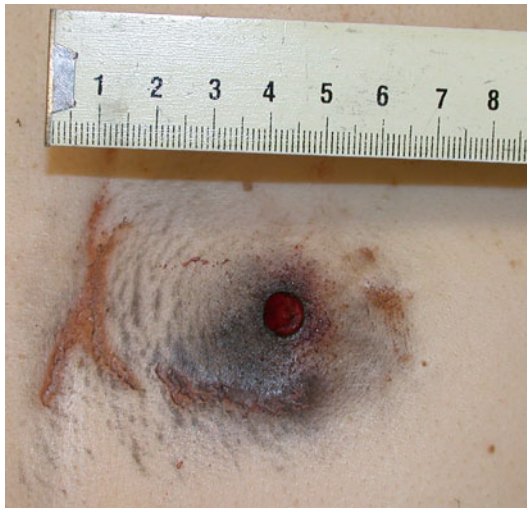

Fig. 10.1 Gunshot entrance wound. Signs of a gunshot entry wound, defect cannot be approximated, mild abrasion ring, absent bullet wipe (clothing!), and a contusion ring

Bullet Wipe. Oil residues on the bullet are deposited at the margin of the central entrance gunshot wound where they form a black ring. Thus, the extent of bullet wipe, or indeed whether this is visible at all, depends on the amount of dirt in the barrel. If a bullet first passes through clothing, bullet wipe may be visible on the outermost layer of clothing rather than on the gunshot entry wound.

Contusion Ring. A contusion ring develops on the skin in response to the temporary wound cavity. It lies adjacent to the abrasion ring and is bluish red in color, fading on the periphery.

Gunshot Entrance Wound to the Skull. A gunshot entrance wound in the skull region produces a round defect in the outer table with cone-shaped beveling through the diploe widening out to the inner table. Corresponding outward beveling from the inner to outer table can be seen at the gunshot exit point (Fig. 10.2).

Bullet Track and Trajectory. Identifying the bullet track and determining a bullet's trajectory is of major relevance in forensic practice; together with the firing distance (see Sect. 10.5), these provide an important basis on which to reconstruct the angle of fire as well as the firing position. A number of types of shot are described according to the bullet's final localization and the bullet track, as well as other possible characterizing features (Table 10.4).

Temporary Wound Cavity. A temporary wound cavity in the bullet track is formed as

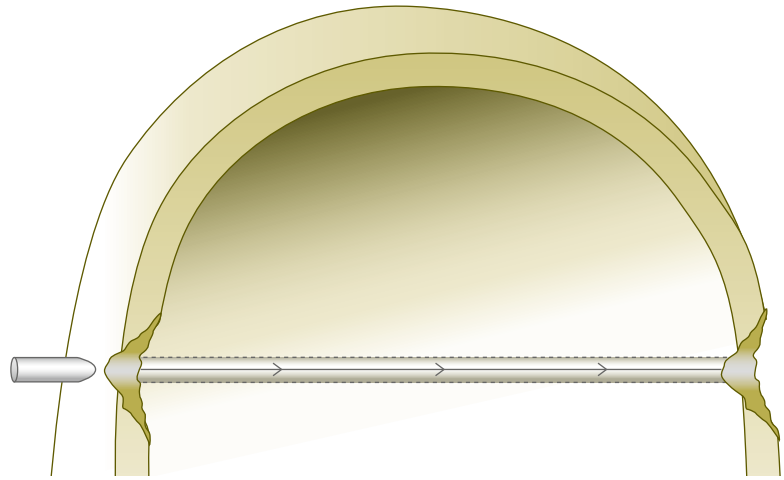

Fig. 10.2 Morphology of gunshot entrance and exit wounds to the skull

Table 10.4 Types of gunshot

Gunshot type	Characteristics
Penetrating	The projectile remains within the body. It can occasionally be felt subcutaneously opposite the entrance site. Possible reasons: bullet has low penetrating force, oblique angle of contact, deceleration due to bone
Perforating (through-and-through)	The projectile has exited the body and can be found externally. In the case of pistol ammunition, usually a of 7.65-mm caliber
Internal ricochet	The bullet changes direction within the body: deflected by tissue of varying densities, e.g., bone
Graze	The projectile grazes the skin. Trough-like skin abrasion, possibly with subcutaneous tissue, additional small oblique radial tears in the skin running in the direction of fire, sometimes difficult to distinguish from a laceration
Tangential	Entrance and exit wounds are located in close proximity. The bullet track travels through skin, subcutaneous tissue, and possibly also deeper soft tissue layers. A distinct oval abrasion ring at the entrance wound oriented away from the direction of fire
Contour	Skull: projectile has only minimal kinetic energy. Inner table contour: projectile follows the contour of the inner table opposite the entrance wound Outer table: projectile penetrates the skull on the opposite side of the entrance wound but not the scalp—projectile moves between the outer table and the scalp
Ricochet	The bullet is deflected from its trajectory by objects it comes in contact with. May cause fatal injury although the shot was actually discharged in another direction, e.g., warning shot. The modified trajectory of the shot (including loss of spin) can produce atypical entrance gunshot wounds lacking: signs of gunshot entrance wound and margins that can be approximated
"Krönlein" shot	Gunshot wound to the skull with a high-velocity bullet. The skull is shattered by the temporary wound cavity with complete evisceration of the brain

kinetic energy is transferred to the contact surface, whereby radial acceleration occurs and deforms the medium in either an elastic or a plastic manner. Both a cavity and a vacuum are thus created behind the bullet. Together, the vacuum and the elastic energy stored in the medium (tissue) cause the temporary cavity to collapse; at high velocities, this can cause organ damage. Since the skull can withstand only minimal expansion, pressure is only able to escape at the entrance or exit gunshot wounds; this process can also involve tissue. Since the entrance wound represents the initial opening, it serves as the primary point of pressure release, producing backspatter, i.e., traces of tissue ejected in the opposite direction to the direction of fire (Fig. 10.3).

Backspatter. The victim's tissue is ejected via the entrance wound in the opposite direction to the direction of fire and, in the case of close-range fire, may be spattered on the firing hand.

High-velocity bullets can cause the skull to shatter, in extreme cases causing complete evisceration of the brain (Fig. 10.4).

Gunshot Wounds to Bone. Due to the expansion of bone tissue and its subsequent collapse, bony defects in the case of perforating gunshot wounds may be smaller than the caliber of the projectile (usually high velocity). On the other hand, if the temporary wound cavity expands or the projectile's trajectory is disrupted, defects may have a larger diameter than the outer diameter of the bullet.

Important: Even in the presence of a circular defect on the outer table in the case of an entrance gunshot wound to the skull, no inferences can be made in relation to the caliber of the weapon used; the actual caliber may be greater or smaller.

10.4 Exit Gunshot Wounds

As a bullet exits, the skin is stretched by the temporary wound cavity and the projectile to the point of rupture; thus, a real "rupture wound" would be seen. Although exit wounds are commonly larger than entrance wounds, this is not always the case.

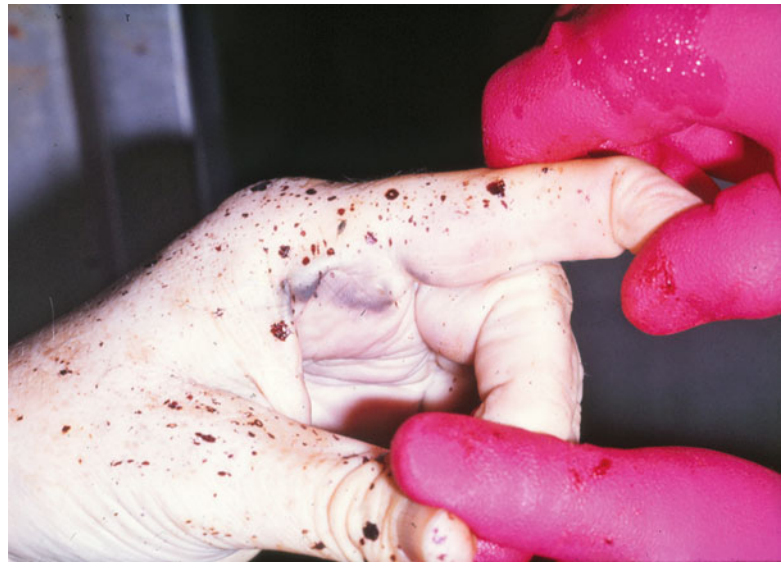

Fig. 10.3 Backspatter. Tissue fragments have been ejected onto the firing hand; injury to the flexor side of the base of the index finger (gun-slide wound) can also be seen

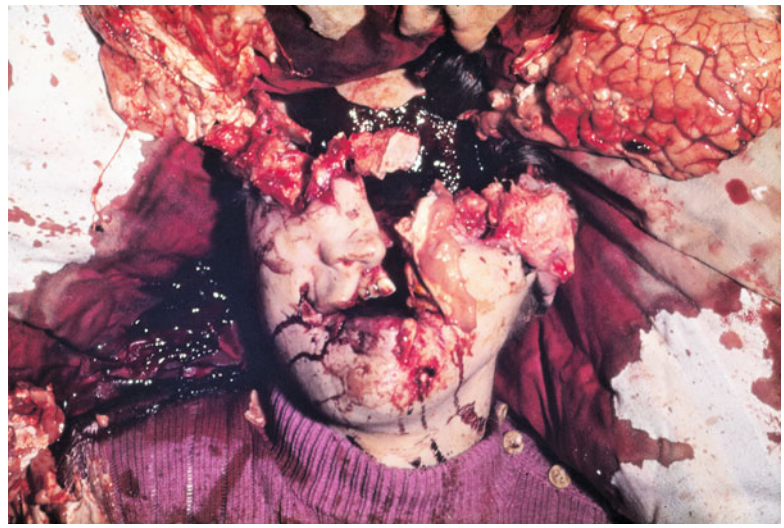

Fig. 10.4 "Krönlein" shot. Complete evisceration of the brain

Important: Gunshot exit wounds are characterized largely by wound margins that can be approximated, as well as the absence of an abrasion ring or other signs of gunshot entrance (Fig. 10.5).

In the case of a burst fracture due to skull rupture in through-and-through gunshot wounds to the head, it may prove challenging to differentiate the actual exit gunshot wound from other skin lacerations in the scalp and facial skin.

Important: Particularly when bone fragments are carried in the direction of fire, fragments of tissue may be ejected through the gunshot exit wound, producing an irregular, generally noncentral tissue defect.

Pseudo Abrasion Ring (Shoring). Close-fitting clothes at the point of gunshot exit may act as an abutment, producing skin abrasions which, on drying, are remarkable. This lesion is susceptible to misinterpretation as an abrasion ring.

Exit Gunshot Wound in Bone. A perforating, or through-and-through, gunshot wound to the skull produces cone-like beveling from the inner table outwards to the outer table. In the

case of a burst skull, this beveling can be reconstructed by reassembling the bone fragments (Fig. 10.6).

Perforating diaphyseal gunshot wounds to long bones near the longitudinal axis can produce a similar picture to that of skull wounds, with beveling in the direction of fire.

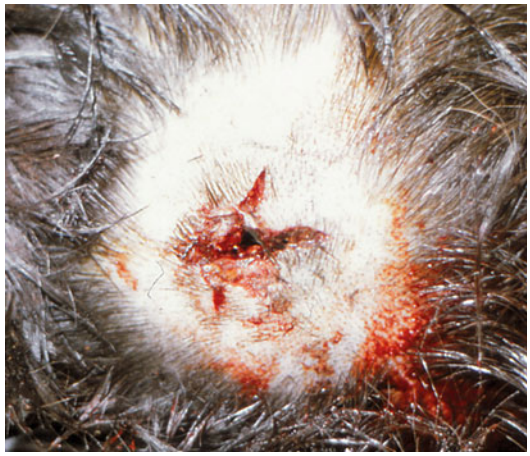

Fig. 10.5 Gunshot exit wound in the upper crown region. Hair in this area has been shaved. Wound margins can be approximated

10.5 Range of Fire

For the purposes of estimating range of fire, morphological wound characteristics are classified into three range of fire categories (Table 10.5):

- Close range (contact wound) (Fig. 10.7)
- Intermediate range
- Distant range

An absolute determination of the range of fire can only be carried out by means of chemical or spectrographic analysis of soot deposition and its density. To this end, an approximately 10 × 10-cm sample of skin with the central defect located in the middle should be taken (stretched over card or cork, marked for orientation, or possibly frozen). If clothing was worn over the entrance gunshot wound, soot deposition can be expected here and clothing should be collected as evidence.

Important: To ensure the reliability of range of fire determination using soot deposition on skin samples or areas of clothing, decedents suspected of having sustained gunshot wounds should not come in contact with water.

Soot deposition found at the margins of an entrance gunshot wound to bone indicates a close-range shot (Fig. 10.8).

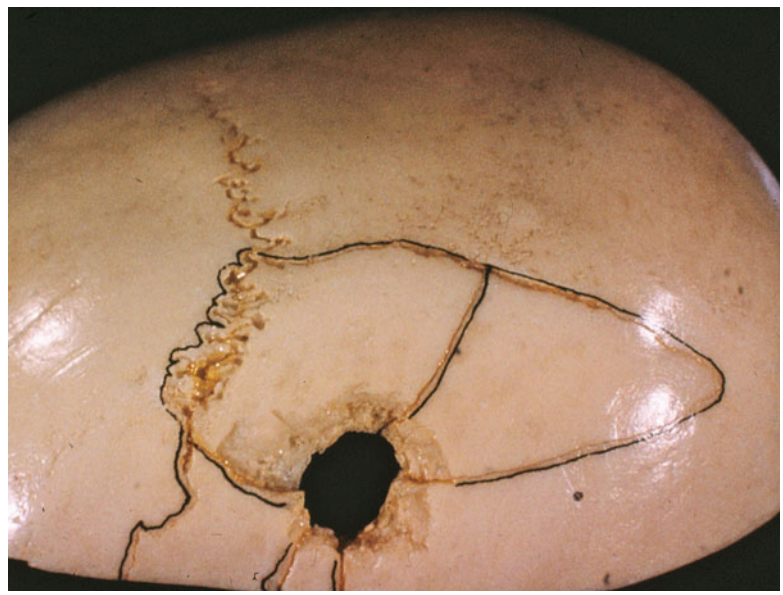

Fig. 10.6 Exit gunshot wound with outward beveling—outer table detachment—and adjacent fractures

Range of fire determinations in Germany are predominantly carried out in police forensic departments. On an international level, many forensic institutes have ballistics departments where investigations of this kind, as well as important research, are carried out. Visualizing traces of soot is made possible using chemical indicators. It is sometimes necessary to fire several test shots against various surfaces and at varying ranges, either with the actual weapon used in the incident under investigation or at least an equivalent weapon, in order to interpret results reliably.

Table 10.5 Categories of range of fire

Category	Morphological criteria at the gunshot entrance site
Close-range (contact) shot (Fig. 10.7)	Soot deposition in and around the wound track, found on the outer table in skull wounds or possibly subperiosteal or on the dura. Stellate laceration in the skin is possible. If a weapon has been pressed tightly against the skin on discharge, an imprint of the muzzle may be visible. CO–myoglobin binding causes salmon-red discoloration of muscles along the bullet track, particularly in the initial segment
Intermediate-range shot	Soot deposition and gunshot residue around the central defect, macroscopically detectable when the muzzle-to-victim distance does not exceed roughly twice the barrel length of the weapon used
Distant-range shot	Signs of close-range fire are absent

The criteria mentioned here are supplementary to signs of gunshot entrance wounds

At the moment of discharge, soot may be deposited to varying degrees on the skin of the firing hand. By comparing hands, typical zones of greater soot deposition can be seen (Fig. 10.9).

Entrance gunshot wound morphology and accompanying findings in relation to the range of fire are shown in Fig. 10.10.

10.6 Special Gunshot Wounds

Special gunshot wounds include wounds not caused by a single projectile fired from a long gun or handgun.

Shotgun Shell Wounds. The shot pellets leave the shotgun barrel together with the plastic "cup" or wad. The pellets begin to disperse beyond the muzzle; the further the pellets move away from the muzzle of the shotgun, the more dispersed the pattern of stippling defects on the skin becomes. At very close range, the plastic cup may perforate the skin, while the still tightly packed shot acts like a

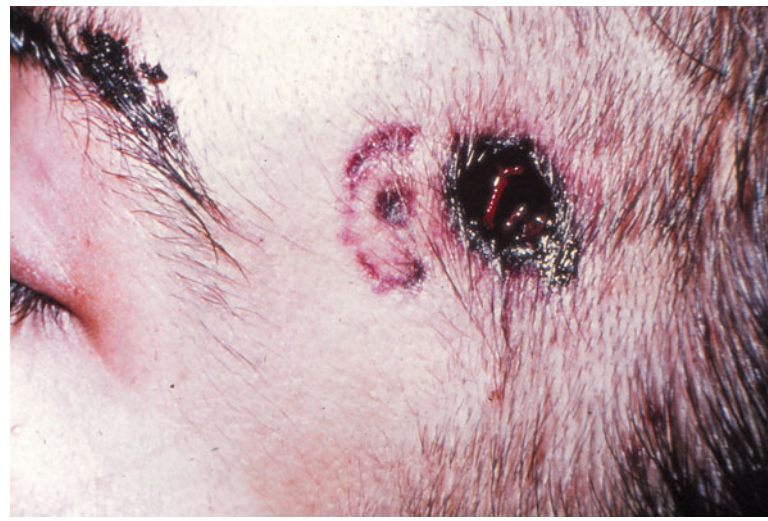

Fig. 10.7 Contact gunshot wound with muzzle imprint and drying in the area of gas escape

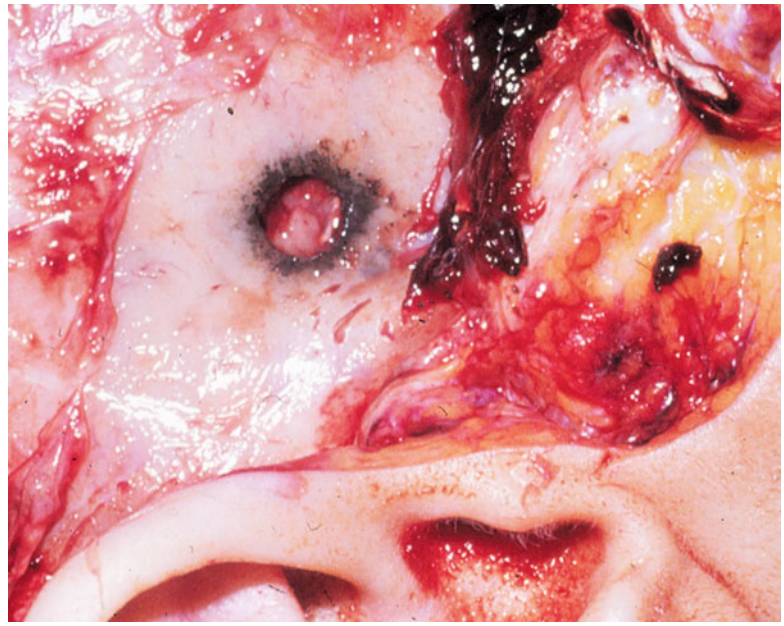

Fig. 10.8 Soot deposition around an entrance gunshot wound to the skull

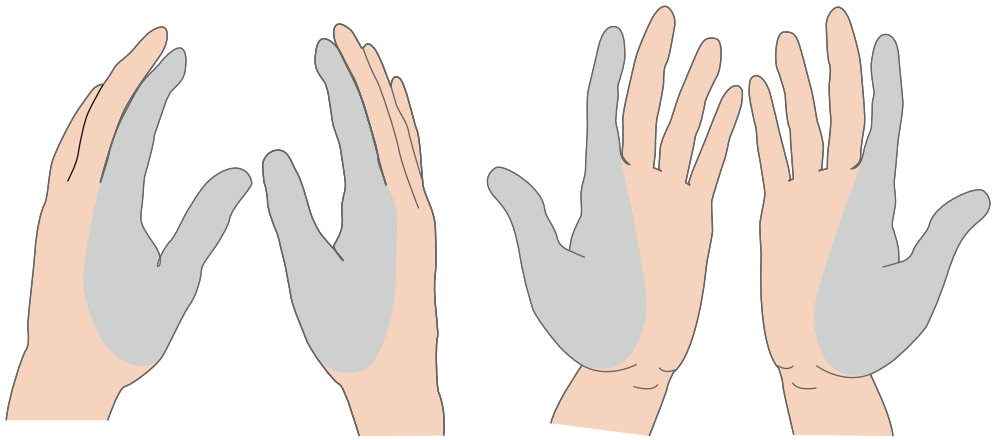

Fig. 10.9 Soot deposition on hands in the determination of the firing hand

single slug. The shot pellets then disperse inside the body according to the so-called billiard effect. In the case of close-range contact wounds from shot ammunition, the expanding muzzle gases are of particular relevance. Depending on localization, soot deposition within the bullet track, internal organ rupture, or skull rupture may result.

Entrance gunshot wounds caused by shotgun shells usually demonstrate a central defect larger than the caliber of the shotgun used, as well as irregular margins (Fig. 10.11). With increasing distance, separate small defects caused by shot around the margins can be seen (Fig. 10.12). Shotgun firing distances in excess of 5 m no longer produce a central defect, but rather extensive stippling defects. The abundant shot pellets can be well visualized using X-ray (Fig. 10.13).

Bolt Gun Wounds. Bolt guns were developed as tools for animal slaughter. A powerful propelling charge is achieved by using a blank cartridge. This drives a metal bolt into the animal's skull. Generally, next to the bolt outlet, there are two opposing openings for the release of excess gas pressure and soot. This constellation produces the typical external wound pattern comprising a central defect and two adjacent rings of soot. A bolt gun pressed against the skull will

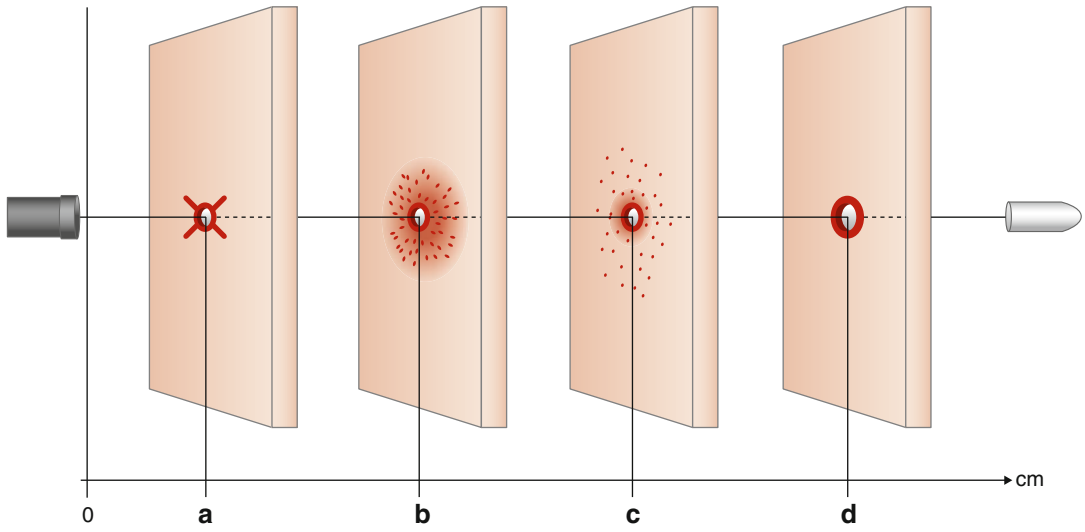

Fig. 10.10 Entrance gunshot wound findings at varying firing ranges. (**a**) Close-range shot. (**b**) Intermediate-range shot fired from a short distance with soot deposition around the entrance wound. (**c**) Intermediate-range shot from a somewhat greater distance with a larger radius of gunshot residue. (**d**) Distant-range shot: no signs of a close-range gunshot entrance wound, bullet wipe, abrasion ring, or contusion ring

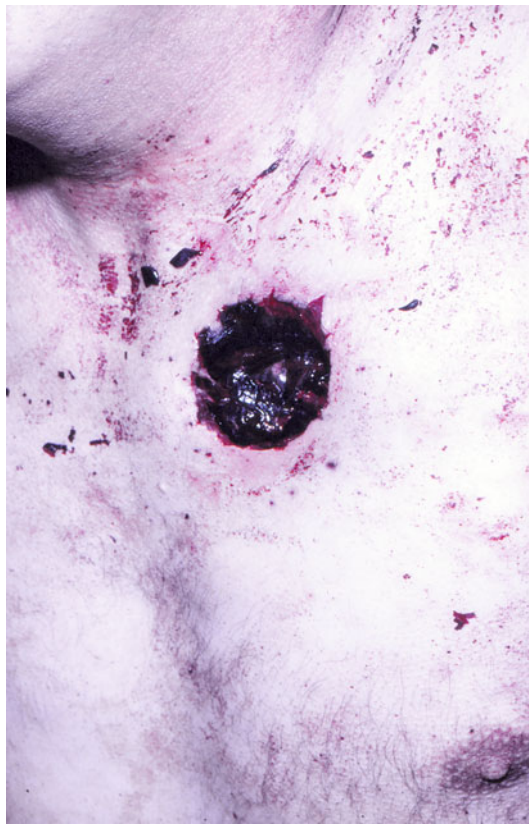

Fig. 10.11 Intermediate-range shotgun shot wound. The central defect is greater than the caliber of the shotgun used and has irregular edges

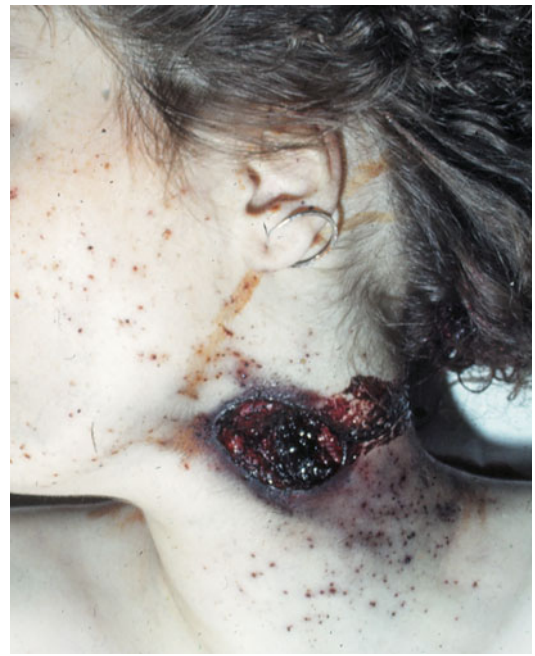

Fig. 10.12 Shotgun shot wound. A large central defect surrounded by smaller shotgun shot wounds in the skin

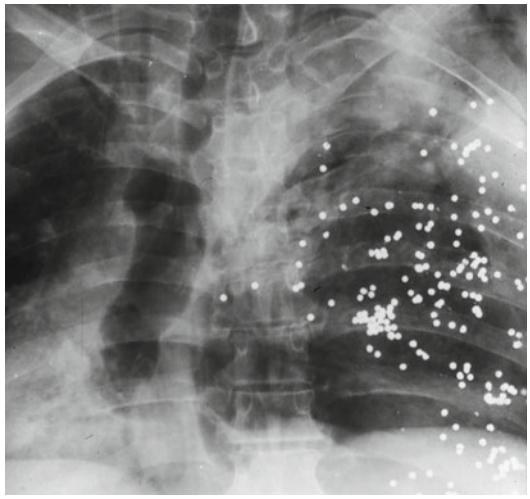

Fig. 10.13 Multiple shot pellets seen on chest X-ray

punch out the scalp and bone, which can then be found embedded in brain tissue at the end of the wound track.

In forensic practice, bolt guns are primarily relevant as a means of committing suicide, being rarely used in homicides. Their use is seen predominantly among occupational groups with access to this type of device, such as butchers and farmers.

Wounds Caused by Blank Firing Pistols. Depending on the type of blank used, the gas blast can produce significant energy flux density. Close- or intermediate-range shots fired from a distance of a few centimeters can cause skin penetration and even internal organ injury. Thin bones may break. Placing the weapon to the head is extremely hazardous, particularly in the area of the temporal bone, which can rupture. Expanding gases and disseminated bone fragments can cause significant brain injury.

Intermediate-range shots striking unclothed skin can cause particles of gunshot residue to scatter, making medical care necessary not solely for cosmetic purposes.

Air Gun Wounds. Air guns (air rifles, air pistols) use mechanically compressed air as a propellant. Individual Diabolo pellets that are inserted at the near end of the barrel are usually used; these pellets are available with a flat, rounded, or conical tip. Depending on the pellet used, the firing range, the point of contact (clothed or unclothed skin), as well as the angle of contact, hematomas, or even skin penetration are seen. Penetration of the orbital cavity or the thin temporal bone can cause fatal brain injury, while other life-threatening injuries may be caused by arrosion of major vessels in the neck area.

Arrow Shot Wounds. Arrows are fired with bows or crossbows by means of a pre-drawn bowstring. These devices are either historic weapons or modern sports devices. Mention should also be made of harpoons, which have an inflatable compressed air cylinder. The harpoon is loaded through the muzzle against the pressure of the compressed air. Modern steel-tipped arrows can be subdivided into two types:

Field Tips. These have a conical blunt shape similar to a truncated cone or round-nose bullet and are used for target shooting in a sporting context. They produce injuries consistent with puncture trauma.

Broadheads. These have two or more razor-like metal blades extending radially from a central shaft, which taper at the tip to form a point. These tips are designed for hunting. Arrows fitted with tips of this kind penetrate the skin with a splitting-like penetration mechanism, producing smooth and often slit-like or possibly radiating linear wound morphology. Rather than being displaced, movable structures such as blood vessels or intestinal loops are cut clean through.

Blast Injuries. These kinds of injuries are typically seen in combat situations or terrorist attacks. The effects of an explosion are categorized into primary, secondary, tertiary, and quaternary injuries (Table 10.6). Explosions produce multiple missile fragments of varying size, as well as a considerable blast wave. Multiple and extensive skin injuries varying in size and form according to the victim's distance from the site of the explosion are characteristic (Fig. 10.14). Thus, multiple missile fragments are often retrieved at autopsy, while internal organs show multiple lacerations and tears; pulmonary lacerations and tears may be caused by the blast wave as well as lacerations of the arterial intima (Fig. 10.15). Amputated extremities and decapitations are also seen.

Table 10.6 Classification of blast injuries

Type	Mechanism of effect	Injury pattern
Primary	Shockwave and overpressure wave: barotrauma	Burst eardrums, pulmonary contusion, gastrointestinal contusion or perforation, mesenteric laceration, liver and spleen rupture
Secondary	Projectile or projectile-like: fragments propelled out of the bomb case or generated in the immediate vicinity	Soft tissue injuries, penetrating injuries with blood loss, pneumothorax, intestinal perforation
Tertiary	Indirect effects of the blast wave: falls, impact trauma, falling or collapsing parts of buildings	Many varied types, particularly blunt, penetrating, and perforating trauma
Quaternary	Miscellaneous: accidental burial, flames, hot flue gases, radioactive elements, biological or chemical toxins	Traumatic amputation, compartment syndrome, crush injuries, burns, inhalation trauma, smoke inhalation, radioactive contamination, biological or chemical intoxication

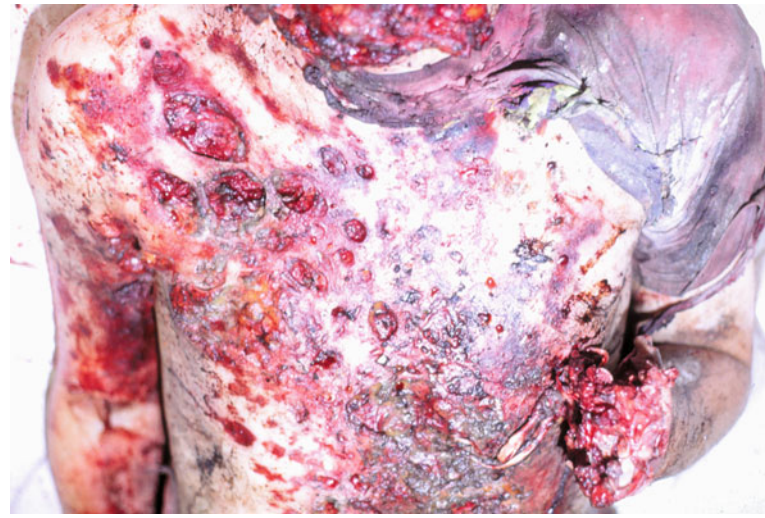

Fig. 10.14 Blast wounds. Multiple splinter injuries of varying depth, amputated hand, and decapitation

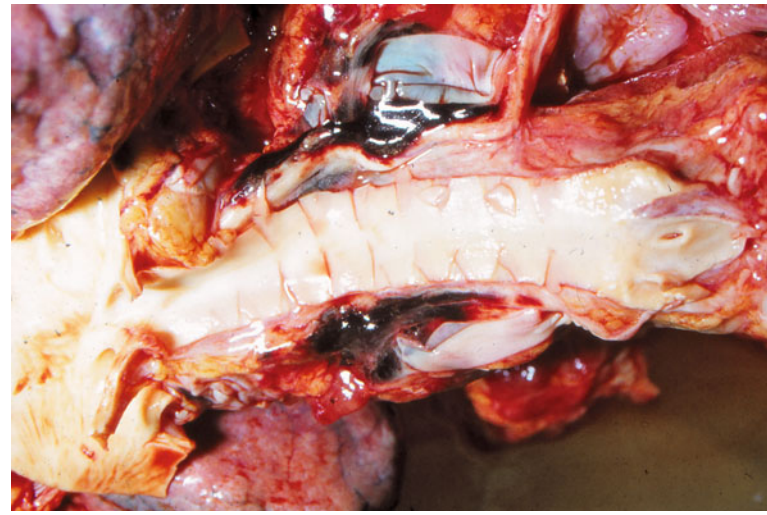

Fig. 10.15 A. carotis communis with intimal lacerations following explosion of a house

10.7 Criminological Aspects of Gunshot Wounds

Suicidal shooting is a widespread phenomenon in Western industrialized nations; naturally, it is seen most commonly in individuals with access to firearms, such as hunters or law enforcement officials. The crucial question to be answered in cases of fatalities involving gunshot wounds is whether injury was self-inflicted or whether another party (or parties) was involved; to this end, Table 10.7 summarizes the most important differential diagnostic criteria.

Much like taking skin samples from around the gunshot entrance wound (range of fire determination), a sample of skin from the suspected firing hand, or even both hands, could be ordered at autopsy for the purposes of detecting or excluding soot deposition. However, quantifying findings is not necessary—detecting soot deposition is sufficient. Therefore, particularly in the case of macroscopically visible soot deposition, invasive measures can be dispensed with and the skin surface simply covered with self-adhesive film for the purposes of collecting soot deposits.

Table 10.7 Differential diagnosis of self-inflicted or third-party involvement in fatal gunshot wounds

Criteria	Self-inflicted	Third-party involvement
Number of gunshot wounds	One	Possibly more than one
Typical localization of the entrance gunshot wound	Temples, mouth	Random
Range of fire	Close-range or close intermediate-range shot	Any range of fire
Angle of fire	Steep angle possible due to the short range of fire	Somewhat flatter to the transverse plane of the victim
Backspatter on the firing hand	May be present	Absent
Soot deposition on the firing hand	Positive	Negative
Gun-slide wounds on the firing hand, typically between the thumb and index finger	Rare	Never
Finding the weapon	In the vicinity of the body	Anywhere and/or cannot be found

The Autopsy Examination Following Gunshot Wounds. Any autopsy examination of gunshot wound victims should establish the number of entrance and exit gunshot wounds, as well as the direction and range of fire. In addition, it should be ascertained which shot or shots were fatal. Before opening the body, X-ray investigations (or ideally a CT scan) of the affected regions should be carried out if penetrating shots are suspected. Imaging all regions for investigation in two planes is important for the purposes of reproducing a spatial orientation (Fig. 10.16). Radiological data available prior to autopsy help in the detection of projectiles and their removal, as well as facilitating the dissection process in general. Investigations to determine range of fire should be completed before the body comes into contact with water. Projectiles lodged in the body should be removed only with plastic forceps in order to avoid compromising ballistic investigations aimed at identifying the weapon.

Causes of Death in Gunshot Wounds

Possible causes of death following gunshot wounds include:

- Suspension of blood circulation due to severe damage to the heart or part of the aorta
- Exsanguination due to large blood vessel injury
- Exsanguination or cardiac tamponade following injury to the myocardium or coronary arteries

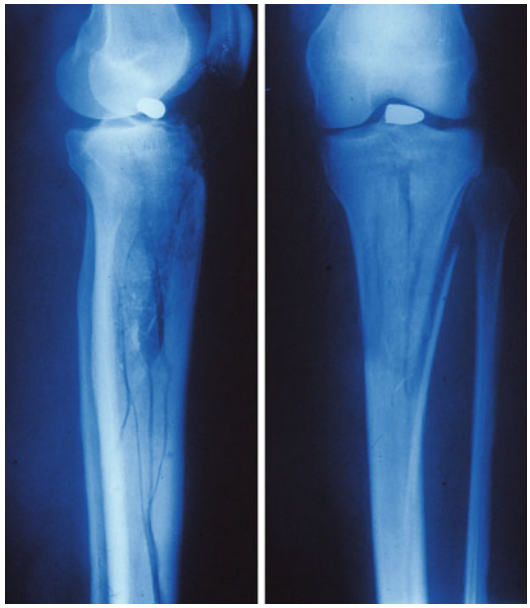

Fig. 10.16 Gunshot wound to the tibia with the projectile reaching its final position in the knee. X-ray of the lower leg in two planes

- Destruction of vital brain areas, in particular the brainstem
- Secondary cerebral swelling following a gunshot wound to the head
- Shock resulting from damage to the central nervous system and subsequent cardiac function impairment

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Neck Trauma

11

Case Study

After a 58-year-old man who lived alone had failed to empty his postbox or contact his daughter for several days, the police gained entry to his home. They found the owner of the home hanging in an upright position above an apparently overturned stool with a 1.2-cm thick rope looped twice around his neck; the rope had been tied into a noose and fastened to a roof beam. The knot was located on the right side of the neck behind the jaw; beneath the ligature, two dried-out brownish furrows could be seen slanting upwards laterally. A fold of pinched skin had formed between the furrows and showed small petechiae. The face was congested and mildly cyanotic, the tongue protruded, and in good light a discrete rivulet of dried saliva could be seen originating from the left corner of the mouth. Abundant petechiae could be seen on the facial skin, particularly in the sclerae, conjunctivae, and oral mucosa. Once the victim's clothes had been removed, livor mortis could be seen in a sock-and-glove distribution on the arms and legs. The patient had been prescribed an antidepressant by his general practitioner some weeks previously. At autopsy, fresh subperiosteal hemorrhage of the right clavicle at the level of the base of the sternocleidomastoid muscle was visible. Hemorrhage could be seen at two points on the ventral ligaments of the lumbar intervertebral discs (Simon's bleeding). There was no indication of violent trauma with second-party involvement.

Neck trauma can be subdivided into compression trauma (strangulation: hanging, ligature strangulation, and manual strangulation), non-compression trauma (blunt trauma: karate chop, a blow to the throat with an object, neck entrapment, or a kick to the neck), a shot wound to the neck, and sharp trauma to the neck (knife, axe, hatchet, bottle, etc.). Indirect trauma to the neck may occur in cervical spine trauma often seen following traffic collisions (see Chap. 21) or in the case of shaken baby syndrome (see Chap. 18). Depending on intensity and duration, all the above forms of violent trauma can be fatal. Whereas a victim can be questioned in the case of survival (see Chap. 17), fatal trauma to the neck raises primarily the following forensic questions:

- What type of violent neck trauma occurred?
- Is the detected neck trauma the cause of death?
- If yes, how intense and for how long did the trauma need to take place in order to cause death?
- Are there any signs of vitality on internal or external autopsy, i.e., can it be proven that the violent trauma occurred while the victim was still alive?

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- Is it possible—or not—that the fatal injuries were self-inflicted?
- Is it possible to make a statement about the victim's capacity to act before, during, and after the event as well as his or her actual actions?
- Is there incident-related violent trauma to other parts of the body beside the neck?

Both compression and non-compression trauma to the neck, as well as severe cervical spine trauma, e.g., a fall downstairs causing fracture of the dens axis, can be fatal, whereby local injury to the neck is not necessarily the cause of death.

11.1 Non-compression Trauma to the Neck

If one disregards road traffic accident-related trauma to the cervical spine (whiplash injury) and shaken baby syndrome, non-compression trauma to the neck primarily includes blows, kicks, and assaults with an object, as well as deep penetrating sharp trauma (stabs and cuts). When investigating stillbirths, birth-related neck injury should be considered in certain cases and carotid sinus syndrome with reflex cardiac arrest in others. Whereas lethal carotid sinus syndrome is considered a rare event and birth-related neck injury can generally be clearly and plausibly explained by events during birth, sharp trauma to the neck is primarily seen in the context of (attempted) homicide and suicide in rare cases.

11.1.1 Stab Wounds and Incised Wounds to the Neck

Stab wounds and incised wounds (cuts) to the neck are differentiated as follows: stab wounds (generally deeper than wide) and incised wounds (generally wider than deep) resulting from the use of a knife, axe, hatchet, broken bottle, or other sharp objects. Causes of death following lethal injury to the neck with a sharp object include:

- Fatal exsanguination due to stab- or cut-induced opening of blood vessels, in particular the carotid arteries (common, internal, and external) or the jugular vein
- Fatal air embolism following stab- or cut-induced opening of the jugular veins (internal and external)
- Fatal blood aspiration following stab- or cut-induced opening of the upper respiratory tract, in particular the pharynx, larynx, and the upper airways
- Fatal stab wound to the neck or back of the neck with injury to the cervical medulla and acute central respiratory and circulatory arrest

If superficial skin incisions are found in addition to one or more deeper wounds, often running parallel to the deeper fatal injury, self-infliction with suicidal intent should be considered (so-called tentative or hesitation wounds; see Chap. 17).

Fatal Exsanguination: Deep stab or incision wounds to the neck may cause fatal blood loss. In such cases, correspondingly significant blood loss at the crime scene can be expected, while stab or incision tracks with vessel injury and signs of blood loss (typically small areas of mild livor mortis, emergence of the color of internal organs, wrinkled splenic capsule, subendocardial hemorrhage at the level of the left ventricular flow path) are demonstrated at autopsy. Likewise, bloodstain pattern analysis at the crime scene may allow inferences about the event to be made. Bloodstains of significant size and reaching considerable distances are often seen following carotid artery injury.

Close inspection of local injuries to the skin of the neck can provide information on the type, number, and depth of stab or incised wounds, their sequence, and possibly also the instrument used, e.g., a broken bottle or a single- or double-edged knife with a smooth or serrated edge. Any appraisal is confounded or made impossible by dried and blackened wound margins. Dissection of the soft tissue of the neck enables the stab track or incision depth to be ascertained. If the instrument used was glass or porcelain, small parts or particles of the instrument may still be found in the wound.

Fatal Air Embolism: Following trauma-related opening of the jugular veins—more likely due to a deep incision than a stab wound—air enters the venous circulation and moves towards the right atrium and right ventricle. If the right ventricle pumps this air to the lungs, pulmonary embolism ensues. As in pulmonary thromboembolism, pressure in the pulmonary circulation increases, the heart is unable to pump, and acute cardiac failure follows. A fatal air embolism can be identified radiologically prior to autopsy, while at autopsy it can be identified using a special dissection technique (cardiac air embolism test according to Richter): following medial fenestration of the chest wall, the pericardial sac is opened ventrally at the level of the anterior ventricular walls with a single incision and, following inspection for pathological changes, flooded with water. Once the epicardium is fully below the surface of the water, a slit is made beneath the water level in the right atrium using the tip of a scalpel. In the case of an air embolism, air bubbles will appear (see Fig. 4.8a), although often only after the incising scalpel has been slightly rotated.

Approximately 70–150 ml of air is sufficient to cause fatal air embolism. Individuals who have survived air embolism report hearing a sucking sound, while a bubbling sound can be heard in the right ventricle on auscultation. Microscopic findings to suggest air embolism can be found in lung and brain tissue, in the latter particularly in the form of ring hemorrhages following embolic spread of air bubbles via collateral circulation (see Fig. 4.8b).

Fatal Blood Aspiration: Should sharp trauma result in an opening of the upper respiratory tract, fatal blood aspiration—as also seen in skull base fractures survived for a short period of time—should always be considered. At autopsy, blood is found in the oral cavity, upper respiratory tract, and peripheral branches of the bronchial tree. Coarse subpleural hemorrhages, occasionally striking due to their "chessboard-like" pattern, can be seen (Fig. 11.1).

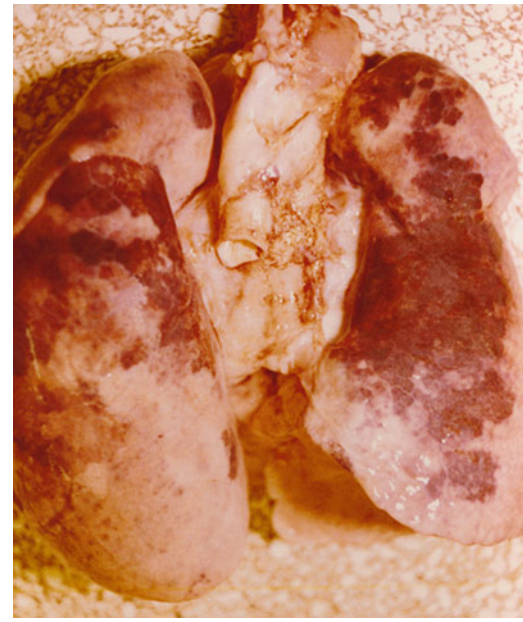

Fig. 11.1 Coarse subpleural "chessboard-like" hemorrhages seen macroscopically in a case of fatal blood aspiration

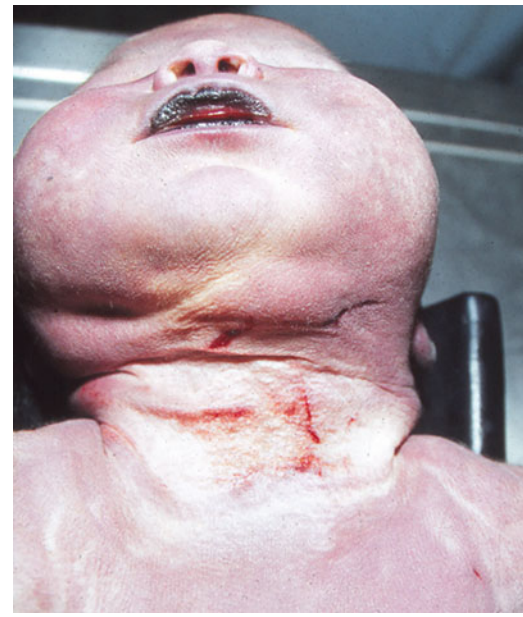

Fig. 11.2 Neck injury in a forceps delivery due to the large size of the child in the setting of previously undiagnosed gestational diabetes

11.1.2 Birth-Related Neck Injury

In addition to strangulation resulting from the umbilical cord wrapping around the neck, injury findings to the neck due to forceps delivery can be seen in the form of linear and sometimes angular epidermal detachment (Fig. 11.2). If the neck is overextended during birth, hemorrhage

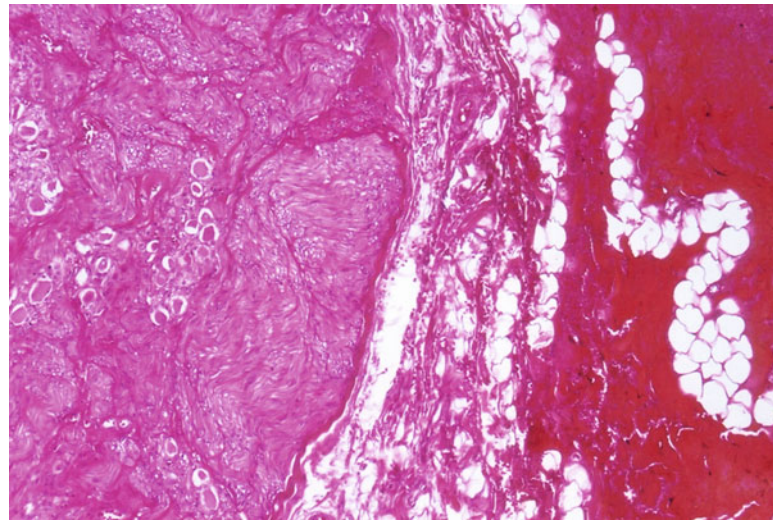

Fig. 11.3 Although areas of hemorrhage can be seen around the carotid body microscopically, neither the event nor the circumstances surrounding the onset of death give reason to suspect fatal carotid sinus reflex

can be seen in the cervical spine ligaments, sometimes also in the soft tissue of the neck. In cases of fatality, this type of hemorrhage is considered a sign of vitality. This phenomenon is seen particularly in stillborn infants with a relatively high birth weight following vaginal delivery (e.g., in the case of gestational diabetes).

11.1.3 Carotid Sinus Syndrome

The carotid body is a small sensory organ that lies at the level of the carotid artery bifurcation; on baroreceptor activation, it can cause a reflex parasympathetic effect via the vagal nerve with acute bradycardia and asystole. Thus in principle, reflex cardiac arrest following vigorous gripping of the throat is possible, i.e., carotid sinus reflex death. A carotid sinus reflex of this kind, although often alleged in court, is generally considered to be a rare occurrence. Alleged cases of carotid sinus reflex often fail to stand up to closer critical scrutiny. Unless the victim has put up significant resistance, only mild injuries and findings are generally observed. Carotid sinus reflex death can occur as a result of a headlock maneuver (stranglehold), as well as other forms of neck entrapment, e.g., with an elbow. So-called karate chops to the neck or other maneuvers causing sudden distension of the common carotid artery bifurcation can cause carotid sinus reflex death. Here, the stimulation of pressoreceptors in the carotid body via increased vagal tone causes decreased cardiac output. Both external and internal findings in the form of local hemorrhage may be absent or discrete, while congestive hemorrhage and petechiae are always absent. Highly excited individuals are at particular risk. Although local injury findings may be visible on the neck, abnormal findings on the carotid body and efferent nerve fibers are often not seen, either macroscopically or microscopically. If, however, hemorrhage (Fig. 11.3) or lymphangiectasias are seen in individual cases, this finding can only be considered an indication of carotid sinus reflex if sudden unexpected cardiac arrest is plausible in the context of the event. Even if the event and local findings are consistent with carotid sinus reflex, this is ultimately a diagnosis of elimination.

11.2 Compression Trauma to the Neck

Excluding intrauterine umbilical cord strangulation (see Chap. 20), which is pathophysiologically classified as ligature strangulation, there are three types of compression trauma to the neck that, by means of external pressure, result in constriction of the neck, i.e., strangulation:

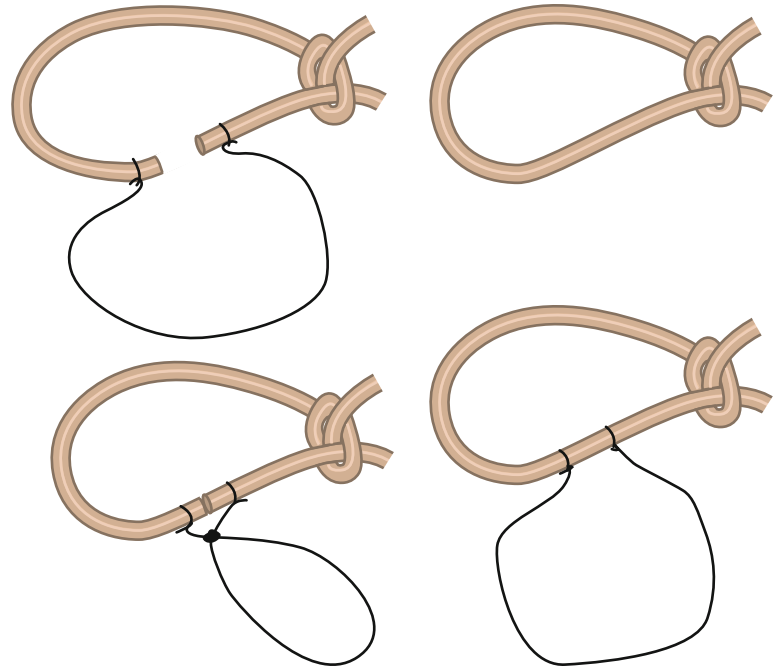

Fig. 11.4 Correct procedure for collecting a ligature

- Hanging
- Manual strangulation
- Ligature strangulation

The pathophysiological effects of strangulation include:

- Compression of cervical veins and arteries
- Carotid sinus pressoreceptor stimulation (carotid sinus reflex), extremely rare (see above)
- Constriction of airways

A self-experiment on neck constriction was described as follows: "The face became red, then blue, vision blurred and there was a whistling in the ears, then we lost our courage" (Nicolas Minovici, Rumanian forensic scientist, 1905).

Whereas hanging is usually with suicidal intent, homicidal intent can always be assumed in manual strangulation and almost always in ligature strangulation. Simulated suicides by means of hanging, self-inflicted ligature strangulation (by tightening a fixed ligature), or accidental ligature strangulation are rare. Accidental hanging is seen in autoerotic accidents and in children playing hazardous "hanging games." As in drowning, for example, there can be a number of general indications of death by strangulation in addition to local findings characteristic of neck compression.

In all three forms of neck compression, postmortem findings on external examination of a body, as well as on autopsy, may vary considerably depending on the type of violent trauma, the intensity with which a victim resisted, as well as the intensity and duration of neck compression. Changes in findings can occur between the onset of death and external examination of the corpse or autopsy and should be taken into account. The following may be used as a ligature: rope, cord, string, scarf, tie, strips of material, cable, etc., usually wrapped once, sometimes twice, more rarely several times, around the neck. Forensic investigation of the body at the scene where it was discovered should include careful documentation of the situation in which the body was found, including whether the head is in a head-down position. Once the corpse has been examined, any changes to the surrounding area should be avoided and the ligature should be left intact on the body until the arrival of the police authorities; the ligature should be removed later, preferably by means of a single cut and the two ends bound (Fig. 11.4).

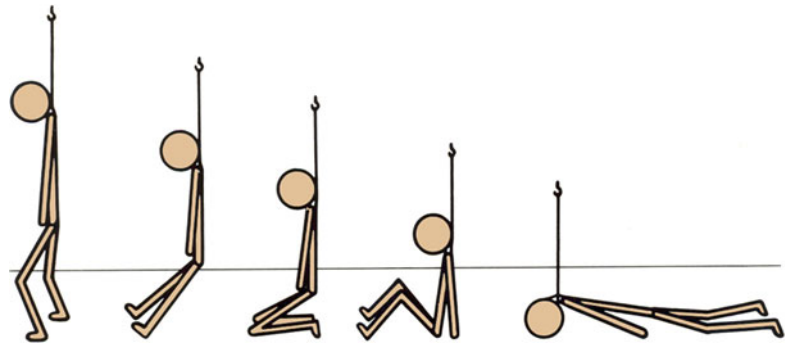

Fig. 11.5 Positions in hanging

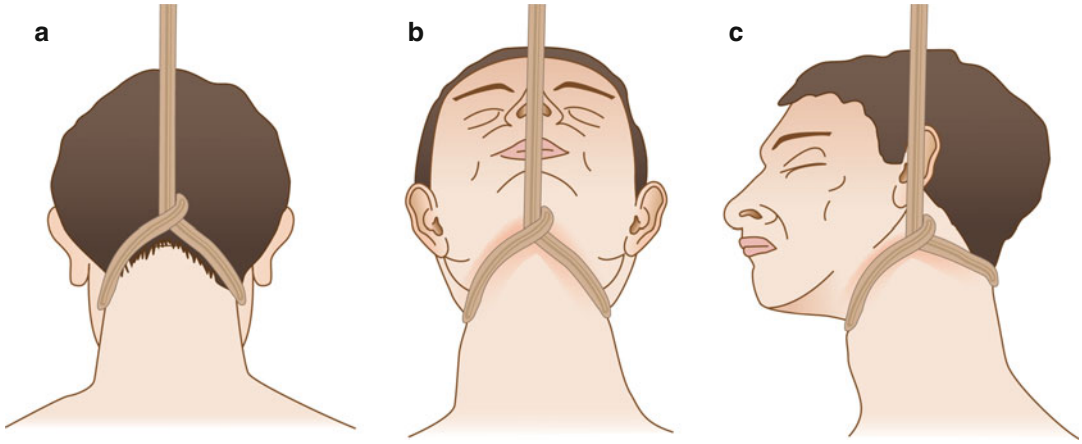

Fig. 11.6 (**a**) Typical and (**b**, **c**) atypical hanging

11.2.1 Hanging

Hanging is defined as constriction of the neck by a ligature whereby the victim's own body weight exerts compressive forces on the neck veins and arteries.

In hanging, it is not necessary for the ligature to fully encircle the neck; frontal constriction of the neck is sufficient to cause death even in a virtually lying or in a sitting/squatting position (Fig. 11.5).

Rather than airway obstruction (trachea and tongue displacement), the primary cause of death is compression-related circulatory arrest to the brain and—if blood vessels in the neck are fully compressed—rapid loss of consciousness or incapacitation. A distinction is made between typical and atypical hanging (Fig. 11.6).

Typical hanging: Symmetrical bilateral ligature furrows slanting upwards, knot or highest point of the ligature at the back of the neck, complete suspension of the body.

Atypical hanging: Asymmetrical ligature furrows, knot or highest point of the ligature at the side of the neck, half-sitting (or other) position.

Typical hanging is less common than atypical hanging. In the former, the knot or highest point of the ligature is generally found above the hairline at the back of the neck in the midline of the body. This causes compression of the carotid arteries and prevents blood circulation to the head; venous blood flow, however, continues to function, and the hyoid bone is often elevated, resulting in rapid loss of consciousness. Petechiae are sometimes seen at the base of the tongue. From a differential diagnostic perspective, it is relevant that often (virtually) no petechiae are seen in typical hanging.

On finding a hanging victim (Fig. 11.7), the following points need to be considered at the scene:

- Is the body completely suspended or partially supported?
- Were devices used to assist the hanging (type of ligature, stool or ladder)?
- Position of the ligature and knot?
- Is the ligature in contact with the skin of the neck?
- Are hairs trapped in the ligature?
- Does the distribution of livor mortis correspond to the position of the body?
- Are there signs of a hemodynamic effect due to neck constriction above the ligature furrow (petechiae, congestion, cyanosis)?
- If there is a rivulet of saliva from one corner of the mouth and, if so, does it correspond to the position of the body?
- Is there any indication of violent trauma with second-party involvement?
- Is there a suicide note?
- Is there a motive or explanation for suicide?
- Is there any indication of other methods of suicide, such as prior severing of arteries, ingestion of tablets, or self-restraining measures?
- Are there scars from tentative wounds on the flexor side of the wrist as an indication of previous suicide attempts?

In terms of the differential diagnosis, the distinction between a largely symmetrical ligature furrow usually slanting upwards laterally as seen in hanging (Fig. 11.8) compared with the horizontal position of the furrow seen in ligature strangulation is relevant. Moreover, swelling, cyanosis, and congestion-induced petechiae are absent in hanging with rapid and virtually complete constriction of the neck arteries, whereas these findings are often present in atypical hanging.

A horizontal ligature furrow in death due to hanging is only possible if the body is in a virtually supine position and even then, in contrast to ligature strangulation, the furrow is not fully circular. If the hanging position cannot be established in such cases, the suspicion of homicide by ligature strangulation should be aroused (Table 11.1).

Fig. 11.7 A characteristic situation in which a suicidal hanging is found, involving a stepladder and no hair trapped in the ligature

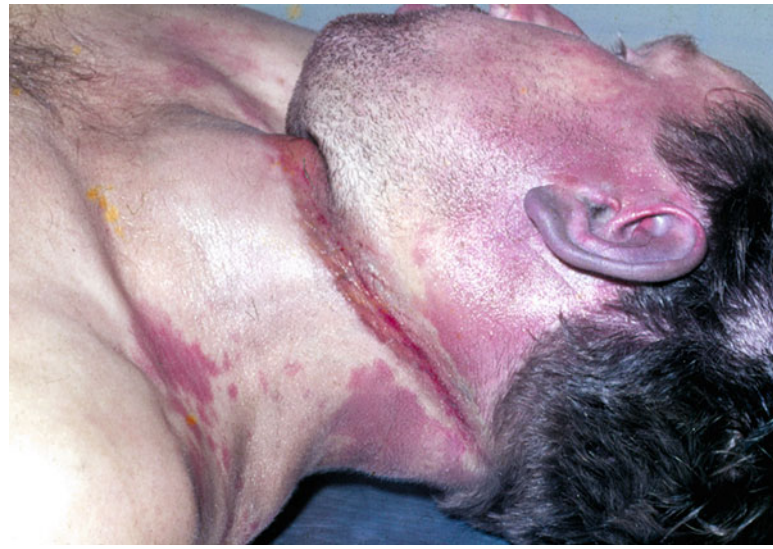

Fig. 11.8 Ligature furrow rising laterally in typical hanging

Table 11.1 Typical and atypical hanging

Finding	Typical hanging	Atypical hanging
Ligature furrow	Ligature furrow or device slants bilaterally and symmetrically upwards to the back of the neck	Asymmetrical position of the ligature furrow or device
Knot	Usually above the hairline at the highest point of the back of the neck in the midline of the body	Usually not in the midline of the back of the neck, e.g., running laterally over the jaw angle or the anterior side of the neck
Position of the body	Body completely suspended	Body in contact with floor/ground (feet on floor/ground, sitting, squatting, almost lying)
Signs of neck constriction	Mild or absent in primarily complete constriction of carotid arteries; possibly slight facial congestion, petechiae usually completely absent	Primarily incomplete constriction of the carotid arteries, hence distinct signs of neck constriction; severe congestion of the face, possibly also abundant petechiae

Since the different forms of neck constriction cause varying findings in the skin of neck, it is possible to differentiate between the ligature-related injuries seen in hanging, ligature strangulation, and manual strangulation.

The Ligature Furrow in Hanging: In the case of hanging, an often dried and brownish imprint or furrow from the ligature can be found on the skin; an imprint of the ligature may be visible in the form of, e.g., the ribbed or weaved structure of a rope. These ligature imprints can disappear if the ligature is removed soon after death; where this is not the case, both the imprints and the width of the furrow should be compared with the ligature (Fig. 11.9).

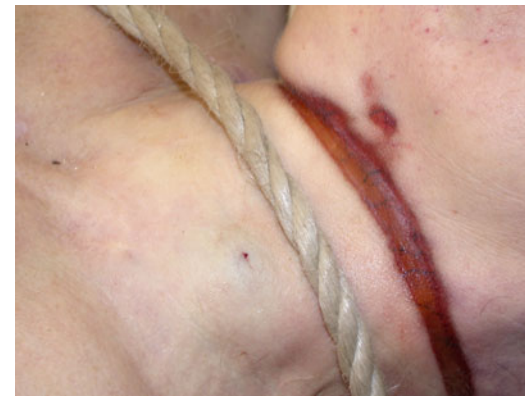

Fig. 11.9 Furrow and corresponding ligature in a case of hanging

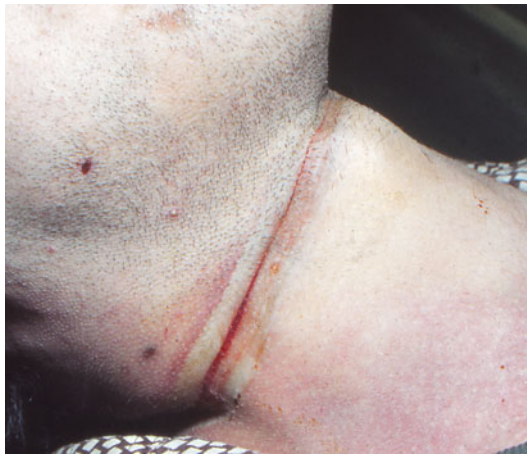

Fig. 11.10 Hemorrhagic strip produced by a double-noosed ligature

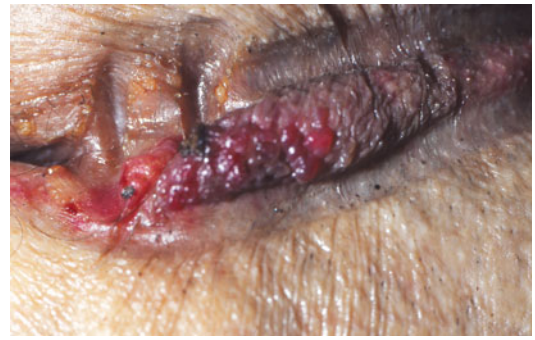

Fig. 11.11 Hemorrhagic strip in a case of typical hanging with a double-noosed ligature. The small epidermal blisters on the strip of skin are rarely seen and are not considered a sign of vitality

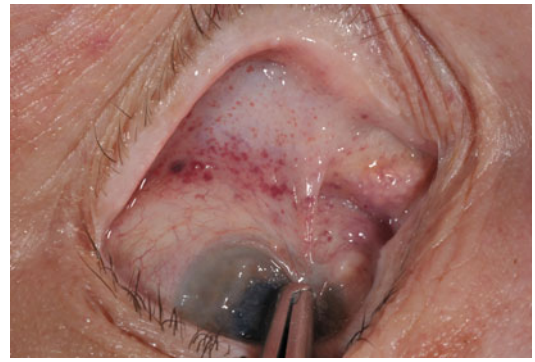

Fig. 11.12 Conjunctival petechiae in atypical hanging

In cases where a wide ligature is used, e.g., a wide soft scarf, it is possible that only discrete findings are present on the neck. If the body is in an upright position, the furrow usually runs above the larynx, while skin abrasions may occur along the lower edge due to the ligature shifting at the time of slipping into the noose (not a sign of vitality!). A furrow as such is also not a sign of vitality, since comparable findings can be inflicted post-mortem, e.g., ligature strangulation followed by hanging in order to mimic suicide.

Subcutaneous Hemorrhage Between a Multiple Ligature: If a ligature encircles the neck twice (or more), the skin may become pinched and produce a fold, the crest of which may show a narrow strip of hemorrhage. This hemorrhagic strip (Fig. 11.10) is interpreted as a sign of vitality. In contrast, the (rare) presence of small areas of blistery skin detachment between a double-noosed ligature cannot be taken as a vital reaction (Fig. 11.11).

The Hemodynamic Effects of Neck Compression: Incomplete compression of the neck in atypical hanging—incomplete arterial closure, relatively little weight in the noose, intermittent compression—causes compression that exceeds the internal pressure in the neck veins. This prevents venous flow from the head back to the heart, while at the same time blood continues to be pumped to the head via the arteries, resulting in acute congestion, swelling, and cyanosis of the face or above the ligature furrow. The first petechiae start to appear after strangulation lasting approximately 20 s, particularly in the eyelids, conjunctiva, and oral mucosa (Fig. 11.12).

In typical hanging involving a fully suspended body, the cervical veins and arteries may be abruptly and completely compromised to the extent that the hemodynamic effects of neck constriction are absent, i.e., no swelling, no petechiae, and facial pallor. Additionally, in both typical and atypical hanging, neurological effects of neck constriction may be seen: increased salivation, as well as involuntary micturition, defecation, and ejaculation.

Self-Rescue Attempts: Since loss of consciousness is rapid in hanging, attempts at self-rescue are rarely seen. Attempts to prevent death from ligature strangulation by grasping the ligature may produce injuries to the cheeks, chin, base of

Table 11.2 Stages of hanging

Time	Pathophysiological event/ symptoms and findings
From approx. 5 s	Loss of consciousness and incapacitation; self-rescue no longer possible; congestion, cyanosis of the face, early swelling
From approx. 20 s	Petechiae appear and may coalesce, followed by possible bleeding from the nose, mouth, and ears
From approx. 30 s	Deep inhalation; contraction of the neck and respiratory muscles; intermittent seizures (asphyxia seizures) every 15–30 s, possibly in the form of convulsive seizures with impact marks on the extremities, e.g., backs of hands and extensor sides of the lower arms; increased salivation, lacrimation, and nasal secretion
From approx. 1 min	Involuntary defecation, micturition, and ejaculation are possible
Approx. 1–2 min	Apnea and agonal respiration (gasping) are possible
After 5–10 min	Irreversible death. Prior to this point rescue is possible, albeit with possible hypoxic brain damage
Up to 20–30 min	Cardiac activity on ECG still possible

the mouth, and neck, while occasionally one or more fingers may remain trapped in the ligature.

The Time Course of Hanging: Depending on the intensity of pressure, compression trauma to the neck can cause rapid loss of consciousness and death according to a staged sequence; approximate times can be given for this sequence (Table 11.2).

Autopsy Findings: These findings can be of crucial importance when establishing vitality at the time of hanging. The anatomical structures of the neck require careful dissection in order to find even discrete injuries. To this end, the soft tissue of the neck, in particular the neck muscles, needs to be dissected under artificial bloodlessness in a layered procedure (Fig. 11.13). Artificial bloodlessness is achieved by prior opening of the skull and removal of the brain, as well as disconnecting the heart from the main afferent and efferent

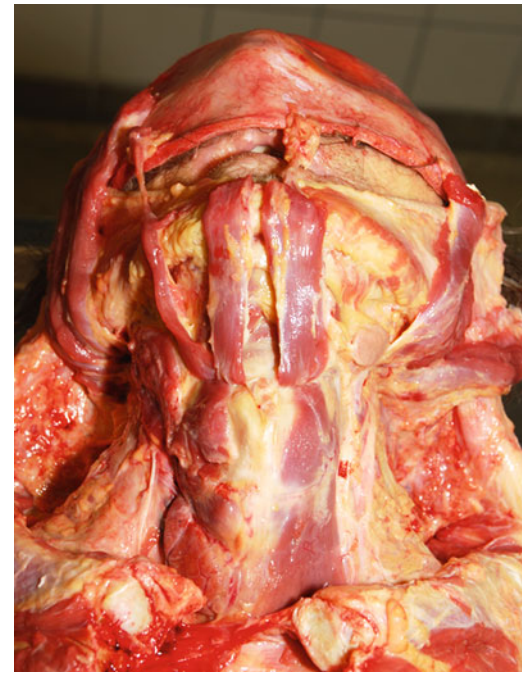

Fig. 11.13 Layered dissection of the anterior neck muscles

blood vessels. Blood present in the neck vessels then flows passively in a cranial direction towards the cranial cavity and in a caudal direction towards the pericardium, thus reducing the risk of dissection-related hemorrhage during the subsequent dissection procedure. Other structures requiring dissection in addition to the neck muscles include the thyroid gland, larynx and hyoid bone, vessels, tongue, tonsils, and clavicles.

Compression trauma of the neck can produce findings resulting from direct local trauma as well as indirect findings depending on the form of neck compression (hanging, ligature strangulation, or manual strangulation). Well-demarcated fresh petechiae are of particular relevance. Not all findings represent evidence of vitality at the time of strangulation (Table 11.3). Small petechiae in the soft tissue of the neck should be interpreted cautiously in the case of autolysis, putrefaction, and a head-down position of the body.

A number of the findings listed in Table 11.3 can be incurred post-mortem and thus do not represent forensic vital signs. Hemorrhage in the neck muscles is often absent in hanging (in contrast to

Table 11.3 Findings on external examination and autopsy in compression trauma to the neck and their validity as signs of vitality

External examination/autopsy findings	Validity
Dried rivulet of saliva: often from one corner of the mouth	Definite vital sign!
Swelling and cyanosis: only above the level of strangulation including the face	Definite vital sign!
Petechiae: in the sclerae, conjunctivae, oral mucosa, and skin above the level of strangulation including the face	Often a definite vital sign! (Caution: petechial vibices in the area of livor mortis in the case of a head-down position)
Hemorrhagic strip of skin between a double or multiple ligature	Definite vital sign!
Internal ligature mark: compression and drying out of subcutaneous fatty tissue at the level of the ligature mark	Not a vital sign!
Subperiosteal hemorrhage of the clavicles at the base of the sternocleidomastoid muscle (possibly unilateral in atypical hanging!)	Definite vital sign!
Neck muscle laceration: only seen in falls in/with the noose from a significant height	A vital sign only with surrounding hemorrhage!
Stretch tears: linear tears in the intima of the carotid arteries	Not a vital sign!
Fractures: of the larynx and hyoid bone structure	A vital sign only with surrounding hemorrhage!
Hemorrhage at the base of the tongue: in compression-related venous blood congestion (Fig. 11.14)	Definite vital sign!
Simon's bleeding: in the ventral ligaments of the lumbar intervertebral discs (Fig.11.15)	Considered a definite vital sign!
Fractures to the dens axis or cervical vertebrae (e.g., "hangman's fracture")	A vital sign only with surrounding hemorrhage!

manual and ligature strangulation). Even fractures of the dens axis and the cervical vertebrae, including injury to the cervical medulla, are possible in hanging, although extremely rare and only seen in falls from a certain minimum height. Falls from a height of 3 m or more can cause severe injury, including complete decapitation.

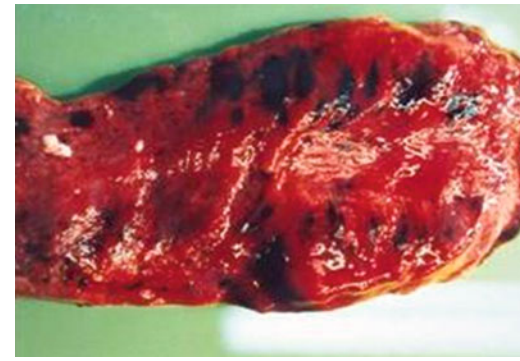

Fig. 11.14 Compression-related hemorrhage in the tongue

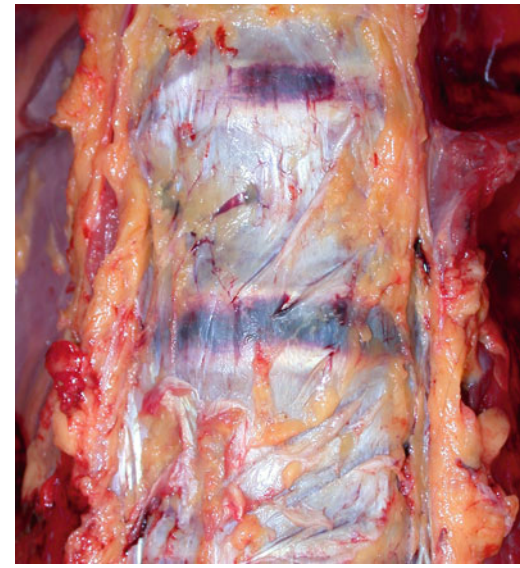

Fig. 11.15 Hemorrhage in the ventral ligaments of the lumbar intervertebral (Simon's bleeding)

Initially, the larynx and hyoid bone are cartilaginous structures, becoming ossified later. Whereas the hyoid bone has a U-shaped configuration, the larynx comprises the thyroid and cricoid cartilages, which are joined posteriorly by ligaments, and the two small arytenoid cartilages, which sit posteriorly on the cricoid cartilage. The posterior edges of the thyroid cartilage run off bilaterally into a superior and an inferior horn. Fractures of the superior horns are seen most commonly, occurring with relatively little trauma. Healed fractures of the superior laryngeal horns are an occasional incidental finding at autopsy,

Table 11.4 Fractures of the larynx and hyoid bone according to age in the setting of compression trauma to the neck (incidence in percent; $n = 194$)

Age in years (no. of cases)	20 (13) (%)	21–30 (23) (%)	31–40 (23) (%)	41–50 (26) (%)	51–60 (31) (%)	61–70 (19) (%)	71–80 (29) (%)	>80 (26) (%)
No fracture	77	49	33	16	19	26	14	8
Horn fracture only	8	39	52	42	55	58	62	42
Thyroid or cricoid cartilage fracture	15	12	15	42	26	16	24	50

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while inferior horn fractures are rare. The cricoid cartilage adjoining the trachea is broadest on its posterior side. Fractures, usually following massive violent trauma, are seen anteriorly at the level of the thinner section of the cricoid cartilage ring. Laryngeal fractures can cause internal bleeding and swelling of the laryngeal mucosa, leading to narrowing of the airways. The likelihood of fractures in the laryngeal and hyoid structures varies according to the degree of ossification and depending on age (Table 11.4).

Besides the laryngeal horns, the head of the hyoid bone is also subject to fractures. Fractures of the cervical vertebrae, although occasionally seen in hanging, are not usual in manual or ligature strangulation. Targeted examination of the cervical vertebrae in fatal strangulation is able to demonstrate hemorrhage in muscles, joints, ligaments, or intervertebral discs. Simon's bleeding (Fig. 11.15) is seen in cases of full-suspension hanging. A further general finding often made at autopsy is overinflation of the lungs resulting from strong agonal respiration, subpleural hemorrhage, as well as other findings supporting the assumption of death by asphyxiation. Where suicide victims have taken measures to prevent self-rescue by the prior application of restraining devices, a careful investigation of the type of restraint, against the background of findings on external examination and autopsy, should be undertaken to exclude intoxication, restraining force, or second-party involvement.

Simulated Suicide by Hanging: In cases where attempts are made to cover up suicide or to simulate suicide by hanging following homicide, findings made at the scene of the hanging can provide valuable information if, e.g.,:

- The distribution of livor mortis is not consistent with the position in which the body is found: Has the body been repositioned?
- The ligature furrow is not consistent with the position of the ligature: Did ligature strangulation precede hanging?
- Other injuries to the skin of the neck are found in addition to the ligature furrow: Did manual or ligature strangulation precede hanging?
- No traces of fiber from the ligature are found on the hands of the victim: Fiber analysis and microscopic trace analysis carried out by the criminal police.
- Hand marks are present on the upper arms where the victim has been carried, given that death or loss of consciousness was caused prior to hanging (it was reported that mild hand marks may be produced in the early postmortem period when cutting the body down!).
- There are indications of previous craniocerebral trauma: Hematomas and head lacerations.
- Objects are found trapped in the ligature or knot, such as parts of a shirt collar, head scarf, other parts of clothing, or hair (due to concerns about pain, suicide victims take care to avoid hair becoming trapped in the ligature).
- Other signs of violent trauma with second-party involvement are found, e.g., self-defense wounds.

Executions: Execution by hanging causes rapid loss of consciousness. The length of the rope and height of the drop are determined—according to the individual's body weight—in such a way as to cause neck fracture ("hangman's fracture") rather than a bleeding wound or even decapitation. The knot is placed lateral to the midline at the back of the neck. Although executions by hanging may take a "quiet" course, violent twitching and convulsions of the body are also seen. Gasping respiration begins almost immediately. The earliest cardiac arrests in executions were recorded after 5 min, while the heart can continue to beat for between 20 and 30 min.

11.2.2 Manual Strangulation

Manual strangulation is anterior or posterior compression of the neck with one or both hands.

Manual strangulation causes incomplete constriction of the carotid arteries with continued carotid artery blood flow when venous blood flow is prevented. At the same time, manual compression of the larynx and trachea occludes the airways. Plethora causes congestion and cyanosis of the face with petechiae above the level of compression. Strangulation marks in the form of planar strips of reddened skin that dry out post-mortem, as well as semicircular skin lacerations possibly attributable to fingernails and which also dry out rapidly, can be found on the skin of the neck depending on the intensity of the victim's resistance and the duration of strangulation. Occasionally, red markings correspond to the position of fingers of the hand used in strangulation. Depending on whether a victim is attacked from the front or from behind, strangulation marks can be found in the skin of the neck and nape. In the case of a slim neck and large hands, strangulation marks may be completely absent when both hands are used, although this is subject to considerable variation (Fig. 11.16).

As in atypical hanging and ligature strangulation, congestion-related hemorrhage may also be apparent in the skin behind the ears, the sclera and conjunctiva, and the epicranial aponeurosis, as well as the soft tissue of the face and mucosa, in particular the oral mucosa. Compression-related hemorrhage in the various layers of the neck musculature can be seen most clearly on dissection under artificial bloodlessness (see above) in manual strangulation (Fig. 11.17). When dissecting the larynx and hyoid bone,

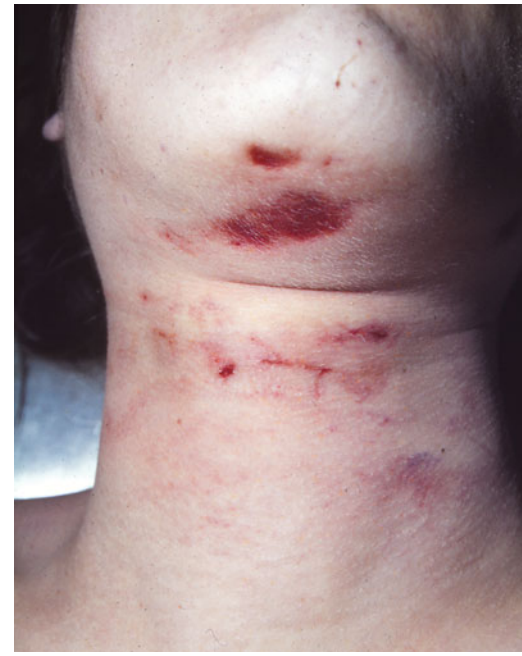

Fig. 11.16 Strangulation marks on the neck and base of the mouth

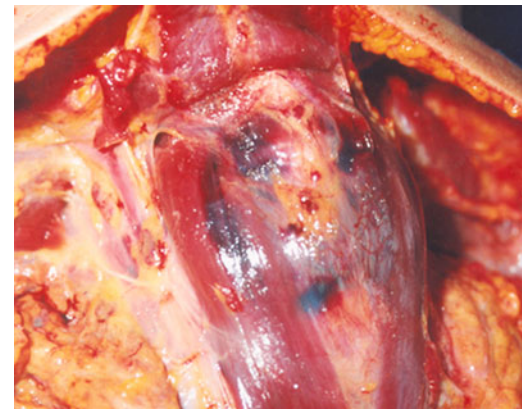

Fig. 11.17 Hemorrhage in the neck muscles in a case of death due to manual strangulation

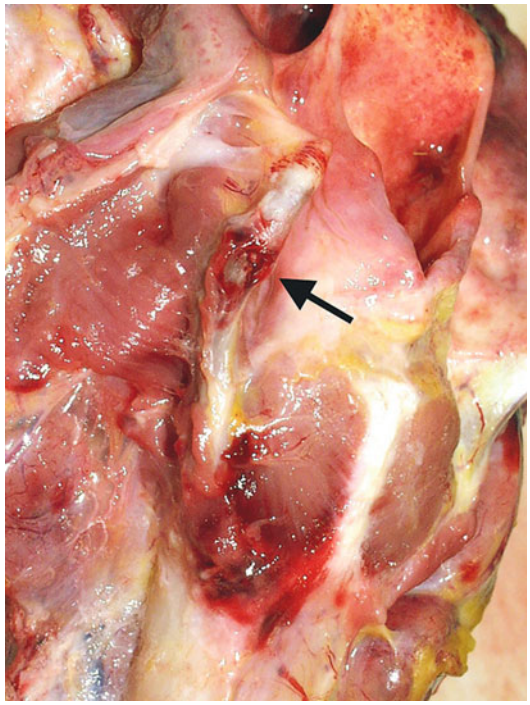

Fig. 11.18 Soft tissue hemorrhage around the head of the hyoid bone (*arrow*) following fatal manual strangulation

attention should be paid to fractures and hemorrhage (Fig. 11.18).

Predilection sites for fractures (with hemorrhage) of the superior horns of the thyroid cartilage and the greater horns of the hyoid bone are shown in Fig. 11.19. Fractures of the larynx and hyoid bone occur as a function of the degree of pressure, the age and sex of the victim, their individual disposition, and the intensity of resistance. In this context, the cartilaginous structures of the larynx and hyoid function as a point of abutment. X-ray diagnosis of laryngeal and hyoid fractures is often superior to autopsy diagnosis, since even fine non-dislocated fractures can be visualized radiographically. At autopsy, soft tissue hemorrhage adjacent to laryngeal and hyoid fractures is interpreted as a sign of vitality in compression trauma to the neck (Fig. 11.20).

Due to the elasticity of the ligaments around the larynx and hyoid bone, as well as the hyoid bone's early stage of ossification, manual strangulation without fracture or cartilage damage is possible in children and young adults, in particular females. Laryngeal and hyoid bone elasticity diminish with increasing age: the older the victim, the likelier compression-related fractures are to occur (Table 11.4). Fractures of the cricoid cartilage at the transition to the esophagus are an indication of particularly intense compression. In addition, edema is often seen in the laryngeal mucosa, while hemorrhage corresponding to the site of strangulation marks is seen in subcutaneous soft tissue, the neck muscles, the base of the tongue, at the level of the palatal arch, the pharynx and epiglottis, and also frequently in the laryngeal mucosa (Fig. 11.21). Hemorrhage at different levels indicates repeated attempts to grasp the neck in the course of strangulation. Hemorrhage of the posterior cricoarytenoid muscle ("posticus sign") warrants particular mention, since this can occur outside the context of neck compression and is seen, e.g., in deceased with a history of myocardial infarction or seizure (Fig. 11.22).

As with all forms of violent trauma involving resistance on the part of the victim, concomitant injuries need to be taken into account, such as self-defense wounds, including self-defense and attempted self-rescue wounds to the neck and hand marks. The absence of clear findings on the neck or soft tissue of the neck in the case of fatal manual strangulation can be attributed to little or no self-defense on the victim's part. Therefore, the prior ingestion or administration of foreign substances (knockout drops, sleeping pills, high-grade alcohol) should be considered. In principle, and particularly in cases of manual strangulation, the intensity of injuries (petechiae, hemorrhage, fractures, etc.) also depends on the intensity of the victim's resistance—the most distinct findings in manual strangulation are found in the case of strenuous resistance (Table 11.5); petechiae and hemorrhage are seen primarily in the sclerae and conjunctivae due to low tissue resistance.

It has also been noted that the probability of external injury or external evidence increases with the number of laryngeal and hyoid bone fractures (Table 11.6).

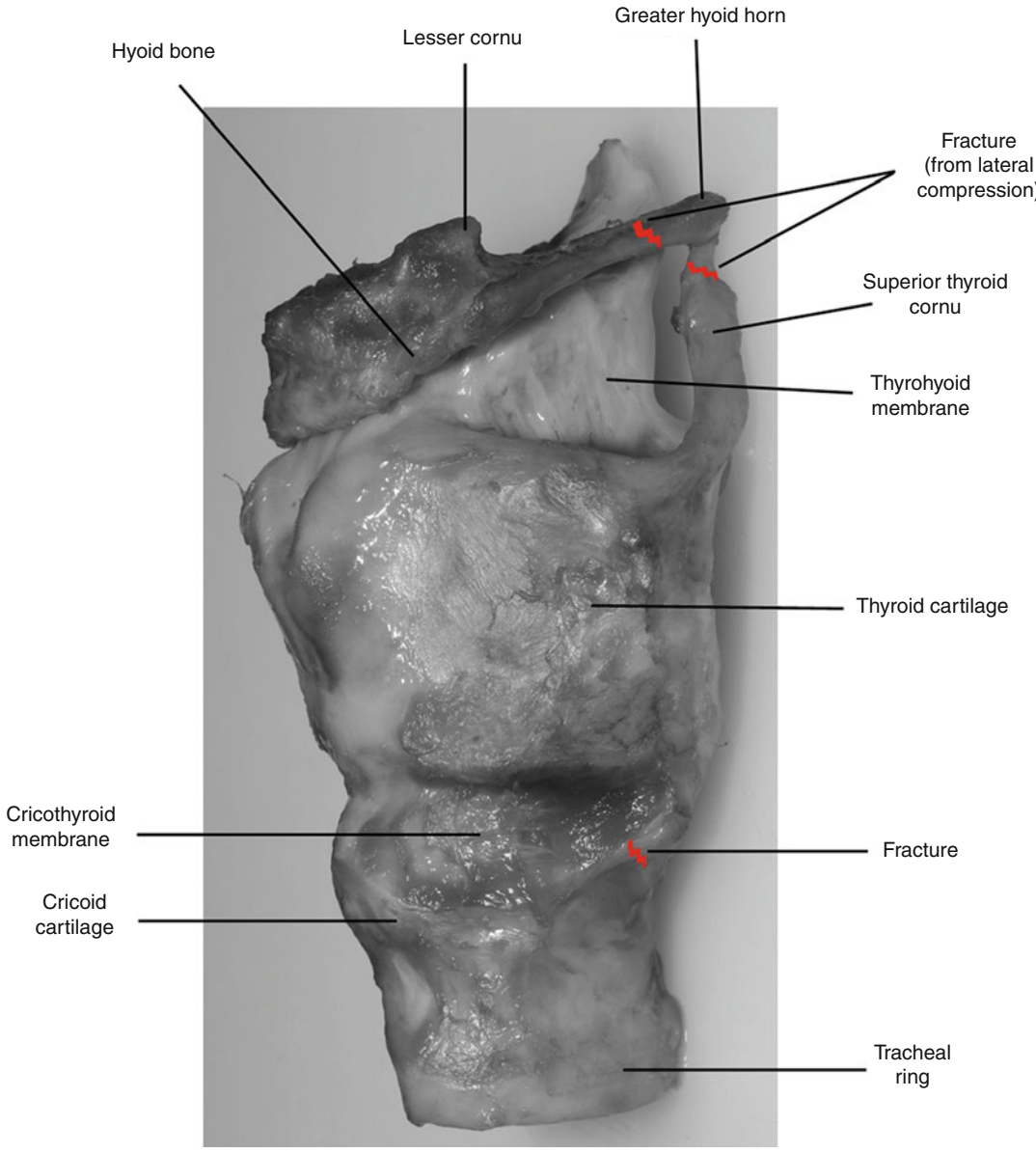

Fig. 11.19 Predilection sites for fractures of the larynx and hyoid bone from lateral compression

11.2.3 Ligature Strangulation

Ligature Strangulation: **Compression to the neck using a ligature tightened by either external physical force or some other device**.

In the case of suicide by ligature strangulation, the constricting force of the ligature is maintained after loss of consciousness by means of knots or twists in the ligature. Local findings in the skin of the neck are determined by the type of ligature used: narrow constriction devices, possibly encircling the neck more than once, often leave distinct strangulation marks, while soft wide ligatures (e.g., a soft scarf) may leave no skin findings. The petechiae seen in individuals who have initially survived ligature strangulation

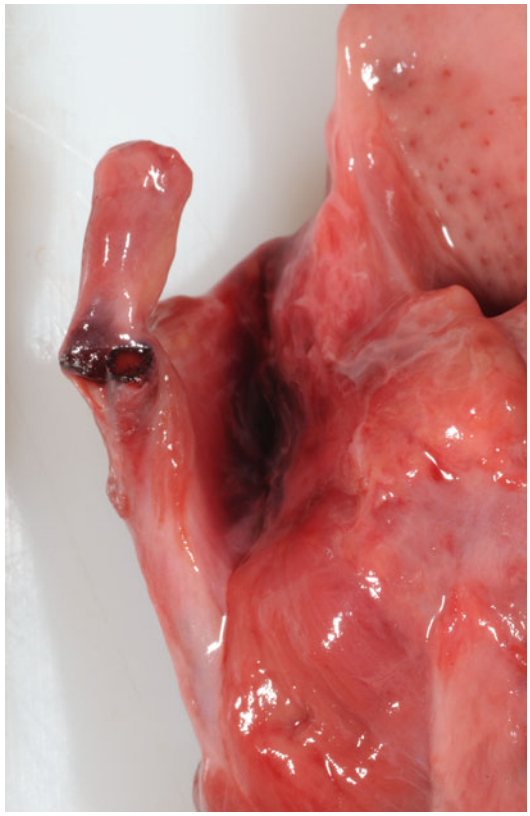

Fig. 11.20 Fracture of the head of the hyoid bone due to manual strangulation where surrounding hemorrhage represents a sign of vitality (freely dissected hyoid head; same case as in Fig. 11.18)

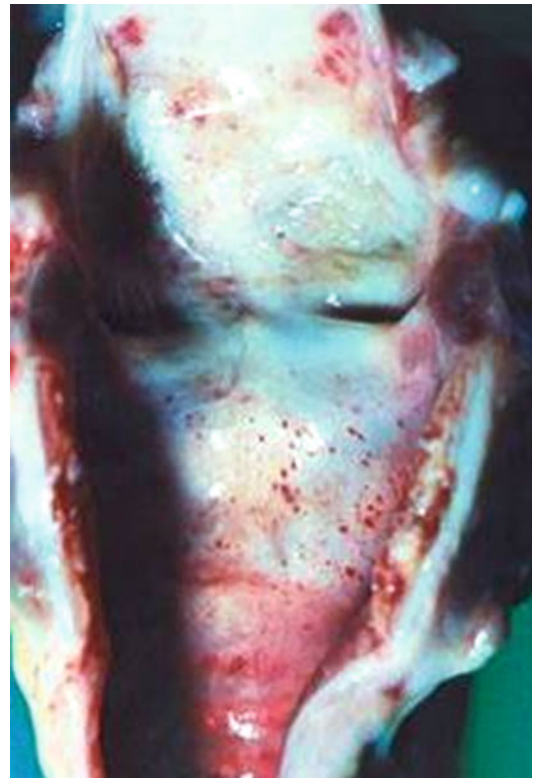

Fig. 11.21 Petechial hemorrhage in laryngeal mucosa following intense strangulation

may fade slowly, although they may remain detectable for up to several days (Figs. 11.23 and 11.24).

Self-inflicted ligature strangulation with suicidal intent is possible using a device capable of "locking in place" and maintaining the constriction exerted by the ligature. Self-inflicted manual strangulation, however, is not possible since muscles slacken on loss of consciousness and constriction is released.

Particularly in ligature strangulation, and depending on its intensity, significant hemorrhage in all layers of the neck muscles beneath the ligature mark can be seen; this is referred to as the "internal strangulation mark." Extensive internal injuries or hemorrhage are not seen in suicidal ligature strangulation. Hemorrhage above the level of the ligature is often caused by congestion. The classical constellations of

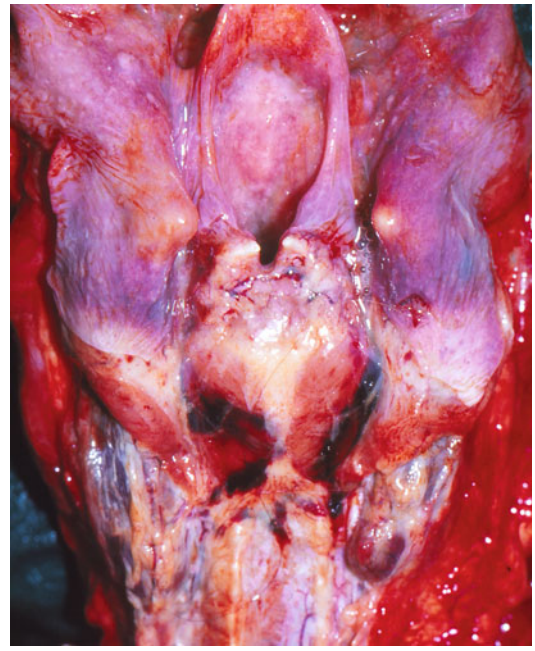

Fig. 11.22 Bilateral hemorrhage of the posterior cricoarytenoid muscle ("posticus sign") in a patient with acute myocardial infarction

Table 11.5 Intensity of injury findings depending on the type of compression trauma to the neck

	Typical hanging	Atypical hanging	Ligature strangulation: suicide	Ligature strangulation: homicide	Manual strangulation
Petechiae: head area	+	++	+++	+++	+++
Bite marks: tongue	+	+	+	+	++
Bleeding: tongue	?	+	+++	+++	+++
Bleeding: skin, ligature furrow	?	?	?	++	+++
Fractures: hyoid bone	++	++	+	+	+++
Fractures: thyroid cartilage	?	?	?	++	++
Fractures: cricoid cartilage	+	?	?	+	++
Bleeding: laryngeal joint	+	+	?	+	+++
Diffuse bleeding: larynx	?	?	?	+	+++

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+ Isolated or discrete, ++ occasional, +++ frequent or very frequent, *?* uncommon, unclear information, requiring individual clarification

Table 11.6 The incidence of external evidence or injury depending on the number of laryngeal and hyoid bone fractures

	Number of fractures					
Type of trauma	0	1	2	3	4	Total
Trauma with external marks	19	22	15	7	5	68
Trauma with no external marks	19	7	1	–	–	27
No trauma detectable	149	17	4	–	–	170
Total	187	46	20	7	5	265

Peters and Umlandt 1973

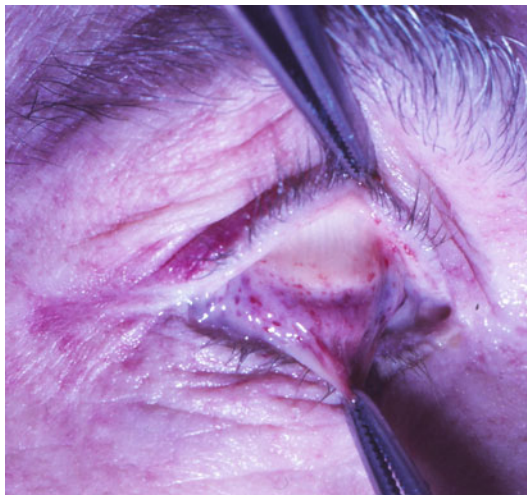

Fig. 11.23 Conjunctival petechiae 2.5 days following a survived attempt at self-inflicted ligature strangulation with a pair of tights

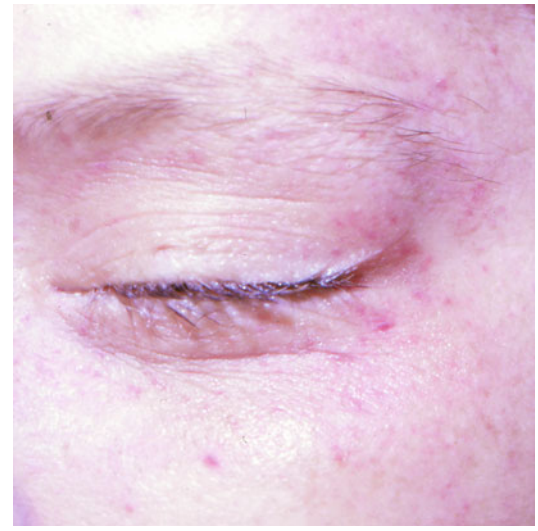

Fig. 11.24 Residual discrete scleral and conjunctival petechiae 1.5 days following a survived attempt at ligature strangulation

findings in all three forms of compression trauma to the neck are given in Table 11.7. The fact that the furrow in ligature strangulation is almost horizontal and circular—and not slanting upwards uni- or bilaterally as in hanging—is of particular significance.

Isolated findings can in principle occur outside the context of compression trauma to the neck and have other causes, e.g., petechiae due to cardiac-related congestion, following intensive resuscitation measures involving cardiac massage, or as a result of the causes given in Table 11.8.

In all forms of trauma, hence also compression of the neck, it should be borne in mind that important diagnostic findings can disappear post-mortem. Thus the signs of blood congestion

Table 11.7 Important characteristics of hanging, manual strangulation, and ligature strangulation

	Hanging	Manual strangulation	Ligature strangulation
Criminological background	Mostly suicide; simulated suicide by hanging after prior homicide (ligature or manual strangulation, intoxication, etc.) is possible	Always homicide; self-inflicted manual strangulation is not possible, since constriction is released immediately on loss of consciousness! Self- defense or concomitant wounds are common	Mostly homicide, rarely suicide (knot at the front of the neck, twisted ligature, laryngeal skeleton usually uninjured), or accidental (in children)
Constricting device	Noose (cord, rope, self-made noose)	Perpetrator uses own hands to constrict the neck	Ligature tightened using physical force
Findings on neck skin	Noose furrow, either symmetrical or asymmetrical, slanting upwards bilaterally towards the back of the neck; incomplete horizontal mark only in hanging in virtually supine position	Either linear or extensive skin redness, possibly comma- or crescent-shaped fingernail marks	Almost circular and horizontal ligature mark, skin imprint of more or less equal depth
Location of findings	In an upright body position, noose furrow usually above the larynx	Clearest findings often at the level of the larynx, laryngeal and hyoid fractures	Ligature mark often not in the upper neck area
Types	*Typical hanging*: knot located on the midline above the hairline at the back of the neck *Atypical hanging*: knot located elsewhere	Strangulation with one or two hands	Thin ligature: imprinted ligature mark Wide soft ligature: possibly no skin findings on the neck
Extent of findings	Possibly double or multiple noose with strip of pinched hemorrhagic skin	Unilateral, localized, or extensive findings possible; intensity of findings depends on victim's resistance	Usually circumscribed ligature mark, multiple ligature marks possible due to repeated attempts
Autopsy findings	Scant hemorrhage in neck soft tissues, possible transverse intimal tear of the carotid arteries, possible unilateral subperiosteal bleeding at the base of the clavicle due to tearing forces	Hemorrhage in subcutaneous fatty tissue and neck muscles, hemorrhagic laryngeal and hyoid fractures	Hemorrhage in subcutaneous fatty tissue and neck soft tissue, hemorrhagic laryngeal and hyoid fractures
Signs of the hemodynamic effects of neck compression	Facial congestion and cyanosis, petechiae in facial skin, sclerae, conjunctivae, and oral mucosa (often absent in typical hanging); livor mortis consistent with the position of the body, possible dried rivulet of saliva from one corner of the mouth. Bleeding from the mouth and nose in the case of extensive venous congestion following mucosal vessel rupture, particularly in lethal manual and ligature strangulation and atypical hanging		
Pathophysiology	In typical hanging, since the prevention of arterial blood flow (ischemia) predominates in the presence of preserved venous blood flow, signs of congestion are absent. Ischemia, blood congestion, and constriction of the airways (asphyxia) are seen in atypical hanging and ligature strangulation. Manual strangulation can cause both constriction of the airways (asphyxia) and predominantly venous congestion		

Table 11.8 Various causes of conjunctival hemorrhage without compression trauma to the neck

Group	No. of cases	Conjunctival hemorrhage	Average age
Childbirth	110	27 (24.5 %)	26.5
Coughing fits	100	3 (3 %)	50.0
Emesis	120	1 (0.8 %)	39.3
Defecation	120	–	–

Prokop and Wabnitz 1970

(swelling, cyanosis, and petechiae) above the level of compression may recede due to repositioning or transportation of a body. Conversely, a congestion syndrome may be simulated if the body is in a head-down position; in fact, this can be due to vibices within the area of livor mortis. Resuscitation measures do not produce massive blood congestion or an increased number of petechiae; however, isolated petechiae due to intensive reanimation, or even lay reanimation, cannot be excluded. Precisely because of this possibility of findings on the body to recede, statements on the duration of trauma in minutes up to the time of death can only be made with great caution. As a rule, death within a low single-digit minute range can only realistically be expected in the case of complete interruption of the cerebral blood circulation, as in typical hanging, while a time period from neck compression with complete, partial, or intermittent (re-)circulation of cerebral blood flow to death can be assumed in the upper single-digit minute range and up to 15 min. Ultimately, the immediate cause of death in neck trauma is not the injuries identified at external or internal autopsy, but rather a series of functional processes leading to brain tissue damage, insufficient "flush function" due to interrupted blood circulation, acidemia, etc., which finally cause central respiratory and cardiac arrest. Thus, as in other causes of death, e.g., drowning and blood aspiration, the cause of death following compression trauma to the neck is generally classified in the first instance as "asphyxial death," even though asphyxia is an umbrella term for a wide variety of causes and mechanisms leading to death (see Chap. 14).

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Thermal Injury

12

Fatal and nonfatal exposure to heat and cold (thermal injury) is seen in the form of accidental injury as burns and scalds, as well as in the case of deliberate exposure with homicidal intent or for the purposes of concealing a homicide, e.g., homicidal arson vs. arson to conceal homicide. Approximately 10–20 % of all abused children demonstrate heat-induced thermal injuries (see Chap. 18). Cold injuries, on the other hand, are rarely the result of a criminal act. In forensic medical practice, hypothermic fatalities raise the question of how the deceased came to be in a situation in which fatal hypothermia could occur. Most forms of thermal injury produce clearly differentiated findings.

12.1 Heat: Burns and Scalds

Case Study

A 16-month-old male presented with scald injuries to the left arm, left anterior chest, abdominal skin on the left side, as well as an injury stretching from the inner left thigh to the left knee. While unsupervised for a short period of time, the child had allegedly poured a steam cooker with boiling water over himself by pulling on the device's cable. Physical examination showed splash-like (left arm) and extensive scald injuries (up to third degree) on approximately 20 % of the body surface area (BSA). The treating physicians were skeptical about the information provided by the parents, suspecting instead child abuse.

General heat injury (heatstroke, sunstroke, heat exhaustion, and heat cramps) occurs as a result of disruption to the organism's thermoregulation mechanism and is distinct from specific localized thermal injury due to exposure to high temperatures. A further distinction is made in the case of the latter between scalds and burns.

Scalds: Exposure to moist heat, either locally or over the whole body, in the form of hot liquids—particularly water—and steam (hair is unaffected)

Burns: Exposure of the body to dry heat, either locally or over the whole body, in the form of high-temperature objects (contact burns) or flames (hair is affected)

Particularly in the case of scald injury to children (more so than burns), child abuse involving the pouring of or forced immersion in (e.g., a hand) a hot liquid needs to be considered. However, scald injuries are also seen in elderly care recipients when carers breach their duty of care, e.g., bathing a person suffering from dementia for an overly long period of time in excessively hot water.

Burns, on the other hand, are seen in the form of contact burns that can leave characteristic injuries (e.g., circular or oval cigarette/cigar burns), as well as in the form of chemical burns

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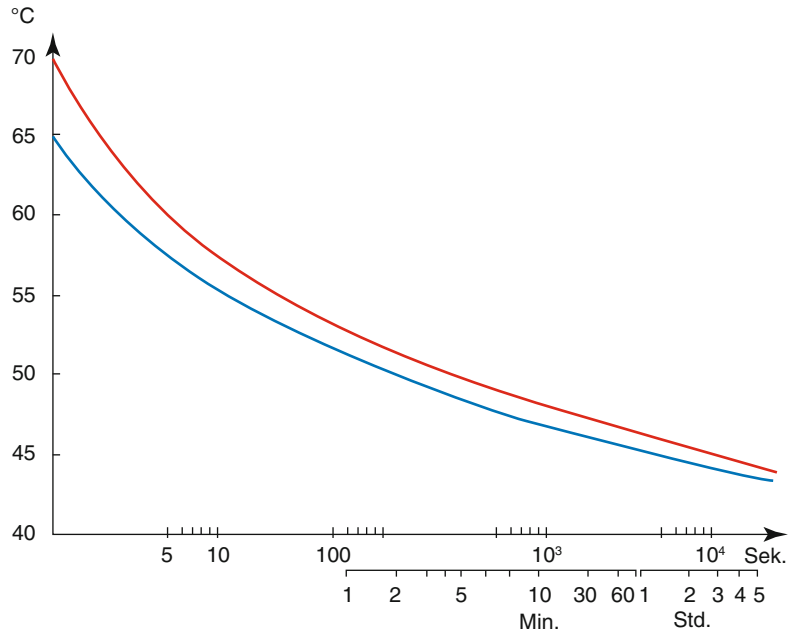

Fig. 12.1 The course of the temperature–time curve in the region of the injury threshold on exposure to heat ranging between 44 and 70 °C (scalding). *Dashed curve*, threshold of reversible heat injury; *solid curve*, threshold of irreversible heat injury

(e.g., caustic lime) or radiation, electrical, and lightning burns (see Chap. 13). In home fires, a smoldering fire that releases toxic gases can cause death without the body showing any visible signs of fire- or heat-induced injury, e.g., carbon monoxide or cyanide poisoning in a heavily alcohol-intoxicated individual in a smoldering apartment fire. In addition, death by fire is seen in the context of accidental death, as well as in the context of arson. Self-immolation with suicidal intent is uncommon. Occasionally, a fire is used to conceal a previous homicide; thus, particular care is required when performing the forensic examination of fire victims in order to differentiate, e.g., between "homicidal arson" and "arson to conceal homicide" (see below). Disposing of a body by means of complete incineration is rare, since this requires—as known from legal cremation—extremely high temperatures and long incineration times, depending on the constitution of the body.

Fires involving naked flames tend to damage primarily unclothed areas of the body (head, hands, and legs). Flames traveling upwards will singe the hair, turning the ends yellowish gray and crinkly. Hot fluids, on the other hand, are absorbed by clothing, causing more severe injury to the skin in those areas where clothing is more fitted (upper body and belt area). Singed hair has never been seen in cases of scalding.

Two factors are critical in terms of local injury to the skin and soft tissue: the level of heat and the duration of exposure (Fig. 12.1).

At an exposure time of 6 h, scalding occurs from a temperature of 44 °C, whereby the type of heat source is relevant. Steam, for example, has far better conductivity and a 4,000-fold greater heat capacity than air. In addition, the intensity of injury is determined by the temperature reached deep in the tissue (i.e., the heat capacity and heat conductivity of the tissue layers), which decreases radially exponentially. The heat conductivity of hot steam and gases is higher than that of solid bodies of the same temperature (Fig. 12.2).

When inhaled, hot steam exceeding 100 °C can cause a temperature rise in the oral cavity, larynx, and trachea (inhalation trauma), leading to severe, microscopically detectable heat-induced injury to the mucosa of the airways. Naked flames cause the most injury to unclothed areas, as do flash fires, e.g., barbecue accident (Fig. 12.3a), including the face (Fig. 12.3b).

Important: **Until the age of five**, **a child's skin is thinner than adult skin**; **thus**, **even low temperatures and short exposure times can cause injury in infants and small children**.

Table 12.1 shows the degree of injury in adult victims in relation to the temperatures given, whereby exposure times always need to be taken into consideration.

In addition to the degree of local thermal injury, the extent of thermal injury—which is calculated according to the "rule of nines" with modifications for neonates, infants, and children—is decisive in terms of survival prognosis (Fig. 12.4).

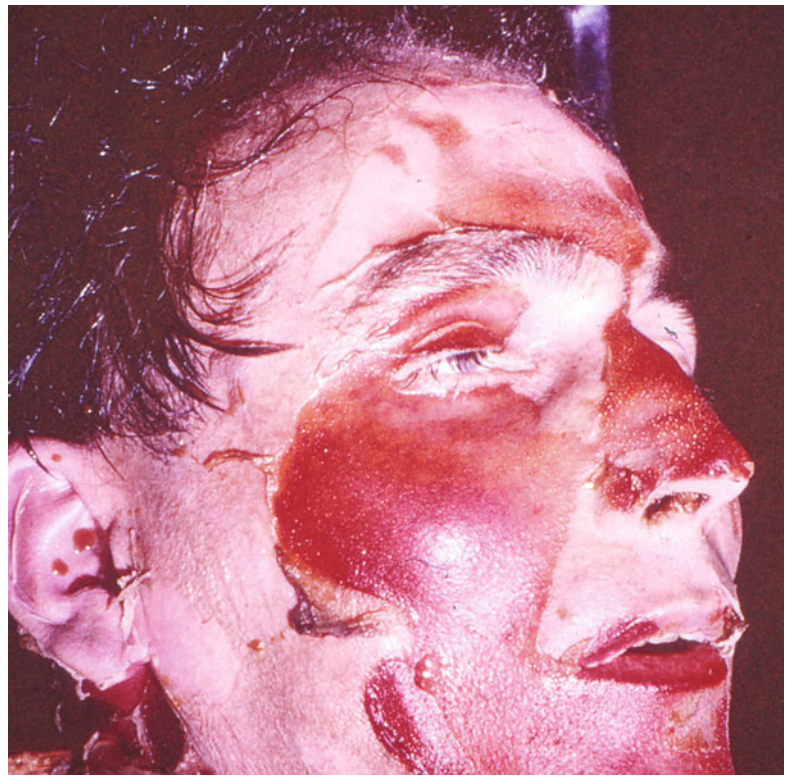

Fig. 12.2 Injury to facial skin from hot steam with sparing of the upper lip and right upper eyelid fold and no damage to eyelashes or eyebrows

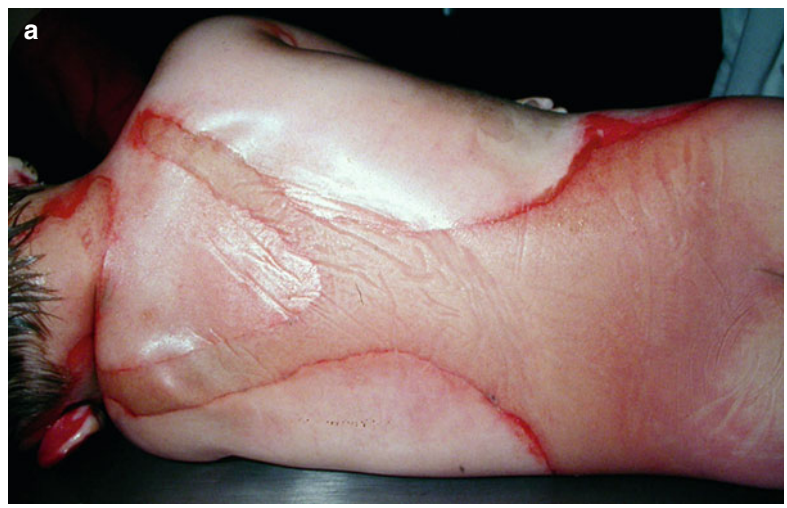

Fig. 12.3 (**a**) Burns due to a flash fire with sparing of clothed body areas (barbecue accident). (**b**) Burns to facial skin from a flash fire with sparing of the nasolabial folds and both eyelids; (same case as in **a**)

Fig. 12.3 (continued)

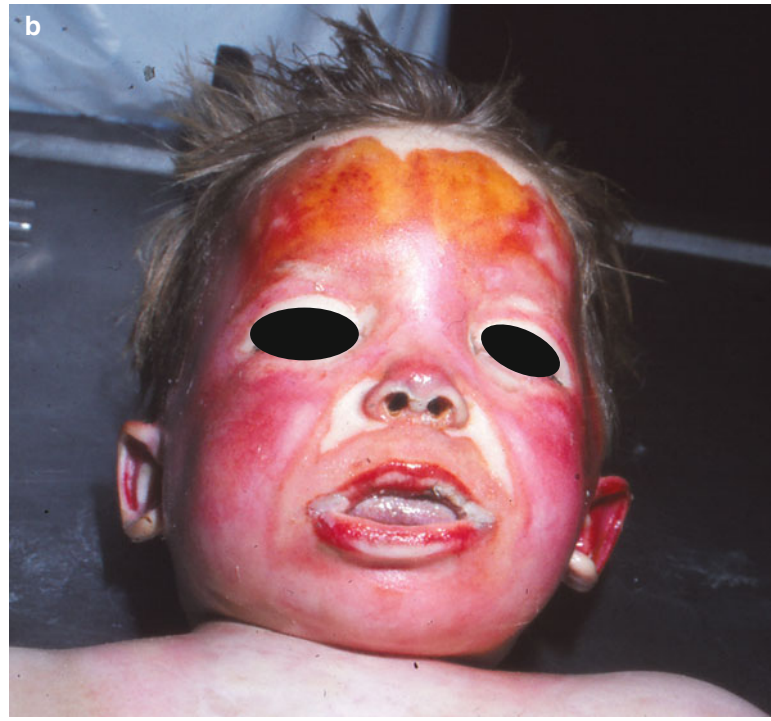

Table 12.1 Degree of injury at respective temperatures

I. Erythema, edema: reversible injury	40–50 °C
II. Blistering serous-hemorrhagic blisters	50–70 °C
III. Necrosis, scab formation, protein coagulation	From 62 °C
IV. Black charring of soft tissue, organs, and bones	From 65 °C and above
V. White charring of bones	Higher (and high) temperatures

Prognosis in thermal injury depends on:

- Degree of burns/scalds (depth of thermal injury)
- The affected portion of the BSA, i.e., the surface area of thermal injury, as a percentage
- Age: Prognosis worse in neonates, infants, and small children, as well as in over-40-year-olds

Important: **The burn index states that where the extent of second- and third-degree burns (in percentage of the BSA) and age add up to 100, survival chances in the case of optimal treatment are maximally 50 %.**

Thus, the following rules of thumb apply to the prognosis in burn victims receiving adequate medical care:

Burn index <80 = low risk of mortality
Burn index 80–120 = acute risk of mortality
Burn index >120 = survival unlikely

Quite apart from the prognostic value of the burn index, other particular factors such as inhalation trauma need to be taken into account, possibly rendering the prognosis worse than initially indicated by the burn index, which relates only to BSA injury (see "Burn Disease" below). The classification and main symptoms of burns to the skin and soft tissue are given in Table 12.2.

First-degree burns (superficial burns) produce erythema with no blister formation, while the epidermis remains intact. Reactive hyperemia in peripheral blood vessels in the superficial corium can be seen microscopically.

In the case of second-degree burns (partial thickness burns), blisters appear and the skin is moist, red, and painful. Microscopically, a blister lumen filled with serous fluid is visible between the keratin lamellae of the epidermis

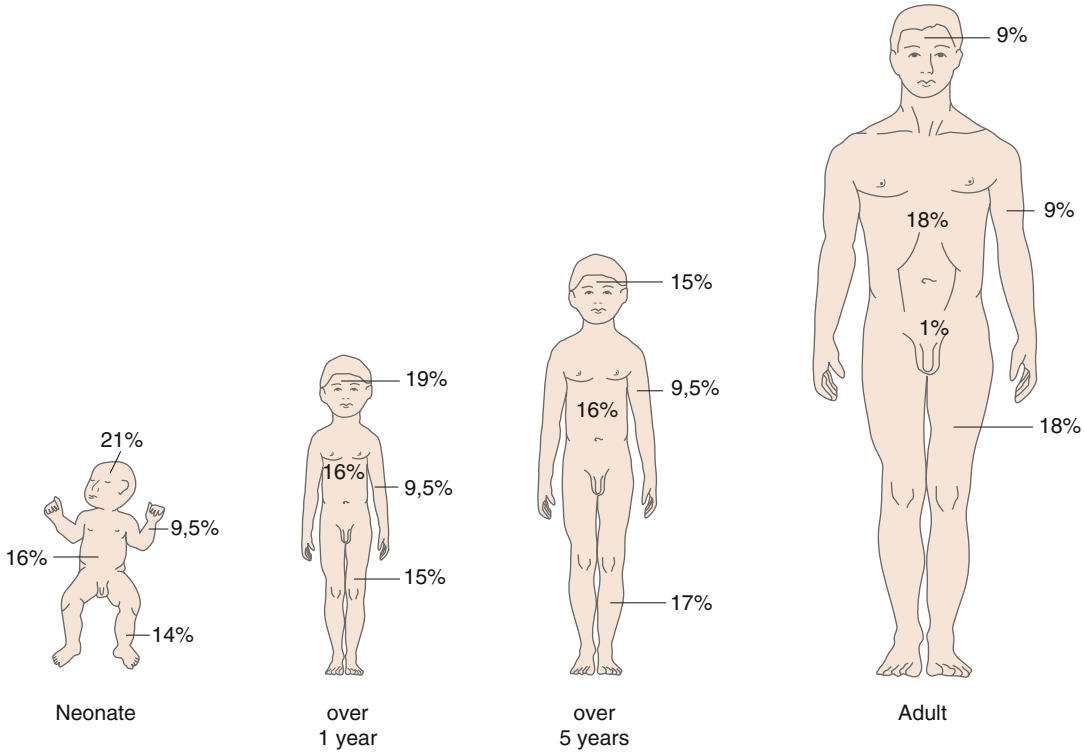

Fig. 12.4 Percentage portion of the skin surface from the head, trunk, and extremities in neonates, children aged over 1 year, and children aged over 5 years, as well as the "rule of nines" applied to adults when estimating survival prognosis—method according to Lund and Browler

Table 12.2 Classifying burns and clinical findings

Degree	Burn depth	Color/appearance	Local finding	Pain	Healing
1	Superficial, epidermis	Red	Intact epidermis, reactive hyperemia and erythema	+	5–10 days
2a and 2b	(a) Superficial: epidermis, corium (b) Superficial: epidermis, dermis down to subcutaneous fatty tissue	(a,b) Red, blister formation (leukocytes in blister fluid is a sign of vitality)	(a) Edematous (b) Swelling	(a) + (b) ±	(a) 10–20 days, scant scarring (b) 25–60 days, scarring
3	Epidermis, dermis, subcutaneous fatty tissue	White to brown	Leathery with coagulation necrosis of the epidermis, dermis, and dermal appendages	–	No spontaneous healing
4	Epidermis, dermis, subcutaneous soft tissue	Black charring	Destroyed epidermis, charring of subcutaneous soft tissue including muscle and bones	–	No spontaneous healing

and the basal layer or, on raising the epidermis, between the basal epidermal layer and the superficial corium. The stratum corneum and stratum granulosum of the epidermis are damaged by heat, while the basal stratum as such remains intact. The blister heals within 2–3 weeks.

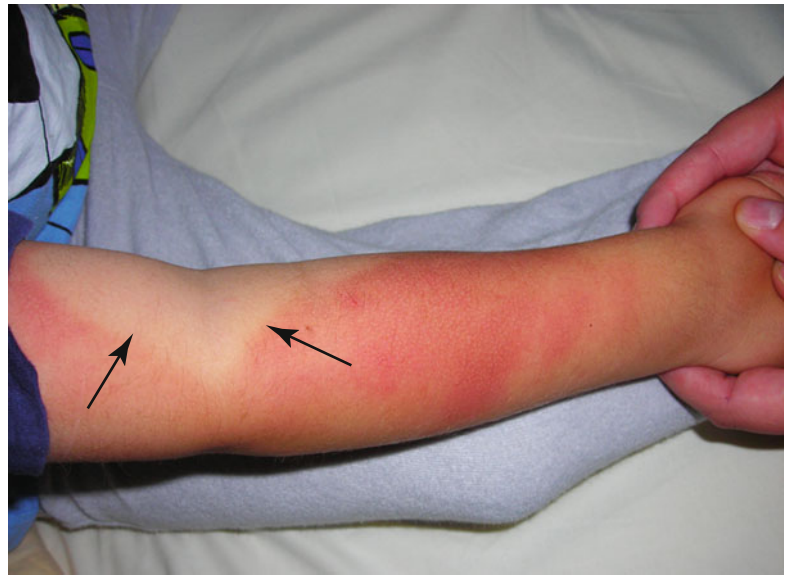

Fig. 12.5 First-degree scalding of the skin with rhomboid-shaped sparing (*arrows*) of the antecubital fossa

Important: **In the case of fire deaths, the detection of leukocytes and proteins in the serous fluid in the blister is considered a sign of vitality**.

From a differential diagnostic perspective, blister formation due to barbiturate intoxication or in the context of Lyell syndrome ("scalded skin syndrome") should be considered; the circumstances of the event or of death are often helpful in the clarification of this point.

Third-degree burns (full-thickness burns) cause coagulation necrosis of the entire cutis (epidermis, dermis, and dermal appendages). Due to heat-induced damage to nerve ends, pain perception is eliminated.

Deeper layers (subcutaneous fatty tissue, muscles, tendons, ligaments, and bones) are affected in the case of fourth-degree burns.

Varying degrees of burns are seen in both accident-related and child abuse-related thermal injury (Figs. 12.5 and 12.6).

In the case of combustion gas inhalation injury, and depending on the temperature of the steam or gases inhaled, thermal damage to the airways is seen: ragged desquamated coagulation necrosis of the mucous membrane of the nasopharynx, larynx, and trachea; reactive submucosal hyperemia; cell and nuclei elongation in a luminal direction in the ciliated respiratory epithelium, where preserved; and possibly also fine soot particles reaching as far as the bronchioli (Fig. 12.7).

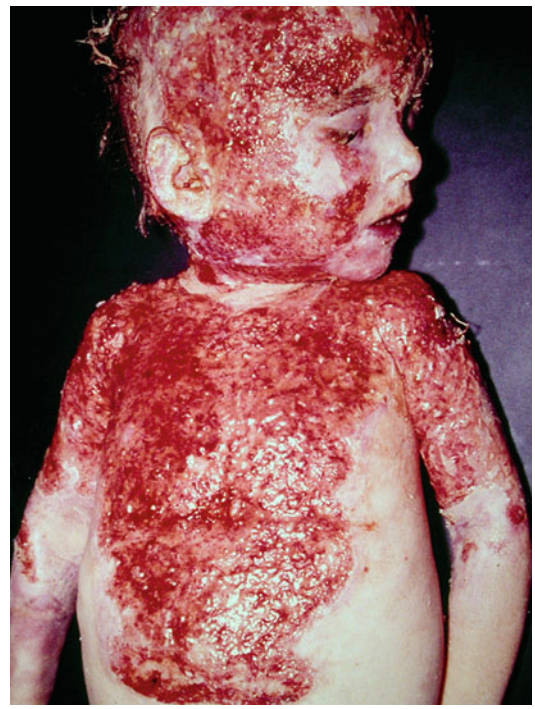

Fig. 12.6 Third-degree scalding in a young child

When examining victims of fire and heat at autopsy, it is important to differentiate between signs that the victim was alive at the time of thermal injury (signs of vitality) and signs of thermal injury that could have been incurred post-mortem. At the same time, it is important to establish

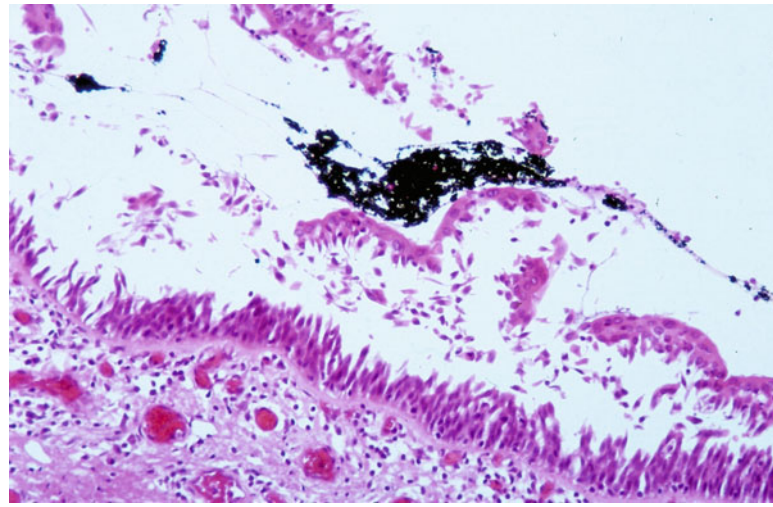

Fig. 12.7 Heat injury to the respiratory epithelium with elongated cylinder epithelia, elongated cell nuclei, reactive hyperemia in the subepithelial tissue, and peripheral soot particles following heat inhalation trauma (H&E × 200)

whether there are traces of prior second-party involvement. The distribution of thermal injury over the body may be relevant in terms of reconstructing the event (Table 12.3). Conceptually, evidence of burning while alive needs to be differentiated from that of burning post-mortem.

Homicidal Arson: Causing death by means of fire (rare).

Arson to Conceal Homicide: Causing death and subsequently burning the body to remove evidence.

In cases where the body is extensively burnt due to direct exposure to fire, often only a charred torso with no extremities remains. Once the boiling point of bodily fluids has been exceeded and interstitial fluid has evaporated, a process of thermal fixation of internal tissues and organs develops, making meaningful findings at autopsy possible. Thus, even in the case of extensively charred bodies, conclusions can be drawn about underlying disease and the cause of death. Identifying victims, however, often requires special analyses such as dental identification or molecular genetic investigations.

12.1.1 Signs of Vitality in Burns and Scalding

One of the most important tasks of visiting the site at which a body was discovered and performing both the external and internal autopsy is to establish whether the victim was alive at the time of heat exposure, i.e., where signs of vitality are present. Particularly noteworthy here are soot aspiration deep into the airways, soot ingestion, the formation of "crow's feet" at the canthi of the eyes, evidence of thermal injury to the deep airways (inhalation trauma), as well as the detection of carbon monoxide (possibly also cyanide) in blood as a result of active combustion gas inhalation.

The Formation of "Crow's Feet." The term "crow's feet" is used to refer to the lines of spared skin at the canthi of the eyes. On sudden exposure to flames or great heat while alive, victims screw their eyes shut tightly, producing a lateral line of skin at the canthi spared from thermal damage. Thus, in fires involving soot formation, the absence of soot particles in the fold formed on squeezing the eyes shut is conspicuous (Fig. 12.8). Crow's feet have also been described in the context of explosion burns, high-voltage accidents (see Chap. 13), and gunshot injury in the form of gunshot deposits. Crow's feet are interpreted as a sign of vitality in all the cases mentioned here.

Once the eyes are tightly shut, exposure to extreme heat or naked flames may cause only the tips of the eyelashes to be scorched.

Soot Particle Inhalation. Soot detected in the deep airways (trachea, main bronchi, and peripheral bronchi) at autopsy can only be explained by active aspiration of soot particles, thus indicating that the victim must have been alive at the time of the fire (Fig. 12.9).

Table 12.3 Vital signs and reactions, as well as thermal injury incurred post-mortem

Vital signs/reactions	Postmortem thermal injury
Deep soot aspiration (unreliable: traces of soot particles only in and around the respiratory orifices!) (Fig. 12.9)	Heat-induced detachment of the skin and subcutaneous soft tissue (heat cracks) (Fig. 12.17)
Soot ingestion with soot particles in the stomach and/or upper small intestine (Fig. 12.10)	"Pugilistic attitude" in the extremities: heat-induced flexion with muscle and tendon shortening
Direct thermal airway injury (heat inhalation trauma)	Heat-induced fractures, e.g., cranial fractures and suture separation (Fig. 12.15)
Smoke inhalation: detection of COHb in postmortem blood using chemical toxicological methods, possibly also cyanide determination	Thermal hematomas: intracranial epidural accumulation of bright red crumbly dried blood following heat-induced shrinkage and detachment of the dura mater from the inner surface of the skull (Fig. 12.14)
Crow's feet: lines of skin at the canthi of the eyes spared from heat injury and showing no traces of soot due to tightly shut eyes (Fig. 12.8)	Heat-induced protrusion of the tongue, traces of soot in the outer airways (Fig. 12.19)
Thermal blisters; serous or gelatinous content with leukocytes and proteins (fibrin) indicates these were incurred while alive	Postmortem thermal blisters contain hemolytic liquid with no leukocytes or fibrin
Fat embolism in the lungs, possibly also in the kidneys: sign of vitality at the start of the fire (DD: other causes of fat embolism)	Heat-induced shrinkage of the epicranial aponeurosis

Soot Particle Ingestion. Similarly, soot particles can only reach the lower section of the esophagus (Fig. 12.10), the stomach, and the duodenum via active ingestion; thus again, vitality at the time of the outbreak of fire can be assumed if soot particles are found in the stomach or intestinal mucosa.

Combustion Gas Inhalation. In cases where combustion gas is actively inhaled, as frequently seen in smoldering fires or fires producing significant volumes of smoke and soot, increased levels of carbon monoxide can be detected in blood using chemical toxicological analysis.

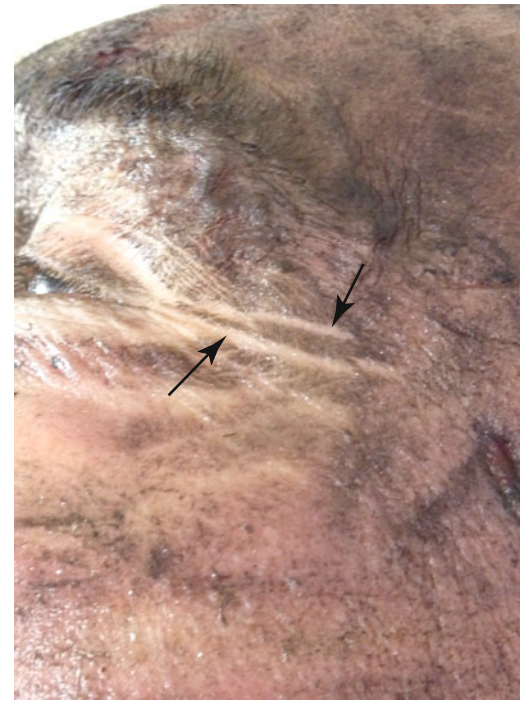

Fig. 12.8 Sparing of the canthi of the eyes (*arrows*) from thermal injury or soot particle deposits as a sign of vitality

Depending on the materials burned in a fire, high concentrations of cyanide are detectable in postmortem blood and tissue samples.

Inhalation Trauma. If dry air with a temperature of around 350 °C is inhaled, the temperature in the laryngeal space will still measure around 159–182 °C, while no elevated temperature levels are detectable at the branching of the main bronchi. However, if the temperature of inhaled dry air measures approximately 500 °C, the temperature in the laryngeal space will reach between 267 and 327 °C and the temperature at the branching of the main bronchi around 50 °C. Inhaling such hot dry air causes injury to the respiratory epithelium, usually in the upper airways. In the case of moist air or steam inhalation, however, significantly lower temperatures are sufficient to cause injury to the lining of the airways due to the greater heat conductivity of these compounds. As a result of reactive hyperemia, distinctly reddened mucosa of the larynx and particularly the upper third of the trachea are seen macroscopically at autopsy. Microscopically,

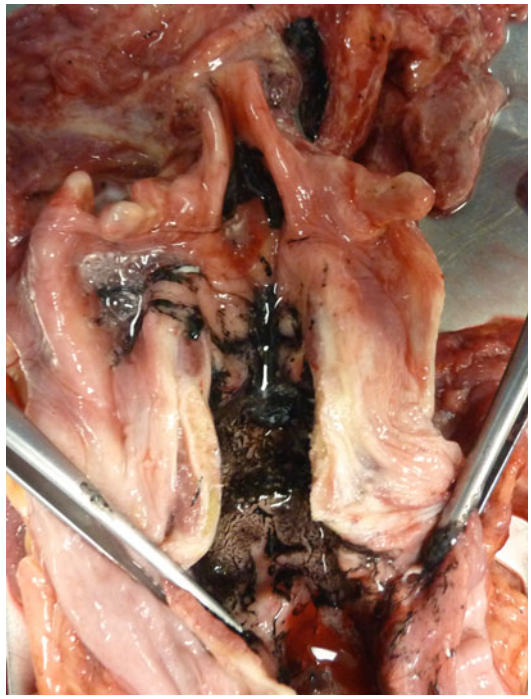

Fig. 12.9 A coating of soot particles on the laryngeal mucosa and neighboring trachea following active inhalation as a sign of vitality at the time of the outbreak of fire

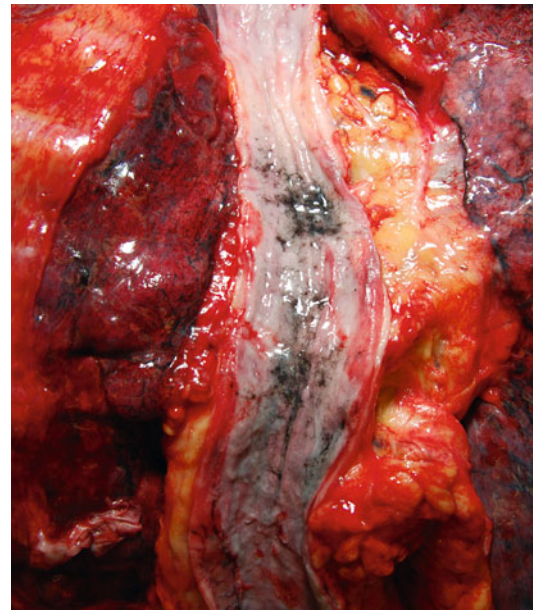

Fig. 12.10 Soot ingestion with a coating of soot in the medial and lower third of the esophagus

characteristic findings include cell nuclei or respiratory epithelium cell elongation in a luminal direction with swollen *goblet cell-like epithelial cells*, vascular hyperemia in subepithelial soft tissue, marked mucosal edema with lymphangiectasis and hemorrhages, as well as frequent soot particle deposits (Fig. 12.7), which can sometimes also be detected microscopically in lung parenchyma. Where inhalation trauma is survived in the first instance, extensive necrosis of the upper airway mucosa may ensue with secondary bacterial colonization, as well as purulent and purulent necrotizing tracheitis, bronchitis, and bronchopneumonia.

Iatrogenic Thermal Injury. Occasionally, thermal injury is incurred in the setting of medical treatment, e.g., electrocautery-induced explosion during eye surgery while the patient receives 100 % oxygen. This type of injury usually affects unconscious patients who, e.g., due to inattention during surgery, incur skin damage from the electrocauterizer or perhaps from excessively long exposure to hot-water bags (Fig. 12.11); defective hot-water bags have also been known to cause injury.

12.1.2 Postmortem Findings and Thermal Injury

Numerous findings may develop post-mortem as the result of a body's exposure to heat; noteworthy among these are:

"*Pugilistic Attitude*" *of the Extremities*: Exposure to heat causes flexion of the extremities on contraction of muscles and tendons (heat rigor). Shoulder abduction, elbow and wrist flexion, as well as incomplete leg extension, sometimes with foot drop and upper ankle separation (Fig. 12.12), are typical, resulting in the so-called pugilistic attitude.

Epidural Heat Hematomas: Exposure to high temperatures between the skull and the hard meninges, i.e., epidural, can cause an accumulation of dried brown, brick-red, or clay-like blood (Figs. 12.13 and 12.14); this is an epidural extravasate, often associated with cranial suture separation, dura mater rupture, and heat-induced fractures of the bony cranial vault

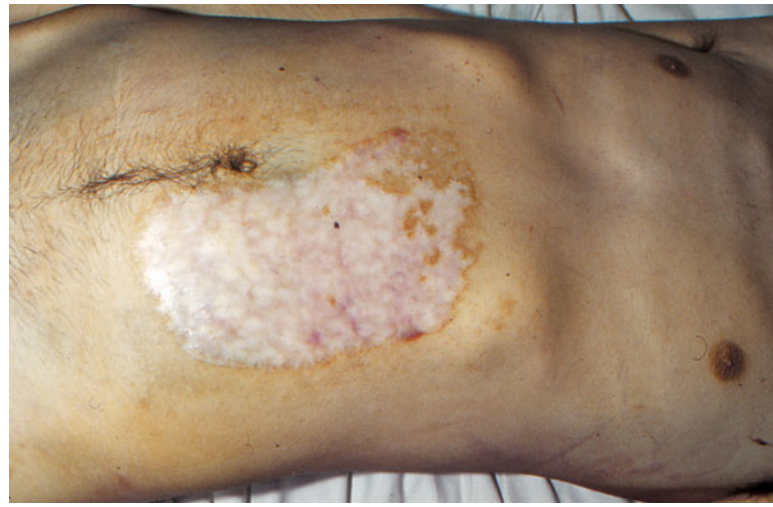

Fig. 12.11 Local iatrogenic thermal injury: skin burn in a cancer patient due to excessively long exposure to a hot-water bag

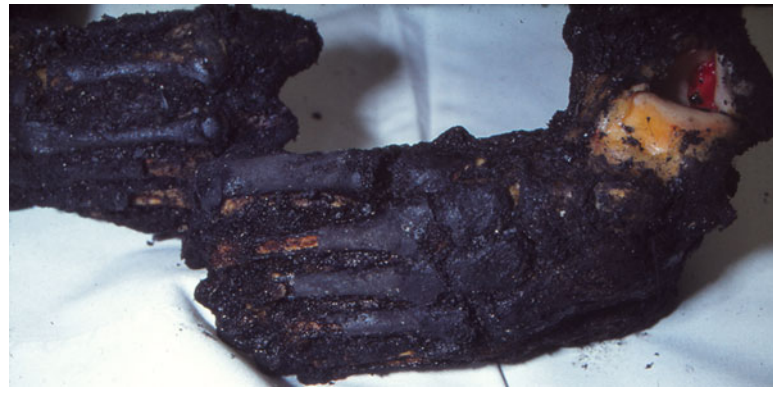

Fig. 12.12 Fire-related foot drop in a charred foot with fire-related upper ankle separation

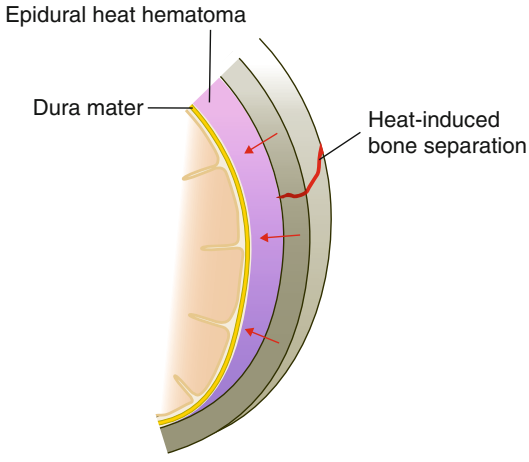

Fig. 12.13 Epidural heat hematoma. Dried out masses between the hard meninges and the inner table of the cranial vault (epidural) following intense exposure of the head to fire and heat-induced bone separation (not a sign of vitality)

(Fig. 12.15). Fractures should be investigated according to localization, type, and course in order to exclude preexisting fractures from blunt trauma to the head prior to the outbreak of fire.

Exposure to heat can also cause blood in the vascular system and heart cavities to transform into dried claylike masses, which in some cases fill the heart cavities (Fig. 12.16).

Heat-Induced Skin Separation: Exposure to heat causes vaporization of the subcutaneous fatty tissue leading to skin separation and subsequent shrinkage. Relatively smooth straight-edged lacerations in the skin (Fig. 12.17) and subcutaneous fatty tissue are visible. Intense exposure to heat causes the chest and abdominal cavities to rupture; this phenomenon can be mistaken for stab or incised wounds in individual cases. Marked shrinkage of the epicranial aponeurosis above the cranial vault can be seen.

Fig. 12.14 Virtually unilateral epidural heat hematoma with dried brick-red blood on the dura mater following removal of the cranial vault (not a sign of vitality)

Fig. 12.15 Heat-induced separation of sutures and bones of the cranial vault (not a sign of vitality)

Fig. 12.16 Heat-induced transformation of blood in the major vessels of the heart to a claylike mass

Soot Particles in the Airways. Neither fine nor coarse soot particles in and around the respiratory orifices (nostrils, mouth) are considered a sign of vitality, since passive spread or deposition of soot particles is always possible (Fig. 12.18).

Heat- or Fire-Induced Protrusion of the Tongue (*Fig*. 12.19). Extremely high temperatures, by vaporizing tissue fluid, cause heat-induced protrusion of the tongue, whereby the charred tip or anterior third of the tongue projects through the likewise charred lips. Microscopically, the tongue tissue has a honeycombed appearance.

Heat Blisters. In contrast to heat blisters incurred ante-mortem, postmortem heat blisters do not contain leukocytes. In case of doubt, a differential diagnosis can only be made on the basis of microscopic analysis of the blister fluid content.

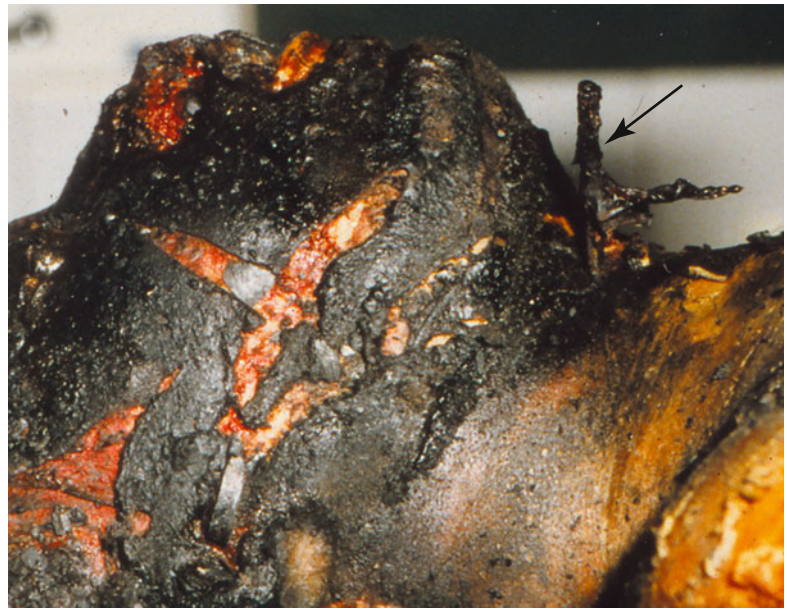

Fig. 12.17 Smooth, straight-edged heat-induced skin rupture (no incised wounds), as well as a screwdriver (*arrow*) in the soft tissue of the neck in a homicidal setting (arson to conceal homicide)

Fig. 12.18 Soot particles in the outer respiratory orifices from a smoldering fire (not a sign of vitality)

Thermal Damage to Hair. Changes in hair color or charring is seen on exposure to dry heat of increasing temperatures (Table 12.4). Hair singed by heat shows crinkling and grayish-yellowish discoloration at the tips (Fig. 12.20).

In the case of antemortem burning or combustion gas inhalation, carboxyhemoglobin levels in blood are determined, usually demonstrating increased (>15 % COHb) or lethal values (from approximately 40 % COHb). Carbon monoxide has an approximately 300-fold greater affinity for hemoglobin than oxygen molecules, which are thus forced out of the Hb bond (anoxic asphyxia). Even low concentrations of CO in ambient air can cause lethal intoxication.

Characteristic findings at external and internal autopsy in the case of lethal carbon monoxide intoxication include:

- Bright red livor mortis (including nail beds)
- Salmon-colored muscles
- Cherry-red postmortem blood

Fig. 12.19 Charred fire victim showing heat-/fire-induced protrusion of the tongue

Table 12.4 Dry heat-induced changes to hair according to temperature

Approximate temperatures (dry heat) (°C)	Heat-induced changes
65	Turns brownish
140	Turns yellow brownish
200–250	Turns yellow whitish, initially at the tips; hair crinkles due to heat, becomes increasingly brittle
300–400	Charring

- Increased COHb level (usually 40 % COHb and above)
- Possible inhalation and ingestion of soot
- Thermal damage particularly to the upper respiratory tract mucosa

In cases where a fire has involved the burning of nitrogenous polymers, the presence of nitrogen oxide due to a lack of oxygen as well as hydrogen cyanide inhalation should be considered. In addition to immediate death due to combustion gas inhalation or local second or third degree burns affecting >50 % of BSA, late fatalities as a consequence of burn disease are seen.

Burn Disease. In cases where burns are (initially) survived, burn disease develops out of the interplay between burn shock, the intoxication-like flood of pyrotoxins out of the heat-damaged tissue, protein loss via wound exudate, as well as secondary infection, particularly of necrotic tissue. In cases such as these—and even at short survival times—fat embolism in pulmonary tissue, a vitality marker, is seen. The extent of fat embolism in burn disease, however, can vary significantly, hence the general rule: always take lung samples from all pulmonary lobes for histological investigation of fat embolism. Occasionally, fat embolism is also seen in the renal glomerular loops of fire victims. Survival in the case of burn disease is unlikely and depends on the degree of injury. The following findings and causes of death are seen in late deaths:

- Hyperkalemia following extensive exposure to high temperatures
- Shock: Toxic organ damage due to protein loss (pyrotoxins)
- Pneumonia and pulmonary edema
- Sepsis
- Acute renal failure
- Thrombosis and embolism (pulmonary and renal fat embolism)
- Stomach and duodenal ulcers (stress ulcers)

Depending on legislation in the country in question, another consideration to be borne in mind in the case of late death is that accidental death or death occurring on the way to work or at work due to heat or fire may need to be reported to the authorities.

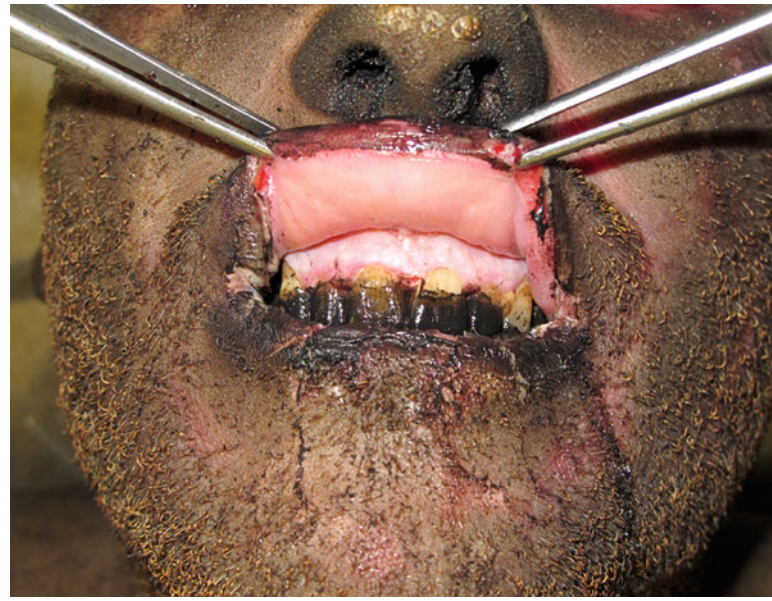

Fig. 12.20 Singed, yellowed, and crinkled beard hair, soot in the nostrils, bright red oral mucosa, and soot deposits on the front of teeth due to slightly opened mouth

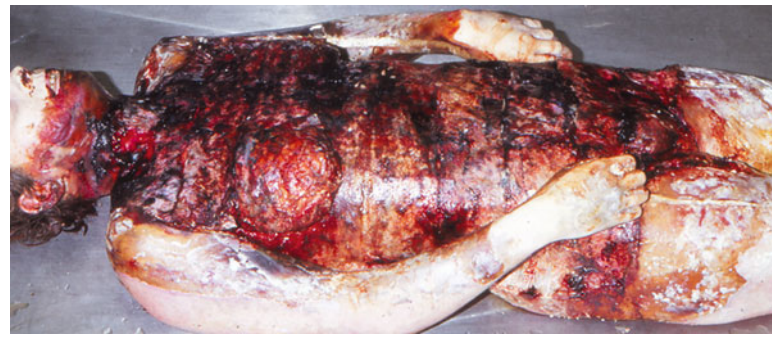

Fig. 12.21 Suicidal self-immolation by burning: 60 % of the BSA, survival time 12 days

Thermal injury is relevant in forensic medical practice in a variety of contexts:

- Homicide (homicidal arson, less common; more commonly arson to cover homicide).
- Suicidal self-immolation (Fig. 12.21): The victim douses himself/herself with a flammable liquid (caution: pay attention to fire-related flow marks!), possibly as a simulated motor vehicle fire.
- Death due to accidental fire, e.g., falling asleep with a burning candle, cigarette (Fig. 12.22), cigar, tea light, or children playing with fire.
- Abuse-related thermal injury: Contact burns from burning cigarettes, forcing a hand into hot water.
- Scalding as a result of placing care-dependent individuals in excessively hot bath water.
- Incidents involving "flash fires," which can cause acute respiratory arrest, laryngo- and/or bronchospasm, as well as acute fatal inhalation heat shock
- In large fires, fire-related oxygen consumption is possible, leading to atmospheric asphyxia in fire victims due to insufficient ambient oxygen but without direct fire- or heat-related injury to the body.
- In cases of arson with thermal injuries on the arms (or the face) of the perpetrator following deflagration of gas (Fig. 12.23).

Homicide in the form of dousing an intoxicated individual with petrol and then igniting a fire, setting the bed (or house) of a sleeping person on fire for the purposes of causing death, as well as dousing an individual with hot or boiling liquid with the intention of causing fatal scalding

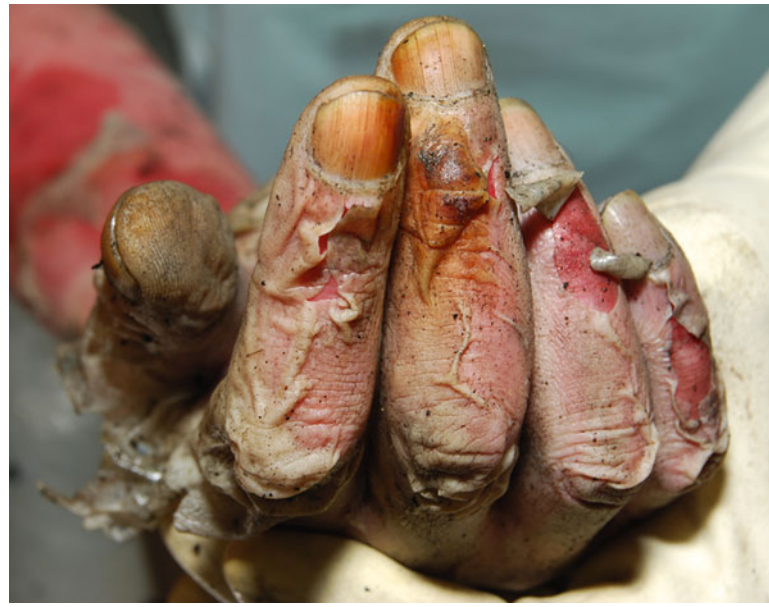

Fig. 12.22 A fire victim with finger and nail discoloration still detectable on the index and middle fingers of the right hand (right-hander) due to chronic nicotine abuse; postmortem formation of washerwoman skin following the effects of exposure to water used by firefighters

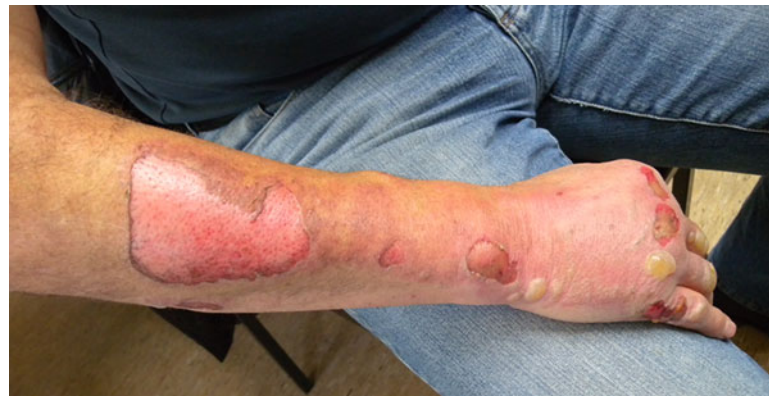

Fig. 12.23 An arsonist with injuries due to deflagration of gas

is known both in the forensic medical literature and in forensic practice.

12.2 Heat Disorders

Heat disorders can be caused by hot humid weather, particularly when the relation between heat production (febrile disease, physical work) and heat loss (tight-fitting clothes) is disrupted. Pathophysiologically, an interaction between electrolyte imbalance (salt loss), a rise in body temperature, exposure of the unprotected cranium to heat (cerebral hyperthermia), and possibly also insufficient fluids (exsiccosis) is seen. Symptoms are characterized as heat exhaustion, heat rigor, sunstroke, heatstroke, and heat cramps:

Heat Collapse/Heat Exhaustion: A breakdown in circulatory regulation while temperature regulation of the organism is initially maintained. Individuals performing heavy physical exertion are primarily affected, e.g., soldiers following a long march in blazing heat. Primary circulatory collapse is a result of a marked increase in blood circulation to the skin, so-called heat exhaustion. When water and electrolyte loss is the primary causal factor in heat collapse, secondary heat collapse (dehydration exhaustion) is referred to.

Heat Rigor: Acute impairment of respiratory excursion due to the sudden onset of heat rigor

over the chest wall, caused primarily by the abrupt occurrence of extremely high temperatures, thus depriving the organism of an adaption phase.

Sunstroke: Direct sunlight to the unprotected cranium affects the central nervous system locally, causing impaired cerebral function, possibly also preceded by neurological deficits. From a pathophysiological perspective, sunstroke is equivalent to heatstroke.

Heatstroke: This involves direct damage to the regulatory center in the brain and a breakdown in temperature regulation due to increased body temperature, particularly cerebral temperature. The body is exposed to greater heat than it is able to dissipate, while sweat secretion decreases. Certain occupational groups, such as those working in high ambient temperatures, are at particular risk.

Heat Cramps: This phenomenon is seen mainly in the case of extreme physical exertion in radiating heat, with an accompanying loss of electrolytes due to perspiration. Dehydration and sodium chloride loss ensue. Tetanic cramps with vasodilation of cutaneous blood vessels and an increase in pulse rate are seen. Cardiac arrhythmias are considered the direct cause of death here.

12.3 Hypothermia, Cold, and Frost

Case Study

The frozen body of a partially clothed woman was found on the outskirts of a large city on a January evening as outdoor temperatures reached −4 °C by day and −11 °C at night. Various items of clothing (jacket, skirt, panties, one shoe, and one stocking) were found strewn about in the immediate proximity, along with empty beer, vodka, and brandy bottles. Due to the woman's partially clothed state, a sexual offense was initially suspected. No papers indicating the identity of the victim could be found. On external examination of the body, large irregularly shaped areas of reddened skin on the extensor side of both knees were apparent, while relatively light red livor mortis could be seen. No injuries suggestive of a physical confrontation were present. On thawing, the body showed small and large irregularly shaped erosions in the gastric mucosa at autopsy; in addition, the body gave off a conspicuous aromatic smell. Blood alcohol determination yielded a value of 1.78 %. The spectrum of congeners (including so-called fusel alcohols) in postmortem blood corresponded to the alcoholic substances found.

Under normal circumstances, warm-blooded (homeothermic) organisms are able to maintain their own body temperature even at significantly lower ambient temperatures. The organism's heat loss is reduced via peripheral vasoconstriction and piloerection, while heat generation is increased by means of shivering and biochemical thermogenesis. If the body's ability to regulate its temperature in a cold environment is impaired, the body temperature decreases and hypothermia ensues.

Hypothermia: A drop in body temperature <35 °C causing hypothermia of varying degrees (mild to severe hypothermia).

The body's ability to compensate for cold decreases ever more rapidly as the difference between body temperature and ambient temperature grows, i.e., significant temperature differences cause rapid cooling, followed by slower cooling as the gap between body and ambient temperatures closes. At the same time, the surface area giving off heat from the cooling organism is an important factor: the greater the surface area, the more rapid the cooling.

Important: **The larger the surface area of thermal exchange**, **the greater the difference in temperature between ambient and body temperature**; **at the same time**, **the lower the body's ability to compensate for heat loss**, **the faster hypothermic death ensues**. **Children are**

Table 12.5 Survival times according to water temperature

Water temperature (°C)	Survival times (approximate)
20	40 h
15	5 h
10	3 h
5	2 h
0	30 min, acute danger of death thereafter

From Lignitz (2007)

more vulnerable to hypothermia since the surface–volume ratio increases with decreasing body size.

Hypothermic deaths are seen primarily during the cold season. In principle, hypothermic death is possible even at room temperature, e.g., in insufficiently (or un-)heated homes at temperatures between 15 and 20 °C, in the case of cooling in unconscious individuals following apoplexy or in a coma, or as a result of long anesthesia times during surgery. Alcohol consumption, wasting diseases, physical exhaustion, and a high wind-chill effect may increase an individual's susceptibility to hypothermia. Elderly people, individuals suffering dementia, as well as confused or disoriented individuals are more commonly affected. A distinction is made between dry hypothermia and hypothermia in water (immersion hypothermia).

In the case of immersion hypothermia, the protective effect of clothing is lost since these are wet. Both the duration of cold exposure and the water temperature are crucial factors in terms of prognosis (Table 12.5). It is assumed that many cases of death by drowning are preceded by hypothermia. Temperatures below 15 °C lead to rapid hypothermia, while the cooling process is accelerated by physical activity in water (e.g., swimming); here, the speed of cooling is increased by 30–50 %. Although early vasoconstriction may reduce heat loss, the influence of alcohol causes vasodilation and hence even faster cooling of the organism.

Core body temperatures between 20 and 25 °C are seen as critical thresholds in hypothermia. Only in a scant number of cases—and after months of therapy—have victims been known to survive cooling to 18–20 °C.

Lethal cold shock following unintentional immersion in cold water not above 10 °C is less common. In some cases, both lack of oxygen and cardiac arrest can be survived significantly longer by hypothermic than by normothermic individuals. Since severe hypothermia can provoke of state "apparent death" (vita minima, vita reducta), all hypothermia victims should be examined with particular care. Muscular rigidity should not be mistaken for rigor mortis. In the case of uncertainty, resuscitation should always be attempted; particularly in cases of hypothermia, successful resuscitation even after prolonged cardiac arrest has been described.

Hypothermia often affects socially disadvantaged individuals and/or addicts (lack of protective clothing, inadequate diet, homelessness, alcoholism, or drug addiction). Poorly supervised individuals suffering from dementia (e.g., getting lost during the winter), as well as immobilized trauma patients, possibly with prior hypothermia, represent vulnerable groups; moreover, exhaustion is also a risk factor for hypothermia. Elderly people are frequent victims. In all cases of fatal hypothermia, the causes leading to a hypothermic state—and whether any of the following factors played a role—needs to be established: alcohol or drug abuse, medication, dementia, cachexia, organic disease, malnutrition, and physical disability.

Paradoxical Undressing: This describes the irrational behavior seen in hypothermia victims whereby, due to a paradoxical feeling of heat, they either completely or partially discard their clothing (frequent removal of clothing from the waist down, urine-soaked clothes). Finding victims in a state of undress can lead to the erroneous assumption that a sex offense has taken place. Occasionally, the phenomenon known as "terminal burrowing" is seen: the body is found in a poorly accessible space, such as an enclosed corner, under a table, or in a wardrobe, which can similarly lead to incorrect assumptions about the circumstances of death. This phenomenon is the result of hypothermia-related sopor and an associated loss of powers of judgement, disorientation, and increasing incapacitation. Central nervous disorders also include impaired decision-making, loss of ability to act, as well as a

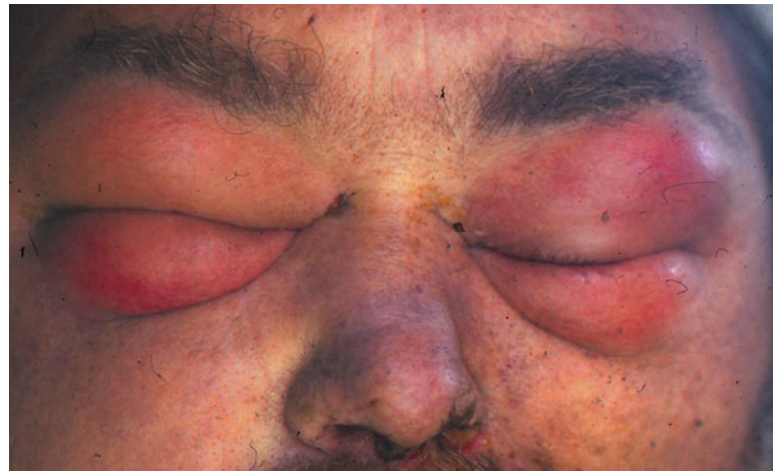

Fig. 12.24 Marked eyelid edema in fatal hypothermia

subjective perception of warmth, all of which are seen even in mild hypothermia.

Important scene-of-death findings and factors in the case of fatal hypothermia may include:

- Victim's clothing (one, two, or three layers).
- Condition of clothing (particularly whether wet or dry).
- Room or ambient temperature (measured at the level of the body).
- Core body temperature (measured as deep rectal temperature with an appropriately calibrated specialized thermometer).
- Ground temperature (particularly where there is a large contact surface between the body and the ground/cold floor).
- Other sources of cold (open window or door, draft).
- Indications of preexisting hypothermia (antemortem febrile disease).
- Perniones (chilblains) on the body.
- Although hypothermia-related eyelid edema (Fig. 12.24) is seen, this could have other causes.

Hypothermia is subdivided into four (partially overlapping) phases according to severity (Table 12.6). In certain cases, there is no strict correlation between body temperature and clinical symptoms.

Death due to hypothermia can occur following varying exposure times ranging from 1.5 to 12 h; body temperature only starts to drop after a plateau phase during which the organism is still able to maintain a constant temperature. Medical condition, state of clothing, and ambient temperature are crucial factors here. The lower the temperature, the shorter the exposure time to death. In the case of dry hypothermia and a temperature of −10 °C, exposure times of between 3 and 6 h are assumed. Due to water's greater thermal conductivity, the agonal period in immersion hypothermia is significantly shorter than in dry hypothermia.

Immersion hypothermia may cause immobility (muscular rigidity), leading to drowning during the agonal period. Below temperatures of around 28 °C, ventricular fibrillation due to further drops in core body temperature becomes increasingly likely. However, lower core body temperatures following relatively rapid cooling (e.g., children falling through sheets of ice) have been survived when treated appropriately (rewarming using extracorporeal circulation). In addition to findings at the scene of death, characteristic morphological findings are generally seen at external and internal autopsy of fatal hypothermia:

1. *Bright red livor mortis*: Although often present, this is not evidence of hypothermia, since this phenomenon is also seen on transferal of a body to a cold ambient temperature and in carbon monoxide poisoning.
2. *Cold erythema*: Bluish-livid or reddish areas of skin, typically on the outer side of the upper thigh, the extensor side of the knee, the wrists, and the extremities.

Table 12.6 Clinical stages of hypothermia

	Phase 1: excitatory phase	Phase 2: adynamic phase	Phase 3: paralytic phase	Phase 4: apparent death (vita reducta)
Body temperature (°C)	36–33	33–30	30–27	<27
Musculature	Shivering (the organism's mechanism of compensatory thermal regulation)	Reduced active muscle tonus	Increased passive muscle rigidity	Caution: muscular rigidity should not be mistaken for rigor mortis
Cardiovascular system	Tachycardia	Sinus bradycardia: increased resistance due to peripheral vasoconstriction	Bradyarrhythmia, further increase in peripheral resistance due to greater blood viscosity	Reduced vital functions, cardiovascular arrest due to ventricular fibrillation
Respiration	Stimulation, possible hyperventilation	Increasing central respiratory depression	Bradypnea, intermittent apnea	Respiratory arrest
Consciousness, pain perception	Increased vigilance, painful extremities, confusion	Disorientation, apathy; pain subsides	Unconsciousness, reduced compliance, loss of reflexes	Unconsciousness, reflexes absent

Modified according to Lignitz (2007)

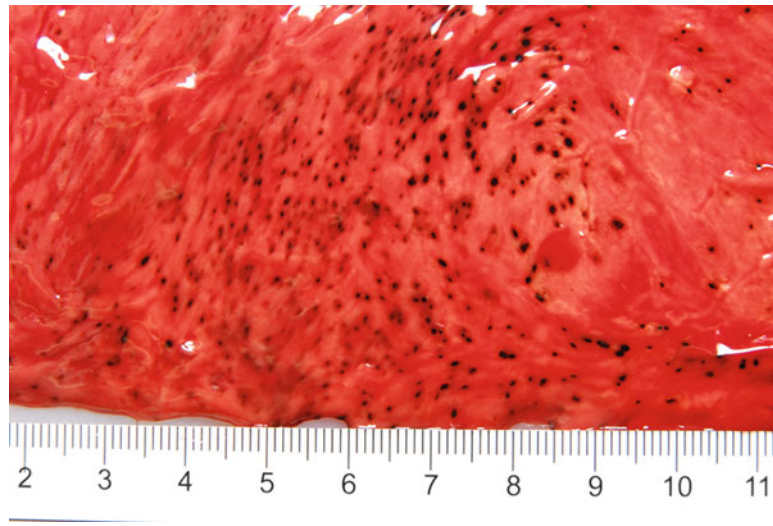

Fig. 12.25 Blackish hemorrhagic erosions in the gastric mucosa (Wischnewsky spots) in a case of fatal hypothermia

3. *Hemorrhagic erosions of the gastric mucosa* (*Wischnewsky spots*): Black spots on the gastric mucosa (Fig. 12.25); histologically, these are wedge-shaped infarct-like erosions of the gastric mucosa, blackish in color due to hematinized blood.
4. *Histological detection of fat vacuoles* in the setting of uniform fatty degeneration, particularly of the renal tubules (Fig. 12.26), where it can be found as frequently as frost erythema and Wischnewsky spots, possibly also fatty degeneration of cardiomyocyte cytoplasm.

A further histological finding made against the background of increased blood viscosity in fatal hypothermia includes microthrombi in peripheral vessels and hemorrhagic infarction in the relevant supply area (e.g., infarction of an

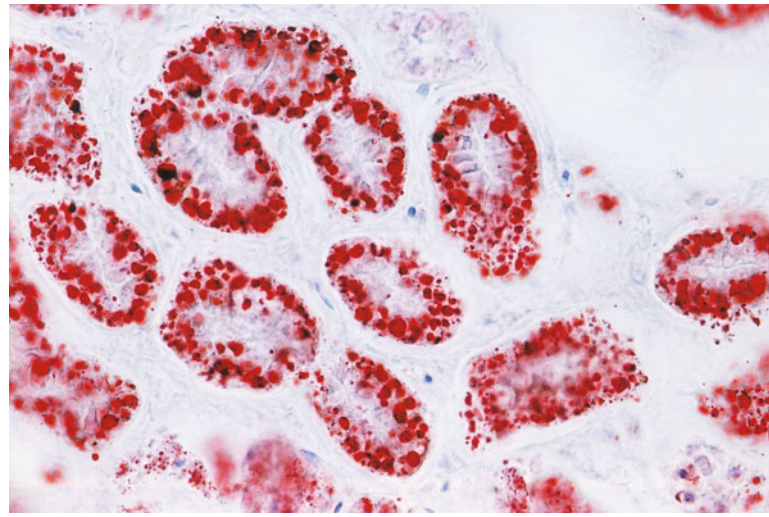

Fig. 12.26 Droplet-like fatty degeneration in the epithelial cells of the renal tubules in a case of fatal hypothermia (Sudan III stain; ×200)

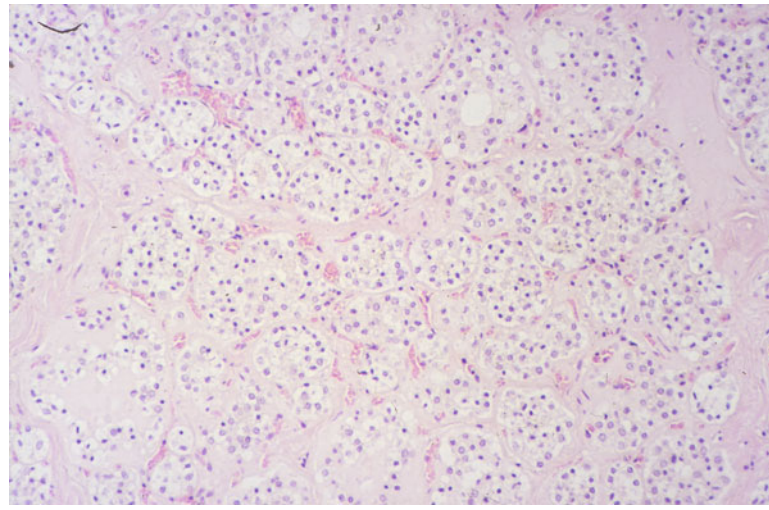

Fig. 12.27 Fatal hypothermia with marked thyroid follicular colloid depletion

intestinal segment). Although pancreatic hemorrhage is rarely detected macroscopically in fatal hypothermia, fine vacuolization of the secretory cells of the exocrine pancreas is occasionally seen microscopically. Hemorrhage in the iliopsoas muscle has been reported in a few cases, while a relatively bloodless spleen and a finely puckered splenic capsule are more commonly seen. In addition, synovial hemorrhage, subendocardial hemorrhage, as well as blood particles in joint fluids as a result of fatal hypothermia have been described. Increased thyroid follicular colloid depletion in the setting of fatal hypothermia has been reported occasionally (Fig. 12.27).

The macroscopically detectable morphological findings described above may be largely absent in the case of rapid fatal hypothermia. Thus an evaluation of the general situation (how and where the body was discovered, autopsy findings, and microscopic findings) is mandatory in all cases of suspected hypothermia. At the same time, chemical toxicological analysis is able to clarify what contributed to an individual reaching a hypothermic state when all other plausible explanations are lacking, e.g., apoplexy, craniocerebral trauma, and when no indication of intoxication is found.

Local Cold Exposure. A distinction is made between general exposure of the organism to cold, resulting in hypothermia, and local cold exposure in the form of frostbite to peripheral parts of the body. Fingers, toes, hands, feet, ears, and the tip of the nose are most commonly affected. Tight-fitting clothes increase the risk of local frostbite. However, frostbite is of lesser forensic medical relevance, occurring primarily in individuals pursuing winter sports, inadequately equipped tourists (e.g., in the Alps), and mountaineers. Frostbite is subdivided into several stages:

1. *First-degree frostbite* (*frostnip*)*—dermatitis congelationis erythematosa*: The skin blanches due to vascular constriction, becoming numb or painful, then reddens and swells, often causing itching.
2. *Second-degree frostbite—dermatitis congelationis bullosa*: Subepidermal blisters containing serous or hemorrhagic fluid form on rewarming areas of skin exposed either to extreme cold or to cold for prolonged periods.
3. *Third-degree frostbite—dermatitis congelationis gangraenosa* (*escharotica*): Bluish-black discoloration of extremities (dry gangrene) with a mummified appearance. The damaged area is demarcated; bacterial infection leads to wet gangrene.

Local frostbite causes tissue damage with microthrombosis and obliterating vessels, while neuritis and neuralgia are also possible.

Chilblains (*Perniones*). Chilblains are bluish-red, edematous swellings with unclear borders. They may appear even at mild drops below room temperature, primarily on the extensor side of fingers, toes, and the knee; the ileac crest area is occasionally also affected. Attention should be paid to chilblains as early as at external examination of the body at the scene of death!

Autopsy examination serves to establish the cause of death and/or provide evidence of fatal hypothermia. Moreover, the cause of hypothermia needs to be established by means of chemical toxicological analysis (e.g., alcohol, drug abuse), postmortem biochemical analysis (e.g., hypoglycemia), and gleaning information about the victim's medical history (age-related dementia, psychiatric disease).

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Electricity, Lightning, and Gases 13

Case Study

The lifeless, fully clothed body of a 78-year-old female was found in the middle of winter by the mobile nursing service on the bathroom floor of the apartment in an old building where the victim lived alone. The bathtub had been filled with water; next to it lays an intact, connected hairdryer, while an old oil heater stood at the side of the room. The emergency physician called to the scene confirmed death (bright red livor mortis, advanced rigor mortis), detected a pacemaker subcutaneously beneath the left clavicula, and found several packs of medication, some opened, some empty: diazepam, a β-blocker, an antidepressant, as well as antithrombosis stockings. The victim's nail beds were also bright red, and an old, healed scar running lengthways along the sternum suggested previous cardiac bypass surgery. Since no further medical intervention was called for, and once external examination was complete, the emergency physician filled out all the necessary forms, giving the cause of death as unclear. The subsequent police investigation, as part of which a forensic autopsy was performed, concluded the cause of death to be carbon monoxide poisoning (COHb = 32 %). A technical expert later determined the oil heater to be defective.

Domestic accidents involving electricity, as well as other types of accident involving power lines, the use of electricity with suicidal intent, and high voltage are seen in medical forensic practice. Electricity is rarely used with homicidal intent. Knowledge about the effects of electricity has been gained from its use in executions (the "electric chair"). Electricity is also used as a weapon to incapacitate an individual for the purposes of detainment (the Taser gun).

Death by electrocution is primarily accidental or work-related; it is occasionally seen in a homicidal context (sometimes falsely interpreted as alleged accidental or suicidal death). In the case of suicide (e.g., hairdryer in the bathtub) and accidental death resulting from autoerotic activity, live conductors (cables) are attached to the genital or anal region, for example, or to the extremities. Incorrectly installed electrical devices and cables or poorly maintained devices can cause death; in such cases, charges of manslaughter are often brought against the responsible party. Moreover, evidence of a fatal electrical accident is relevant in terms of insurance law. Individuals with some knowledge of electricity have been known to use it as a means to commit suicide. Fatal injury due to mobile phones exploding—a rare phenomenon seen mainly in Asia—has been reported; the cause here is believed to be the use of cheap batteries, possibly in combination with extreme heat. Electric cushions and blankets, e.g., in a child's bed, can cause burns.

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13.1 Fatal Electrocution

Contact with current-carrying conductors can have various effects on the body:

- Electrical injury due to electrical stimulation: Muscles, nerves, and the cardiac conduction system
- Electrothermal injury due to local current-induced heat damage, e.g., local charring or cutaneous metallization
- Indirect injury, e.g., fall injuries following electrocution

In practice, a distinction is made between four voltage ranges: extra-low, low, high, and extra-high voltage. Household voltage is usually 220 V (USA, 110 V) with a frequency of 50 Hz (USA, 60 Hz). The low-voltage range goes up to 600 V, while the high-voltage range is between 600 and 1,000 V and higher.

However, the duration of current flow (contact time) and the strength of current passing through the body are the crucial factors in electrical injury. The current (I) is determined by the voltage (U) and the body's resistance (R) according to Ohm's law (($U=R\times I$). If the hand comes in contact with a current-carrying conductor, mild muscle contraction will ensue at a current of 1 mA; from 5 mA, the lower arm will be affected; up to 15 mA it is still possible to break contact, which is no longer the case from 25 mA. Currents of between 25 and 80 mA cause a rise in blood pressure and possibly also cardiac arrhythmias.

Skin resistance at the current entry and exit points can vary depending on corneal thickness, skin moisture (reduced resistance due to perspiration!), or protective clothing: gum boots serve a protective function, while walking barefoot on wet ground is particularly hazardous. In individual cases, the question of injury or fatal cardiac arrhythmia (generally ventricular fibrillation) depends on several factors. In addition to the current itself, its path and duration, the level of skin resistance, and the age of the victim, the crucial factor in electrocution is the point in time of the current surge in relation to the vulnerable cardiac phase. The "let-go current" is also a crucial factor: firstly, the current excites nerves causing sustained muscle contraction. Once local muscle control is lost, the conducting object can no longer be actively released, and the current continues to flow through the body. The "let-go" threshold is exceeded beyond 15 mA, the risk of fatal cardiac arrhythmia is increased from 50 mA, and values >80 mA cause loss of consciousness and ventricular fibrillation.

The alternating current usually found in households has a frequency of 50 Hz. The likelihood that a current alternating direction 50 times/s triggers fatal cardiac arrhythmia (by reaching the heart during the vulnerable phase) is significantly higher compared to the effects of direct current. Very high-frequency currents, however, penetrate the organism only slightly, while the thermal effects at the interface to the current-carrying conductor or electrode are greater. For this reason, high-frequency surgery is carried out in the 300- to 2,000-kHz range. Frequencies over 100,000 Hz are harmless, since nerves are no longer excited; at even higher frequencies, the current runs off body layers close to the surface and internal body structures remain unaffected.

The effects of current strength and voltage on the heart are shown in Table 13.1.

Skin resistance determines current strength in the body, whereby a distinction is made between various alternating-current ranges (Table 13.2). Alternating-current frequencies between 40 and 150 Hz are hazardous. Ventricular fibrillation is generally the cause of death in electrical accidents. The risk of fatality is greater when the current path between contact sites goes via the heart (arm–arm or arm–foot). However, the direction of the current is also relevant; in the case of a transverse current flowing from hand to hand, a lower proportion of the current flows through the heart compared with a longitudinal current flow. Current flow to other sites, such as between two fingers, usually only causes local thermal injury.

Electrical Burns. In the case of electrical fatalities, electrical burns at the point of contact with the conductor need to be sought on the body. Points of contact can be extremely small, uncharacteristic in appearance, or completely absent. Broad and moist contact surfaces in particular often develop no electrical burns at all. If electrical

Table 13.1 The effects of current strength and voltage on the heart

Current strength	Voltage	Effects on the heart in particular
<25 mA	Up to 65 V	Extra-low voltage, harmless
<25 mA	100–130 V	Low voltage: electrical effect; short muscle cramps, no injury
25–80 mA	110–380 V (usual domestic alternating current)	Low voltage: electrical effect; brief asystole and arrhythmia or (reversible) ventricular fibrillation, potentially life-threatening
80 mA–8 A	110–380 V	Low voltage: electrical effect; ventricular fibrillation (reversible), potentially life-threatening
>8 A	2,000–3,000 V (up to 100,000 V)	High voltage: electrothermal burns and acute asystole
>8 A	>100,000 V	Extra-high voltage: severe electrothermal burns (temperature up to 4,000 °C), charring, acute asystole; current transfer possible even in the absence of contact with the conductor ("electric arc")

Table 13.2 The effects of alternating current

Amperage	Effect
<0.5 mA	Perceptible with the tongue at most
From 0.5 mA	Perceptible tingling
From ca. 5 mA	Muscle excitation
From ca. 15 mA	Muscle cramps, flexion contractions, no longer possible to let go of the contact site independently
25–50 mA	Cardiac arrhythmia, possible loss of consciousness in the case of longer contact times
Ca. 50–80 mA	Risk of acute ventricular fibrillation
From 80 mA	Short contact times are sufficient for ventricular fibrillation; respiratory arrest due to respiratory muscle paralysis
From ca. 3 A	Electrothermal effect with tissue charring at the contact site

Note: In the case of direct current, limit values can be increased by a factor or four

burns are present, the following findings of electrothermal injury are usually made:

- Crater-like elevation of the skin around a central depression (sunken center) (Fig. 13.1).
- Pale, porcelain-like, or alabaster-colored borders.
- Particles of metal from the conductor may be "burnt into" the skin (so-called metallization), although this is not always the case. Metal particles can be detected with the help of spectrographic or histochemical analysis.
- Blister formation in the raised border of the electrical burn (honeycomb pattern) can be seen histologically.
- Fishbone-like deformation of epidermal basal cells and nuclear elongation can also be detected histologically (Fig. 13.2).

Although electrical burns are often specific for electrocution, they are not a sign of vitality unless there is microscopic evidence of a cellular reaction and/or local hemorrhage. From a differential diagnostic point of view, it may be necessary in individual cases to distinguish between a burn mark (contact burn) and an electrical burn. One important criterion here is the formation of an elevated border, as well as possible discoloration at the center of the lesion (Fig. 13.3).

Depending on the intensity of the local current, blackish charred contact sites can be seen on the skin, frequently on the fingers, and involving metallization that is often only detectable using microscopic analysis (Fig. 13.4). However, additional skin samples taken adjacent to the affected area should also be investigated, since metal particles (e.g., copper) in the skin of electricians' hands, for example, are found relatively often even in the absence of electrocution.

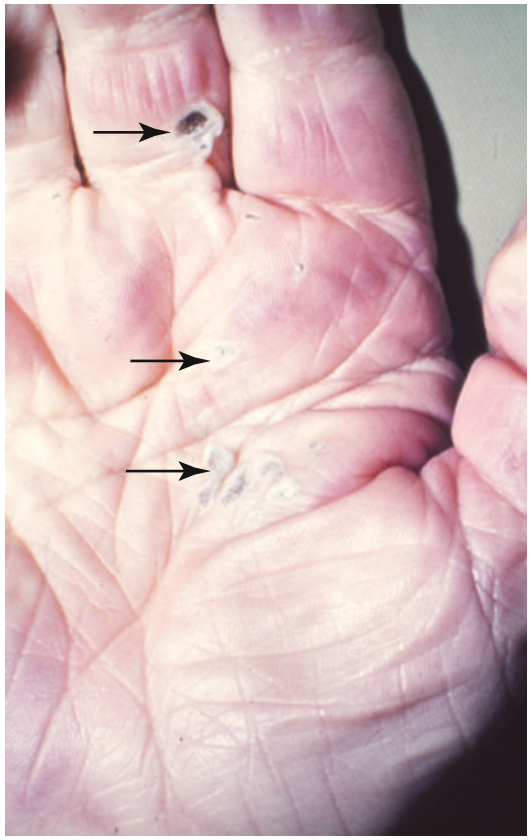

Fig. 13.1 Electrical burn. Trough-like electrical burn on the palm of the hand with multiple sunken areas

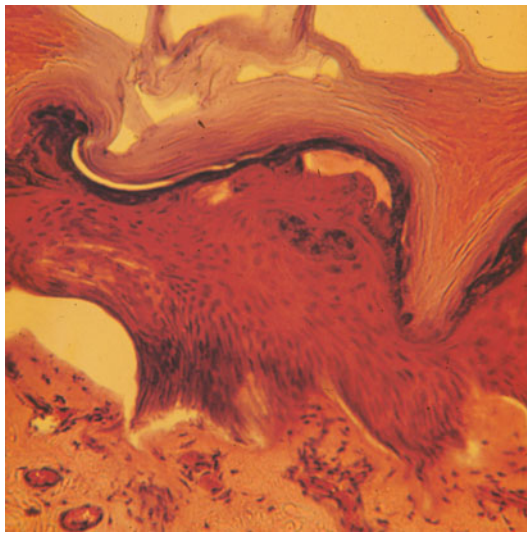

Fig. 13.2 Histological finding in an electrical burn: basal cell deformation, elongation, as well as palisade and fishbone-like arrangement of cell nuclei

Alongside electrical fatalities in the context of occupational accidents (subject to reporting requirements), electrocution in the bath merits particular attention in forensic medical practice.

Electrocution in the Bath. Occasionally, a fine linear reddening of the skin can be seen at the level of the water in the case of electrocution in the bath (Fig. 13.5).

Even in the absence of any findings, a fatality in the bath should always arouse the suspicion of electrocution. Since electrical burns can be incurred post-mortem, they do not represent a sign of vitality. Thus the diagnosis "electrocution" is often a *diagnosis of exclusion*. In equivocal cases, a technical expert needs to be consulted, not least to prevent further cases of electrocution. Acute asystole may occur up to some minutes following contact with a current. Even when electrocution is initially survived, electrothermal injury to the skeletal and heart muscles (infarct-like pattern of damage) can cause late fatalities in the setting of multiorgan failure (MOF), including kidney failure ("crush kidneys"). Whenever an individual is found lifeless in a filled or unfilled bathtub, particular attention should be paid to finding and identifying an electrical burn. Suspicious skin lesions should be investigated histologically. All conductors and current sources at the scene need to undergo technical inspection, while a careful record of the scene itself needs to be made. Cases are known where bodies have been "cooked" in the bathtub due to bathwater being heated by a hairdryer falling into the water.

When the diagnosis "death by electrocution" appears plausible only as a diagnosis of exclusion, the goal of any criminal investigation is to establish whether an accident, a suicide, or a homicide has taken place. In forensic medical practice, electrocution is usually accidental in nature; electricity is rarely used to commit suicide. It is essential to weigh up findings carefully while taking the background history, findings at the scene of death, as well as autopsy results into consideration. This may provide information about tetanic muscle cramps or a hand "getting stuck" on a conductor. Emitting a sudden scream followed by loss of consciousness and pulse is consistent with electrocution. Finding a body in

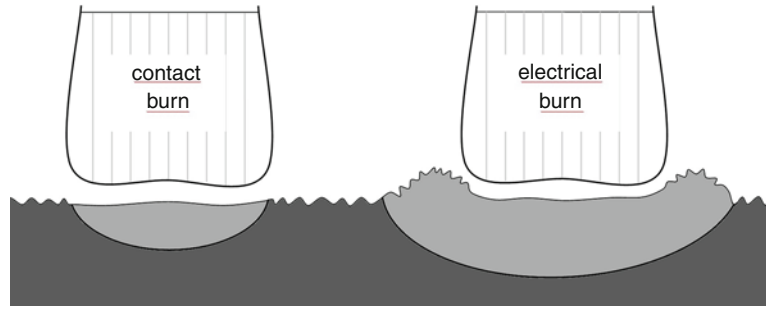

Fig. 13.3 Differential diagnosis between a burn mark (*contact burn*) and an electrical burn: skin burn mark *without* a raised border, electrical skin burn *with* a raised border

Fig. 13.4 Blackish charred electrical contact site on the skin of the finger

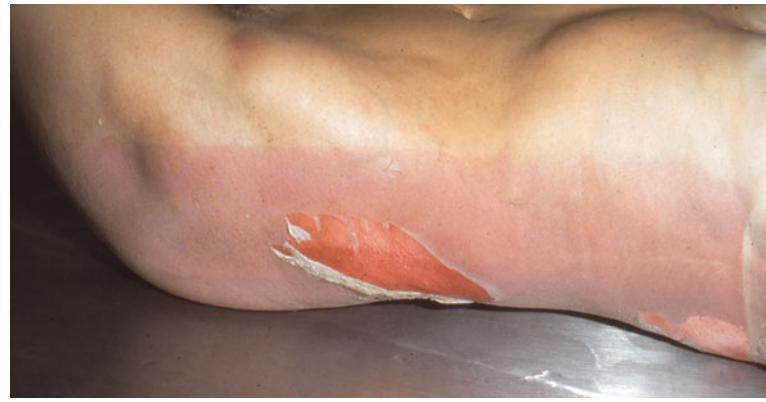

Fig. 13.5 Fine linear reddening of the skin at the level of the water in the case of electrocution in the bath

the vicinity of a power source should suggest the possibility of electrocution.

High-Voltage Accidents. In the case of high voltage, no direct contact is needed for current to pass through the body—being at a certain distance from the conductor is sufficient, e.g., a few centimeters from a 10,000-V conductor and several decimeters from a 100,000-V conductor. An electric arc with high current, extreme heat, and intense light is formed between the conductor and the body. Victims show charring on the conducting extremities and convulsive contortion of the facial muscles; eyelids may also be closed. When there is no electric arc and *flashover*

occurs, multiple brownish-blackish, densely arranged skin perforations can be seen.

Most high-voltage accidents occur in an occupational setting, almost always affecting males (working on overhead power lines, substations, transformers, and tall cranes that come into contact with high-voltage current, etc.). Brief contact with a current is often followed by a fall and associated fall injuries, which are sometimes erroneously interpreted as the cause of death. In the majority of cases, high-voltage accidents cause immediate death—direct contact with a power line is not necessary for this to occur, since an electric arc can be formed from an electric arc flashover. Temperatures within electric arcs of this kind reach several thousand degrees Celsius. Such cases are usually accidental, where the victim came too close to high-voltage power lines. The flashover distance for 100 kV is approximately 3.5 cm, increasing with increasing voltage. Contact may also be caused, for example, by urinating from a bridge onto high-voltage power lines. Suicide should be considered in individual cases (climbing up a high-voltage mast). Although most high-voltage accidents produce clear findings on the body and clothing, short but intense contact with a high-voltage conductor has been known to cause fatalities with only scant findings. A small electrical burn, as seen in low-voltage accidents, is often the only finding, and even this may be overlooked if localized on feet covered by shoes. However, high-voltage accidents generally produce the following classic constellation of findings, the intensity of which depends on the duration of current flow:

1. Mostly third-degree burns to the face and other exposed sites (Fig. 13.6). The formation of "crow's feet" (see Fig. 12.8) is possible, i.e., lines of spared skin at the canthi of the eyes in otherwise burned facial skin. Other lines of skin spared from burning suggest a final contraction of the facial mimic muscles. Linear sparing of this kind is also interpreted as a sign of vitality in high-voltage and electric arc accidents.
2. Possible metallization along skinfolds similar to "crow's feet." This finding is not necessarily a sign of vitality in high-voltage accidents, since electrically induced muscle contraction can also cause increased skinfold formation.
3. Scalp hair, eyebrows, eyelashes, and facial hair are often singed.
4. Depending on the duration of current flow, both direct and indirect burn injuries (Fig. 13.7) including extensive charring, articular dehiscence, and an "overcooked" appearance of skeletal muscles have also been described.
5. An electric arc can cause "molten beads" to form on bone from molten calcium phosphate.
6. In addition, petechial hemorrhage of the conjunctiva, facial skin, mucosa of the upper respiratory tract, as well as beneath the pleura and epicardium, are occasionally seen. These findings are suggestive of current-induced tetanic respiratory muscle contraction and an intrathoracic pressure increase ("electrical asphyxia").

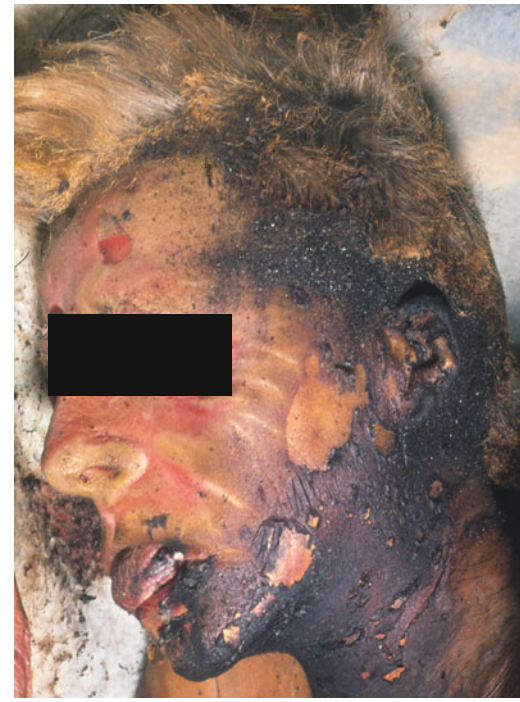

Fig. 13.6 High-voltage accident with facial burns. Protrusion of the tongue and singed hair

Extensive skin burns running lengthways over the body are seen in electrical arc accidents, the

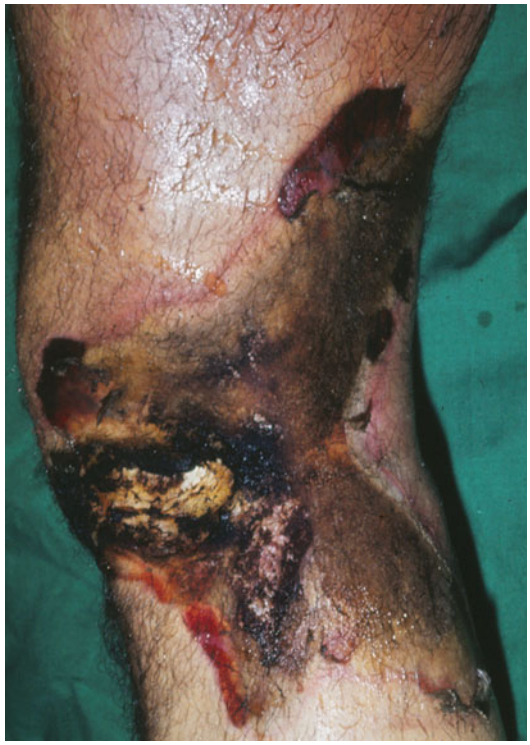

Fig. 13.7 Extensive blackish charring of the skin and subcutaneous soft tissue at the point of current entry on the knee

burns sometimes ending abruptly where they meet insulating clothing, e.g., sparing of the skin of the foot if shoes are worn (Fig. 13.8).

In high-voltage accidents, damage to clothing needs to be correlated with findings on the body. Clothing can be badly torn (Fig. 13.9) and charred black, while particles of molten metal may be detected. Damage to clothing and shoes may help to identify the current entry point.

The feet or soles are the most commonly seen point of current exit, demonstrating extensive or patchy charring of the skin (Fig. 13.10).

13.2 Lightning

Although rare, lightning-related accidents occur and are associated with a mortality rate of up to 40 % in cases of extremely high voltage delivered within less than a millionth of a second. A lightning discharge can carry a current of several million volts. Fluid in moist objects vaporizes along the lightning pathway, while mechanical and thermal injury can occur.

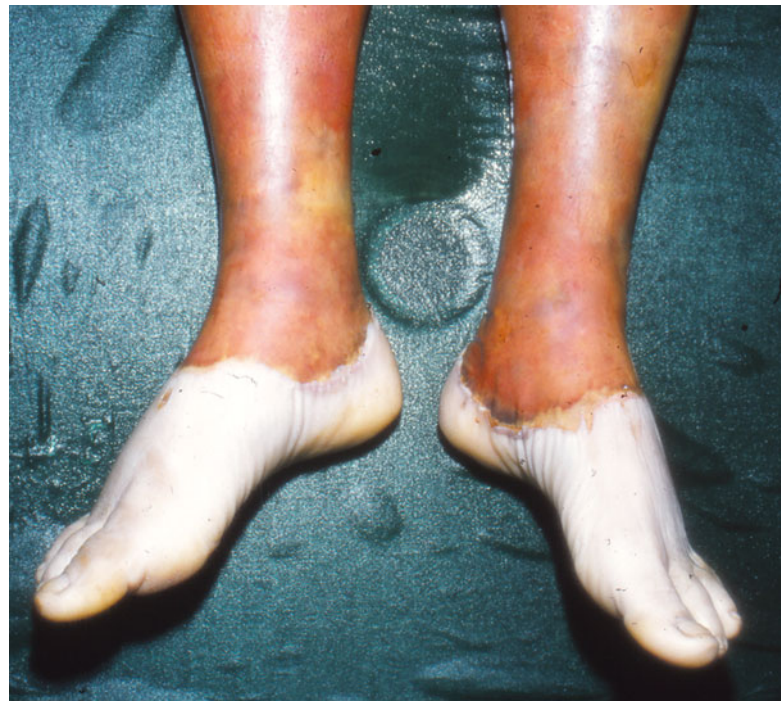

Fig. 13.8 Shoes spare the feet from burning in the case of an electric arc accident

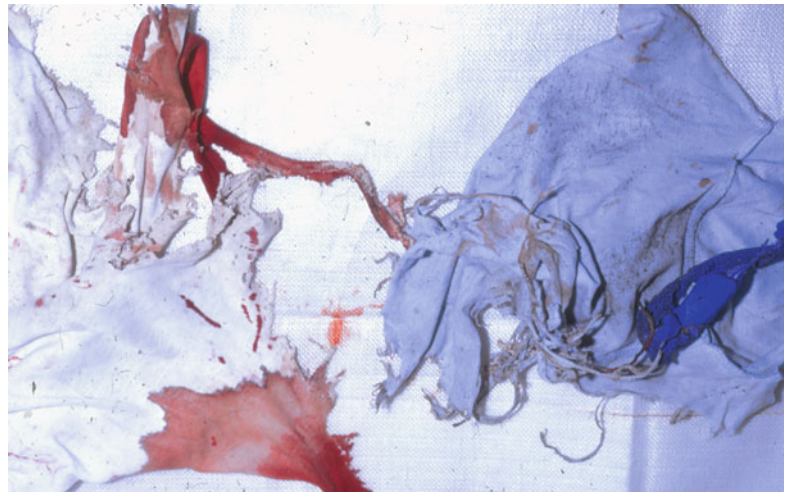

Fig. 13.9 Badly torn and partially blood-soaked clothing following a high-voltage accident involving an electric arc

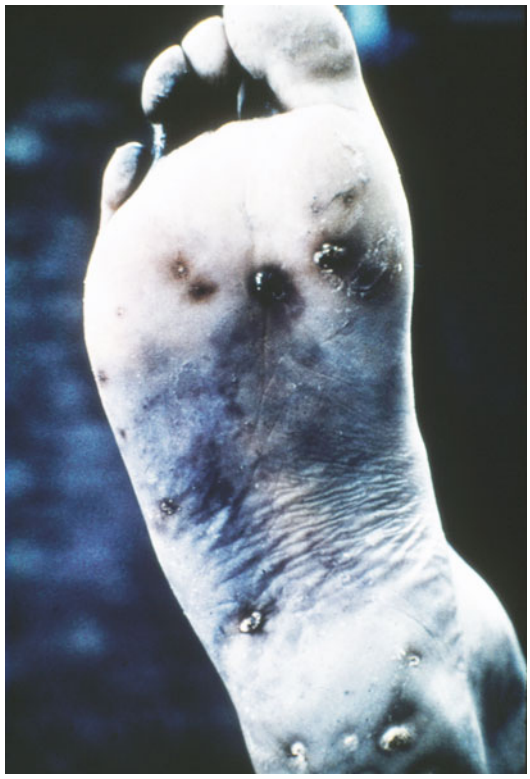

Fig. 13.10 Patchy blackish charring at the point of current exit on the skin of the foot sole

Lightning-Induced Injury. The mechanisms by which a bolt of lightning may cause injury are differentiated as follows:

- Direct stroke.
- Step voltage: A lightning bolt initially strikes the ground or possibly a tree.
- Flash discharge: A lightning bolt "jumps" from one object to another.
- Contact strike: The victim comes in contact with an object hit by lightning.
- Blunt trauma: Secondary injury, e.g., a victim falls as a result of a bolt of lightning.

As with high-voltage accidents, findings on the body and clothing should be identified and compared.

Fernlike Injury. Fernlike injury (arborescent erythema or feathering burns; Fig. 13.11), which forms part of the injury pattern typically seen in lightning strikes, is due to small-vessel hyperemia in the corium and can disappear within hours post-mortem and with increasing postmortem interval. In addition, epidermal cells in an elongated and palisade-like arrangement can sometimes be seen at the same level. Singed hair along the lightning pathway, charred and torn clothing, metal particles with signs of melting, as well as torn shoes are also observed. The entry point of lightning in the case of fatality is typically located on the head (crown area). Although not always the case, extensive linear skin burns may be observed along the trunk and on the lower extremities. Contact burns and metallization may occur wherever the skin comes in contact with metal objects. Clothing is subject to varying degrees of mechanical and thermal damage, including hole formation and melting of metal parts (belt buckles, wristwatches, earrings, buttons, etc.). As in high-voltage accidents, holes and tearing may be seen in footwear (shoes, socks/stocking).

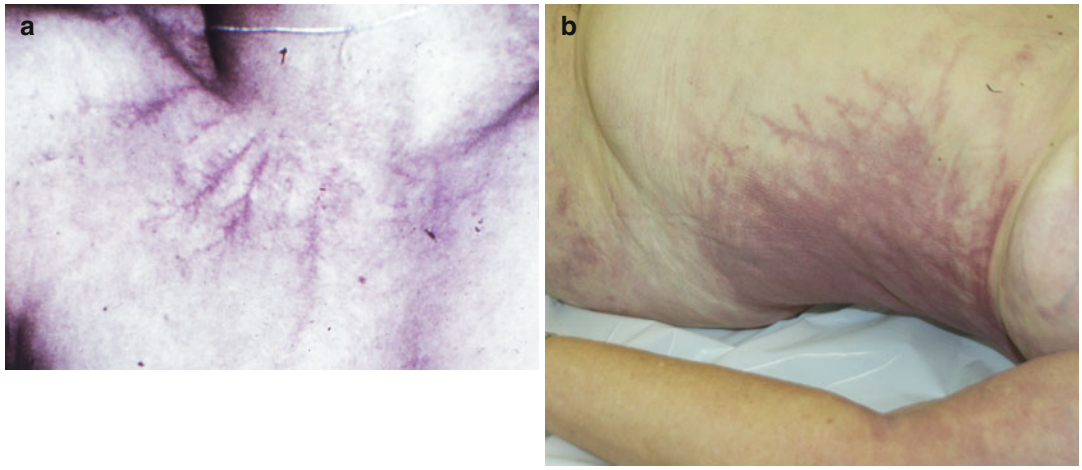

Fig. 13.11 (**a**, **b**) Two cases with fernlike injury in lightning strikes

Ground Strike. The term "ground strike" or "step voltage" describes the partial flow of current from a lightning bolt through the body (from leg to leg) of an individual or animal located near the point at which a lightning bolt strikes the ground. Ground strikes of this kind form a discharge voltage pattern, whereby voltage reduces as it moves out from the center towards the periphery. Thus when the legs are in a striding position, the potential difference on the ground may be picked up ("stride potential") and cause death. In this way, bolts of lightning are able to kill entire herds of cattle.

Although immediate resuscitation following a lightning strike may be successful, the extent of burns as well as injury to internal organs will determine the further course. Neurological deficits of varying degree and duration dominate the clinical picture in the case of survival. In addition to acute cardiac and cerebral death following a lightning strike, myocardial necrosis or fulminant bronchopneumonia may lead to late fatality despite initially successful resuscitation. Acute kidney failure may develop depending on the degree of tissue injury. Survivors of lightning strikes often demonstrate neurological deficits, including aphasia, temporary blindness, swallowing and speech impairments, spastic paraplegia affecting the legs, cerebellar injury that is reversible for up to 1 year following the insult, as well as temporary paralysis.

13.3 Electric Shock Devices and Taser Guns (Stun Guns and Remote Electrical Discharge Weapons)

Electric Shock Devices. Electric shock devices are gun- or prod-like devices designed to incapacitate a subject by means of an electrical discharge—without causing significant injury. When pressed against the skin, the high-voltage circuit is reduced by skin resistance to the extent that the current at the electrodes drops significantly. Commercially available devices produce an unpleasant perception of pain without impairing consciousness. Skin reddening (erythema) in a paired arrangement, approximately 5 mm in diameter and at intervals corresponding to the distance between the electrodes, are possible; local pain has been reported. Local erythema is temporary and fades within several hours. The paired arrangement of erythema is the most significant indication of the use of an electric shock device.

The Taser Gun. "Taser" is an acronym of "*Thomas A. Swift's electric rifle*" from the book by V. Appleton (1911). In contrast to electric shock devices, two projectiles (Taser darts) are fired out of a cartridge from long range and at a speed of up to 50 m/s. The darts are fitted with insulated wires that deliver electrical impulses from the Taser gun to the subject. The wires are several meters long, while the darts have small

barbs capable of penetrating several layers of clothing. The device has a range of up to 10 m. Although each cartridge can only be fired once, the operator is able to deliver several electric shocks to a subject by pressing the trigger. Taser guns are considered appropriate weapons for use at an escalation level below that of firearms and are supposed to confer a lower risk of mortality. At the same time, Taser guns are used by police officials as a means of self-defense. They should only be used when all other means (physical force, baton, pepper spray) have proved unsuccessful; moreover, the subject should be given prior warning of their use. In practice, the effects of the gun depend on various factors:

- The point at which the electrodes penetrate the body
- The distance of the electrodes from one another
- The length of the current path between the electrodes
- The nerves and muscles affected by the current path (is the heart affected?)
- The penetration depth of the electrodes
- The current intensity delivered (should be 3 A, but can go up to 5 A)

According to the manufacturers of the Taser gun, which can fire two or four barbed darts, an electric shock of approximately 17,500–500,000 V (open-circuit voltage) is delivered via the barbed hooks attached to the wires only when the trigger is activated or for up to 5 s. The current surge delivered by the Taser gun causes immediate loss of neuromuscular control, i.e., the subject is immediately incapacitated or defenseless and can be apprehended by police officials. The Taser gun is now available worldwide and has been in use by British and US police authorities since 2001. Although the immediate electric shock causes brief, intense pain, local skin findings at the point of impact of the live mini-darts show scant morphological changes.

From a medical perspective, however, the electric shocks delivered in this context are not entirely without hazard. In principle, electric shocks of this kind may trigger fatal ventricular fibrillation if delivered during the vulnerable phase of the cardiac cycle. Thus, there are an increasing number of reported fatalities associated with the use of Taser guns. The recommendation that police officials keep a defibrillator ready for use when deploying Taser guns has been discussed. However, from a critical point of view, it must be borne in mind that a proportion of the subjects to be apprehended—as we know from experiences with so-called positional asphyxia during or following police apprehension—are in a psychologically disturbed state, highly agitated due to psychiatric disease (schizophrenia, psychosis, delusions, etc.), and/or under the influence of alcohol or drugs. Long-term abusers of neuroleptics as well as numerous types of drugs (in particular cocaine, amphetamines, heroin) may have occult cardiac damage that predisposes them to sudden fatal ventricular fibrillation triggered by the electric shock delivered by a Taser gun. An inflammatory cardiac response is not impossible, whether due to chronic infection, e.g., hepatitis, or due to an often undetected yet acute viral infection with mild cardiac involvement, which would normally follow an unremarkable and often symptom-free course before resolving. Given the known risk of preexisting cardiac damage in drug abusers and individuals with psychiatric disease, it is to be expected that the wider availability and use of Taser guns will be accompanied by a corresponding increase in related fatalities. Precisely for this reason, police officials in some countries are obliged to summon emergency medical services to examine victims following deployment of Taser guns. There is also a regulation specifying that Taser darts should only be removed from the body of the victim by a physician.

In many countries where Taser guns are in use, particularly in the USA, detailed forensic investigations or autopsies are only rarely performed in the case of Taser-related deaths. From a forensic point of view, investigations are required and should include microscopic analysis of the heart and cardiac conduction system. Of 167 individuals who died following the use of Taser guns between 1999 and 2005, coroners stated that Taser deployment was the cause of death, contributed to death, or could not be excluded as the cause of death in 27 cases (from

a report by "Arizona Republic," http://www.raidh.org/RAIDH-devoile-la-liste-des-167.html). Since June 2001, Amnesty International has reported more than 330 fatalities associated with Taser deployment in the USA alone. In more than 50 cases, coroners maintained that the electric shocks were the direct or indirect cause of death. In many cases, alone the proximity in time of death to the use of a Taser gun suggests a causal link. For this reason, Taser guns should be classified at best as "less lethal weapons" than firearms. Particular caution should potentially be exercised when using Taser guns in the vicinity of flammable materials or fumes, such as gasoline fumes.

In turn, evidence that the use of Taser guns results in fewer fatalities than the use of conventional firearms has hitherto been unconvincing. If anything, there are fears that the purported harmlessness of the Taser gun has encouraged its premature and unnecessary deployment. There is criticism that Taser guns are used in situations where a firearm or even a baton would not have been warranted. In many countries, the Taser gun has not yet been classified according to firearm regulations or remains the subject of controversy. However, it is conceivable that in countries with relatively high numbers of firearm deaths (approximately 30,000/year in the USA), consistently substituting a firearm with a Taser gun indeed results in an overall reduction in fatalities.

The potential use of Taser guns as a method of torture is cause for concern, given that intense pain can be inflicted in a traceless manner (so-called white torture). The UN Committee Against Torture expressed its concern in this regard in November 2007. Also of concern is the use of Taser guns by laypersons for self-defense purposes or their misuse as a means of intimidation to compel sexual activity, for example.

13.4 Gas Fatalities

Gas fatalities are classified as death by asphyxia. Carbon monoxide (CO) intoxication is the most commonly encountered accident involving gas. Other gases, such as hydrogen sulfide and putrefaction gases, are found in sewage plants and silos, among others.

Carbon Monoxide Intoxication. Carbon monoxide has a 300-fold greater affinity for hemoglobin than oxygen; as a result, the latter is displaced. Thus even small ambient concentrations are enough to cause fatal intoxication. While COHb values of around 15 % are possible in heavy smokers, COHb values above 40 % are considered a plausible cause of death. Values <40 % combined with a fatal course are suggestive of preexisting (cardiac) damage to the organism or additional intoxication with a poison such as cyanide. The most commonly seen causes include house fires, defective gas pipes, smoldering fires, or poor room ventilation. Suicides are occasionally seen involving the diversion of car fumes into the interior of a car or into a closed garage, resulting in fatal carbon monoxide intoxication. On external forensic examination, bright red livor mortis (Fig. 13.12) and pinkish finger- and toenails are conspicuous, while salmon-colored muscles and cherry-red blood are apparent at autopsy. Internal organs may also show a striking bright reddish color. It is always essential to find the source of carbon monoxide; where necessary, a technical expert should be consulted.

Decomposition and Fermentation Gases. In addition to carbon monoxide poisoning, intoxication by gases that are heavier than oxygen—and thus settle and accumulate at the bottom of structures such as silos, septic tanks, and digestion towers—is also possible. Without the victim subjectively perceiving any symptoms, anoxic asphyxiation due to lack of oxygen as well as sudden loss of consciousness may ensue. This is caused by so-called fermentation gases, which have a significantly higher carbon dioxide (CO_2) content than normal ambient air, which is usually around 0.04 %. CO_2 is formed during the alcohol fermentation of mash, must, and feeding stuffs. Decomposition gases can build up at the bottom of silos and in wine cellars. Odorless, the gas goes undetected. The risk of symptoms and a fatal course increase according to the concentration of CO_2 in ambient air (Table 13.3).

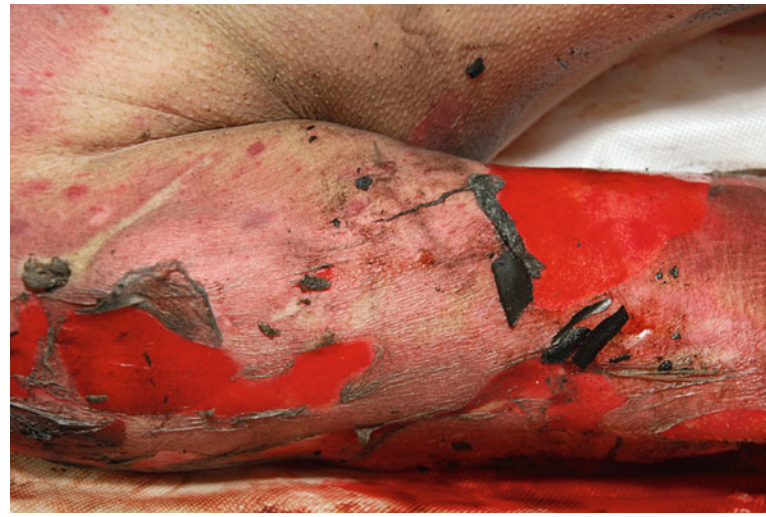

Fig. 13.12 Bright red livor mortis in the partially charred body of a domestic smoldering fire victim (COHb level in postmortem blood, 52 %)

Table 13.3 CO_2 concentrations in ambient air and their effects

CO_2 content (%)	Effect
0.5	Maximum workplace concentration
1	Symptoms apparent after several hours
4	Increased respiratory frequency, impaired concentration, cardiac symptoms, reduced vigilance, drowsiness
9	Fatal within 5–10 min, faster in the case of preexisting cardiac damage
14	Sufficient to extinguish a candle flame
20	Rapid death

Thus, unprotected individuals attempting to help already unconscious victims occasionally become victims of asphyxia themselves.

There is a danger that individuals entering a room where there is an increased level of CO_2 will initially feel a sensation of mild intoxication; however, victims rapidly collapse and lose consciousness, exposing the respiratory organs to even greater levels of fermentation gases due to the higher concentrations of CO_2 at ground level and thereby increasing the gas' noxious effects. Since silos need to be entered by a ladder, they often present the additional hazard that, on losing consciousness, the victim falls from the ladder into the silo.

In addition to its destructive force, fire also produces fumes; in addition to carbon monoxide, these fumes often contain other elements in varying concentrations, such as aldehydes, acrolein, cyanides, sulfur dioxide, ammonia, hydrogen chloride, and phosgene. The concentrations of individual elements of smoke depend largely on the type of material burnt. In cases of fatality, samples should be taken from each pulmonary lobe and stored in headspace vials for the toxicological detection of volatile gases.

Caisson Disease (Decompression Sickness). The air we breathe is made up of 78 % nitrogen (N), 22 % oxygen (O_2), and 1 % other gases. All gases dissolve in blood and tissue. Once oxygen has been used up, mainly nitrogen remains in blood and tissue. Just like oxygen dissolved in blood, nitrogen dissolved in blood can also be released and form bubbles at depths of around 13 m. The deeper the diving depth, the more nitrogen is released with each breath due to water pressure. Depending on the temperature and water pressure, blood and tissue absorb nitrogen; the maximum absorbable volume is referred to as the saturation limit, which is reached after a certain length of time at particular depths. As pressure reduces while the diver's return to the surface, the "stored" nitrogen is delivered to the lungs via the bloodstream. In order for the nitrogen to be safely expelled from the lungs, particular ascent rates need to be observed. Failure to

adhere to decompression times, combined with rapid ascent, leads to the formation of nitrogen bubbles in blood and tissue (caisson disease), causing damage to small blood vessels and tissue.

Caisson disease, which has varying degrees of severity, is not seen at depths of less than 9 m. Early symptoms may appear within minutes or after several hours, with the spectrum ranging from pruritus to death and including skin irritation (formication); fatigue; exhaustion; back pain; joint and extremity pain; sensory, visual, and auditory impairment; speech disorders; and paralysis. In extreme cases, embolic spread of nitrogen bubbles can cause fatal pulmonary artery embolism. At autopsy, attention should be paid to identifying gas bubbles in the capillaries and fatty tissue, in addition to cardiac, pulmonary, and cerebral gas embolism. Osteoarthropathies represent the most common late effect in survivors of caisson disease. However, fatalities associated with caisson disease are rare among divers, with drowning representing a more frequent cause of death, sometimes due to claustrophobia or panic reactions.

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Asphyxia

14

Essentially any episode of cardiac or circulatory arrest and/or respiratory arrest interrupts oxygen supply to the organism and its various organs and tissues. Since the causes are myriad, a classification of the various forms of asphyxia has been undertaken.

Asphyxia is defined as an interruption of any stage of the supply or transport of oxygen between the environment and the organs, e.g., by obstructing the airways or respiration in general, the oxygen-transporting medium blood, blood circulation, and the environment.

Firstly, a distinction is made between external and internal asphyxia:

External asphyxia: Due to insufficient atmospheric oxygen, oxygen is unable to reach the pulmonary alveoli. Possible causes include:

- Generally insufficient levels of atmospheric oxygen (referred to as environmental asphyxia).
- The respiratory excursions of the chest are obstructed (e.g., in the case of burking or positional asphyxia).
- The afferent airways are obstructed, either due to external mechanical compression, as in manual strangulation, or due to obturation, e.g., food aspiration ("bolus death").

Internal asphyxia: Although atmospheric oxygen supply and oxygen transport to the pulmonary alveoli are both intact, oxygen–hemoglobin binding and/or the delivery of hemoglobin-bound oxygen to the organs and tissues is impaired, e.g., due to carbon monoxide or cyanide poisoning.

Signs of external asphyxia include facial congestion and cyanosis, petechiae in the facial skin and conjunctivae, reddening and swelling of the base of the tongue and pharyngeal wall, as well as rapid and intensive onset of dark blue-violet livor mortis.

Signs of internal asphyxia include hyperemia of internal organs, small bloodless spleen, pulmonary hyperinflation, dilatation of the right ventricle, as well as ecchymosis beneath the serous membranes of the visceral pleura, referred to as Tardieu spots when occurring beneath the pleura. Fluidity of postmortem blood is often seen due to increased fibrinolysis, while microscopic investigations have described vacuoles and fat droplets in hepatocytes, blood extravasation into the heart, kidneys, and liver, as well as ganglion cell necrosis, all of which depend on the type and duration of the mechanism causing death.

Important: **None of the signs of asphyxia in internal organs is conclusive for asphyxia—they merely have indicative value!**

Any differential consideration of the pathophysiology of asphyxia sets knowledge of a number of terms as a prerequisite; these terms are defined in Table 14.1.

Mechanical trauma involving occlusion of the airways usually occurs in the case of external asphyxia:

1. Occluding the external airways with a soft cover, e.g., infants or elderly care-dependent individuals unable to put up resistance.
2. Aspiration of fluids, in particular water, i.e., drowning, but possibly also aspiration of blood in the case of a skull base fracture with loss of gag reflexes following craniocerebral trauma.

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Table 14.1 Terms and pathophysiology of asphyxia

Term	Definition
Dyspnea	Difficulty in breathing, respiratory distress
Apnea	Respiratory arrest
Hypoxia	Diminished oxygen concentration in blood, organs, and tissues
Anoxia	Absence of oxygen
Hypercapnia	Increased carbon dioxide partial pressure (considerable respiratory stimulus)
Asphyxia	Simultaneous hypoxia and hypercapnia
Ischemia	Restricted blood supply resulting in a lack of oxygen, increase in, e.g., carbon dioxide and lactate levels

3. Aspiration of solid material resulting in airway occlusion, e.g., aspiration of sand after submersion in sand.
4. Placing a tightly fitting plastic bag over the head, seen in an accidental context in children and an accidental autoerotic context in adults.
5. Foreign body airway obstruction ("bolus death"), e.g., a piece of sausage occludes the laryngeal inlet. However, in the case of bolus death, death due to a vasovagal reflex has also been discussed.
6. External obstruction of respiratory excursions of the chest and simultaneous airway obstruction (nostrils and mouth), referred to as "burking."
7. Death due to positional asphyxia (or postural asphyxia), particularly in individuals in a highly excited state due to psychiatric disease or following drug abuse (excited delirium), restrained in a prone (or "hog-tied") position; deaths while in police custody are seen.
8. Gagging, i.e., introducing a foreign object into the mouth and throat, thereby causing either airway obstruction due to displacement of the posterior airway space by the gag or by simultaneously occluding the external airways.

Cases of accidental strangulation, for example, in children (Fig. 14.1a, b) or belt-restrained patients in hospitals or psychiatric units, are also seen. Cases of this kind usually demonstrate varying degrees of strangulation-related injury to the neck, while hospital fatalities may additionally give rise to an autopsy investigation on the grounds of allegations of errors in treatment or care.

If neck compression is caused by a wide, soft ligature or a wide belt, findings in the neck area may be absent or only discrete. Neck compression persisting after death combined with a particular position of the body can lead to compression-related sparing from livor mortis, e.g., due to the pressure of the compressing collar (Fig. 14.2). For a more detailed discussion of compression trauma to the neck, see Chap. 11.

14.1 General Pathophysiology of Asphyxia

External asphyxia refers primarily to an interruption in external oxygen supply, which—in the absence of mechanical obstruction—includes lack of oxygen in small enclosed spaces, inhalation of gases, and high-altitude death. In the majority of these cases, asphyxia is associated with hypercapnia and an extremely unpleasant sensation of dyspnea (difficulty in breathing, temporary tachycardia, high catecholamine release, and fear of suffocation). However, if carbon dioxide can still be inhaled, one refers to hypoxic asphyxia with a final reduction in respiratory drive, an euphoric state, and an acute loss of consciousness.

The question of how long the process of asphyxia lasts is often raised in forensic practice; this is given as 3–5 min and subdivided into several phases, each lasting 1–2 min. Depending on the concrete pathomechanism or intermittent free respiration in the case of neck compression, asphyxia can last significantly longer. The general stages of asphyxia are shown in Table 14.2.

Findings in external asphyxia may include petechiae in the sclerae, conjunctivae, face, and oral mucosa on external autopsy examination and beneath the serous membranes above the diaphragm, i.e., under the thymus capsule, as well as subpleural and subepicardial petechiae on internal autopsy examination (Figs. 14.3 and 14.4).

If asphyxia is caused by strangulation, congestive petechiae and fine hemorrhages on the inner surface of the epicranial aponeurosis and in the

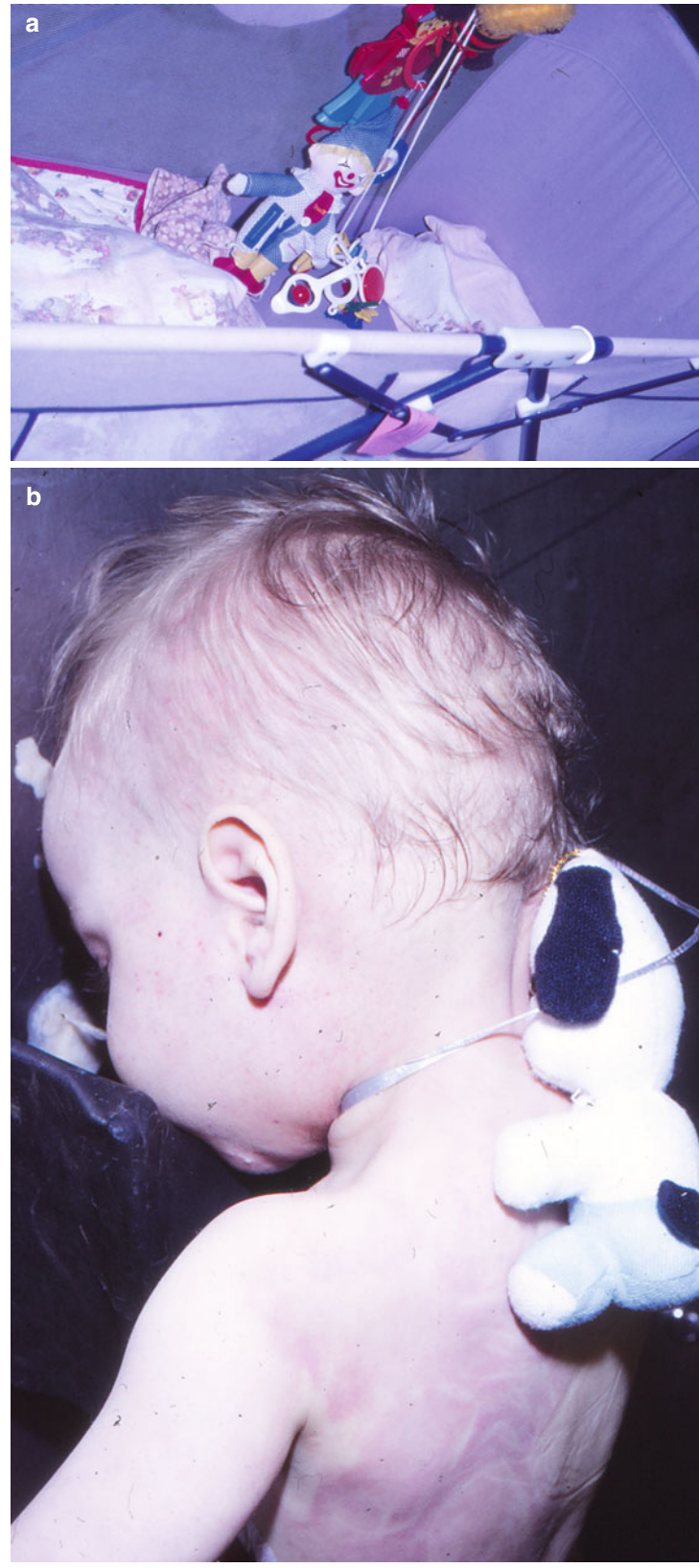

Fig. 14.1 Accidental strangulation of an infant with a thin cord: in its cot (**a**) and following reconstruction with a soft toy constricted between the neck and ligature (**b**)

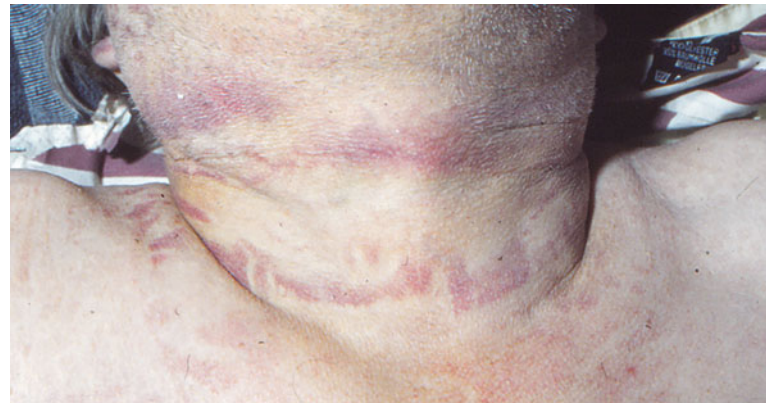

Fig. 14.2 Compressive force of the collar persisting post-mortem with sparing from livor mortis in a case of asphyxial death otherwise presenting scant findings

Table 14.2 General stages of asphyxia

Stage	Pathophysiology
Dyspnea	Increased respiratory activity, inspiratory dyspnea (stridor), increasing cyanosis (e.g., if a cushion is pressed against the mouth causing incomplete airway obstruction)
Neurological effects of oxygen deficiency	Tonic–clonic convulsions (asphyxial convulsions), tachycardia, high release of catecholamines from the adrenal medulla (particularly in asphyxial suffocation with hypercapnia), hypotension, loss of consciousness ("blacking out"), possibly also involuntary defecation and urination, ejaculation (e.g., in ligature or manual strangulation until loss of consciousness)
Preterminal respiratory pause	Apnea, hypotension, continued tachycardia
Terminal apnea	Gasping with no effective movement of air, final respiratory arrest

The heart can continue beating for a number of minutes following respiratory arrest; increasing bradycardia!

temporal muscles may be found in addition to petechial hemorrhage (Fig. 14.5). However, petechiae of this kind may also be seen when the body is in a head-down position, making it important to interpret this particular finding in the context of the situation in which the body was found.

Assuming autopsy is carried out shortly after death, acute epiglottitis, a rare cause of asphyxial death, produces findings of severe epiglottic swelling with distinct narrowing of the laryngeal lumen (Fig. 14.6). If a finding of epiglottitis is made, its inflammatory etiology should be confirmed histologically.

14.2 Particular Constellations in Asphyxial Deaths

In addition to the general pathophysiological process of asphyxial death, there are—more or less irrespective of the type of asphyxia—certain autopsy findings that are indicative of asphyxia. Taken in isolation, these findings are insufficiently characteristic and need to be consistent with a concrete sequence of events, such as gagging, aspiration death, placing a plastic bag over the head, suffocation with a soft cover, bolus death, and positional asphyxia.

14.2.1 Positional Asphyxia (Physical Restraint)

This includes fatalities in abnormal physical positions that cause causing constriction of breathing and cardiac circulation. In addition to positional asphyxia, fatalities caused while restraining highly excited individuals, known as restraint asphyxiation in excited delirium, are seen, e.g., in police custody, in hospitals, particularly among psychiatric patients, and in care facilities. Death in an upright position, on hyperflexion of the neck and obstruction of the airways, in a head-down position, and in a crucifixion

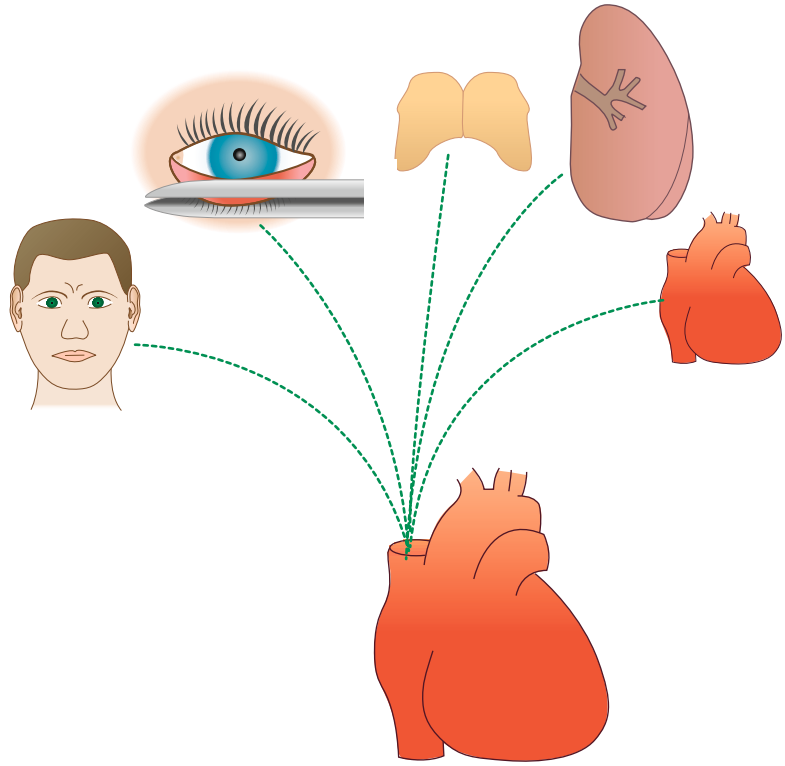

Fig. 14.3 "Asphyxial petechiae." Localization of primarily petechial or fine hemorrhages

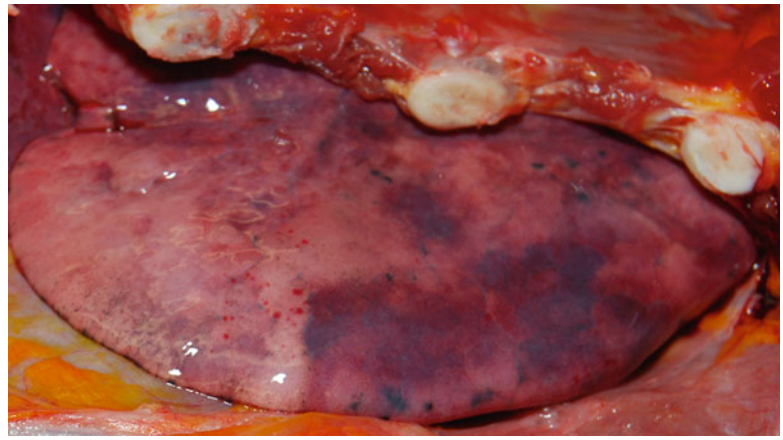

Fig. 14.4 Asphyxial death with multiple subpleural "asphyxial petechiae," also referred to as Tardieu spots

position all belong to the category of positional asphyxia. Cases of restraint-related compression of the chest have been reported in the context of accidents with wheelchairs.

Therefore, positional asphyxia can affect, among others, highly excited and/or intoxicated individuals restrained in a prone position while partial or complete obstruction of respiratory excursions of the chest is caused by compression (e.g., a knee pressed against the back!) as well as partial covering of the airways, while simultaneously a maximum release of catecholamines takes place. In cases such as these, acute cardiac and respiratory arrest may ensue, particularly following prior cocaine and amphetamine consumption. Positional asphyxia is seen in

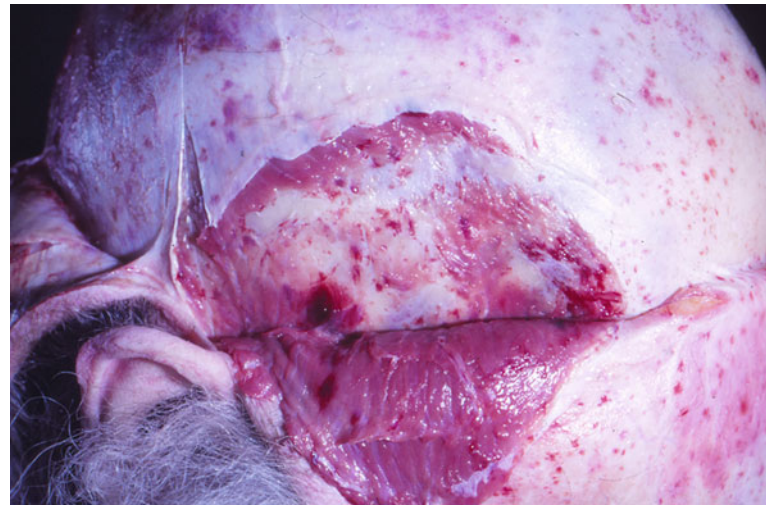

Fig. 14.5 Strangulation- and congestion-related petechiae and fine hemorrhages on the inner surface of the epicranial aponeurosis and in the temporal muscles

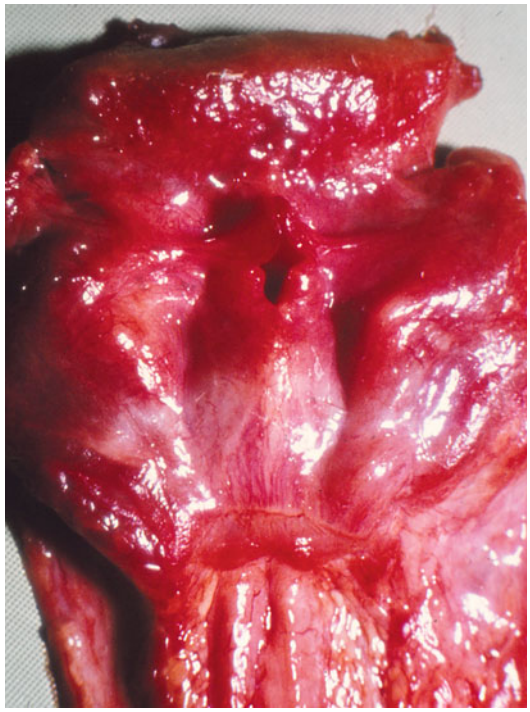

Fig. 14.6 Acute epiglottitis with severe mucosal swelling and narrowing of the laryngeal lumen

association with individuals being apprehended by the police and restrained in a prone position due to resistance, in restrained patients in psychiatric and geriatric units and care facilities, as well as in highly intoxicated individuals. Accidental positional asphyxia is rarely seen in children.

Fatalities during and shortly after police restraint play a quantitatively significant role in the USA. Although highly excited individuals under the influence of cocaine are the most commonly affected group, the widely used form of restraint involving a prone position with hands and feet all tied together ("hog-tying")—and which is banned in some states in the USA—generally increases the risk of positional asphyxia (positional restraint). The cases of positional asphyxia described in the literature show a number of characteristics; these are listed in Table 14.3.

Positional asphyxia affects almost exclusively men, in particular those under the influence of alcohol, drug abusers, and individuals suffering from psychosis. Victims demonstrate highly aggressive and threatening behavior, often necessitating that several law enforcement officers are required in the restraining process. Even the deployment of aids such as tear gas or Taser guns produces no radical effect. During the process of apprehension or restraint, considerable pressure may be exerted on the subject's back as he is pressed to the ground. In the case of violent resistance, this process will be repeated until cardiopulmonary and muscle exhaustion result in the usually heavily perspiring subject pausing due to exhaustion. Evidently, this "exhaustion pause" is sometimes misinterpreted as a conscious cessation of resistance, only to be shortly followed by

Table 14.3 Positional asphyxia: risk factors and symptoms

State of high excitement
Significant alcohol and/or drug intoxication, perhaps involving states of confusion or disorientation
Restraint in a prone position
Respiratory obstruction due to partial occlusion of the airways
Constriction of respiratory excursions of the chest
Massive perspiration
Increased/excessive body temperature
Strong respiration with the mouth opened widely
Unusual respiratory sounds
Gasping
Targeted and untargeted resistance
Abrupt cessation of resistance
Pallor or bluish skin discoloration
Sudden circulatory arrest
Impaired consciousness
Convulsions
Involuntary defecation and urination
Increased salivation

a further bout of violent resistance. The next time resistance ceases, the law enforcement officers assume the subject to be feigning submission and continue restraint, including compression of the chest and possibly also at least partial external airway obstruction. Even the subject's claims that he is experiencing respiratory distress, as well as indications of imminent asphyxia—increasing cyanosis, audible stertorous breathing, salivation, etc.—are interpreted as a renewed attempt by the subject to feign more passive behavior rather than as a warning sign of a deterioration in the subject's condition. In cases such as the one described here, respiratory and circulatory arrest follow; if these are falsely interpreted as a "simulated pause in fighting" and the subject's vital parameters (heart rate, respiration, blood pressure) remain unchecked, cardiac arrest will be recognized too late and resuscitation will be unsuccessful. Although a few cases of successful resuscitation have been reported, the victims had already suffered hypoxic cerebral damage with, in some cases, significant neurological deficits and even apallic syndrome.

From a pathophysiological perspective, this represents a stress situation with maximum catecholamine blood concentrations in the highly excited delinquent. At the same time, increased muscle activity to the point of exhaustion means high oxygen requirements, while respiration or oxygen uptake may be significantly impaired due to chest compression and possibly also partial airway occlusion. A situation such as this causes lactate concentrations to rise and possibly also causes carbon dioxide levels in blood to increase, causing acidosis. If the cardiac muscle has already been sensitized by additional local catecholamine release in the myocardium itself, this will be compounded by the negative effects of acidosis. At the same time, an association between intense muscle activity and increased heat production is suspected, while hyperthermia is more likely to occur in high ambient temperatures.

Important: **Acute psychiatric patients under mechanical restraint while in an excited delirious state have been known to suffer cardiac arrest even when no medication has been previously administered**. **Restraint does not always produce the expected immobilization**, **exacerbating instead an existing state of excitement**.

In the majority of cases reported in the literature, sudden respiratory and cardiac arrest occur directly during police intervention; subsequent resuscitation efforts are unsuccessful. However, there are also reports in which subjects in a prone position experienced respiratory and cardiac arrest once police intervention had ceased and following a certain interval, thereby additionally causing delayed detection. One must assume that leaving a subject in a prone position in itself represents an unacceptable risk factor. Thus, the following applies:

Important: **Once actual restraining measures have ceased**, **highly excited detainees should be immediately moved out of a prone position into a lateral or upright position permitting unrestricted respiration**. **Thereafter**, **vital parameters** (**respiration**, **pulse**, **facial color**, **responsiveness**, **and reactions**) **should be closely monitored in those subjects where adverse restraining measures have been used**.

Table 14.4 Measures to avoid positional asphyxia

Provide training for police, police physicians, physicians, and care personnel
Avoid the prone position
Fix subjects in a stable lateral position, where possible in an upright position
Avoid any form of respiratory obstruction
Note warning signs: difficulty in breathing, sudden loss of muscle strength (tone), and perspiration
Monitor to check: the jugular vein for pulse, respiratory activity, facial color, and responsiveness

Attention should be paid to preexisting conditions considered to represent risk factors; quite apart from the adverse effects of intoxication, these may include preexisting heart damage, either as a result of coronary artery arteriosclerosis, chronic drug abuse, the effects of cocaine (cocaine cardiomyopathy), as well as cardiac hypertrophy, e.g., due to undetected or untreated hypertension. This type of preexisting damage, particularly in combination with catecholamines or cocaine, is believed to be a predisposing factor to life-threatening cardiac arrhythmia.

Educating police trainees on the risk factors for sudden positional asphyxia, as well as on preventive measures, should form an integral part of police training (Table 14.4).

If police restraining measures are used in the presence of a physician, the latter should be appropriately qualified and call for measures to reduce the risk of positional asphyxia and encourage appropriate monitoring to be carried out subsequently. In the absence of these measures, the police officers and physician could face prosecution for manslaughter or bodily injury in the case of fatality or hypoxic brain damage, respectively.

14.2.2 Autoerotic Accidents

In the case of autoerotic accidents, the circumstances under which the body is found may initially be misinterpreted as those of a suicide or more rarely a homicide. Fatal electric shock, fatal hanging and strangulation, death following toxic substance (narcotics) abuse, as well as induced asphyxia all come into consideration depending

Fig. 14.7 A death scene following an autoerotic accident involving a plastic back placed over the head

on the mechanism of accidental death. In general, these cases are clearly of an accidental nature. Victims are generally alone and certain circumstances can be reconstructed that indicate a mechanism of sexual gratification (Fig. 14.7). Fatalities during masturbation with no other adverse circumstances are not classified as autoerotic accidents.

When examining the place where the body was discovered, attention should paid to paraphernalia indicating exhibitionistic, masochistic, or sadistic tendencies (whips, chains, barbed wire, fetters, leather clothing, etc.); transvestite and fetishistic components may also be seen. Bondage (Fig. 14.8) may include "hog-tying" in either a horizontal or vertical position.

Pornographic material, nude photographs, women's erotic underwear, or apparatus for the purposes of self-photography are frequently found. The lower body or genitals are often exposed and signs of ejaculation or condoms are present. If electrical current has been used, wires, clamps, or metal foil serving as electrodes may be attached to the genitals or erogenous zones (nipples, anal region). In cases such as these, attention should be paid to electrical marks.

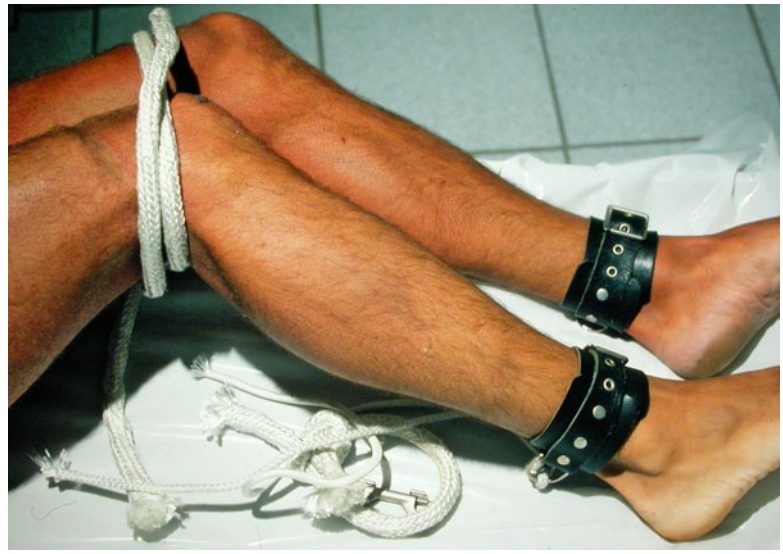

Fig. 14.8 Fatal autoerotic accident involving prior self-bondage

Perforations in the intestinal wall may be present if objects have been inserted in the rectum; this, however, can only be established at autopsy.

A situation becomes life-threatening when (supposedly carefully metered) asphyxial techniques are applied in the context of autoerotic activity, such as strangulation or placing a plastic bag over the head or airways. Although without pathophysiological basis, a certain degree of hypoxia is believed to be sexually arousing. The plastic bag placed over the head is sealed around the neck; asphyxia ensues as oxygen is consumed and carbon dioxide builds up. Findings consistent with external asphyxia are often scant and uncharacteristic. In cases where toxins are inhaled, these generally include acetones, alcohols, benzine, chlorethyl, chloroform, ether, nitrous oxide, or carbon tetrachloride, sometimes in products such as paint thinner, adhesives, or nail polish remover.

Autoerotic activity of this kind has been classified to a certain extent under the umbrella term "sexual preference disorder" or "paraphilia." The vast majority of victims are male. Case reports predominate in the literature and a high number of unreported cases are assumed. Strangulation deaths and asphyxia involving plastic bags are both relatively frequent; fatal mechanical injury is rarely seen.

14.2.3 Aspiration

Foreign material aspiration, in particular chyme, but also blood (craniofacial or skull base fractures!), can cause partial or complete airway displacement. Impaired swallowing and gag reflexes, either due to alcohol (or some other form of) intoxication or neurological deficit following craniocerebral trauma, often represent a causal factor here. Fatal amniotic fluid aspiration is occasionally seen in neonates, as is foreign substance aspiration in occupational accidents. Finally, vomiting followed by chyme aspiration during the agonal phase is possible, whereby in this case chyme aspiration would not represent the cause death. In some cases, the (iatrogenic) spread of, e.g., chyme to the peripheral branches of the bronchial tree during resuscitation should be considered. Vegetable matter, among others, can be detected microscopically in the peripheral bronchi following chyme aspiration.

14.2.4 Gagging

Gagging is a subtype of asphyxia caused by forcing a foreign object, such as a rolled-up cloth or other piece of (usually) fabric material, into the mouth and throat. Depending on the size and position of the gag, the nasopharyngeal space

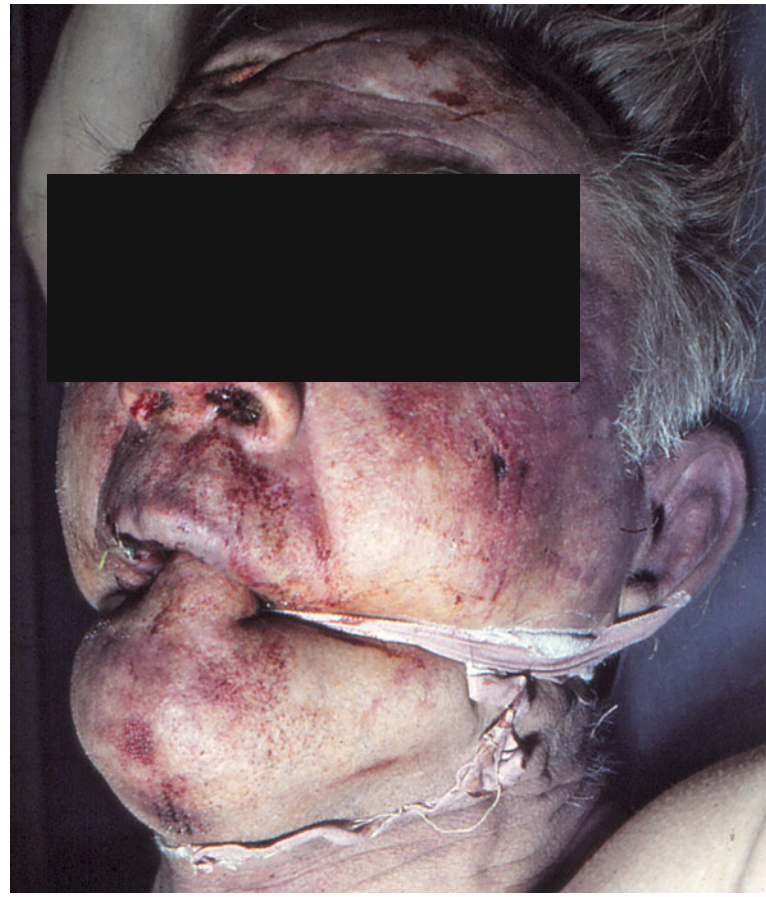

Fig. 14.9 A case of gagging involving a gag fixed with adhesive plaster

may be displaced, either by the gag itself or by the base of the tongue as it presses upwards. While volumes of up to around 100 ml may be tolerated, anything over 150–200 ml can obstruct breathing by closing the internal airways. A life-threatening situation ensues rapidly if nasal respiration is additionally obstructed. Cases of self-gagging are seen, e.g., in the context of autoerotic activity or with suicidal intent. In cases where the gag has been removed following death, distinct but generally small injuries to the mouth region, including the oral mucosa, may support the suspicion of gagging. In some cases, smear samples taken from the oral cavity yield evidence of textile fibers. If the gag has been fixed with material such as adhesive tape or plaster (Fig. 14.9), imprints running outwards from the corners of the mouth can sometimes be seen on the face.

Distinct signs of asphyxial suffocation are usually found: petechiae, hemorrhages in the auxiliary respiratory muscles and at the base of the tongue, as well as facial congestion. If a gag is placed in the mouth or throat with significant force and then fixed with some form of device such as adhesive tape, perioral abrasions and gag marks at the corners of the mouth and on the cheeks may be visible (Fig. 14.10). Concomitant injuries may be present in the case of violent gagging.

If the victim is unable to remove the gag or gagging material themselves, for example, in the case of infants, fatal gagging without gag fixation using adhesive tape may occur (Fig. 14.11).

In other cases, the gagging material is chosen spontaneously, such as grass and leaves, and pressed into the victim's mouth and throat without fixing the gagging material in any way until

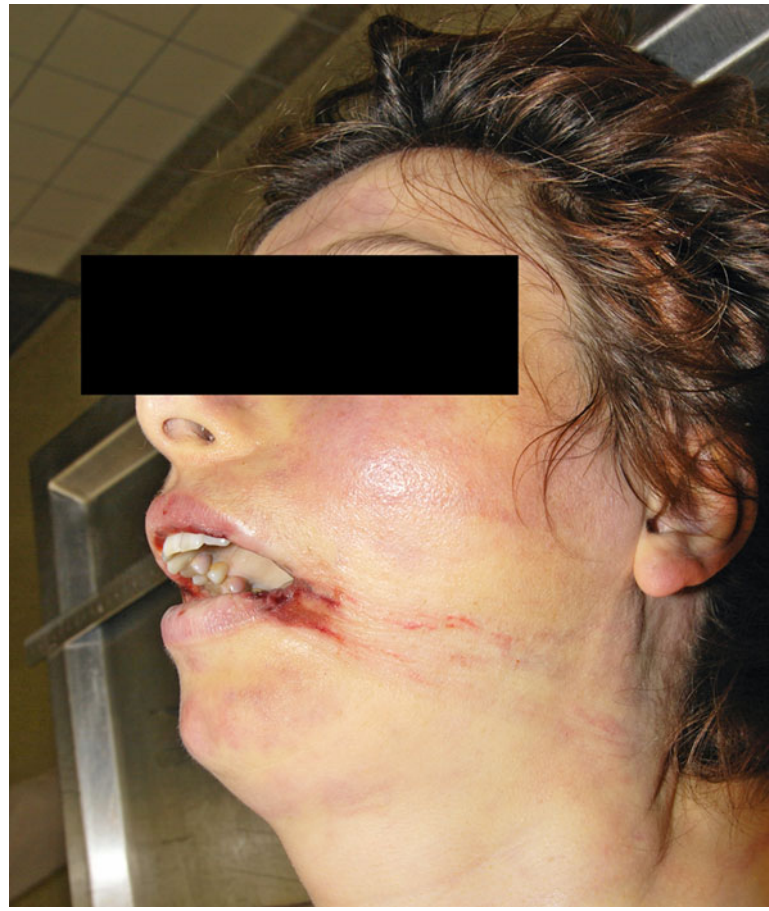

Fig. 14.10 A gagging victim: the gag has been removed and evidence of gag fixation using adhesive tape can be seen at the corners of the mouth and on the cheeks

death occurs. In all cases of gagging, additional signs of trauma, such as strangulation marks or evidence of attempts to cover the airways, should be sought (Fig. 14.12).

14.2.5 Other Forms of Asphyxia

In addition to the asphyxial forms discussed above, there are other constellations that can lead to death by asphyxia; a number of these represent forms of homicide that leave little or no evidence.

Placing a Plastic Bag over the Head. Here anoxic asphyxia occurs following oxygen consumption with a low dead-space volume of air in the plastic bag (Fig. 14.7). Asphyxia can occur even if the plastic bag is not completely sealed around the neck, since sufficient air circulation is no longer possible. As, in principle, exhalation remains possible, asphyxia symptoms are believed to be less dramatic. If the plastic bag is removed following asphyxia, it is possible that no findings indicating the cause of death can be made. Fatalities of this kind are seen in the context of autoerotic accidents (see above), suicide, and accidental death in children.

Placing a plastic bag over the head in the context of assisted suicide ("exit bag") or "killing on request" similarly causes asphyxial death. The bag is placed over the head either by the individual wishing to end their life or by an assisting person; oxygen is then prevented from entering the bag by sealing it at the neck with, e.g., an elastic band. Assisted suicide organizations, such as "The Right to Die Society of Canada" or "Exit International," have been known to work with this type of "exit bag," leading to strong controversy.

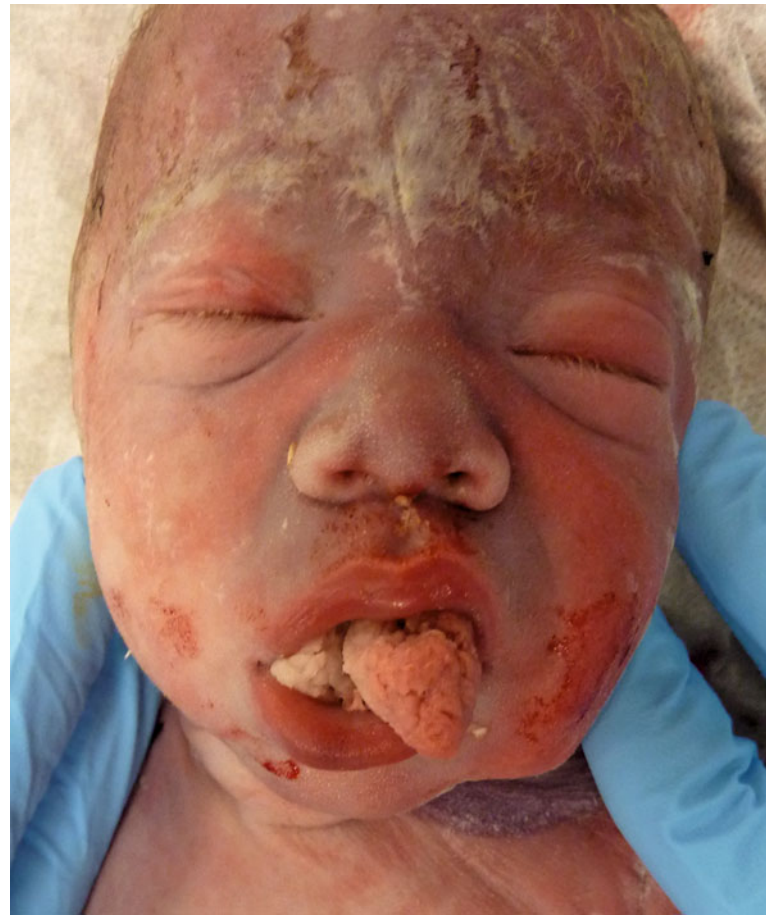

Fig. 14.11 Fatal gagging of a neonate minutes after birth (positive hydrostatic test; lungs only; see Fig. 20.2) without external fixation of the gagging material (toilet tissue), as reported by the perpetrator

Exit bags are considered by the disabled community as a threat to the lives of care-dependent individuals; they argue that "the production and distribution of the Exit Bags directly threatens people with disabilities (…) who are pressured by "caregivers" to commit suicide, or killed without their consent, because they are considered a burden" (from the Canadian journal *Abilities*, 2002, edition 52, p. 9). In some countries, such as Ireland, the use and provision of exit bags is a criminal offense.

Suffocation with a Soft Cover (*Smothering*). Causing death by obstructing the airways with a soft object, such as a pillow or clothing, is seen primarily where the perpetrator is physically stronger than his/her weak victim (infants and young children, sick, elderly, and care-dependent individuals, as well as unconscious individuals, e.g., following intoxication). There may be no, or only discrete, findings of asphyxia, as in the case of infants suffocated with a soft cover to simulate sudden infant death syndrome (SIDS). Attention should be paid to discrete petechiae in the facial skin and conjunctivae, fine tears at the corners of the mouth, and discrete lesions to the lips and oral mucosa. Small reddish-brown areas of drying around the mouth and nostrils, as well as lung expansion, may be seen. These scant findings are often—understandably—misinterpreted as the result of intensive resuscitation efforts. It is not unusual for a homicide to be discovered only as a result of the perpetrator's confession.

Bolus Death. This is seen when a foreign body is lodged at, above, or in the tracheal space adjacent to the laryngeal inlet; the object is usually a fragment of under-chewed food such as a small piece of sausage or meat (Fig. 14.13), very rarely a dislodged or poorly fitting dental prosthesis.

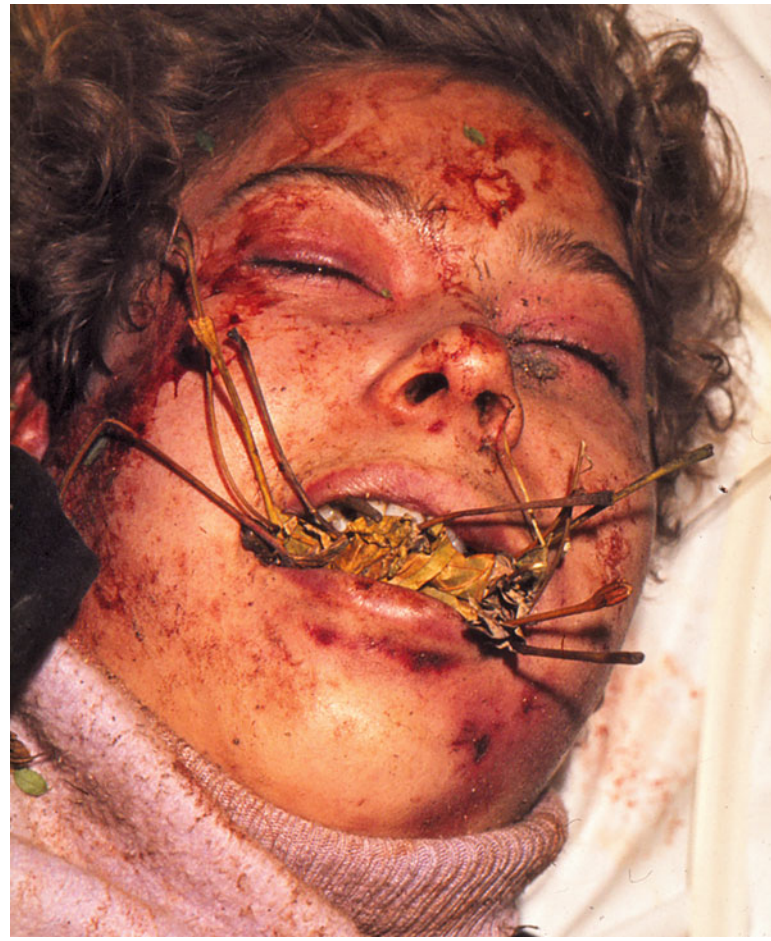

Fig. 14.12 Foliage used as the gagging material and pressed into the mouth and throat by the perpetrator; additional dried-out skin seen periorally is a sign of attempted obstruction of the airways

Extensive occlusion of the airways causes asphyxial suffocation or reflex cardiac arrest (vasovagal reflex evoked by stimulation of the autonomic nerve plexus of the laryngeal inlet). Sudden unresponsiveness, collapse, and rapid death have been reported in the case of a vasovagal episode, thereby precluding the more dramatic symptoms of asphyxia such as respiratory distress, facial cyanosis, convulsions.

Heavily intoxicated individuals as well as craniocerebral trauma patients with organic brain injury and resultant swallowing and gag reflex disorders are commonly affected. However, overly hasty food intake can also play a causal role.

Perthes Syndrome. This describes thoracic compression and fixation, usually in an expiration position, e.g., persons trapped or buried under rubble or due to an occupational accident, mass panic in a crowd, or a fallen tree; rapid compression-related increase in blood circulation and copious dense petechiae in the head, neck, and shoulder area, as well as in the conjunctivae and possibly also below the level of compression in the lower extremities (around the ankle and sole), are seen. A similar mechanism causes death in the case of burking.

Burking. This form of asphyxial homicide takes its name from the serial offender Burke: the physically stronger perpetrator obstructs respiratory excursions by sitting or kneeling on the victim's chest while simultaneously occluding the external airways (mouth and nose). Burking produces little or no external signs of violent trauma. The mass murderer Burke killed 16 people in this manner in the early nineteenth century, leaving little trace of his crimes. He then sold the bodies

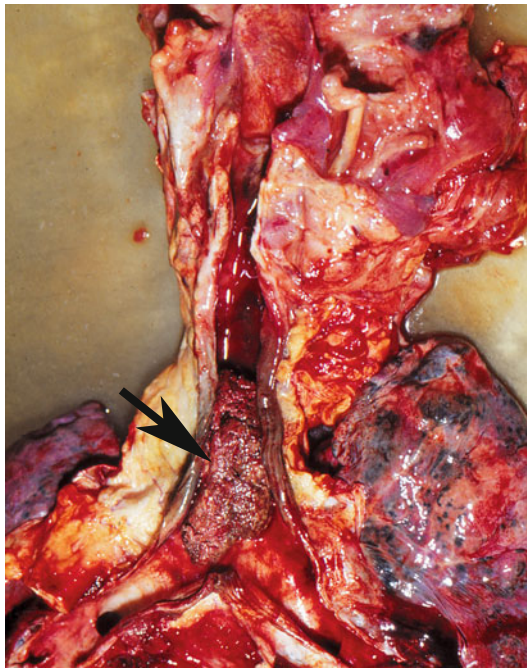

Fig. 14.13 Bolus death after a piece of steak (*arrow*) became lodged in the trachea

to Edinburgh's institute of anatomy. The mechanism of death is similar to that seen in traumatic asphyxia caused by a constricting snake, although this latter form involves no airway occlusion.

High-Altitude Illness (*Mountain Sickness*)/*High-Altitude Death*. The composition of air at approximately 21 % O_2, around 78 % N_2 (+ noble gases), and 0.03 % CO_2 barely changes up to an altitude of 100 m. However, at higher altitudes, air pressure drops and inspiratory pO_2 decreases. Hyperventilation is able to compensate for arterial hypoxia to only a certain degree. Hemoglobin oxygen saturation drops due to the altitude-related reduction in O_2 partial pressure. The following approximate classification applies in this setting:

1,600–2,000 m ASL = mild hypoxia
3,000 m ASL = moderate hypoxia
5,000 m ASL = severe hypoxia
7,500 m ASL = 50 % of unacclimatized individuals lose consciousness within minutes

Acute mountain sickness causes the following symptoms: headache, nausea, dry cough, tachycardia, and chest pain. Altitude adaption primarily affects erythropoiesis, causing increased erythrocyte production and the development of polycythemia. Acute pulmonary edema is sometimes seen at altitudes above 2,500 m even in individuals with healthy heart and lung parameters. Due to the insufficient supply of oxygen to the brain, altitude illness can lead to symptoms such as euphoria, impaired concentration, languor, convulsions, as well as loss of consciousness, and ultimately to death.

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Water-Related Deaths

15

Case Study

A healthy 28-year-man arranged to meet three men and four women at a lake on a day in summer. The 28-year-old drank a bottle of beer with food. At around 14.30 pm, after sunbathing for a significant length of time, the men decided to have a race to the bathing island located approximately 350 m into the lake. After sprinting about 30 m to the edge of the lake, all four men dived headfirst into the water and started swimming. On reaching their target, three of the men heard the women calling from the side of the lake; although unable to understand what the women were saying, they quickly swam back. Around 20 m from the shore, the lifeless body of their 28-year-old friend was floating beneath the surface of the water. The men carried him to the shore and initiated resuscitation immediately; this measure was then continued by the emergency physician called to the scene, but finally discontinued.

No macroscopic pathological findings were made at the autopsy investigation ordered by the court. The injuries that were found could be explained by resuscitation. No signs of drowning were visible. As a precaution, samples were taken for diatom investigation. Marked stomach filling (primarily meat and sausage) was remarkable. Histological investigations showed no pathological organ findings and, in particular, no indication of infection such as myocarditis. Blood alcohol determination in blood taken from the femoral vein yielded a value of 0.28 %. Apart from small amounts of the substance paracetamol, detected by means of toxicological analysis of cardiac blood, gastric content, and urine, no other foreign substances were found. Diatom investigation was negative.

In forensic appraisal, the absence of pathological organ lesions, signs of injury, drowning, or poisoning is interpreted in the sense of a diagnosis of exclusion. Death due to vagal inhibition, classified as immersion syndrome, remained the only explanation. Details reported by witnesses, i.e., that the victim entered the water rapidly after an extended period in the sun and without previously cooling down, on a full stomach, and following mild alcohol consumption all supported the diagnosis of immersion syndrome.

Finding a body in water always demands a differentiated collection and interpretation of findings. Distinguishing between a natural and an unnatural death can be challenging. Deaths in water are primarily accidental in nature, often involving children who either are unable to swim or, due to their age, are unable to save themselves.

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Table 15.1 Possible constellations that help establish the cause of death in water fatalities

	Natural death	Accident	Suicide	Third-party involvement
Death in water	Death due to vagal inhibition (immersion syndrome)—essentially also possible as an accident and suicide and with third-party involvement	Drowning, e.g., a swimmer experiences muscle cramps or a passenger falls into the water	Drowning by "submersion," "entering the water," e.g., shooting or cutting wrists, possibly to ensure success	Homicidal drowning
	Sudden death due to an intrinsic cause, e.g., myocardial infarct, cerebral insult, myocarditis, or epileptic seizure	Hypothermia (ventricular fibrillation) following a fall into water or the sinking of a vessel	Intentional	Drowning or hypothermia after being thrown into the water
		Falling into water from a significant height	Autointoxication, usually prior to entering the water	Death caused by any form of violence, possibly involving the use of weapons typically used in water, e.g., harpoon
		Injury caused by jumping into water (e.g., diving)	Jumping into the water from a significant height (e.g., bridge)	Intentional homicide of a bather by means of a watercraft
		Injury to a bather caused by a vessel	Deliberately causing an "accident-type" situation	
		Accidents involving watercraft		
		Injury from aquatic animals, e.g., sharks, piranhas, or water snakes		
Death out of the water	Sudden death due to an intrinsic cause, e.g., myocardial infarction, cerebral insult near a bank or shore or when leaning over a bridge		All forms of suicide near a bank or shore with postmortem entry into the water	Causing death by any form of trauma or poisoning and postmortem immersion in water to simulate death in water (typically drowning)
			Decomposition-related contact with water, e.g., animals take parts of a body into water or a hanging victim falls from a bridge into water	Immersion of (possibly individual) body parts in water following defensive dismemberment of a body

Thus, fatalities in children are seen in swimming pools (public and private), garden ponds, lakes, and waterways.

In the case of unnatural death, accidental causes need to be distinguished from third-party involvement. Significant insight into the possibility of homicide can be gained by clarifying whether the death occurred in or out of the water. Table 15.1 shows various possible constellations supporting the differential diagnosis between natural and unnatural death. Particular mention should be made here of the comparatively rare phenomenon "immersion syndrome," as well as "atypical drowning." The risk of death by drowning is significantly increased in epileptics.

15.1 Drowning and Homicidal Drowning

Case Study

The body of man dressed only in bathing trunks was spotted in a facedown position on the bank of a slow-flowing river in branches on a morning in August. Once recovered, the body showed hemorrhagic edema fluid around the airways as well as skin abrasions on the forehead, backs of the hands, extensor side of the knees, and on the dorsum of the feet. Ragged skin detachment was seen in places and the abdomen was bloated. Whereas scalp hair and eyebrows could be easily pulled out, finger- and toenails were still firmly attached. At autopsy, watery fluid was found in the sphenoid sinus and stomach. The inflated pulmonary lobes met in the midline behind the sternum. Due to the body's aromatic odor, blood alcohol determination was performed using femoral vein blood (3.46 %). In addition, clearly definable symmetrical hematomas on the inner and outer sides of both upper arms were visible. It was possible to establish the man's identity using dental charts. According to the police investigation, the man had been "pumped" with alcohol by two other men a few days previously. One of these men had scratches to his face and neck. According to these two men, the deceased had been determined to go swimming in the river by his own volition.

Typical (primary) drowning is caused by occlusion of the respiratory orifices or inner airways by water. Although it is not incorrect to classify the mechanism as asphyxia in water, this classification is incomplete. Drowning with and without aspiration causes death due to submersion of the respiratory orifices (mouth and nose) in fluid and subsequent hypoxia and hypercapnia. One speaks of "near drowning" when a victim at least temporarily survives drowning—survival times are reported to be longer in cold water. In the case of drowning, the drowning medium is aspirated, most commonly water (fresh- or saltwater), in some cases together with its constituents, e.g., bath salts. From a pathophysiological perspective, death by drowning is classified as asphyxiation; however, it has some particular features. Following "surprise" respiration on entering the water, four stages of drowning are identified (Table 15.2 and Fig. 15.1).

Table 15.2 Stages of drowning

Stage 1	*Voluntary breath-holding* Duration: 30 s–1 min, up to max. 2 min in trained individuals
Stage 2	*Dyspnea:* due to respiratory reflex mediated by the respiratory center as a result of increased CO_2, respiration can no longer be voluntarily suppressed. Inspiration takes place, followed by cough-like expiration. As unconsciousness deepens, the need to cough reduces; respiratory movements vary in intensity. Duration: 1–3 min
Stage 3	*Seizures:* tonic–clonic seizures; respiratory activity continues Duration: up to 90 s
Stage 4	*Involuntary breath-holding and terminal gasping:* initial preterminal apnea, with circulation still functioning, is followed by the final stage of drowning, which ends with cardiac arrest

Depending on the duration of immersion, the degree of putrefaction can make diagnosis in a body recovered from the water challenging. A body found in a (filled) bathtub should prompt investigation into the possibility of exposure to electricity.

15.1.1 Postmortem Interval

Since the postmortem interval between drowning and the time a body is found can vary, the body's state of preservation is subject to wide variation. Initially, the body sinks to the bed of the body of water; its reappearance is determined largely by the buildup of gases during putrefaction and is therefore temperature-dependent. At temperatures from 18 °C, a body can be expected to

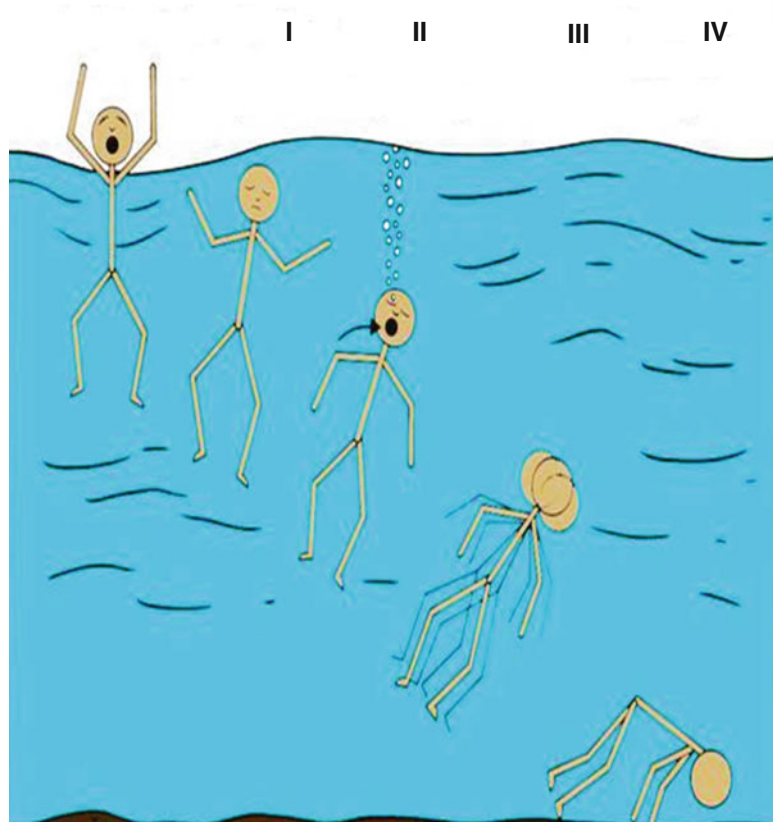

Fig. 15.1 Phases of death by drowning: following deep inspiration, breath is held after submersion below the water level in *stage I*; dyspnea and involuntary respiration in response to increased CO_2 occur in *stage II*, asphyxial seizures in *stage III*, and, following a respiratory pause, terminal gasping and death occur in *stage IV*

appear after 2–3 days, unless the body has become attached by clothing. However, at water temperatures below 10 °C, weeks or months may elapse before a body surfaces. The buildup of gases in the body that causes it to resurface may be absent in the case of severe penetrating wounds. Although the processes of putrefaction are comparable to those in bodies out of water, the speed at which they take place varies. Since these processes are strongly temperature-dependent, the body of a victim drowned in the autumn/winter usually takes significantly longer to find than a body drowned in spring/summer. Again, Casper's dictum should be borne in mind: the extent of putrefaction after 1 week in air is equivalent to 2 weeks in water and 8 weeks buried in soil.

The body of a recently drowned victim usually assumes a head-down position. If the body of water is moving, characteristic abrasions on the forehead, backs of hands, knees, and toes are seen (Figs. 15.2 and 15.3); these are not considered a sign of vitality.

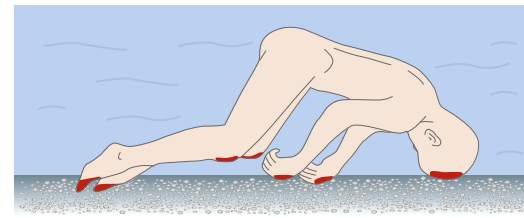

Fig. 15.2 Localization of drift injuries incurred in the typical drift position in flowing waters: upper forehead, back of hands, extensor side of the knees, dorsum of the feet, and extensor side of the toes

Subject to the postmortem interval in water, varying degrees of postmortem changes are seen. "Washerwoman's" skin can develop after only a few hours, initially on the hands and feet, eventually causing glove-like skin detachment (Figs. 15.4, 15.5, and 15.6). Washerwoman's skin starts at the fingertips, subsequently spreading to the inner surface of the hand; however, this progression is strongly temperature-dependent. Longer postmortem intervals in water can cause circumscribed and extensive discoloration of the body,

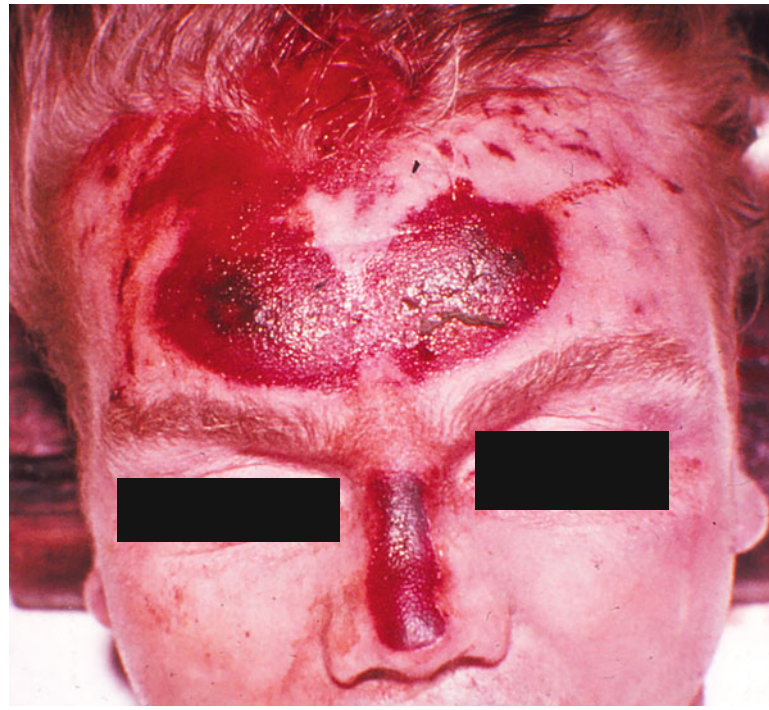

Fig. 15.3 Postmortem drift injuries to the forehead in a body recovered from a flowing body of water

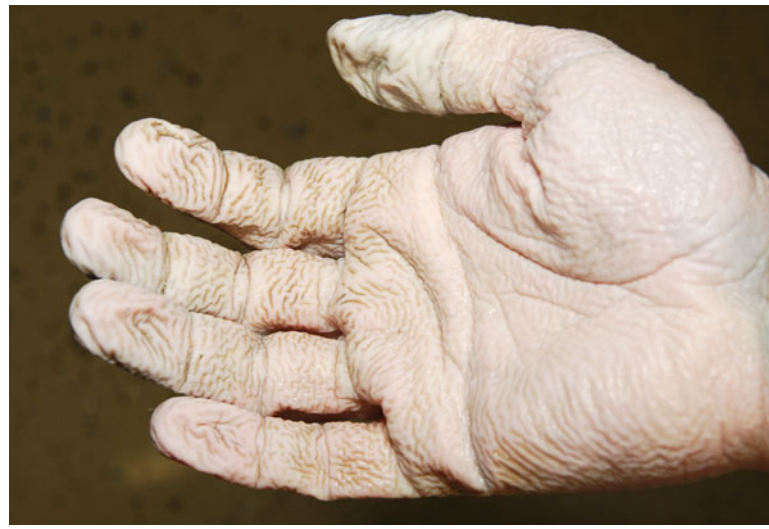

Fig. 15.4 Marked washerwoman's skin on the hands following a postmortem interval in water of approximately 15 days (at a water temperature of 5–7 °C)

primarily red or violet, as a result of colonization by pigment-producing bacteria such as *B. prodigiosum* and *B. violaceum*.

Depending on the type of water (e.g., still lake, stagnant pool, or flowing current), other postmortem changes are seen: soil deposits, colonization with algae (Fig. 15.7), epidermal detachment, buildup of gases in the abdomen, easily detachable hair, increasing bloodlessness of the heart chambers, and putrefaction fluids in body cavities.

Table 15.3 provides an overview of the expected findings in a body found in water depending on water temperature and following recovery and subsequent storage for 2–3 days at +4 °C in a morgue refrigeration unit.

In addition, injuries due to aquatic animal predation are seen, as well as possible propeller

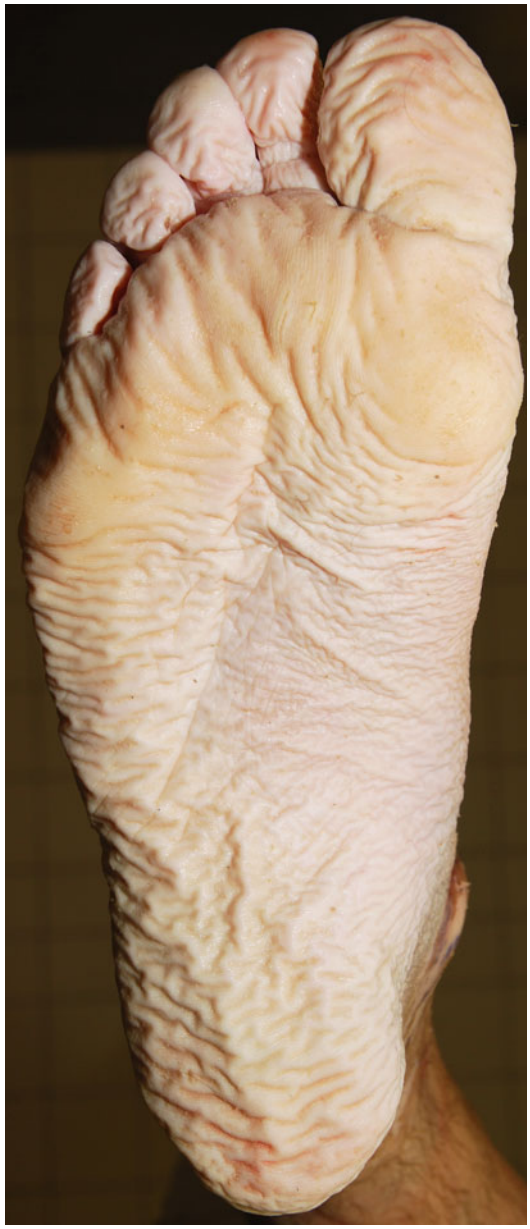

Fig. 15.5 Distinct washerwoman's skin on the soles of the feet following a postmortem interval in water of approximately 15 days (at a water temperature of 5–7 °C)

injuries, some of which may require differentiation from antemortem injury. Besides skin and soft tissue damage (Fig. 15.8), propellers can cause fractures, e.g., of the bony skullcap (Fig. 15.9). In this setting, parallel smooth-edged injuries are evident. The use of mechanical equipment in difficult-to-recover bodies may cause injury during the recovery process.

15.1.2 Osmolarity of the Drowning Medium

In relation to the osmolarity of the human organism, freshwater is hypotonic and seawater hypertonic. However, not only seawater is hypertonic: there are certain bodies of water, usually small streams, which take up water from heavily overfertilized fields, thereby reaching particle concentrations similar to those of seawater. Significant differences are seen between fresh- and seawater drowning:

Freshwater drowning: Freshwater causes hypotonic hyperhydration and rapid hemolysis. In the final stage, water is drawn out of the already sharply overexpanded lungs by osmotic pressure, producing dry overdistended lungs macroscopically, referred to as "emphysema aquosum." At autopsy, the lungs meet at the body midline behind the sternum or even overlap (Fig. 15.10); lung emphysema of this type is relatively stable postmortem. The lungs are often remarkably light in weight, with the total weight of both lungs often not exceeding 1,000 g, while simultaneously anemic. Blunt pressure often causes imprints on lung tissue (Fig. 15.11). Differentiating between this form of acute lung emphysema and chronic lung emphysema is possible by making fresh cross-sections of lung tissue: in the case of chronic lung emphysema, the sectioned bronchial tubes and vessels protrude at the cut surface, but not so in emphysema aquosum. Histologically, and in addition to anemic and blood-rich areas, alveolar distension, tearing of the alveolar septa, as well as edematous areas are seen in lung tissue.

Particularly in the case of freshwater aspiration, diatoms (silica algae) and impurities in the aspirated water, such as amorphous foreign particles or plant constituents (Fig. 15.12), are often observed.

Saltwater drowning: Saltwater drowning takes longer than freshwater drowning. Aspirating saltwater causes an influx of NaCl from the lungs

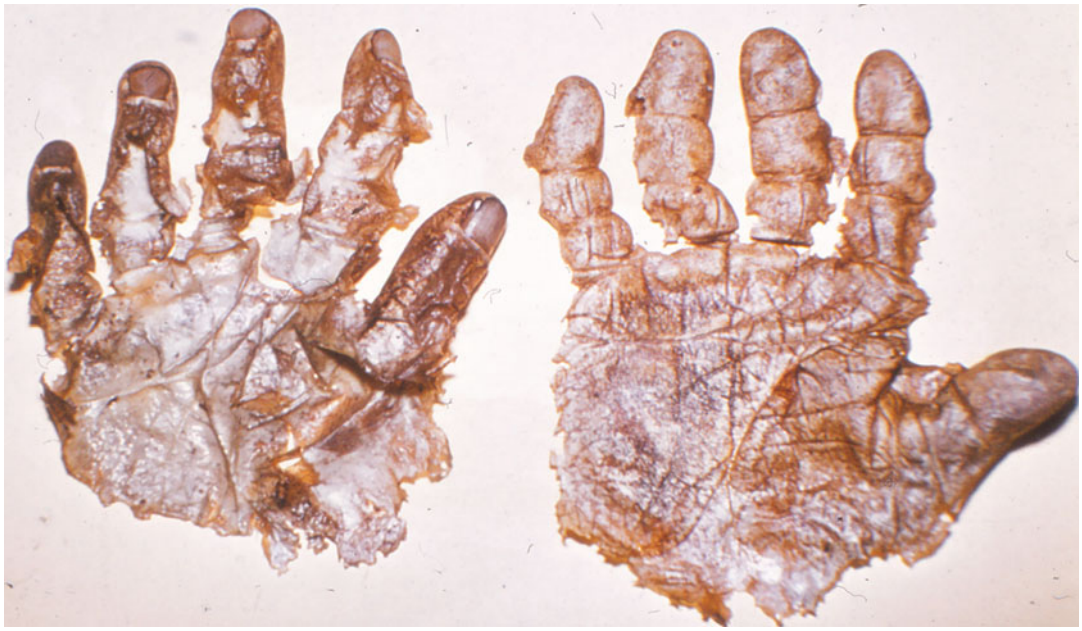

Fig. 15.6 Glove-like skin detachment on both hands following an extended postmortem interval in water

Fig. 15.7 A body found in the water with large areas of epidermal detachment and extensive algae and soil deposits

into the blood, while proteins and fluid are transported in the opposite direction, causing hypertonic hyperhydration. Thus, in addition to the aspirated water, the lungs also collect fluid from surrounding tissue, producing the finding of "edema aquosum": macroscopically, the lungs are massively distended, heavy, and waterlogged. Overextension of lung tissue may cause subpleural hemorrhage per rhexis (Fig. 15.13).

15.1.3 Diagnosing Death by Drowning

The external finding of cyanosis of the face, neck, and shoulders is occasionally made after short postmortem intervals; however, these are as unspecific as "goose flesh," shrinking of the penis or nipples, excretion, or biting of the tongue. Right heart dilatation, the absence of postmortem blood coagulation, and anemic spleen are seen at autopsy in the case of death by drowning. Linear subfascial hemorrhage in the superficial neck and chest muscles have been described and are interpreted as the result of convulsive respiration and resistance.

Table 15.3 Changes seen at minimum postmortem intervals in water (in days) to bodies found in Central European waters and stored at +4 °C in a morgue refrigeration unit following recovery

Month	January	February	March	April	May	June	July	August	September	October	November	December
Median water temperature (°C)	3.2	3.9	5.8	9.9	13.0	17.4	18.6	18.6	17.3	13.2	8.8	4.7
Marbling	35	25	16 (23)	9–10	4–5	2	1–2	2	3	4–5	10	17
Tissue distension due to gas	35	25	16 (23)	10	4–5	2–3	2	3	3–4	7	10	17
Discoloration of the body	35	25	16 (23)	(14)	4–5	2	2	3	3–4	7	10	17
Peeling of the epidermis	35	25	16 (23)	(16)	4–5	3	2	3	3–4	7	10	17
Hair loss	35	25	16 (23)	10–12	4–5	2–3	2–3	3	3–4	7	10	17
Hands: early wrinkling	(1)	(1)	(12 h)			(6 h)			2 h		2 h	(1)
Nails become loose	35	28–30 (40)	23	16	5	2–3	3	3	3–4	11	17	28
Peeling of skin in glove form	Over 35	30–32 (45)	23	16	10	3	3	3–4	4	7	20	28
Nails lost	Over 35	45	30 (40)	21	14	8	3	4	10	Over 11	20	Over 35
Feet: early wrinkling	(1)	(1)	(12 h)	(1)		(6 h)	0.5 h		2 h		2 h	(1)
Nails become loose	Over 53	40	26 (35)	17	10	5	3	4	8	12	17	28
Peeling of skin	Over 53	60	35	16	10	5	3	5–6	8–9	Over 11 (14)	20	28
Nails lost	Over 53	Over 60	53	Over 35	Over 28	Over 10	3	Over 10	Over 10	Over 11	Over 20	Over 35
Transudate in pleural cavity[a]	35	25 (40)	18 (35)	10	5	3–4	3	3	5	11	Over 20	
Heart without blood	Over 39	32–34 (40)	23	14–15	9	4	3	3	5	11	20	28
Brain liquefied	35	30 (40)	(23)	14–15	5	3–4	3	3	6	10	17	28

According to Reh (1969)
Values given in parentheses represent maximum times
[a]>500 ml in adults

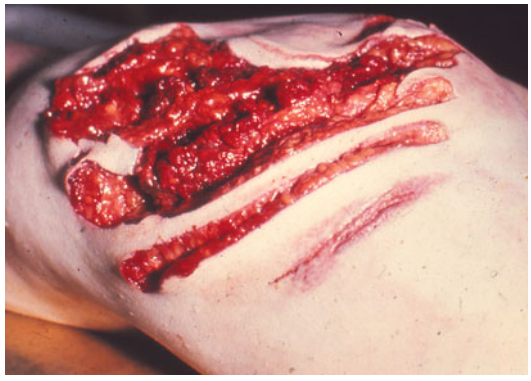

Fig. 15.8 Parallel smooth-edged propeller wounds, deep in places, with no apparent hemorrhage at the margins

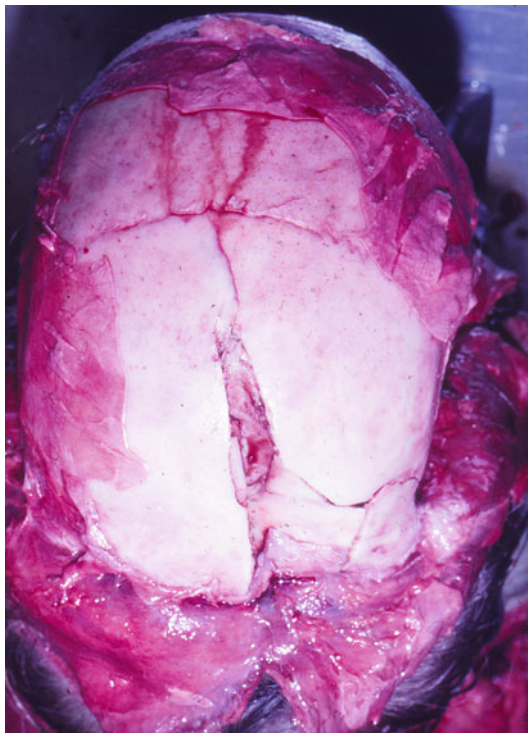

Fig. 15.9 Largely smooth-edged postmortem fracture to the bony skullcap by a propeller following death by drowning

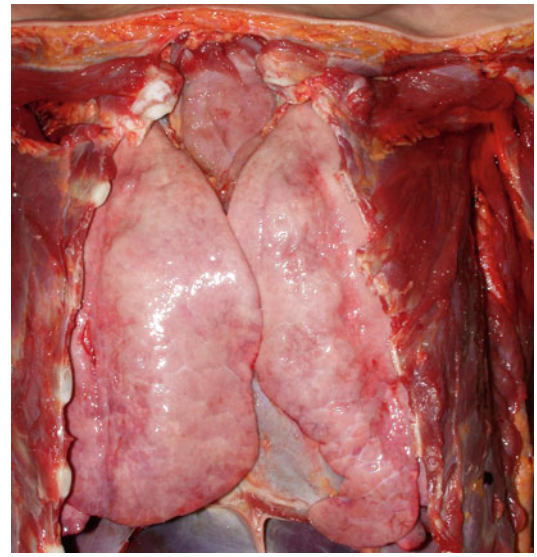

Fig. 15.10 Emphysema aquosum with sharply overexpanded lungs that overlap at the body midline

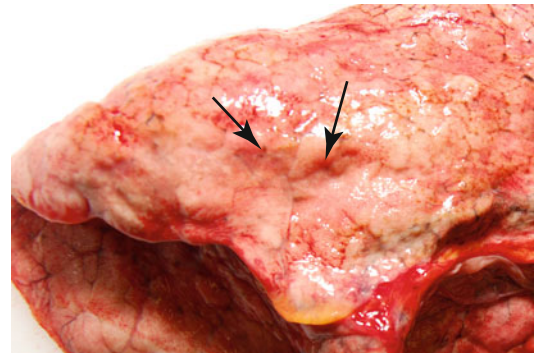

Fig. 15.11 Distinct emphysema aquosum with residual imprints following blunt pressure (*arrows*)

The signs of drowning as set out in Table 15.4 represent crucial diagnostic criteria; the individual signs are seen with varying frequency following death by drowning. Thus, the most significant morphological indications of drowning fatalities include foam around the mouth and nostrils, lung distension (emphysema aquosum), subpleural hemorrhage (Paltauf's spots), foamy content in the airways, as well as increased blood hemolysis, particularly in the arterial circulatory system.

The identification of watery fluid in the upper gastrointestinal tract is less conclusive, as is watery fluid (ca. 1–4 ml) in the sphenoid sinuses. The latter, which can hold up to 9 ml of fluid, can be easily punctured from the skull base at autopsy. Emphysema aquosum is considered the most reliable indication of death by drowning, while laryngeal edema is of no significance in terms of establishing the cause of death, being considered a postmortem phenomenon.

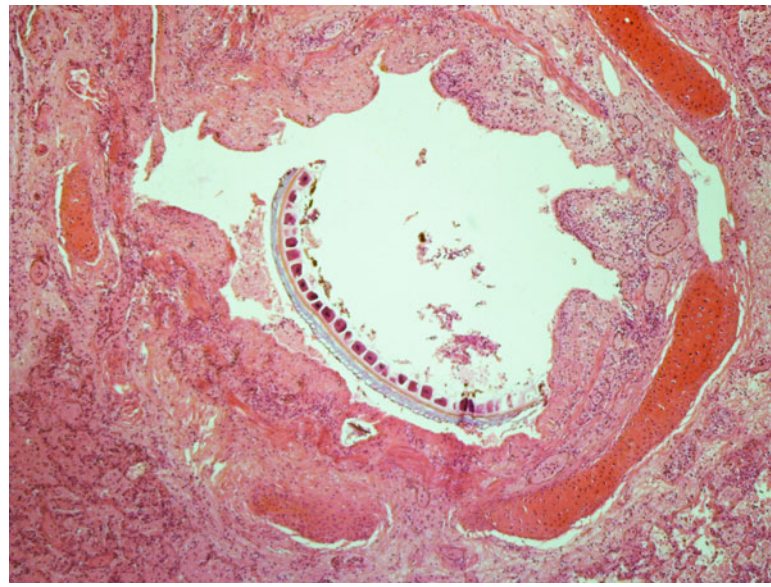

Fig. 15.12 Aspiration of plant constituents in a case of freshwater drowning (H&E×40)

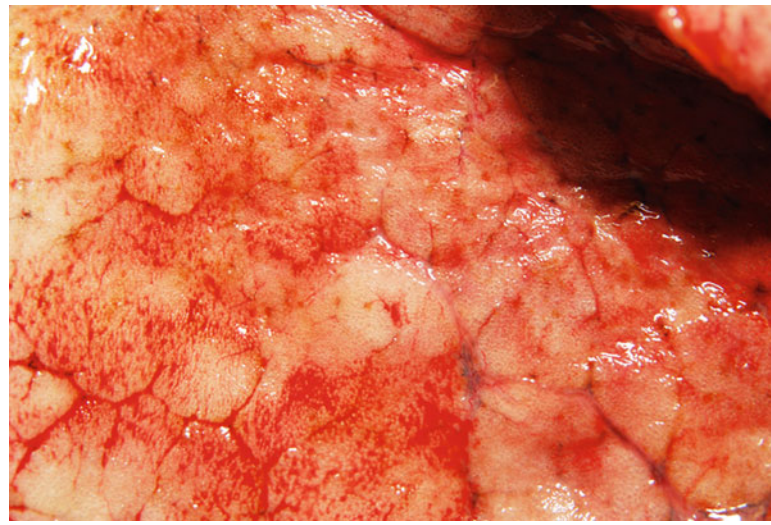

Fig. 15.13 Subpleural hemorrhage per rhexis in a case of saltwater drowning

In the case of freshwater drowning, algae (diatoms) present in the water are aspirated and can be subsequently detected not only in the lungs but also in internal organs as a result of hematogenous spread. At the same time, final strong respiratory efforts lead to the overinflation of lung tissue (emphysema aquosum), which is more pronounced in the peripheral region. This results in the rupture of narrow interalveolar septa, which coalesce to small blister-like cavities. Pulmonary alveoli are acutely dilated, while septal capillaries are compressed and contain scant erythrocytes (Fig. 15.14). However, the histological picture of a "drowned lung" can also be produced by the continuous water pressure a body is exposed to during long postmortem intervals at a water depth of at least 5 m.

Water is swallowed during the drowning process, along with watery foam from the pharyngeal region. In some cases, substantial volumes of water enter the stomach; occasionally this causes the gastric wall to distend and lacerations are seen in the gastric mucosa (Fig. 15.15). Lacerations of this kind are generally seen in the form of radial

Table 15.4 Typical signs of drowning

Sign of drowning	Morphology	Etiology
Foam (Fig. 15.17a)	Foam made up of fine bubbles (like shaving foam) at the mouth and nostrils, possibly hemorrhagic; also found in the airways (Fig. 15.17b) DD: foam made up of larger bubbles in cardiac lung edema, drug fatalities	Develops shortly after recovery as a result of the ensuing reduction in lung volume due to a mixture of air, water, edema fluid, and bronchial mucus
Emphysema aquosum (Fig. 15.10)	Sharply distended lungs, macroscopically dry, meeting or overlapping in the body midline; an imprint remains following palpation	Freshwater drowning, aspirated water is diffused to the surrounding area
Edema aquosum	Distinctly swollen lungs, macroscopically moist and heavy, meeting or overlapping in the body midline	Seawater drowning, tissue fluids are additionally diffused to the lungs
Paltauf's spots	Subpleural, particularly in the middle lobe fissure, pale red to brownish, can be fingernail size, blurring	Subpleural hemorrhage per rhexis following tearing of the capillary walls caused by massive overdistension, additional hemolysis, particularly in freshwater drowning
Detection of fluid in the frontal or sphenoidal sinuses (Svechnikov's sign)	Visible following removal of the ethmoid plate or puncture with a syringe: clear watery fluid	Penetration of water to the sinuses, not a sign of vitality in prolonged postmortem intervals
Wydler's sign (Fig. 15.16)	Gastric contents separate into three layers when left to stand in a glass: top layer is foam, middle layer clear watery fluid, more solid components gather at the bottom of the glass	Swallowing of drowning medium (foam), water (middle layer), and original gastric contents bottommost
Diatom detection (Fig. 15.17)	Enzymatic digestion of tissue to be analyzed (lung, kidney, liver, bone), followed by microscopic diatom detection	Aspirated and, in the presence of intact circulation, transported via vessels to internal organs

fissures in the lesser curvature of the stomach. Water and foam are sometimes also found in the duodenum and upper small intestines. No systematic investigations have been carried out into the frequency and intensity with which water is swallowed. It should be borne in mind, however, that the drowning victim may have drunk water prior to death. Depending on the postmortem interval in water, passive penetration of water to the gastrointestinal cavity cannot be excluded.

Important: Assuming a relevant case history, at least one reliable sign of drowning should be sought if possible in order to establish a diagnosis of death by drowning.

Since a significant postmortem interval may elapse between drowning and the time a body is found, it should be borne in mind that putrefaction processes involving the formation of gases in particular can lead to some, or in extreme cases all, signs of drowning undergoing changes or being destroyed.

15.2 Immersion Syndrome and Atypical Drowning

Although it can be assumed that the "bathing rules" are flouted on a grand scale every year, immersion syndrome remains a rare phenomenon. This is also true of the special form of drowning referred to as atypical drowning. A distinction is made between these two forms of sudden death.

15.2.1 Immersion Syndrome (Hydrocution)

This phenomenon primarily affects young individuals entering the water voluntarily. Victims sink suddenly and silently, even in objectively safe waters and without manifest agitation or excitement. Autopsy yields no signs of drowning or indications of preexisting underlying disease. Vagal

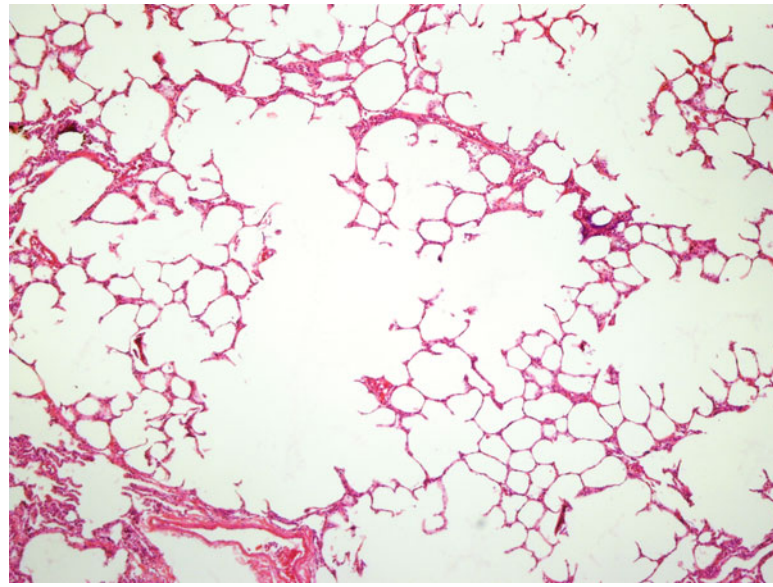

Fig. 15.14 Histological correlation of emphysema aquosum in a case of death by drowning: extremely narrow or flattened interalveolar septa, occasionally stump-like at the margins of blister-like cavities (H&E×40)

reflexes are believed to cause sudden and unexpected submersion in water while producing no, or only scant, distinct signs of drowning. Controversy exists as to whether immersion syndrome can be classified as death by natural causes. Immersion in water represents a vital predisposing factor in terms of external events; however, a number of internal predisposing factors need to be present for this form of death due to vagal inhibition to occur; in addition to acute factors such as hyperthermia, a full stomach, prior alcohol consumption, and sudden cooling down, a certain predisposition to death by vagal inhibition must be present.

Immersion syndrome is an umbrella term for various forms of death in water due to vagal inhibition. In principle, it represents a diagnosis of exclusion that can only be made when a body found in the water shows no signs of drowning, injury, or pathological changes to explain the cause the death. The absence of signs of water aspiration in particular has led to the use of the term "dry drowning."

A number of vagal reflexes have been proposed as the cause of death:

The Ebbecke reflex: Facial skin becomes a reflex zone (second trigeminal branch), swallowing reflex following immersion in cold water, bradycardia, respiratory arrest ("diving reflex")

Aschner reflex (oculocardiac reflex): Bradycardia due to compression of the eyeball

Hering reflex (carotid sinus reflex): Bradycardia due to chemical or thermal stimulation of the nasal mucosa. Possibly relevant in bathtub homicides following sudden forced submersion of the head in water by pulling on the legs

It is assumed that, due to prior ingestion of a significant volume of food, blood flow in the gastrointestinal tract is redistributed, possibly due to peripheral hyperthermia. A lack of blood ensues if vagal stimulation causes additional strain on the circulatory system. Alcohol may lead to further peripheral vasodilation, while alcohol-related changes in cardiac stimulus conduction could be relevant in terms of cause of death.

Furthermore, other reactions in terms of acute "shock states" have been associated with sudden death following immersion in water:

Cold shock: Massive redistribution of blood due to peripheral vasoconstriction.

Pain shock: Intense stimulation of, e.g., the solar plexus in the case of a "belly flop"; blood sinks to the visceral vessels, slackened due to vagal inhibition.

Laryngeal shock: Laryngospasm in response to water coming in contact with the larynx.

Shock due to acute perforation of the eardrum: Water penetrating to the middle ear triggers vertigo and disorientation.

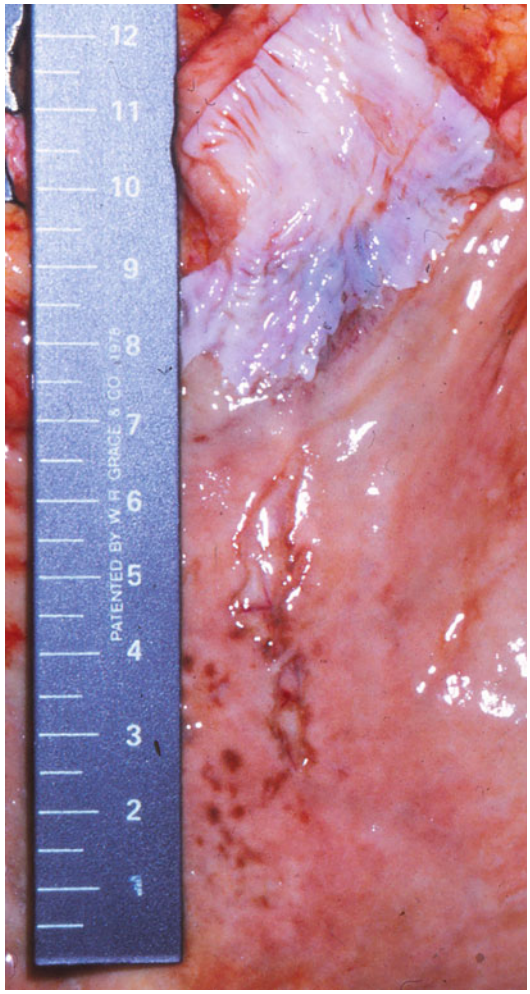

Fig. 15.15 Lacerations in the gastric mucosa after swallowing of water in a case of fatal drowning

Immersion syndrome remains at best a partially understood phenomenon, possibly of multifactorial etiology. It can also be assumed that preexisting cardiovascular disease plays a predisposing role alongside acute strain on the circulatory system.

15.2.2 Atypical (Dry) Drowning

The term "atypical drowning" describes drowning due to dysregulation unrelated to water. Dysregulation of this kind may be the result of natural or unnatural events, such as loss of consciousness following myocardial infarction or

Fig. 15.16 Wydler's sign: layer with clear watery fluid (*arrow*)

cerebral insult, as well as due to intoxication, craniocerebral trauma, or electrocution. For this reason, "atypical drowning" fits only awkwardly into the classification shown in Table 15.1. In particular, drowning in a bathtub or in a "puddle" is commonly classified as atypical drowning.

15.2.3 "Near Drowning" and Mycotic Infection

"Near drowning" describes cases where a previously submerged individual dies within at least 24 h of being rescued. Fungal infections can be a rare late effect in near drowning with aspiration of water, in particular involving cerebral infection with *Pseudallescheria boydii* or *Scedosporium apiospermum*. Mycotic encephalitis and intracerebral abscesses following initial survival of near drowning have been reported.

The pathophysiological mechanisms, symptoms, and histological findings in freshwater drowning, saltwater drowning, and near drowning are listed in Table 15.5.

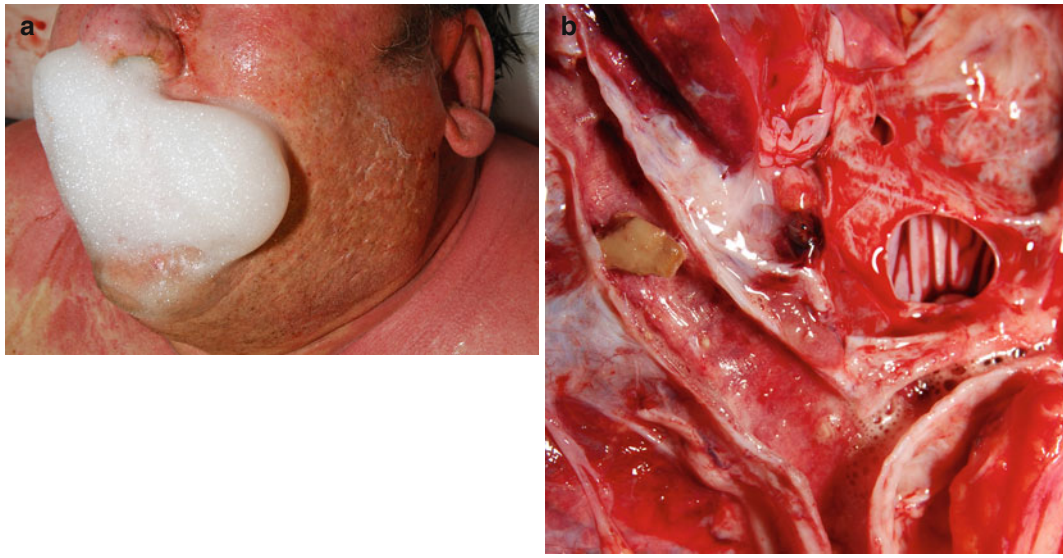

Fig. 15.17 (**a**) Foam at the mouth and nostrils in a case of death by drowning. (**b**) Aspiration of chyme particles up to the upper half of the trachea and watery/foamy content in the airways

Table 15.5 The pathophysiological mechanisms, symptoms, and histological findings in freshwater drowning, saltwater drowning, and "near drowning"

Freshwater	Saltwater	Near drowning
Hypotonic	Hypertonic	–
Large volumes of water pass rapidly through the alveoli	Plasma is drawn osmotically into pulmonary alveoli	–
Increase in blood volume	Decrease in blood volume	–
Hemolysis with potassium release	Hemoconcentration	–
Denaturation of pulmonary surfactant	Dilution of pulmonary surfactant	Pneumonitis, fever, sepsis
Emphysema aquosum ("dry lung")	Pulmonary edema	Pulmonary edema
Expansion, thinning, and occasionally tearing of alveolar septa; ruptured elastic fibers (EvG staining)	–	Hemoglobinuria
Rupture of capillary walls with hemorrhage (Paltauf's spots)—extravascular detection of erythrocytes	–	Signs of cerebral hypoxia: amnesia, convulsions, confusion, coma
Possibly aspirated foreign bodies in water, rarely aspiration of chyme	Rarely aspiration of chyme	Sudden development of cerebral edema

In the case of longer postmortem intervals, autolysis and putrefaction may alter findings to the extent that death by drowning is no longer morphologically detectable. In some cases, staining of alveolar reticular fiber structures may be helpful in the detection of alveolar expansion.

15.2.4 The Detection of Diatoms in Death by Drowning

The diagnosis of death by drowning can be very challenging. It must be proven that covering of the airways by fluid has occurred by active aspiration of the drowning medium. Numerous

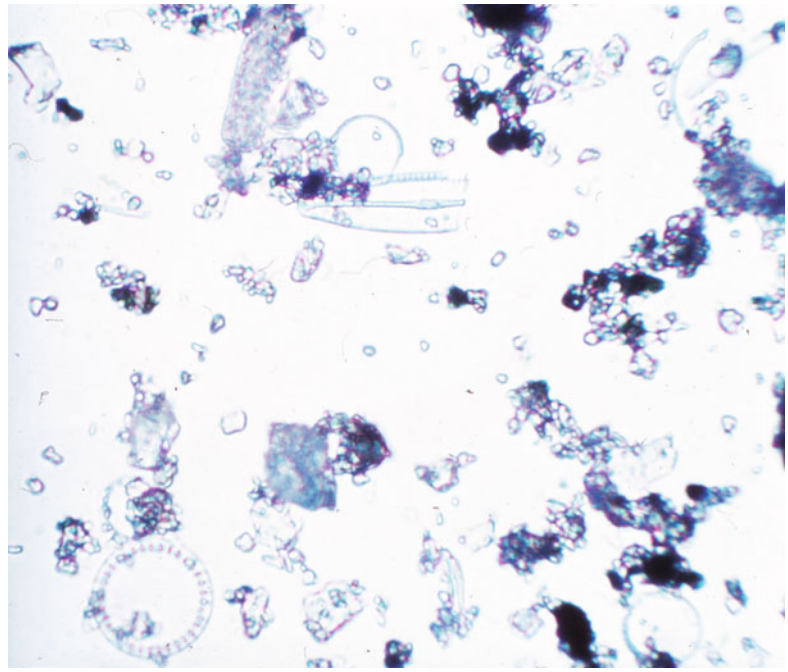

Fig. 15.18 Microscopic detection of diatoms in drowning-medium sediment

corpuscular elements are present in water, including brown, green, red, and blue algae and diatoms, as well as pollen, putrefaction products, dirt particles, and small animal organisms. The detection of diatoms in the drowning medium (water), as well as samples taken at autopsy (blood, lung tissue, bone marrow, etc.) can be used to diagnose death by drowning. However, the spectrum of diatom types detected in both types of samples does not always concur exactly. Diatoms (Fig. 15.18) are eukaryotic unicellular or colonial algae that are ubiquitous in water, air, and soil. In cases where a diatom-containing drowning medium is aspirated, some diatoms may reach the blood circulation via the airways following pre-final rupture of pulmonary capillaries and spread through the organism. Small diatoms (e.g., *Melosira*, *Synedra*, *Cyclotella*, *Stephanodiscus*, *Navicula*, *Fragilaria*, *Nitzschia*, *Amphipleura*) cross the pulmonary alveoli into the blood circulation, thereby reaching organs such as the brain, kidneys, liver, and bone marrow. However, in principle, diatoms may even be detected in organs of non-drowning victims, since they are also found in pulmonary dust. By virtue of their robust resistance to putrefaction, acids, and heat, diatoms can essentially be detected even in adipocerous or markedly putrefied bodies. As a basic principle, diatoms found in the liver, kidneys, or bone marrow point to death by drowning as the cause of death. However, the diagnosis of death by drowning requires: 20 diatoms/100 µl of sediment taken from 10 g of lung tissue and five diatoms/100 µl of sediment taken from 10 g tissue from at least one other organ. Using these reference values, false-positive findings can be avoided, assuming that sources of contamination have been excluded and that the diatoms have been accurately identified. In principle, diatoms can also penetrate post-mortem to the peripheral bronchials via the upper respiratory tract. In particular, in cases of submersion at depths of over 2 m and a postmortem interval of at least 5–6 h, the possibility of postmortem penetration of diatoms should be considered.

15.3 Suicide and Homicide in Water

In order to assume that death occurred in water, either signs of drowning must be present or, at the very least, indications of postmortem immersion in water should be absent. Eyewitness accounts may

be helpful in the differentiation between homicide and suicide; unfortunately, these are often not available in the case of a body found in water. Thus, an autopsy examination is vital in order to differentiate between homicide and suicide in water.

15.3.1 Suicide in Water

Signs of antemortem injury are absent in the case of suicide. However, the postmortem picture may be different if an individual has entered the water with suicidal intent, only to then panic and struggle, thus causing antemortem impact injuries. Suicides in water account for 10–20 % of all suicides, women being more commonly affected than men. The use of self-restraining devices is sometimes seen, as is weighing-down with objects, such as attaching stones to the body. Scars on the flexor side of the wrists, for example, can be interpreted as an indication of previously attempted suicide. Once a body has been identified, the hypothesis of suicide may be supported by information gathered from the victim's relatives or acquaintances in the course police investigations, or the discovery of a suicide note. In the case of combined suicide, a secondary suicide method is chosen, such as cutting the main arteries in water or prior ingestion of tablets, with the aim of drowning following blood loss- or medication-induced loss of consciousness.

Blood Alcohol Determination and Toxicology. Since suicide victims often take "liquid courage" prior to a suicide, moderate blood alcohol concentrations in postmortem femoral vein blood are common; distinguishing these from significant alcohol consumption, which is more suggestive of accidental entry into the water, depends on an individual's alcohol tolerance. According to animal studies, in the case of death by drowning and hemodilution, one can assume an approximately 10 % lower blood alcohol concentration than would normally be expected; in some cases the difference may be even higher.

Foreign substances over and above prescription drugs, possibly in combination with alcohol and which can be identified using forensic toxicological analysis, may indicate attempts to "guarantee" successful suicide or a combined suicide.

15.3.2 Homicide in Water

Evidence of homicide in water can be established by detecting antemortem injuries. These may permit a better assessment of an incident or mechanism of injury, as well as enabling a differentiation between homicide and accidental death. Injury detection may be impossible in fatalities presenting little or no evidence or in the case of advanced decomposition. For example, if a passenger leaning over the railing of a ship is pushed into the water by another individual, homicide with no external signs of injury may result. However, minimal injury to the anterior lower leg can be caused as the victim is pushed against the railing.

Bathtub Homicide. Bathtub fatalities are treated as a special case. A homicidal act that is apparently often successfully used consists of pulling both legs upwards simultaneously, thus forcing respiratory orifices under water. The Hering reflex (see above) seems to play an important role here in terms of the cause of death. A further, possibly evenly more commonly used, mechanism of homicide is electrocution in the bathtub, e.g., by throwing a hairdryer (which does not need to be switched on) into the water. However, the FI circuit breakers required by law today frequently prevent success in an act of this kind. In individual cases, an activated hairdryer may continue to function, thereby heating the water to a point at which a body is, so to speak, "cooked."

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Death by Starvation and Dehydration

16

Case Study

A 22-year-old mother presented at her family physician in a small rural town one evening in March and handed over an evidently deceased infant wearing an old, soiled diaper and dressed in a snowsuit. The mother reported that she had given her 14-month-old daughter a bottle of milk only 1 h or so earlier, but later stated that this had been around lunchtime. Due to the condition of the body, the family physician informed the police.

When questioned, the young mother claimed that she had not been able to cope with caring for her daughter and had not been looking after her in recent weeks. She had repeatedly told her husband that the child was asleep and that he should not wake her. The husband had last seen the child 3 weeks previously. The mother had not appealed for assistance to any of the people who could have helped her, since she feared that her child would be taken away from her. More recently, due to the squalid condition of her home, she had not allowed anyone entrance.

On-site investigations confirmed the squalid condition of the house. "It smelled like a pet shop," reported one of the police officers. The level of humidity inside the house was high, almost comparable to a subtropical climate. Every conceivable surface area was filled with aquariums. Two well-nourished dogs were also found in the home; the father of the child had been walking the dogs while the mother had brought the dead infant to the home physician. Both parents were drug users.

At autopsy, the 14-month-old infant weighed 5,996 g at a crown–heel length of 73 cm. Extreme dehydration was observed, alongside subcutaneous and pararenal fatty tissue depletion, muscle atrophy, facial wrinkling, straw-like hair, nutritional edema on the upper and lower extremities, and extensive diaper dermatitis. In addition, massive cerebral edema (cerebral weight, 1,021 g), a thin parchment-like pericardium, and thickened blood were all striking.

Although death by starvation and/or dehydration is relatively rare in industrialized countries, chronic malnutrition and death due to starvation are not uncommon when seen from a global perspective. Fatalities of this kind occur in the setting of extreme cachexia due to, for example, tumor cachexia, anorexia nervosa, hunger strike, age-related cachexia, accident-related burial, malabsorption and malassimilation diseases, congenital cardiac defects and anomalies, and other consumptive diseases such as infections, tuberculosis, and thyroid dysfunction. If malnutrition is the

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cause of death in fatal cachexia, one refers to actual death by starvation. Fatalities due to starvation are seen in forensic medicine in the case of:

- Neglect of infants and young children
- Psychiatric patients
- Anorexia nervosa patients (women > men)
- Accident victims cut off from food supplies
- Prisoners left to starve to death
- Hunger strikes (usually for political reasons)
- Refusal to eat for other reasons, e.g., elderly, sick persons
- Withholding food from elderly care-dependent individuals: Homicide by omission

In addition to identifying death by starvation, and irrespective of whether the immediate cause of death is purulent bronchopneumonia for instance, a number of other questions require answers:

- Starvation persisted over what period of time?
- Is it possible to classify the starvation in terms of severity?
- Was the gradually worsening and possibly life-threatening condition of the victim recognizable to family members, parents, the authorities, etc., prior to death?

There is no clear differentiation between the terms "cachexia" and "marasmus" mentioned here. Common terms and definitions used in relation to starvation and death by starvation include:

Cachexia: Generalized atrophy of the organism involving more than 20 % weight loss (may vary according to initial weight).

Marasmus: Generalized muscle atrophy associated with severe emaciation due to starvation dystrophy (undernutrition).

Kwashiorkor: A tropical form of protein–energy malnutrition (PEM) due to protein deficiency despite an otherwise sufficient intake of calories in the form of carbohydrates.

Inanition: Depletion of the body's energy reserves. Although the process is reversible for a long period of time, it is not possible to reliably determine the point at which it becomes irreversible.

Nutritional edema can be seen in advanced stages of starvation.

Nutritional edema: Extracellular buildup of water in tissue and body cavities (ascites) due to malnutrition or in the context of nutritional dystrophy resulting from protein deficiency.

A state of chronic malnutrition can also develop in the case of regular or intermittent yet quantitatively and qualitatively insufficient nutrition. This can make assertions about the duration of starvation up to the time of death equally as challenging as absent or unreliable information about body weight prior to the period of starvation. Nevertheless, using the body weight determined at either forensic physical examination or autopsy and applying age-appropriate standard values as well as classifications proposed in the literature (see below), an estimate of the duration of starvation can be made by way of extrapolation. However, it is important to clarify here whether complete nutritional abstention or rather occasional but insufficient food intake has taken place. Of note is also the fact that marked weight loss, or failure to gain weight contrary to expectations, should prompt the responsible persons or guardians to take action, irrespective of whether these individuals are medical lay people!

16.1 Death by Starvation

As a basic principle, starvation and ultimately death by starvation result when an individual's daily calorie intake falls below daily calorie requirements.

Important: **The World Health Organization (WHO) defines malnutrition as the cellular imbalance between the supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions**.

However, death by starvation can only be diagnosed when—taking histological and chemical–toxicological analyses into consideration—all other causes of death can be reliably ruled out, and moreover, the circumstances and findings of the specific case do not present any precluding factors.

16.1.1 Starvation and Death by Starvation in Adults

In the early phase of starvation, i.e., the first 24 h, rapidly available energy reserves from stored carbohydrates, most notably glycogen, are primarily used up. If no food intake takes place after this

period, glucose needs are then met by means of proteolysis and gluconeogenesis, which can persist for some weeks and produces a negative nitrogen balance. The body responds to insufficient food intake by adapting to a catabolic metabolism (starvation adaption). The organism's basal metabolic rate drops after approximately 8–10 days. The body continues to call upon its own energy reserves, fats (lipolysis) with accompanying ketogenesis, as well as protein, leading to increased levels of acetone in urine. Since ketone bodies are released during starvation, metabolic acidosis develops (starvation acidosis). Reduced metabolic function results in a drop in body temperature of around 1 °C. Starvation-related depletion produces a number of findings:

- Marked weight loss depending on the duration of starvation or malnutrition.
- Rarefied subcutaneous fat tissue.
- Rarefaction of fatty tissues within the body, most notably fatty tissue of the greater omentum, the mesentery, and the adipose capsule of the kidney.
- In some cases, gelatinous transformation of fatty tissue.
- Atrophic skeletal muscles.
- Narrow and largely empty (at most, bilious mucous and scant stool) gastrointestinal lumen due to contraction, thin gastric wall.
- Fecaliths in the gastrointestinal lumen.
- The gallbladder is frequently filled with greenish bile.
- Atrophy and weight loss in internal organs (except the brain).
- Atrophy also seen in endocrine organs and lymphatic tissue.
- In extreme cases, even subepicardial fatty tissue is strongly rarefied and hepatocyte vacuolization may be observed in the liver.

Death occurs after weight loss of up to 40 % of the body's initial weight, possibly more in the case of preexisting obesity.

In principle, the extent of starvation dystrophy can be estimated by determining the body mass index (BMI), even though this value is typically used to classify overweight (Table 16.1). However, the BMI does not permit any assertions regarding the severity or duration of starvation to be made—it merely describes a state.

Table 16.1 Determining nutritional status using body mass index (BMI)

Classification	BMI (kg/m²)
Underweight	<18.5
Normal	18.5–24.9
Grade I: overweight	25.0 bis 29.9
Grade II: obese	30.0 bis 39.9
Grade III: extreme obesity	>40.0

The main focus of attention in cases of death by starvation (and dehydration) is on macroscopic findings. However, there are histological findings that correlate with macroscopic findings and patient history: subcutaneous fatty tissue with gelatinous atrophy (Fig. 16.1).

Although a final aspiration may occasionally be seen, infection (bronchial pneumonia, urogenital infection, etc.) should also be considered as a possible direct cause of death. Bone tissue demonstrates increased resorption lacunae and osteoclasts in the case of advanced rickets, while frontal lobe atrophy and acute renal tubular necrosis have both been described.

Anorexia Nervosa: A psychiatric disease associated with a severe eating disorder. Although girls and young women are most commonly affected, anorexia nervosa is also seen in men (ration of women to men, ca. 10:1). Patients control their eating habits to the point of rigid dieting or long periods of starvation (restrictive form of anorexia nervosa). Sometimes, the sensation of hunger is suppressed by drinking large volumes of fluids, or weight loss is accelerated by self-induced vomiting, taking diuretics/appetite suppressants, etc. (active form of anorexia nervosa, binge-purging type). These patients, who often undertake excessive physical or sporting activity, are frequently achievement-oriented and show a tendency towards perfectionism. Anorexia nervosa patients are distinctly underweight, often to the extent of complete cachexia. Not infrequently, body weight is more than 25 % below age-specific norms (in adults, a BMI of <17.5). Once a certain weight threshold has been fallen below, extensive disruption to the endocrine system is seen, including amenorrhea and loss of libido. Psychological and psychiatric comorbidities manifest more frequently (depression as well as anxiety, personality, and addictive disorders).

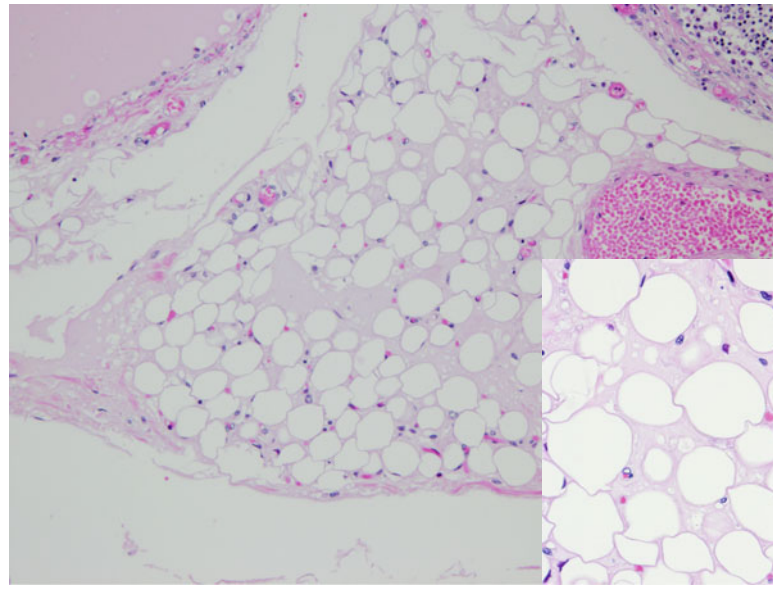

Fig. 16.1 Gelatinous atrophy in fatty tissue due to hunger striking (77 days) (H&E×100; H&E×400)

Furthermore, an electrolyte and vitamin imbalance occurs, albumin and overall protein levels in blood are reduced, and skin becomes dry and flaky; anemia and liver dysfunction are sometimes concomitant. Next, liver enzymes are increased (transaminase, lipase), and the onset of osteoporosis and kidney dysfunction is seen in later stages, as well as possible peripheral neuronal damage and seizures. In this setting, the sudden death of a relatively young patient, likely in a state of extreme chronic malnutrition, occasionally prompts a forensic autopsy to determine the cause of death. In contrast to other cases of chronic malnutrition due to neglect (in children, care-dependent persons, etc.) or hunger strike, the patient history can be followed regularly over a long time period in the case of anorexia nervosa, while the condition of the body at the time of death also suggests the diagnosis. Anorexia nervosa has a mortality rate of 10 %.

16.1.2 Starvation and Death by Starvation in Children

Alongside the abovementioned findings, additional signs seen in children in terms of age and initial weight range from growth retardation to short stature. Significantly reduced body weight is also seen in children at autopsy (Fig. 16.2; see Fig. 20.13).

Adverse effects are classified by applying anthropometric parameters, such as those specified by the WHO. According to the WHO classification, the extent and duration of PEM are subdivided into three groups:

Stunting: Insufficient height relative to age. This anthropometric parameter reflects growth in height and is an indicator of growth failure as a result of long-term malnutrition.

Wasting: Insufficient weight relative to height, an indicator of acute weight loss.

Underweight: Insufficient weight relative to age.

According to the WHO definition, one of the above forms of malnutrition is present if the corresponding anthropometric finding in a child falls below two standard deviations from the median of the same-age reference group.

In order to better assess the extent of starvation or chronic malnutrition in children, PEM classifications were developed in the field of pediatrics on the basis of anthropometric data. Alongside the Wellcome classification of PEM (Table 16.2) and the Gomez classification (Table 16.3), the Waterlow classification is the most commonly used to estimate nutritional status (Table 16.4).

The Gomez classification has the disadvantage that smaller children are still classified as

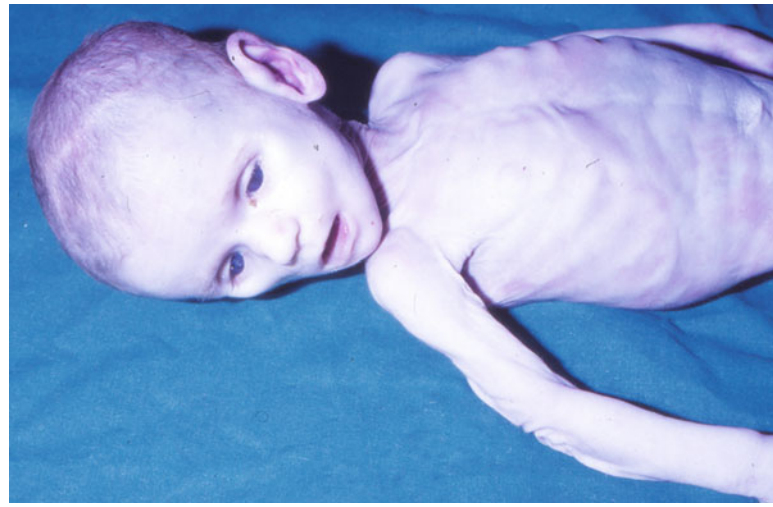

Fig. 16.2 Death by starvation, female, 9 weeks, significantly reduced body weight

Table 16.2 The Wellcome classification of protein–energy malnutrition (PEM)

	Percentage of benchmark values for weight/age (%)	Edema
Subclinical PEM	80–60	–
Marasmus	<60	–
Kwashiorkor	>60	+
Marasmic kwashiorkor	<60	+

Table 16.3 Classification of protein–energy malnutrition (PEM) according to Gomez

	Percentage of standard weight (age) (%)
Gomez I	90–76
Gomez II	75–61
Gomez III	<61

malnourished even if their weight is adequate relative to their height. Thus, if there is a high percentage of children of shorter height in a particular region compared to children in industrialized countries, applying the Gomez classification will result in a considerable overestimation of malnutrition.

The Waterlow classification uses the age-independent weight-to-height relationship as well as the child's height. Growth retardation is interpreted in the classification as a sign of chronic PEM, i.e., a previous state of starvation. However, the best indicator of acute PEM is insufficient weight relative to height (wasting). Severe acute malnutrition is present in the case of a weight-to-height relationship of less than 70 % of the standard value. The Waterlow classification estimates chronic starvation dystrophy and retardation by using weight-to-height relationships and height-to-age relationships as follows:

Table 16.4 Waterlow classification for the estimation of malnutrition

Weight/height Height/age	Value > 80 %	Value < 80 %
Value >80 %	Normal	Acutely malnourished
Value <80 %	Chronically malnourished	Chronically and acutely malnourished

Weight/height–height/age

Two fields are defined for each parameter—above two standard deviations and below two standard deviations, altogether yielding four defined categories (Table 16.4):

- Normal nutrition
- Acute malnutrition
- Chronic malnutrition
- Acute and chronic malnutrition

Other interpretations of this classification additionally differentiate on the basis of:

1. *Degree of retardation*: >95 %, normal; 95–87.5 %, mild; 87.5–80 %, moderate; <80 %, severe
2. *Degree of acute malnutrition*: >90 %, normal; 90–80 %, mild; 80–70 %, moderate; <70 %, severe

Table 16.5 Classification of malnutrition in children

	Mild malnutrition	Moderate malnutrition	Severe malnutrition
Percent ideal body weight (%)	80–90	70–79	<70
Percent of usual body weight (%)	90–95	80–89	<80
Albumin (g/dl)	2.8–3.4	2.1–2.7	<2.1
Transferrin (mg/dl)	150–200	100–149	<100
Total lymphocyte count (per µl)	1,200–2,000	800–1,199	<800

Weight and height are not the only parameters used to assess the degree of chronic malnutrition in children–variations in certain laboratory parameters are also correlated with the severity of malnutrition (Table 16.5).

Duration of Starvation. According to data in the literature, if there is a complete lack of food intake, a weight reduction of 0.7–1 %/day of a child's initial weight can be assumed when estimating the duration of starvation or death by starvation. Using the weight measured at the time of examination or autopsy should yield a time period that approximately corresponds to the duration of starvation.

16.2 Death by Dehydration

Whereas an individual can survive a lack of nutrition for a relatively long time period (see below), fluid deprivation or insufficient water intake can cause death in a considerably shorter period of time.

Although daily fluid requirements depend on ambient temperature and level of physical activity, a person requires a minimum of between 1 and 2 l/day. The feeling of thirst becomes apparent from 0.5 to 3 % loss of body fluids. The body needs water in the blood vessels to transport nutrients and cells, as well as in individual cells to maintain cell metabolism and regulate temperature. Failure to drink water causes dehydration.

Hypertonic Dehydration (or exsiccosis): Reduction in body water. Death by dehydration occurs as a result of hypertonic dehydration, i.e., primarily Na levels in plasma remain almost unchanged despite loss of water.

Severe states of dehydration are associated with central nervous disorders, most notably impaired consciousness and reduced vigilance. Hypertonic dehydration causes hypovolemia, a drop in cardiac output and blood pressure, as well as hypovolemic shock.

Loss of skin turgor and raised skin folds are striking at autopsy (Fig. 20.13). The speed of death by dehydration is highly temperature-dependent, occurring within 1 day in a hot desert or after many days under cool and shady conditions, whereby perspiration is also a relevant factor.

16.3 Causes of Death

In the case of death due to starvation or dehydration, death is caused by a variety of pathologic changes or diseases.

Lack of fluids (death by dehydration) can lead to an electrolyte imbalance accompanied by ventricular arrhythmia, as well as to hypovolemic shock and subsequent cardiovascular failure.

Death due to lack of nutrition can occur either early on in the course of starvation or at a later stage depending on specific circumstances such as age, preexisting diseases, preexisting nutritional status, fluid intake, and ambient temperature. Morgulis (1923) reported a time period of 17–76 days (median 40 days), while Prokop (1966) reported 60 days for nutrition deprivation only and 8–21 days for combined nutrition and fluid deprivation.

In the case of death due to chronic malnutrition (death by starvation), acute lethal hypoglycemia is the most likely immediate cause of death alongside secondary infections, e.g., purulent bronchopneumonia, ascending urethritis, and pyelonephritis. In addition to these findings, hepatic steatosis of varying degree has been reported histologically.

In certain cases, postmortem biochemical investigations can help in the diagnosis of hypoglycemia:

Hypoglycemia. As with the diagnosis of hyperglycemia or diabetic coma as the cause of death, measuring glucose and lactate levels in cerebrospinal fluid or vitreous humor can also help in the setting of hypoglycemia. Although postmortem values are subject to significant variation depending on the postmortem interval and ambient temperature, it is still helpful to calculate the sum value of glucose and lactate measured in cerebrospinal fluid or vitreous humor (sum value in mg/dl according to Traub). Thus, lower-range sum values of 50–80 mg/dl in cerebrospinal fluid and 100–160 mg/dl in vitreous humor are given for fatal hypoglycemia. However, there is no systematic investigation that involves determining glucose and lactate values in cerebrospinal fluid and vitreous humor in the case of death by starvation or dehydration. This should be borne in mind when interpreting the values measured, since their reliability is limited in cases of fatal starvation/dehydration.

Hyperglycemia and Diabetic Coma. Values exceeding 362 mg/dl in cerebrospinal fluid indicate a high likelihood of lethal diabetic coma once other causes of death have been ruled out; an upper value of 410 mg/dl has been reported in the literature for vitreous humor. Both values are believed to remain relatively stable for up to 10 days post-mortem under cold-storage conditions. HbA-1c determination can be used to estimate antemortem glucose levels, whereby an increased value is an indication of preexisting hypoglycemia of long standing.

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Clinical Forensic Medicine

17

Case Study

A 13-year-old girl was brought to the hospital by the police and her mother for examination. According to the girl's report, her older girlfriend's intoxicated boyfriend had attempted to rape her 7 h earlier. The incident had taken place at the home of the girlfriend as the victim came out of the shower wearing only a towel. Although the girl had resisted to the best of her ability, her assailant was the stronger party. The man had gripped her by the throat with his left hand, unfastened his trousers with his right, and forced himself between the victim's legs. After being forced to the ground, the girl screamed loudly, prompting her assailant to cover her mouth with his right hand. At that moment, the girlfriend returned home, upon which the assailant abruptly desisted in his attempts.

The young girl, who was very slight of build, showed patches of skin reddening on the neck and a small crescent-shaped skin laceration on the left side. Individual conjunctival petechiae were visible. Two small contusions could be seen on the mid-third of the inner side of the thigh, as well as two further small contusions on the spinous processes of two vertebral bodies of the lower thoracic spine, respectively. The contusions were bluish in color and well demarcated.

Clinical forensic medicine is the application of medical knowledge for the assessment of injuries in living persons for the purposes of administering justice.

The forensic examination of living individuals, although a comparatively neglected field of forensic medicine in some countries, is gaining in relevance. Affected subjects are often—but not exclusively—examined by order of the relevant authorities for the purposes of making and interpreting findings in the context of a specific diagnostic task:

- Examination of victims and suspected perpetrators (victim and defendant examinations) in the case of offenses involving bodily injury and attempted homicide, e.g., cases of survived strangulation.
- Abuse, sexual abuse, and neglect of children.
- Victims of violence in the home (domestic violence) or in the workplace, e.g., violence against physicians and nursing personnel.
- Violence against elderly (forensic gerontology) and care-dependent individuals.
- Victims and suspects/defendants in sexual offense cases. Since strong resistance is often used in the case of trauma to the neck, it is desirable from a forensic medical perspective to examine a suspect/defendant where possible in order to reconstruct the facts of an offense more accurately where necessary.
- Examinations for the purposes of forensic age diagnosis.

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- Traffic accident victims, whether as pedestrians, drivers, or passengers, etc. (including appraisals for civil proceedings to obtain damages or compensation, such as in cases of whiplash injury).
- Cases of self-harm, e.g., to simulate a criminal offense.
- Self-mutilation, e.g., for the purposes of committing insurance fraud.
- Assessment and interpretation of forensic psychiatric findings (see Chap. 18).
- Evaluation of an individual's fitness to understand a police caution, undergo questioning, be held in custody, stand trial, serve a custodial sentence, travel, or undergo deportation, e.g., in the context of asylum procedures.
- Assessment of findings in torture victims (see Chap. 26).

Although injury patterns can vary greatly, they need to be consistent with an alleged offense. It is sometimes possible to make inferences about a particular form of trauma on the basis of marks on the skin, such as finger marks following blows from the flat of a hand (Fig. 17.1) or cigarette/cigar burns (Fig. 17.2).

In this context, it is important to distinguish between various forms of trauma; of particular importance here is the differentiation between injuries caused by blows and injuries caused by falls. The localization of an injury often aids in its interpretation (Table 17.1).

Not infrequently, a forensic medical appraisal is initially only supported by a pattern of injury; ultimately, however, any appraisal should be formed on the basis of all available information: reported history, injury pattern seen on

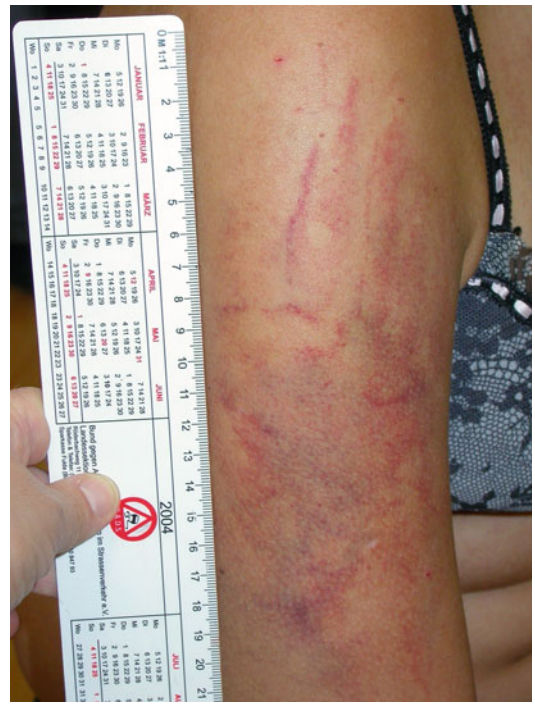


Fig. 17.1 Finger marks following a blow from the flat of a hand

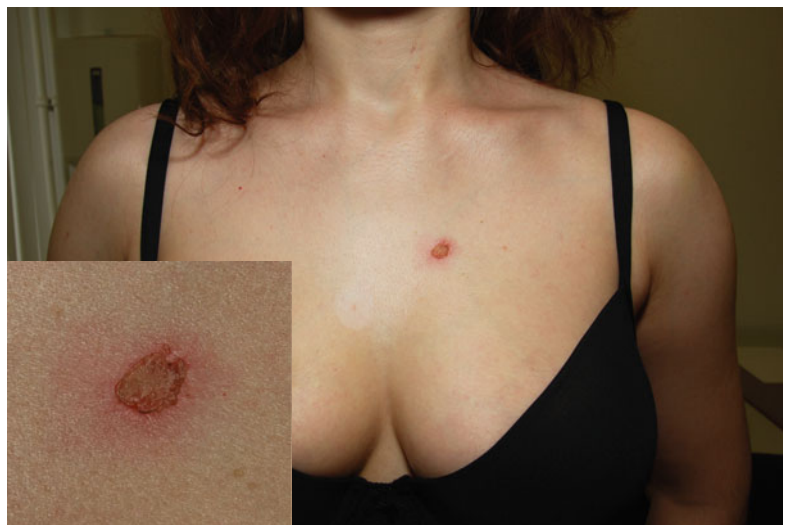


Fig. 17.2 Circular cigarette burn in a case of domestic violence

Table 17.1 Interpreting injuries according to their localization

Injury localization (selection)	Interpretation[a]
Head (above the hat line)	Likely a blow (with the exception of falls downstairs)
Head (below the hat line)	Typical of falls: frontal protuberance, eyebrows, tip of the nose, tip of the chin (with the exception of a blow to the face)
Monocle hematoma	Usually a blow (rarely: blood that has descended following a fall on the eyebrow; orbital roof fracture with monocle hematoma after a fall on the back of the head)
Contusions on the upper arm (inner and outer), possible also bilaterally symmetrical	Hand marks
Contusions on the extensor side of the lower arms	Self-defense injury
Back of the hand	Passive self-defense injury
Palms of the hand	Injury incurred while trying to support oneself during a fall; active self-defense against sharp trauma
Contusions over the hip bones	Generally caused by impact trauma
Outside of the elbow	Typical of a fall
Extensor side of the knee	Typical of a fall
Back, buttocks	Typical of corporal punishment
Oral mucosa	Likely a blow but possibly also a fall
Over the spinous processes of vertebral bodies	Abutment injury due to anterior pressure (victim lying with their back on a hard floor)
Orbital fracture	Blow (blow-out fracture)
Horizontal ligature marks	Ligature strangulation
Ligature marks slanting upwards	Hanging
Parallel contusions	Injury due to blows
Extensive skin reddening on the neck and small superficial skin lacerations	Manual strangulation (one or two hands)
Small patches of skin reddening on the neck	Manual strangulation (DD "hickey"/"love bite")
Incision wounds to the flexor side of the wrist	Self-inflicted: (attempted) suicide
Bullet entry wound to the hard/soft palate	Generally suicide
Contusions to the inner side of the upper thigh	Self-defense injury in (attempted) rape (Fig. 17.3)
Often multiple, parallel scars on the flexor side of the forearms	Self-inflicted (borderline syndrome?)

[a]Note: Any interpretation should encompass the whole pattern of injury; in some cases, an alternative explanation or unusual sequence of events may account for injuries

examination, laboratory findings, and possibly also radiological findings.

Important: Having as much information as possible to support injury assessment, as well as a familiarity with other findings to be included in the overall picture, is of critical importance to any forensic medical appraisal.

Human bite wounds are occasionally seen and must not be associated with sexual offenses (Fig. 17.4; see Figs. 9.14 and 9.15).

Petechiae in the sclerae, conjunctivae, and oral mucosa or extensive hemorrhage beneath the conjunctivae (Fig. 17.5) suggest compression trauma to the neck (ligature or manual strangulation, as well as atypical hanging possibly involving tearing of the ligature).

The average resuscitation time for individual organs following interrupted oxygen supply to the organism is crucial for prognosis; however, these are subject to considerable variation depending on the age and temperature of the organism. The following times are given as a guideline:

- Brain: approx. (3) 5–8 min (followed by irreversible damage)
- Heart: approx. 15–30 min
- Liver: approx. 30 min

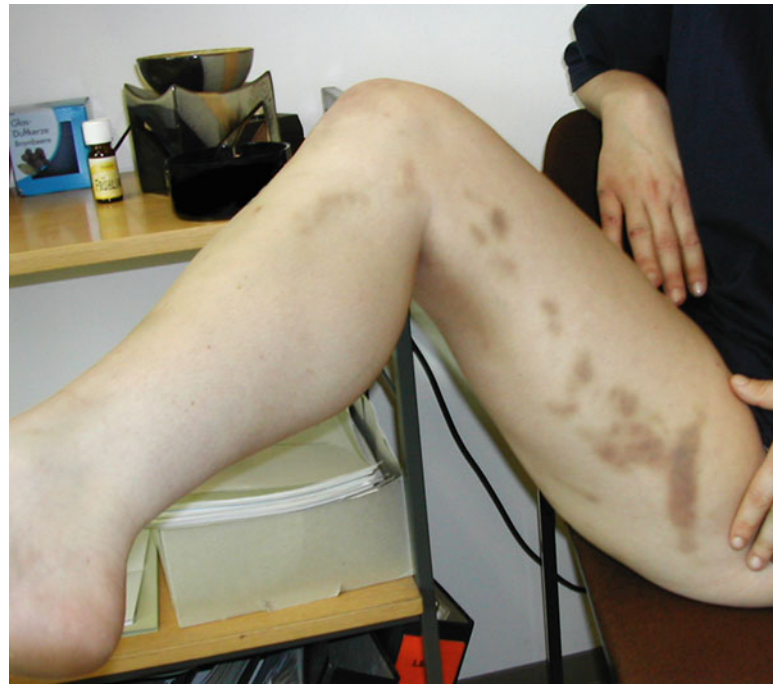
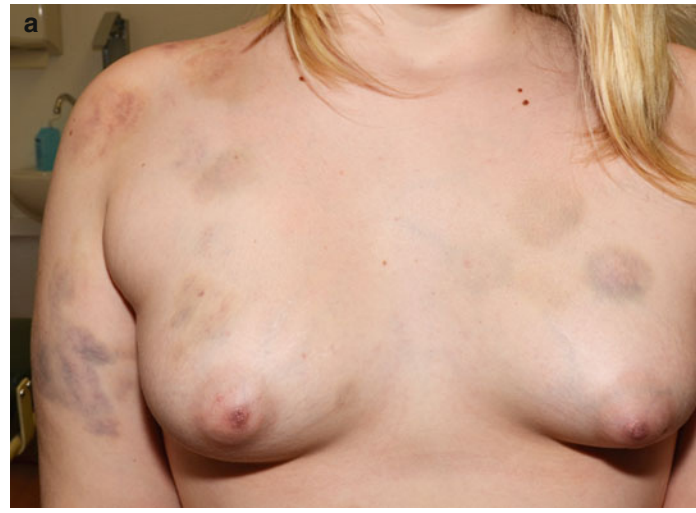


Fig. 17.3 Wounds in a case of (attempted) rape



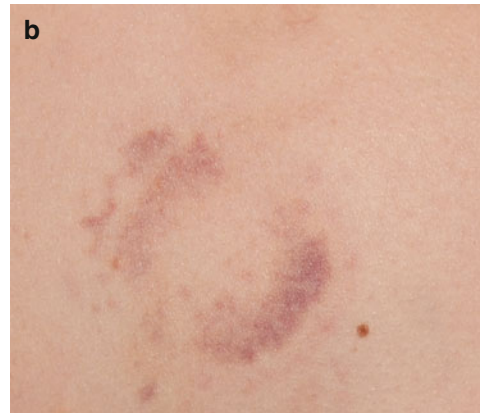


Fig. 17.4 (**a**, **b**) A young woman with multiple bite wounds of characteristic shape in a case of domestic violence

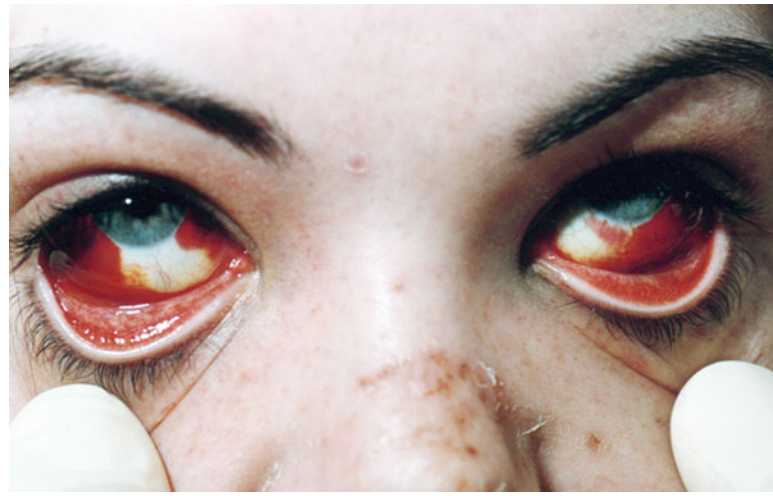


Fig. 17.5 Extensive hemorrhage beneath the conjunctivae in a case of survived manual strangulation

Table 17.2 Findings on the hemodynamic and neurological effects of neck compression

Hemodynamic effects	Neurological effects
Congestion above the level of compression?	Loss of consciousness?
Facial cyanosis?	"Blacking out?"
Petechiae?	Memory gaps?
Hemorrhage under the skin of the neck?	Involuntary defecation and urination?
Swelling of the laryngeal mucosa causing luminal narrowing?	Impaired vision?
Pain on swallowing (dysphagia)?	
Changes in voice pitch (dysphonia)?	

- Lungs: approx. 60 min
- Kidneys: approx. 90–120 min
- Muscles: approx. 2–8 h

Any forensic medical appraisal also needs to address the question of injury severity. Particularly in the case of compression trauma to the neck, information on duration, intensity, and lethality is sought. To this end, signs of the hemodynamic and neurological effects of neck compression need to be established (Table 17.2).

In addition to signs of the hemodynamic and neurological effects of neck compression, attention should be paid to other injuries in the appraisal of injury severity or when making conclusions on the lethality of neck compression. Under the case law rulings of Germany's highest criminal court, laryngeal cartilage fracture, prolonged respiratory distress, as well as overall duration and intensity are relevant to the question of lethality. Injuries and symptoms permit inferences to be made about duration and intensity, as do the approximate time periods to loss of consciousness:

(a) Complete interruption of arterial blood supply to the brain: Loss of consciousness within a few seconds (6–10 s), e.g., typical hanging.
(b) In experiments, loss of consciousness for up to 100 s caused by interrupting blood supply left no residual damage.
(c) Isolated occlusion of the airways, on the other hand, may be withstood for somewhat longer before loss of consciousness, while blood circulation is maintained, e.g., gagging causes airway displacement by displacing the tongue to the posterior pharynx.

The severity of injuries and symptoms tends to be greater in victims having suffered a loss of consciousness (Table 17.3).

Lethality and Severity of Strangulation. Injuries are considered to be lethal if death would be imminent without (medical) intervention, e.g., a victim bleeding to death in the absence of surgery or a blood transfusion. The injuries inflicted by the perpetrator may (not "must") cause death without the perpetrator being able to influence the further outcome in any way (the further course is ineluctably predetermined)—this is assumed in the case of neck compression where an acute onset of neurological symptoms

Table 17.3 Congestive petechiae and symptoms in 82 survivors of manual or ligature strangulation

	Victims who remained conscious ($n=42$) (%)	Victims who lost consciousness ($n=32$) (%)
Congestive facial petechiae	55	78
Urination	7	31
Defecation	0	16
Amnesia	0	25
Dyspnea (difficulty in breathing)	29	13
Dysphagia	71	78
Dysphonia	5	6

From Strauch et al. (1990)

is seen, e.g., loss of consciousness, memory gaps, as well as involuntary defecation and urination. Affirming the actual lethality of neck compression from a medical perspective in the presence of facial and conjunctival petechiae but in the absence of neurological symptoms is controversial and certainly requires careful pathophysiological consideration. Thus, from a medical point of view, lethality can be affirmed when, following trauma-related injury or damage, death would have been imminent in the absence of external intervention or when the further course of injury had reached a stage where the perpetrator no longer had any influence on whether the victim survived or not (an inevitable course following a chain of events caused by the perpetrator): in particular, irreversible damage to brain tissue with increasing brain edema due to damage to the central nervous system as a result of oxygen deprivation (CNS; diffuse axonal injury, DAI). Although at this point death and survival (with or without complications) are both possible outcomes, the perpetrator is no longer able to influence the outcome, since his/her actions have progressed too far.

Classifying Severity in Strangulation Survivors. Attempts have been made to classify the severity of trauma; the following classification has been proposed for strangulation (from Plattner et al. 2005):

"*Severe*" *form*: Only lethal if petechial hemorrhage is present, with or without loss of consciousness

"*Moderate*" *form*: Injury to the soft tissue of the neck or larynx, sore throat, dysphagia, hoarseness, and hemorrhage beneath the skin

"*Mild*" *form*: Only superficial skin findings

A more recently proposed classification assumes lethality if at least one of the following three criteria is fulfilled (from Parzeller et al. 2008):

- Venous congestion above the level of strangulation and detection of facial petechiae (including conjunctivae and mucosa), which cannot be explained in any other manner
- Loss of consciousness, either supported by witness evidence or credibly self-reported, at the time of neck trauma, which cannot be explained in any other manner
- Evidence of severe injury to the upper neck and pharyngeal soft tissue or the upper respiratory tract capable of causing secondary obstruction or decreased cerebral perfusion/hypoxia

The literature gives the following times to the onset of loss of consciousness, inability to act, and irreversible cardiac arrest:

(a) Loss of consciousness and inability to act due to complete compression of the large vessels in the neck: 6–14 s (Rossen et al. 1943; Denk and Missliwetz 1988).
(b) Ligature strangulation times of up to 100 s have been survived without complication (Rossen et al. 1943).
(c) Petechiae appear after between 20 s and several minutes (Maxeiner 2001).
(d) Irreversible loss of brain function after 3 min and more (Henßge 1990).
(e) Ultimate cardiac arrest after 5–18 min (Rossen et al. 1943).

Data on the speed at which petechiae appear vary from 20 s to approximately 3 min. At low

pressures (35 mmHg), petechiae appear after 15 min, whereas at higher pressures (90 mmHg) they appear after around 3 min (Prokop und Göhler 1976). Petechiae could be produced after around 3 min in experiments on human skin with suction cups. However, an abrupt rise in pressure has been reported to cause petechiae in significantly shorter times; moreover, particularly in the case of homicide, additional pressure increases in the chest area due to strong respiratory movements serve as a compounding factor. Thus, data on precise time courses between neck compression, loss of consciousness, and death should be disregarded, since sequences of events relating to crimes are often complex and the pathophysiological processes subject to great variation.

Therefore, the duration of asphyxia can vary considerably depending on its cause. Shorter times are given for uninterrupted suppression of blood supply and blood flow to and from the brain (as in typical hanging) with total arterial and venous compression. Somewhat longer times are seen in partial suppression of oxygen supply and/or blood flow to and from the brain (atypical hanging, ligature strangulation). Longer times are given when oxygen supply to the lungs is obstructed but brain perfusion maintained, as in manual strangulation or other forms of respiratory obstruction (e.g., suffocation with a soft cover, gagging) without compression of the blood vessels of the neck.

The time to the onset of damage and unavoidable death in the case of total suppression of blood flow to the brain (typical hanging, uninterrupted massive ligature, or manual strangulation) is generally in the low single-digit minute range (at room temperature). The time to the onset of damage and unavoidable death in the case of interrupted oxygen supply but maintained blood supply is likely to be somewhere between the upper single-digit minute range and 10 min or slightly more (a "flushing effect" prevents acidosis).

Thus, in cases where victims of violent trauma survive, it is important from a forensic medical perspective to answer the following questions:

- What type of trauma has taken place? Particularly in the case of neck trauma: hanging or manual/ligature strangulation?
- Which injuries can be identified as resulting from the effects of trauma?
- Can self-inflicted injury be ruled out?
- Can the intensity of injuries and trauma be measured in terms of degree from a medical point of view?
- Can it be assumed from a medical point of view that acute lethal trauma has taken place? If so, on what criteria is this statement based and how can the lethality of the trauma be established?
- Is it possible to draw any conclusions about the duration of trauma?
- Is it possible to draw any conclusions about the victim's ability to act, or their actual actions, during the incident?
- In addition to the principle injury, were any further injuries incurred during the course of the incident, such as facial contusions, active and passive self-defense wounds (on the flexor side of the forearms or palms of the hand), hand marks (contusions on the upper arm), lacerations at the corners of the mouth, small injuries to the lips and oral mucosa (due to the mouth being held shut to prevent the victim from screaming), tooth marks (due to a blow to the lips), possibly also contusions/injuries and findings consistent with an attempted sexual offense?

17.1 Self-Inflicted Injuries in Suicides and Attempted Suicides

Mention should be made here of tentative, or hesitation, wounds—the frequently encountered scars running either laterally or longitudinally across the flexor side of the wrist—which are occasionally fresh incision wounds made in the context of an acute suicide or an attempted suicide (Fig. 17.6). Tentative wounds of this kind are also seen in the form of deeper incision wounds to the neck with suicidal intent, in which case they are largely superficial (epidermis and subepidermal dermis), straight parallel incision wounds, which rarely go as deep as the subcutaneous fatty tissue. In some cases, the victim fails to make deeper incisions and the incision wounds heal (Fig. 17.7).

Fig. 17.6 Self-injurious behavior involving tentative wounds made to the flexor side of the wrist

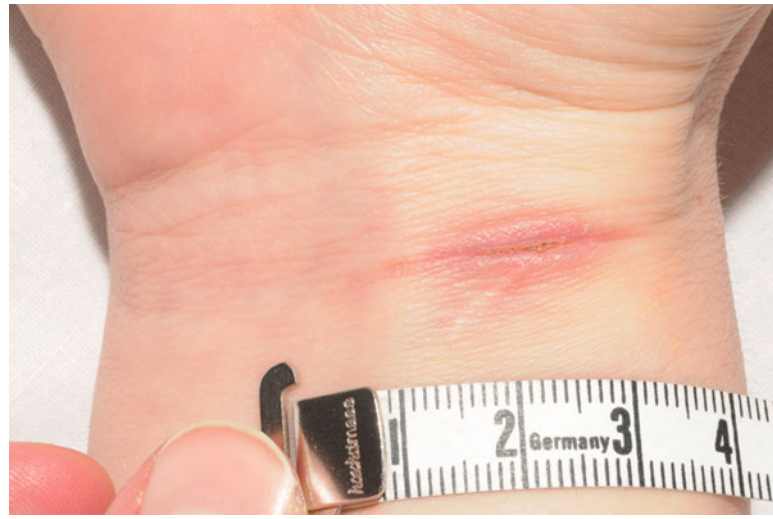


Fig. 17.7 A single older tentative wound made in the context of a non-genuine attempt at suicide with overtones of an "appeal for help"

Although tentative wounds on the flexor side of the wrists can generally be attributed to (attempted) suicide, injury patterns that are inconsistent with an alleged course of events are sometimes seen, thereby necessitating a differentiation between self-inflicted injuries and injuries inflicted by others.

17.2 Self-Harm and Self-Mutilation

The Anglo-American literature reports the prevalence of self-injurious behavior at 0.6–0.8 % of the population; according to statistics, there is a marked prevalence in the 15- to 35-year-old age group; moreover, women are between two and five times more frequently affected.

Some cases of self-injurious and self-mutilating behavior prompt the relevant authorities to seek clarification on the causes or circumstances behind the injuries. Although self-harm is not in itself a criminal offense, a lack of further information may arouse the suspicion of a simulated criminal offense. Where an individual is suspected of having committed an offense that was in fact simulated by another, not only may the wrong person be suspected but an instance of libel may be considered. If a third person is used for the purposes of self-harm or self-mutilation,

this may represent an offense against common decency, which can also be a criminal offense. In practice, self-harm should also be considered in the context of simulating a self-defense situation or with the intention of incriminating another person, such as a law enforcement officer following an arrest. There are also cases where the perpetrator self-inflicts injury in order to simulate a robbery, whereas he in fact stole the goods himself. Occasionally, fatal accidents, such as traffic accidents, are simulated in order to ensure that a life-insurance payout will provide financial security for surviving dependents. Self-mutilation is also seen in the context of making a claim against an accident insurance or in order to evade military service, e.g., a "million-dollar wound" or "blighty wound."

Self-harm (synonyms: self-injury, self-injurious behavior, self-inflicted injuries) describes direct, self-inflicted injury, usually of a nonserious nature. Injury is not inflicted with the intention of causing a life-threatening situation.

Self-mutilation describes deliberate, self-inflicted, substantial loss of peripheral body parts.

Although injuries incurred in the course of suicide or attempted suicide could also be seen as self-harm or, in some cases, self-mutilation, they are excluded in this context. The following causes form the basis of self-injurious behavior:

- Self-injury to simulate a criminal offense
- Psychiatric disorders
  - Psychoses
  - Neuroses
  - Borderline personality disorder
  - Compulsive self-injurious behavior
- As a "test of courage"
- For sexual reasons
- For the purposes of committing insurance fraud, e.g., amputating a finger and claiming for accidental injury while working in the garden
- Munchausen syndrome
- For religious or political reasons, e.g., self-immolation or hunger strike
- Self-harm while in custody
- Self-mutilation to evade compulsory military service
- Self-harm in asylum seekers to prevent deportation
- To follow fashion, e.g., tattoos, piercings, and other forms of body modification

In cases such as the above, a forensic physical examination and expert correlation with an alleged incident or event are usually able to contribute significantly to establishing the veracity of victims' claims.

17.2.1 Differentiating Between Self-Inflicted Injury and Injury Inflicted by Others in the Case of Alleged Assault

In order to assess injuries following an alleged but otherwise unverified assault, physical findings of injury need to be consistent with the findings made by the investigating authorities (see Table 17.4, Fig. 17.8).

A somewhat "fantastical" account of an assault is often given in the case of self-inflicted injuries. The presence of superficial, mostly parallel cuts or scratches to the skin on areas of the body that are easy for the victim to reach, e.g., forearms (Fig. 17.9a, b) or the anterior upper body (Fig. 17.9c), is striking.

17.2.2 Self-Harm and Psychiatric Disorders

Self-harm is primarily seen in borderline patients, less frequently in other histrionic, dissocial, or paranoid personality disorders. The injuries encountered can usually be explained by checking the psychiatric diagnosis of the underlying disease. Patients with psychoses and neuroses are capable of inflicting severe or even life-threatening injury on themselves and causing significant blood loss. Cases of self-immolation—otherwise occasionally seen in the context of political or religious protest to highlight a particular plight—are also seen in psychiatric patients. Psychiatric disorders may lead to suicide or attempted suicide involving self-inflicted injury; however, here again, an assault is occasionally reported as the alleged cause of injury.

Table 17.4 A comparison of the characteristics of self-inflicted injury and injury inflicted by others

Characteristic	Injured inflicted by others	Self-inflicted injury
Type of sharp injury	Usually stab wounds, some cuts, occasional amputations	Predominantly cuts, as well as scratches and transitional forms
Distribution	Irregular distribution over the body	In groups, largely parallel, occasionally in rows, symmetrical distribution
Localization	All body regions, without sparing of sensitive areas	Chest, pubic region, and unclothed areas are more often affected (arms, chest, abdomen); sensitive areas (nipples and lips) and functional areas (eyes, ears) are spared; the back and inaccessible areas are unaffected; emphasis on the contralateral side to the dominant hand
Type of individual injuries	Generally short, irregular, and distinctly curved in shape	Often long, constant, mildly curved, and uniform in shape
Intensity of individual injuries	Highly variable; often deep	Almost always constant; always superficial; uniform injury depth even on non-flat areas of the body
Number of individual injuries	Rarely multiple	Remarkable frequency of multiple injuries; signs of previous self-harm possible
Overall injury severity	Generally (very) severe	Consistently mild
Concomitant injuries	Generally multiple concomitant injuries of varying type	Occasional concomitant injuries of varying type (self-inflicted)
Clothing	Involved in the injury; bears multiple signs of physical conflict	Generally not involved; occasional signs of physical conflict (self-produced)
Self-defense wounds	Often typical, deep incisions on the flexor side of the fingers, palm of the hand, and forearms	No self-defense wounds or untypical, consistently superficial incisions on fingers, hands, and forearms

From König et al. (1987), Pollak et al. (1987)

Borderline Personality Disorder. A compulsive urge to self-harm as a means of releasing inner tension is seen predominantly in young women (aged around 15–35 years), who take care to conceal their self-inflicted injuries. The frequency of scratches in upper- and forearm skin is often remarkable in borderline personality disorder patients (usually females, but occasionally also males). On physical examination, straight, mostly fine scars in either a parallel or an intersecting formation can be seen.

Munchausen Syndrome. This patient group simulates disease symptoms, self-harms, exacerbates existing symptoms, and/or delays healing of existing wounds by deliberately introducing foreign substances into the wound. These patients show an abnormal willingness to undergo diagnostic and therapeutic measures, seek contact with physicians and nursing personnel, gain superficial medical knowledge over time, and frequently demonstrate a worsening of symptoms immediately prior to discharge from hospital. The desire for a renewed stay in hospital can become obsessive, while at the same time these patients demonstrate a certain indifference to the course of their disease and show little interest in making a recovery. Symptoms may be contradictory; moreover, these patients often discharge themselves from hospital and switch physicians and hospitals regularly.

The number of unreported cases of Munchausen syndrome is estimated to be high. In addition to up to 80 % of patients being female, individuals in the medical profession appear to be more commonly affected.

17.2.3 Self-Harm in Custody

Detention facility inmates, both with and without psychiatric disorders, show a tendency to self-harm. Some form of "incarceration shock" on initial incarceration may represent a trigger for self-injurious behavior. In some cases, fellow cell

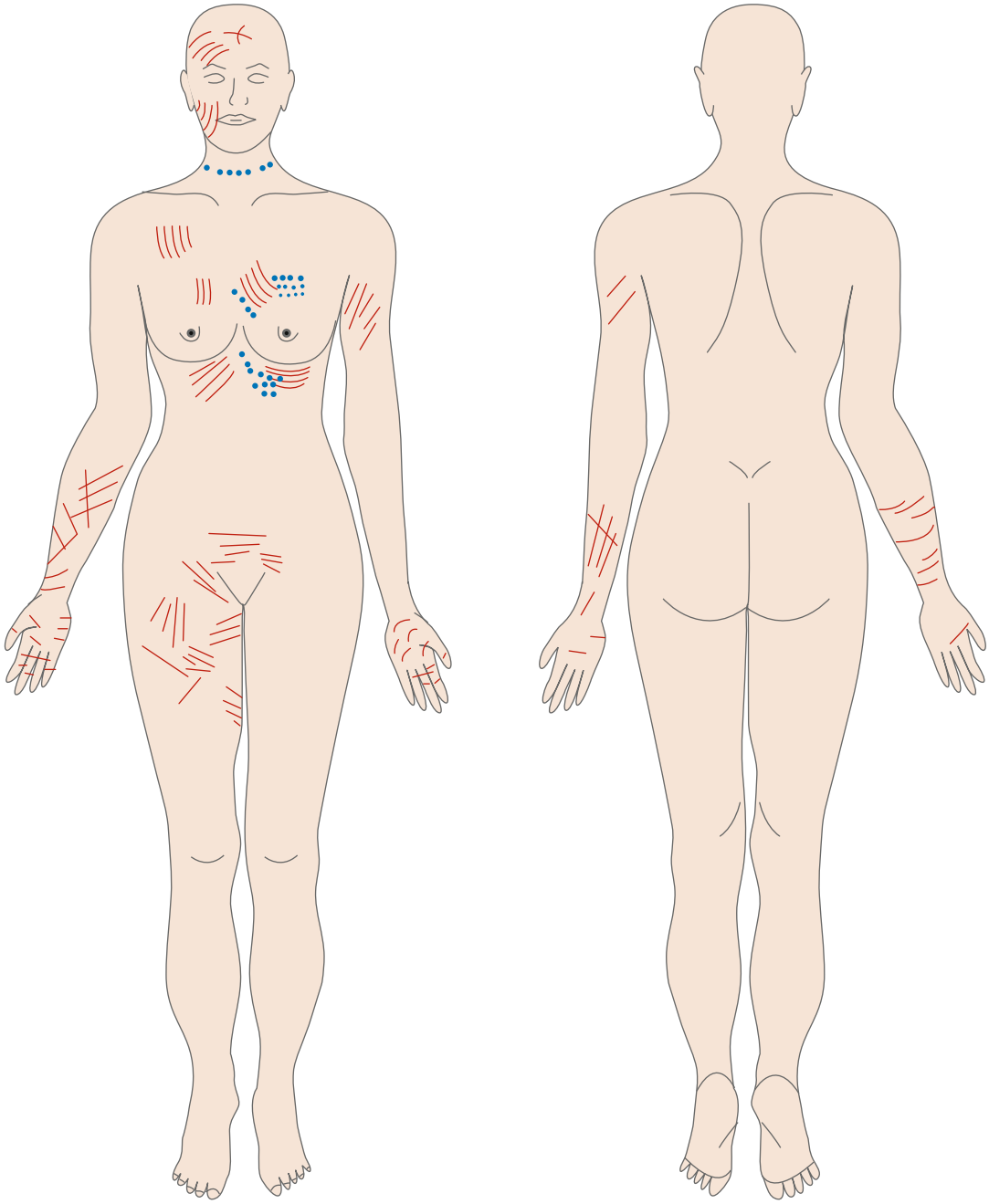


Fig. 17.8 Areas of the body affected in typical self-inflicted injury (From König et al. (1987))

inmates may be a triggering factor for anxiety and self-injurious behavior. In general, specific objectives are pursued through self-harm: increased opportunities for escape, transfer to a hospital, better detention conditions, different cell mates, etc.

Cases of self-harm predominantly involve intoxications and superficial stab wounds or cuts; the ingestion of corrosive substances is sometimes seen when inmates have access to such materials. In addition, foreign bodies may be

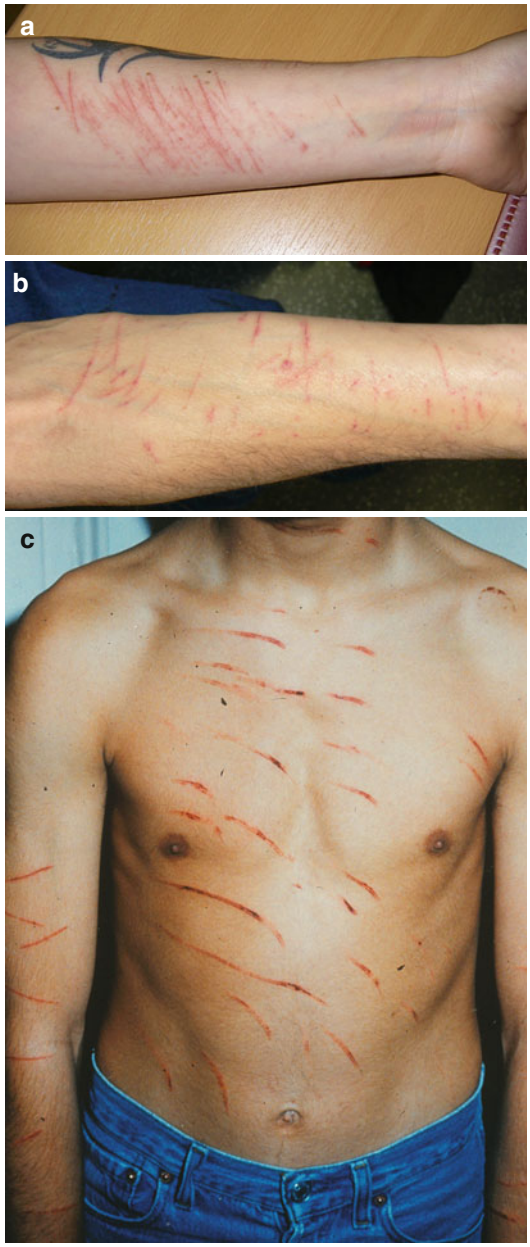


Fig. 17.9 (**a**, **b**) Self-injurious behavior with parallel superficial scratches on the forearm. (**c**) Self-inflicted superficial scratches or cuts

swallowed to induce disease symptoms, sometimes leading to life-threatening situations. Small objects found in the cell may be swallowed, including parts of cutlery, batteries, rarely razor blades, and occasionally drugs illicitly obtained in detention facilities. Life-threatening situations arising from the above, e.g., infections, perforations, and hemorrhages, are generally not intentional—hanging is the prevalent method of suicide in prisons.

17.2.4 Body Modification

The term "body modification" is used to describe varying degrees of physical modification undertaken on the explicit wish of the affected individual. The spectrum ranges from tattoos and relatively harmless piercings to severe injury associated with the risk of infection, such as implanting foreign material subcutaneously. Voluntarily inflicted burns ("branding") and scars in particular patterns or shapes produced by making incision wounds ("cutting") are also known. In some cases, the boundary between modifications that represent normal variants of acceptable behavior and unethical self-harm, possibly involving third parties, becomes blurred. Perspectives may be influenced by the cultural background of a particular country, as well as the cultural or social subgroup to which an individual belongs.

17.2.5 Self-Harm and Insurance Fraud

In addition to examining and interpreting injuries in surviving victims—both adult and pediatric—of violent trauma and the associated collection of evidence, clinical forensic medicine deals with assessing mechanisms of injury that give rise to the suspicion of self-harm or self-mutilation for the purposes of making insurance claims. Cases of this kind involve, for instance, amputations of extremities (thumb or finger) that have been either staged or carried out by the accident victim themselves in an alleged accident while performing farming or building activities. In some cases, claims are then filed with several accident insurance institutions. Self-mutilation is sometimes performed for the purposes of insurance fraud, in particular by amputating the distal phalanx of fingers, toes, whole fingers, and more rarely

hands and feet. There are a number of characteristic features of self-mutilation (Table 17.3). Deliberate self-mutilation usually affects one or two fingers, only rarely three or more. Total hand amputations are rare. The fact that genuine accidental finger and hand injuries often occur in the course of everyday life should always be borne in mind.

Table 17.3 Characteristics of suspected self-harm using finger injury as an example (from Rastrup (1992))

- Recently acquired insurance policy.
- Disparity between the insurance sum and the economic situation of the insured person.
- Failure to disclose double or multiple insurances.
- A lack of eye witnesses or witnesses are subsequently influenced for the purposes of eliminating conflicting information about the time and circumstances of the "accident."
- The description of the accident is primitive, emphasis is put on irrelevant details, and a description of the immediate sequelae is omitted.
- Untruthful claims about left- or right-handedness in order to make injury to the other hand more plausible.
- Disposal of amputated digit(s), implements, and evidence.
- Discrepancies between the reported course of events and the objective findings.
- Distinct injury abnormality (total loss of the finger, short finger stump, transverse line of transected edges, finger injury as the sole injury).
- Abnormal concomitants, such as pain relief and hemostasis prior to the accident.
- Abnormal behavior in the insured and their relatives when examined by the insurance physician.
- Immediate enthusiasm for and psychological disinhibition at the mention of proposed insurance settlements.

In some cases, attempts to reconstruct an accident for the purposes of clarifying how an injury was incurred can be helpful, particularly where complicated manual procedures involving machinery are concerned.

The distinction between an accident and self-harm is rarely achieved on the basis of a few abnormal features or types of behavior; generally, a great number of indicators need to be collated and considered jointly. The following require consideration from a forensic medical perspective:

- Type and localization of the injury, e.g., injury above the level of the sole of the foot
- Type and demarcation of injury to soft tissue and bone
- Direction of injury
- The ratio of injured to non-injured areas
- The work procedure
- The implement(s) used
- The workpiece
- Physical conditions, including lighting among others
- Traces of evidence

Once the results of an investigation have been evaluated, it is necessary to establish whether the injury could have been:

- Caused by the implement reported, in the position the implement was reported to be in
- In the reported posture
- Caused to the limb actually injured
- Caused by a physiologically and mechanically plausible mechanism

It must then be determined whether or not the injury is consistent with the sequence of events described by the injured party or witnesses, whether injury could have occurred spontaneously in the course of performing common and familiar activities, or whether it could only be explained if some form of contrived posture was adopted. It is also necessary to establish whether information given about the accident is consistent with the objective findings. For example, accidental amputation of the left thumb or a finger on the left hand (in right-handed individuals) allegedly due to poor aim while using an axe or hatchet is reported relatively frequently. In cases such as these, a poor aim that happens to strike the thumb is only possible if the left hand had been placed hazardously close to the point the axe was intended to strike in the first place. Where this is the case, the direction of amputation deducible from X-ray findings should be consistent with the

alleged initial position, that is to say, for example, from the ulnar side to the radial side and not from the extensor side to the flexor side.

17.3 Fitness to Undergo Questioning, Be Held in Custody, Stand Trial, and Receive a Custodial Sentence

Particularly in the context of judicial proceedings, there are a number of circumstances that require a (forensic) medical assessment of witnesses (victims) and defendants (suspects) to establish their status: from the time of arrest, police caution, and questioning (capacity to understand a caution, fitness to undergo questioning) to detention and trial (fitness to be held in custody, capacity to stand trial, and fitness to serve a custodial sentence).

Fitness to Undergo Questioning. Witnesses (including victims) and defendants should be questioned as soon as possible following an incident. A defendant must be given the opportunity to comment on the charges brought against him/her. Since, following caution, the information or description given by the defendant will be used in subsequent legal proceedings, the defendant must be fit to undergo questioning.

Fitness to undergo questioning is the capacity to understand the meaning of and answer questions posed during a hearing (or questioning) by the investigating authorities (police, public prosecutors) and the court. The defendant possesses sufficient ability to communicate in a contextually ordered manner.

During questioning, the interrogee's capacity to make decisions should not be impaired by disease, intoxication, etc. Impairment to the suspect as a result of prohibited interrogation methods (§ 136a, German code of criminal procedure), such as threats of violence (torture), is not permitted. A suspect is not fit for questioning, for example, when his/her consciousness, ability to reason and make decisions, or memory is severely impaired. Acute severe alcohol, drug, and/or medication intoxication, as well as acute withdrawal symptoms and severe exhaustion or fatigue, can cause a suspect to be unfit for questioning. Thus, it is quite possible that the procedural admissibility of statements made by suspects under the influence of alcohol or drugs requires subsequent examination. If, following questioning, the suspect remains under suspicion, he may be detained in police custody until arraignment, provided fitness to be detained in custody is proven.

Fitness to Be Detained in Custody. A distinction is made between fitness to be detained in custody and fitness to serve a custodial sentence. An individual may be detained in custody until arraignment or for the purposes of sobering up. Where doubt exists, fitness to be detained in custody needs to be medically checked or established.

Important: Fitness to be detained in custody relates to the question of whether the detention of a person in temporary police custody is medically tenable.

Crucial here is the fact that a person is (initially) held in temporary custody in areas or cells specified by the police. Restrictions to an individual's fitness to be detained in custody may arise in the case of:

- Acute (psychophysical) disease requiring therapy or surgery
- Medical conditions, such as diabetes mellitus, hypertensive crisis, epileptic seizure, and cardiovascular disease
- Alcohol, drug, or medication intoxication
- Psychiatric disorders such as acute psychosis (claustrophobia, etc.), whereby in such cases, the question of forced detention may need to be examined, depending on the legal situation

If the investigating authorities are of the opinion that grounds for detention are present, the individual held in custody must be brought before a committing magistrate, who then decides whether the individual will be, at least initially, remanded in custody—on the provision that the person is fit to be detained in custody.

Fitness to Serve a Custodial Sentence. Whenever a person is to be placed in custody, such as remand or prison, it is necessary to establish whether this form of long-term detention is

medically tenable. The following represent possible restrictions to fitness to serve a custodial sentence:

- Acute life-threatening disease
- Severe psychiatric disease
- Chronic depleting diseases such as anemia or advanced cancer
- Conditions associated with hunger strike

As with other concepts, there is no legal definition for fitness to serve a custodial sentence. Prison physicians may be called upon to establish whether a person is fit to serve their sentence or an examination may be carried out in a prison hospital.

Important: Unfitness to serve a custodial sentence presupposes an immediate disease-related risk of death, a severe psychological deterioration, or severe and usually chronic health impairment.

An individual detained in remand will only be transferred to a regular prison once their sentence has come into force following trial. Attendance at trial presupposes that the detained individual is able to travel to court and attend court sessions. In general, fitness to travel and attend court sessions can only be impaired on the basis of severe impairments to health, which need to be medically established. The question of whether an individual is fit to stand trial is more commonly raised.

Fitness to Stand Trial. Occasionally, the question of whether a person is fit to stand trial or participate in legal proceedings and conduct his/her defense (legal capacity to sue and be sued) requires clarification. According to the German Federal Constitutional Court (*BVerfG NJW* 1995, 1951), a defendant should have the capacity, both within and outside of court proceedings, to defend his/her interests in a reasoned way, put forward his/her defense in an informed and understandable manner, as well as make and understand procedural declarations.

To ensure that these requirements are fulfilled, it is the task of the medical expert, usually called upon by the court, to provide information on an individual's fitness to stand trial while taking the following points into consideration

- Type of disease?
  - Organic disease?
  - Psychiatric disease?
- Acute or chronic intoxication?
- Withdrawal symptoms?
- Is there a temporary disease-related impairment to an individual's fitness to stand trial?
- Where relevant, is unfitness to stand trial total or partial?
- Can the disease causing total or partial unfitness to stand trial be medically treated with curative therapy? If yes, when can a restoration to fitness to stand trial be expected?
- If the disease cannot be treated curatively, would palliative therapy be able to prognostically guarantee fitness to stand trial until proceedings are completed?
- Could a disease only susceptible to palliative therapy cause unfitness to stand trial in the foreseeable future? If so, within what timescale?
- If a disease can be either curatively or palliatively treated, how much risk is associated with the required therapy as administered according to standard medical practice?

In practice, it should be borne in mind that defendants sometimes deliberately cause their unfitness to stand trial. This may be achieved by abusive ingestion of alcohol, drugs, and medication, a deliberate failure to take advantage of treatment options, deliberately bringing on a psychological emergency, attempting suicide or inflicting some other form of self-harm, and occasionally hunger strike.

17.4 Radiological Diagnosis

Alongside the clinical and physical examination of an injured person, in particular victims of violent crime, radiologically detected findings may also be very helpful. They are often able to make the diagnosis of internal findings in the neck and throat region easier (hemorrhage under the skin, in the soft tissue of the neck and in the salivary glands, as well as fractures). It may be helpful to examine a patient more than once, and at least twice, at intervals of a few days. Radiological

examinations, however, require the victim's consent and possibly an order from the court or authorities.

Radiological diagnosis plays an important role in the collection and documentation of evidence in clinical forensic medicine. In addition to classic X-rays and computed tomography, radiation-free methods such as magnetic resonance imaging and ultrasound are increasingly used in forensic diagnosis.

Since bone fractures, for example, represent sufficient clinical indication for X-ray examinations, existing X-rays can be used for the purposes of forensic assessment. The same is true for CT datasets produced for the clinical diagnosis of injuries (e.g., involvement of body cavities in sharp trauma or complex fractures of the midface). These datasets permit the visualization of actual wounds, as well possibly enabling a reconstruction of, for example, puncture wounds to the skin and wound tracks.

Methods associated with radiation exposure are rarely used in patients purely on the basis of forensic indications, given that this would contravene the principles of protection against radiation. By way of exception, a child's skeletal system may be investigated for the purposes of detecting or excluding older, healed bone fractures in the case of suspected child abuse. Forensic age determination in living individuals is only sufficiently conclusive when X-rays of the teeth, medial clavicular epiphysis, or carpal bones are performed; but again, the indication must be made strictly on the basis of diagnostic relevance and according to the basic principles of radiation protection.

Ultrasound can have an important role to play in the estimation of extent of superficial soft tissue hematomas; together with the color of a hematoma, estimations on hematoma age are possible to a limited extent.

External signs of injury may be absent even in the case of genuine injury to the neck. Although clinically reported difficulty in swallowing and hoarseness may be further indications of injury, these symptoms are highly subjective. In such cases, detecting trauma to the neck and throat may be possible by means of soft tissue visualization using magnetic resonance imaging. These methods and their applications are discussed in greater detail in Chap. 24.

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Child Abuse

18

Case Study

Accompanied by her boyfriend, a 22-year-old mother presented at an outpatient clinic with her 3-year-old son who, she reported, had fallen from a climbing frame on a playground the day before, hitting the edge of the sandpit and then striking his head on a slab of stone. Although the child had only cried a bit at the time, large contusions were now visible. Striped red marks were visible on the somewhat lachrymose child's left cheek, as well as two parallel red striae about 1 cm apart on his back. When questioned, the mother reported that her son had struck his back on the sandpit and his cheek on the slab of stone. However, she admitted that she had not been present at the time, but rather her boyfriend had been looking after the child. The 27-year-old boyfriend claimed to have been looking away at the time of the fall. The forensic expert called to the case classified the injuries as a strike on the back with, e.g., a belt, and the striped red marks on the cheeks as a slap with an open hand, while the vomiting was the result of concussion caused by these blows. The treating physician informed the mother (who had sole custody of the child) in confidence that there were grounds to suspect child abuse. The mother was indignant at this claim and wanted to leave the outpatient clinic immediately. The physician insisted on further inpatient tests, in particular to exclude the possibility of intracranial bleeding.

18.1 Introduction

Varying degrees of violence against children and adolescents is an age-old phenomenon in many countries and cultures. The boundary between acceptable violence in the context of so-called necessary educational measures by parents or as part of accepted tradition, such as genital mutilation, and unacceptable violence leading to death or severe injury is not always clear. The association between chronic suffering in adults as a result of abuse suffered in childhood and adolescence (WHO 2002) is well known. A definition of child abuse could be formulated as follows:

Child abuse is non-random physical and/or mental damage inflicted on a child, either wilfully or through neglect, within the family or institutions, which causes injury and/or impaired development and which, in individual cases, may cause death.

Although the term "battered child" is often used to describe physical child abuse, the recent literature refers to non-accidental injury (NAI) and abusive or inflicted injury. The spectrum of violence against children covers blunt force (blows), thermal injury (burns, scalds), particular forms such as shaken baby syndrome, as well as the effects of psychological violence and neglect. The forensic clinical examination of children and adolescents (including the preservation of evidence) is subject to the same requirements as those for the examination of adults. Although police criminal statistics in numerous countries show several thousand cases of child abuse every

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year, the number of unknown cases is estimated to be far higher. This raises the question of which factors should arouse the suspicion of child abuse. Infants aged between 2 and 4 years are at particular risk, as well as unwanted, developmentally impaired, handicapped, and neglected children. "Actively" abusive perpetrators are usually young adults, predominantly fathers, stepfathers, mothers' partners, or uncles, while women tend to commit "passive" abuse. However, violence against children—including sexual abuse—is found at all social levels!

Abused children are often conspicuous by their behavior in hospital: some may be hyperactive or aggressive, showing antisocial or destructive behavioral patterns, while others are abnormally anxious, inhibited, and passive, enduring medical care without complaint. Some abused children appear insecure and tense with a sad facial expression, referred to as "frozen watchfulness." Other *indications of child abuse* can be inferred from the overall circumstances in combination with medical findings:

- The patient history and/or alleged sequence of events do not correspond to injury findings.
- Injuries other than those for which the patient primarily presented are found.
- Information on the preceding events varies and/or is vague.
- Details on patient history gathered from several carers vary considerably.
- The accident alleged to be responsible for the pattern of injuries seen does not correspond to the child's age.
- Medical care is sought only after a significant time delay.
- Visits are made to several physicians and/or hospitals.
- The child's injuries are alleged to be self-inflicted or to have been inflicted by a sibling.
- The child has been an inpatient in the past due to nonspecific disorders (e.g., failure to thrive, refusal to eat).
- Abnormally high incidence of "accidents."
- Vague or implausible explanations for severe injuries, such as fractured ribs in an infant.
- Previous contact with governmental child protection bodies.
- Abnormal social or family history; carers also have a history of domestic violence and abuse.
- Carers have a history of alcohol and drug abuse.

In cases where children are injured in genuine accidents, medical care is almost always sought immediately, and the accident represents a plausible explanation for the injuries. Injury to the following sites (typical of non-accidental injury) arouses the suspicion of child abuse:

- Inner lip
- Lip and tongue frenulum
- Gum and cheek mucosa
- Retroauricular, hairy scalp, and buttocks

The typical localization of injuries following abuse compared with fall-related injuries is shown in Fig. 18.1.

In addition to injury localization, injury type, e.g., parallel striae, can also arouse the suspicion of child abuse. The requirements for a comprehensive clinical examination in the case of suspected physical child abuse are given in Table 18.1.

Depending on findings, instrument-based or imaging investigations can be considered for further diagnostic testing:

- X-ray skeletal screening in all children <2–3 years with suspected physical abuse (possible repeat investigation after 14 days) and in unexplained deaths.
- In principle, the following can be used (with limitations): Ultrasonography of the head, conventional X-ray, skeletal scintigraphy, cerebral computed tomography (CCT), and magnetic resonance tomography (MRT), as well as funduscopy and laboratory investigations.

The diagnostic reliability of injuries in terms of identifying possible child abuse varies (Table 18.2).

As with other injuries, fractures need to be seen in the context of the overall picture before the question of child abuse is addressed. Even when fractures are detected in a child, these can have varying degrees of reliability in terms of identifying child abuse (Table 18.3).

It is particularly important in the case of fractures to ask for detailed information about an accident or the circumstances of an incident and assess whether the account is plausible in terms

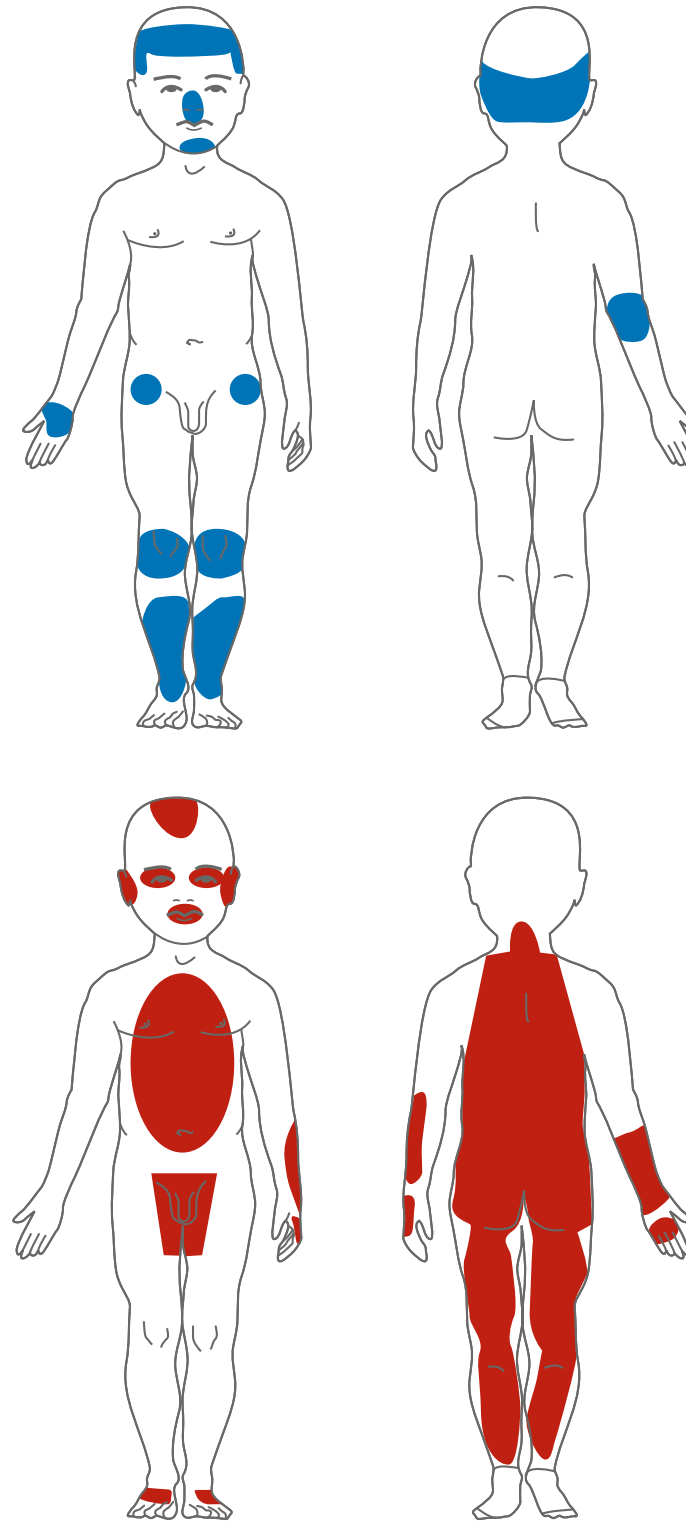

Fig. 18.1 Localization of injury or bruising in accidents (*blue*) and abuse (*red*) (From Herrmann et al. (2010))

Table 18.1 Clinical examination in the case of suspected physical child abuse

Finding/parameter	Measures
Growth parameters	Assess height/length, weight, head circumference, percentiles
Whole-body examination	Thorough physical examination, including anogenital region, of fully undressed child, neurological examination, including attention to predilection sites
Description and documentation of findings	Localization, type, color, size, form or shape, grouping, signs of wound healing. All injuries should be measured and photographically documented with a reference scale on the images (overview and detailed images); annotate all sketches with measurements
Fresh bite marks	Sterile swab for forensic DNA evidence; allow to air-dry
Behavior/statements	Avoid all suggestive questions! Document statements verbatim!
Siblings	Where necessary, siblings should be examined due to increased risk of abuse

From Hermann et al. (2010)

Table 18.2 Specificity of injuries in the identification of non-accidental causes (excluding fractures)

High reliability	
CNS	Subdural hematomas with retinal bleeding and brain damage, retinal bleeding, retinoschisis, vitreous hemorrhage
Skin	Bite wounds, contusions of particular shape (hand and finger marks, striae, belt and strap marks, loop configurations, stick marks) Burns of particular shape (cigarette, stove, iron, heater, etc.), immersion injuries (glove or stocking pattern)
Abdomen	Intramural duodenal hematomas, hollow organ perforation
Medium reliability	
CNS	Subdural hematomas (over the convexity in particular, interhemispheric, subarachnoidal)
Skin	Multiple contusions (unusual localization), contusions in infants, retroauricular contusions, torn frenulum of the lip or tongue, burns/scalds to hands, feet, anogenital region
ENT	Hypopharynx perforation
Abdomen	Injury to the left liver lobe, kidneys, and pancreas, pancreatic pseudocysts
Other	Recurrent apnea (ALTE, apparent life-threatening event)
Low reliability	
CNS	Epidural hematomas
Abdomen	Spleen injury
Skin	Multiple contusions on "leading" areas of the body at toddler age, contusions of varying color, burns/scalds in irregular "spray" or "droplet" pattern
It is always important to verify a plausible accident or preexisting disease on a case-by-case basis!	

Modified from Herrmann (2002)

of the child's age. Inconsistencies in an account or an unclear mechanism of injury even in the case of long bone fractures should prompt child abuse to be considered (Fig. 18.2). Protrusions or evidence of bony calluses are occasionally found in the case of previous or old rib fractures, while evidence of periosteal calcification (Fig. 18.3) should similarly suggest child abuse in the past.

18.2 Blunt Force and Child Abuse

Blunt force, particularly in the form of blows, is the predominant form of child abuse seen in routine forensic practice. Fractures are seen mainly in children <3 years; in addition, subperiosteal hematomas are more frequently seen in this group due to the ease with which the periosteum is separated from underlying bone. Skull fractures in infants cannot generally be caused by a fall from up to 150 cm, although an accident should always be considered in the differential diagnosis. Injuries vary according to the type of blunt force used.

Table 18.3 Radiological specificity of fractures in the identification of child abuse

Specificity	Findings
High specificity	Classic metaphyseal fracture, rib fractures (in particular dorsal), as well as scapula, spinous process, and sternal fractures. Fractures in the first 6 months of life and in premobile infants
Medium specificity	Multiple, in particular bilateral, fractures, fractures of varying age, Salter-Harris fractures, vertebral body fractures or subluxation, finger, hand, or foot fractures, complex skull fractures, mandibular fractures, periosteal reactions, fractures in infants
Low specificity	Clavicular fractures, long bone shaft fractures, simple linear skull fractures, diaphyseal fractures
It is always important to verify a plausible accident or preexisting disease on a case-by-case basis!	

From Herrmann et al. (2010)

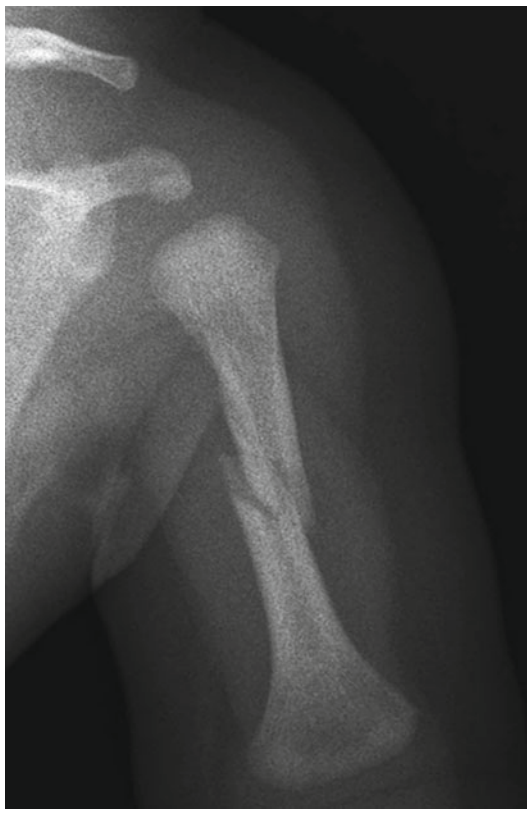

Fig. 18.2 Left upper arm fracture with displacement of the lower fracture segment towards the body following a blow with a blunt object, as well as a contusion on the outer side of the upper arm at the level of the fracture (4-month-old boy)

18.2.1 Blows and Parallel Contusions

Parallel rows of linear contusions on the skin of a child are the result of blows from a stick or rope, for example. These marks represent anemic impact marks, pale in the central region and bordered by thin parallel contusions. Marks or this kind can be found following similar blows in adults.

It is not unusual to find multiple parallel contusions, particularly on the buttocks, back, shoulders, upper arms, and backs of the legs. Patterned injuries from specific objects used to inflict a blow, such as belt buckles, may be seen on the skin (Fig. 18.4).

18.2.2 Fist Blows

Blows to the face from a fist can result in monocle or spectacle hematomas of varying degree (Fig. 18.5) and may cause eye injury. Blows with a fist to the mouth area produce hematomas in the oral mucosa, as well as mucosal lacerations when the child's teeth have represented a point of abutment. Fist blows or kicks to the abdominal area can cause intra-abdominal organ laceration involving injury to the liver, spleen, pancreas, and gastrointestinal tract.

Blows from the flat of a hand can leave striped finger marks, particularly on the cheeks. Retroauricular bruising as well as ruptured eardrums may also be seen, in which case a child should be examined additionally by an ear, nose, and throat specialist if abuse is suspected. Blows from the knuckles of a clenched fist may produce a row of roundish contusions.

18.2.3 Other Forms of Blunt Force

Depending on the object used to strike a blow (belt, ashtray, etc.), patterned contusions corresponding to the object in question may be seen; however, extensive bruising is also seen (typically on the back and buttocks). Older children sometimes show self-defense or "parrying" injuries to the extensor surface of the lower arm.

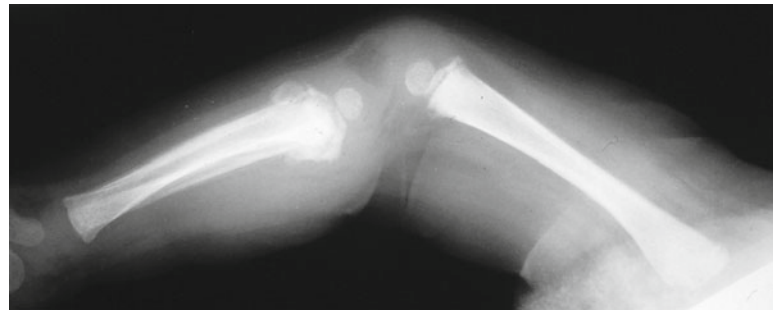

Fig. 18.3 Status following old bony trauma with periosteal calcification and metaphyseal chipping in an 8-month-old boy

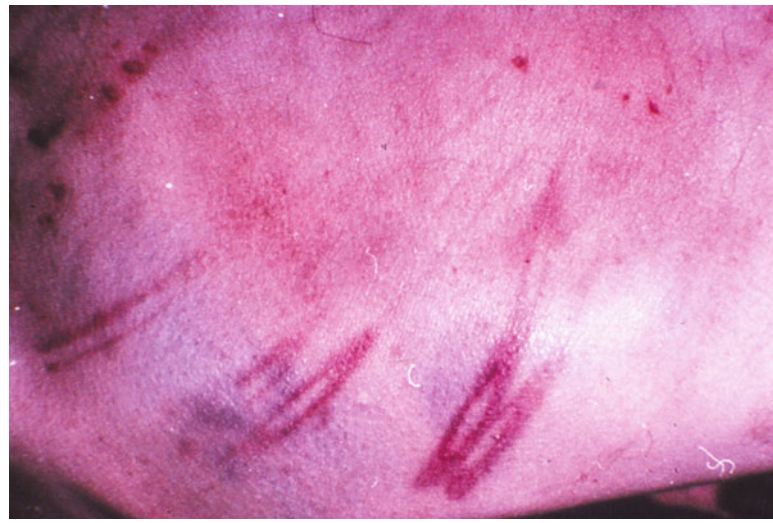

Fig. 18.4 Multiple parallel contusions with central paleness following blows from a piece of cord

Blows to the head cause contusions, lacerations, and corresponding scarring. A kick from a shod foot may leave a recognizable shoe imprint. However, it should be noted that, although blows and kicks to the abdominal wall of clothed victims do not necessarily produce outwardly visible signs of injury, serious internal injury and hemorrhage may nevertheless be present!

Blunt force to the upper abdomen (a blow to the solar plexus) can cause intra-abdominal injury including laceration of the left hepatic lobe and/or pancreatic head, as well as duodenal hemorrhage (Fig. 18.6); child abuse-related rupture of the stomach wall (Fig. 18.7) and tearing or laceration of the intestinal wall (Fig. 18.8) are less commonly seen. In cases where a child is seized or handled, hand marks can be seen; these are often symmetrical contusions on the upper arms—often thumb marks on the inner side of the upper arm or hand marks on the chest of the

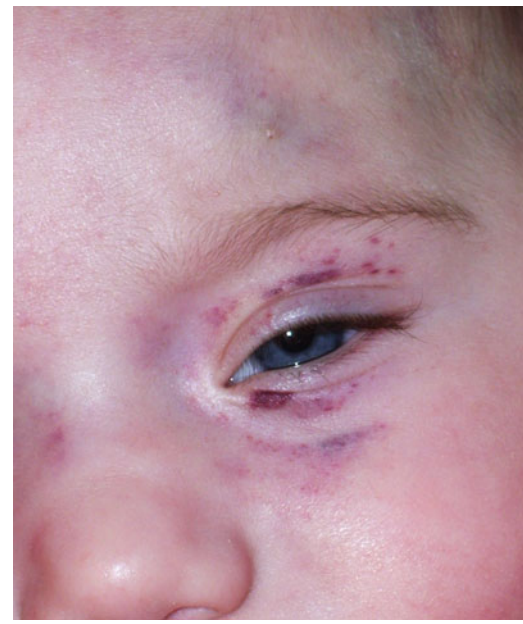

Fig. 18.5 Incomplete monocle hematoma resulting from a blow to the face

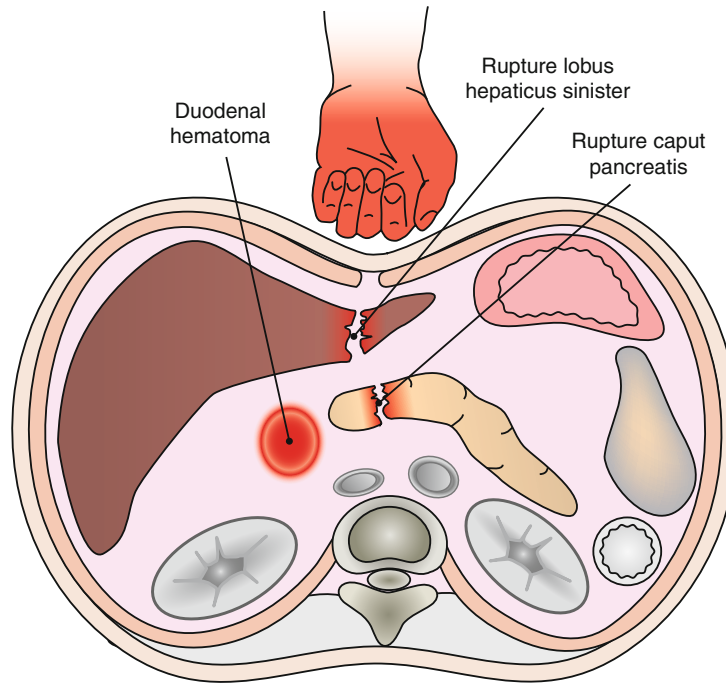

Fig. 18.6 Abdominal injuries frequently resulting from child abuse (From Herrmann et al. (2010))

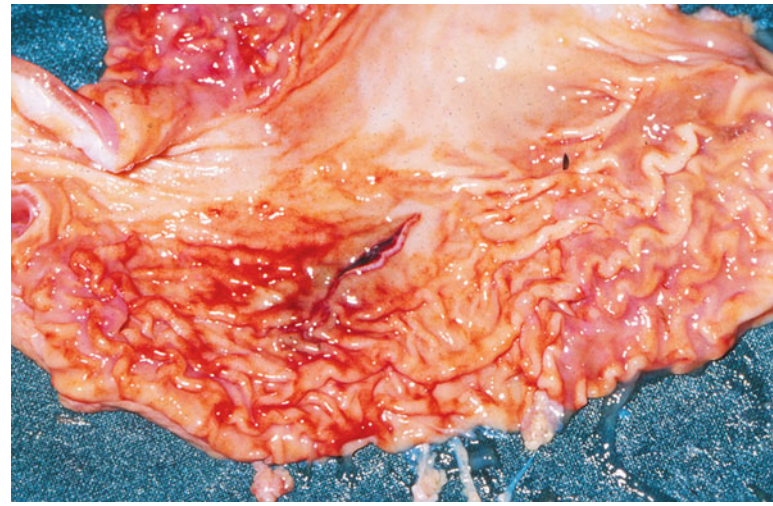

Fig. 18.7 Ruptured stomach wall following a kick to the upper abdomen in a 21-month-old boy

infant or toddler in the context of shaken baby syndrome. Pinching can produce uncharacteristic contusions, occasionally also involving abrasions from fingernails. Contusions around the base of the ear or on the ear suggest pulling or tearing of the ear, while bald patches on the skull suggest pulling out of hair (epilation). Signs of restraint are typically found on the wrists and ankles in the form of circular/linear skin lesions of varying width and severity.

18.2.4 Bite Marks

Small contusions and teeth marks running in a curved line and forming an oval or half-moon pattern are suggestive of bite marks. Occasionally, fresh bite marks even demonstrate the position of individual teeth in relation to one another. In the majority of cases, however, marks are already partially faded (Fig. 18.9a). Bite injuries inflicted post-mortem are rare (Fig. 18.9b). Measuring the

diameter of contusions and the distance between each one indicates whether an adult or a sibling—as occasionally alleged—is responsible for the bite injury.

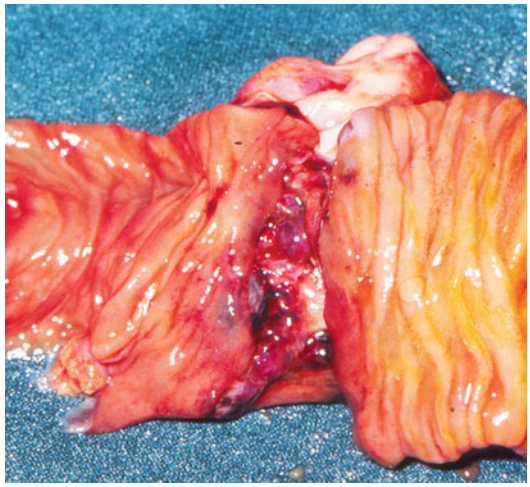

Fig. 18.8 Torn small intestine following a kick to the upper abdomen in a 21-month-old boy

18.2.5 Throwing or Dropping an Infant or Toddler

When an infant or toddler is thrown against a solid object or strikes the floor or an object, extensive bruising is usually caused. Fractures are also possible in some cases, whereby cranial bones are most commonly involved. In the case of gross blunt trauma, suspected fractures, and substantiated suspicion of repeated child abuse, radiological investigations are indicated; these findings may require assessment particularly in terms of whether a fracture is accident related.

18.3 Thermal Injuries and Child Abuse

While injuries resulting from sharp or penetrating trauma play a minor role in child abuse, abuse-related burns and scalds are seen relatively often. Cold-related injuries, e.g., due to confinement in

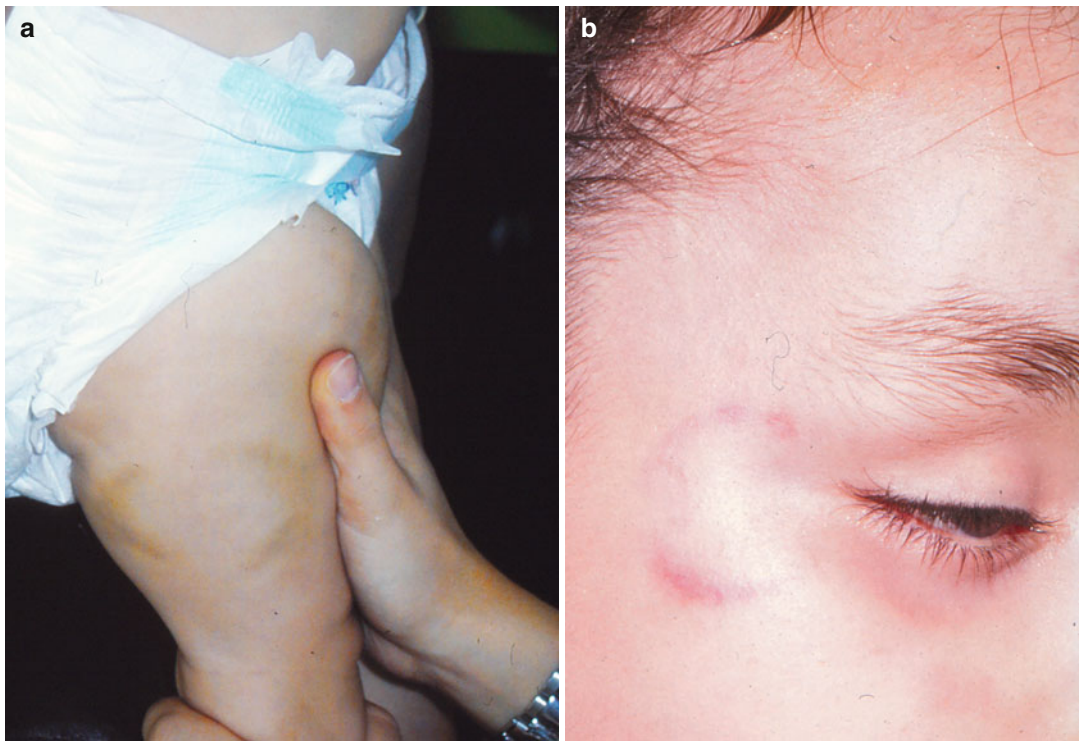

Fig. 18.9 (**a**) Old bite marks on the outer side of the right thigh in a 16-month-old boy. (**b**) Bite marks inflicted post-mortem adjacent to the right eye in a 6-year-old girl

a cold room or immersion in cold water, are rare and seen primarily in the context of neglect. In the case of burns and scalds, it should be noted that significantly shorter exposure times are sufficient to damage young skin compared with adult skin. Typical objects used to inflict contact burns include cigarettes, cigars, cigarette lighters, irons, heaters, hairdryers, curlers, car hoods, hot plates, and ovens.

Scalds: Exposure to moist heat, usually a hot liquid, most commonly water. Preservation of skin appendages, e.g., hair, is characteristic.

Burns: Exposure to dry heat, i.e., contact burns.

Direct exposure to flames is infrequently seen in the context of child abuse. Additional damage to hair and skin appendages is seen in the case of dry heat.

Accidental thermal injury also needs to be considered in the differential diagnosis of burns (Table 18.4).

Abuse-related scalding in children often involves the hands (Fig. 18.10), feet, or buttocks (Fig. 18.11) and shows conspicuous water-level demarcation lines. Splashes of hot water sometimes come in contact with the eyes in the course of an incident (Fig. 18.12).

In the case of accidental scalding, on the other hand, irregular, splash-like injuries

Table 18.4 Differential diagnosis of accidental vs. abuse-related thermal injuries

Accidental scalds/burns	Abuse-related scalds (immersion) and burns
Irregular splash-like distribution of injuries, possibly also flow marks	Injury pattern, often including water-level marks
No clear demarcation from healthy skin	Sharply demarcated from healthy skin
Scalding: arrow-like configuration on the chest	Contact traces are absent in the case of forced immersion of the face
Irregular, splash-like distribution of scalds on the extremities	Stocking or glove pattern of scalds following hand/foot immersion
Unclear demarcation of hot contact surfaces	Often clear pattern of the hot object applied: cigarette, iron, hotplate, hairdryer
Smaller injuries of varying depth since the body or the object is not fixed in mechanisms of accidental injury	Relatively homogenous injury depth due to pressure on or fixation of the object or the child's body

Modified according to Yeoh et al. (1994)

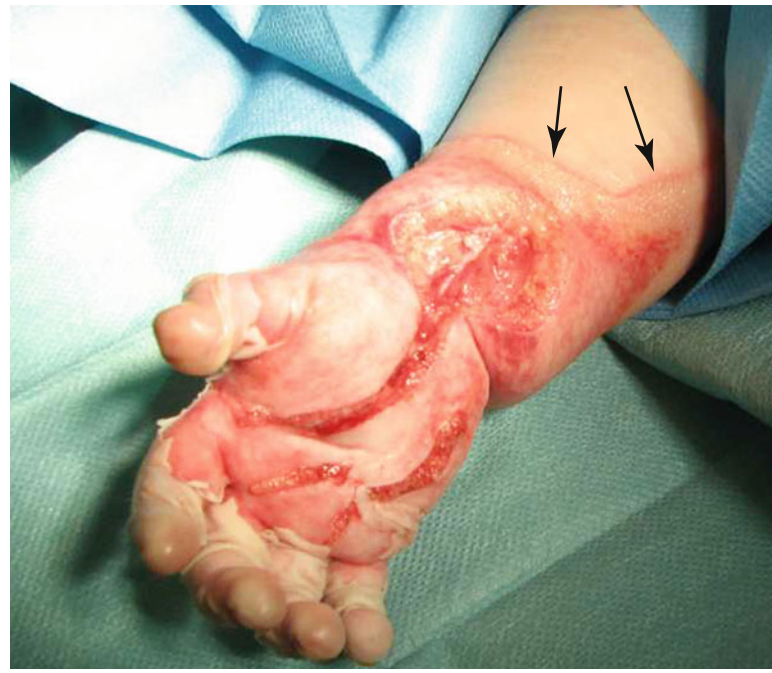

Fig. 18.10 Scald injuries (seen here postoperatively) on the hands of an infant following forced immersion in hot water with clearly identifiable watermark lines (*arrows*)

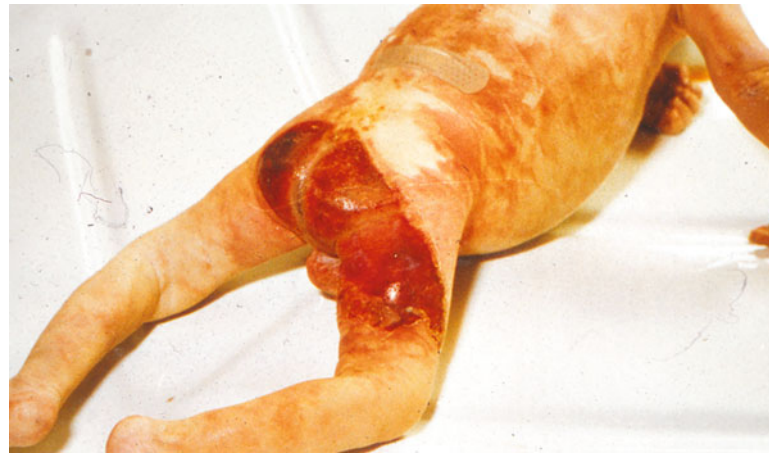

Fig. 18.11 Clearly delineated scald injuries to the buttocks after the child was seated in hot water

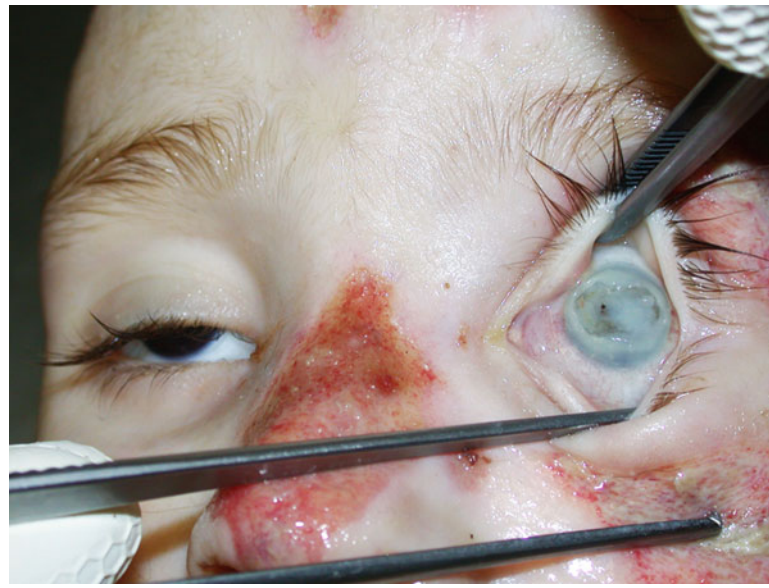

Fig. 18.12 Abuse-related scald injuries including involvement of the left eye

showing flow lines and a less symmetrical distribution over the body are seen, typically, for example, when a child tips a saucepan of hot water off the stove and the hot water pours down one side of the child's body (Fig. 18.13a–c).

18.4 Shaken Baby Syndrome

Up to 95 % of severe head injuries in the first year of life are believed to be the result of abuse. Of particular note here is shaken baby syndrome (SBS), a specific form of gross blunt trauma; impact injuries to the head (shaken impact syndrome) in this context may also explain skull fractures.

SBS primarily involves infants but may also be seen in children aged up to 2 years. An infant's peak crying phase between the ages of 2 and 5 months represents a predisposing factor. An infant may be seized by the arms or around the chest (Figs. 18.14 and 18.15), also occasionally by the shoulders or extremities.

Gripping tightly around the chest can cause paravertebral rib fractures, while the head is shaken backwards and forwards in a whiplash-like action.

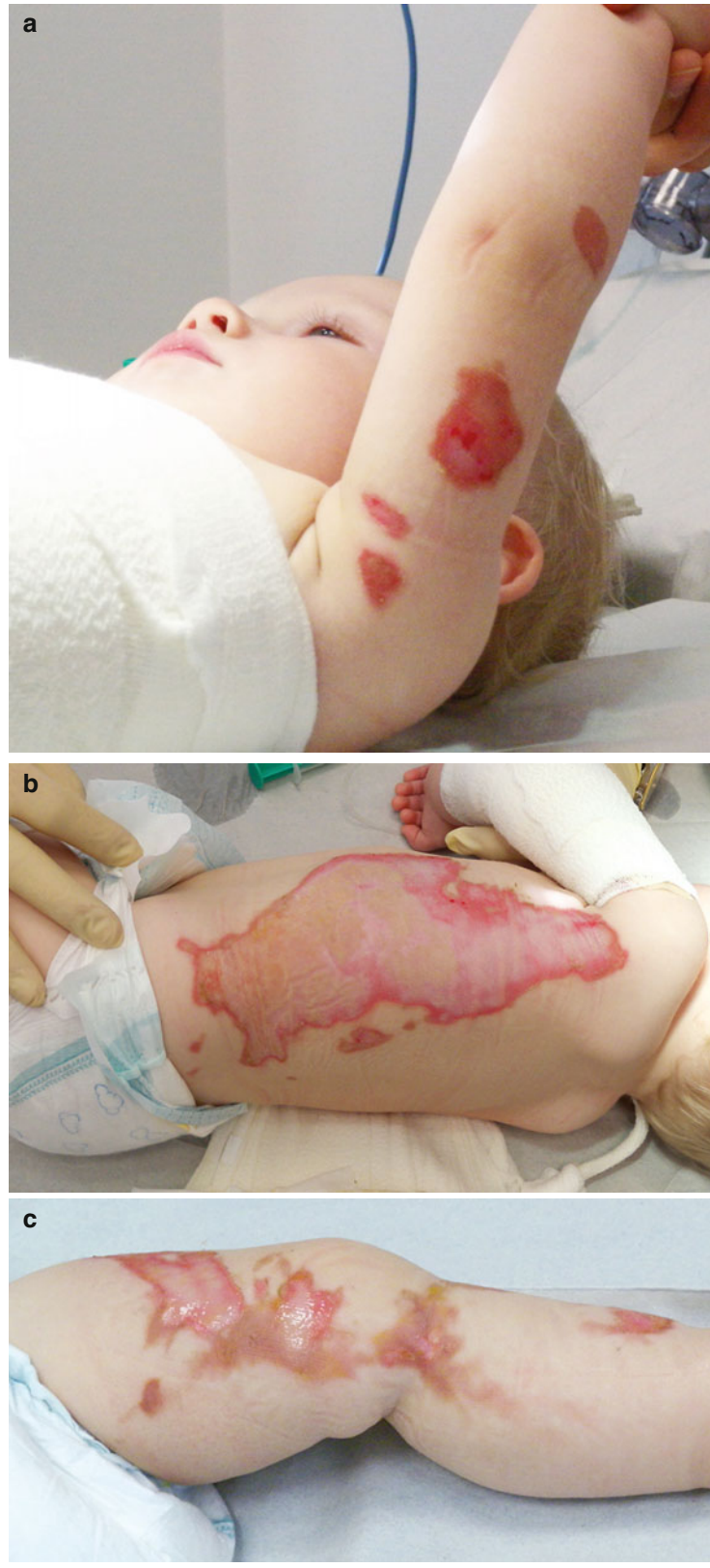

Fig. 18.13 (**a**–**c**) Irregular distribution of accidental scald injuries showing a splash-like pattern with flow marks on the left upper arm (**a**), trunk (**b**), and left leg (**c**) of a toddler who tipped a saucepan of boiling water off the stove

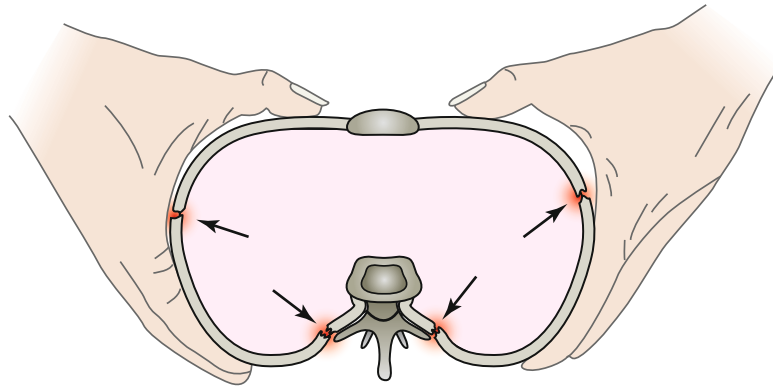

Fig. 18.14 Grip position around the chest in shaken baby syndrome with serial rib fractures due to tightness of grip

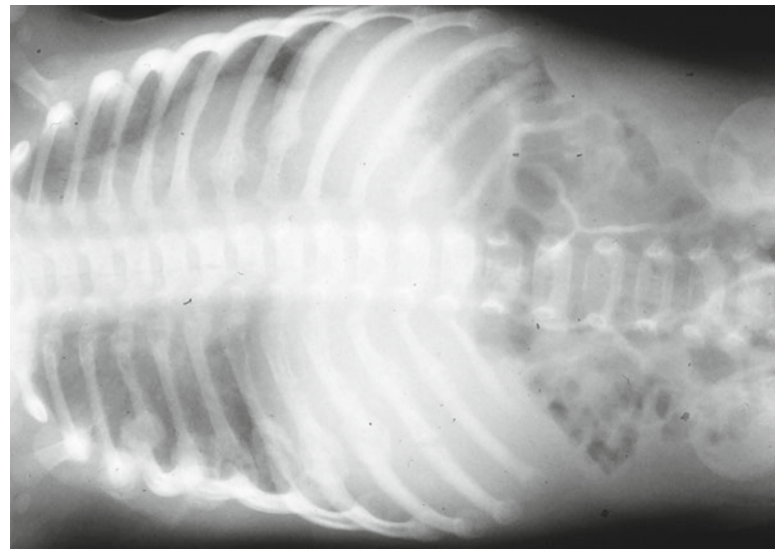

Fig. 18.15 Old serial rib fractures, 8-week-old female with typical callus formations

Important: Shaken baby syndrome is of such severity that even individuals with no medical knowledge cannot help but be aware of the damaging and life-threatening outcome of violent shaking of this kind.

Violent shaking results in clinical symptoms and injuries. In general, there is no, or only a very short, interval free of neurological symptoms. Clinical signs of non-accidental head injury may be evident in shaken baby syndrome:

- Poor general condition
- Weak feeding, unwillingness to feed
- Refusal to feed
- Irritability
- Drowsiness
- Vomiting (intracranial pressure!)
- Muscular hypertonia
- Cerebral seizures
- Apnea
- Impaired temperature regulation
- Bradycardia
- Somnolence, apathy, coma, death

Clinical symptoms vary; moreover, severe neurological symptoms may be present despite the absence of externally visible injuries. Internal injuries of varying degrees of severity can include:

- Subdural (Fig. 18.16) and subarachnoid hematoma, often of a non-space-consuming nature
- Uni- or bilateral retinal hemorrhage (Fig. 18.17)
- Retinal detachment
- Optic nerve hemorrhage (Fig. 18.18)
- Vitreous hemorrhage

- Hand marks (upper arms, chest)
- Fractures, including dorsal serial rib fractures
- Diffuse axonal injury (DAI) with extensive damage to cerebral parenchyma
- Possible impact injuries to the head
- Subperiosteal bleeding at the base of the clavicle/neck muscles due to tearing forces

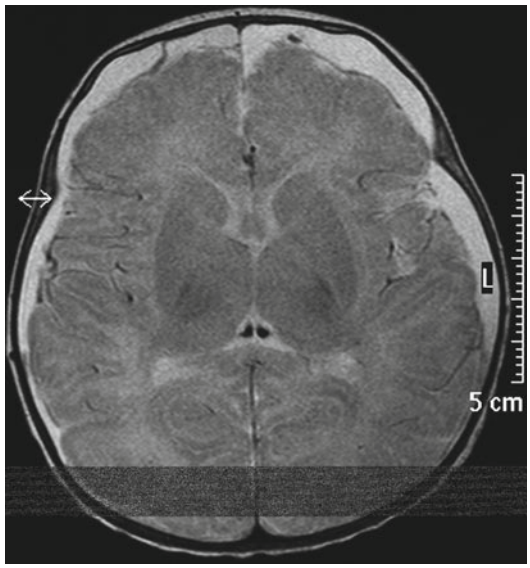

Fig. 18.16 Acute subdural hematoma (*double-ended arrow*) seen on MRI in a case of shaken baby syndrome; no relevant intracranial mass (From Herrmann et al. (2010))

Important: An ophthalmological examination is mandatory in the case of suspected shaken baby syndrome, particularly in infants <4 years. As a rule, cerebral seizures, coughing fits, and resuscitation do not cause retinal hemorrhage.

Shaken baby syndrome is associated with a mortality rate of 12–27 %. Two thirds of survivors are left with varying degrees of neurological damage including cerebral atrophy, subdural hygroma, multicystic encephalopathy, cerebral seizures, mental retardation, and optic nerve atrophy.

18.5 Special Forms of Child Abuse

Case Study

Due to persistent diarrhea in her infant, a mother sought consultation with her family physician and a hospital pediatrician. The ensuing inpatient investigations yielded no pathological findings, and the infant showed no signs of diarrhea during the inpatient period. When the mother reported renewed symptoms several weeks later, a chemical-toxicological investigation was carried out on a urine sample from the infant. This investigation showed that the infant had been given laxatives.

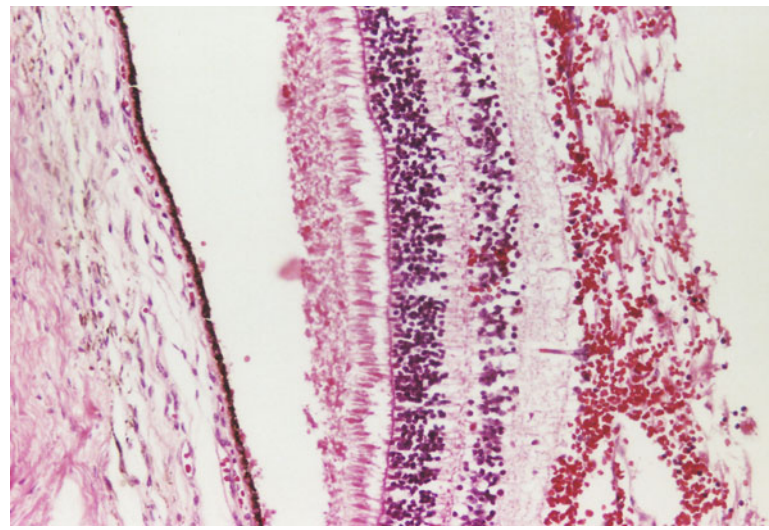

Fig. 18.17 Microscopically detected retinal hemorrhage in shaken baby syndrome (HE×200)

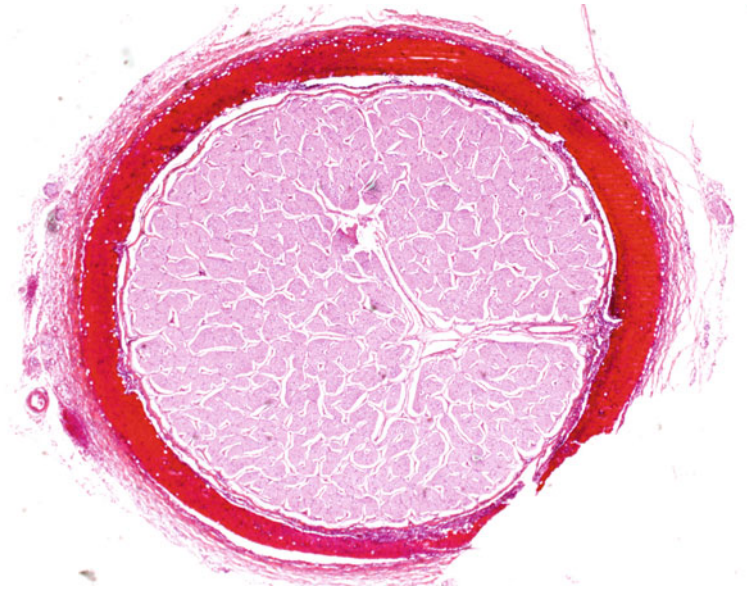

Fig. 18.18 Shaken baby syndrome with circular hemorrhages around the exit of the optic nerve (H&E×40)

A second mother presented with her infant reporting bloodied diapers. On inspection, the diaper was found to indeed contain blood, arousing the suspicion of hemorrhagic urocystitis; however, clinical and laboratory findings did not support this diagnosis. Molecular genetic analysis of the blood demonstrated that it had originated from the mother rather than from the child and that it had been placed in the diaper by the mother.

Special forms of child abuse comprising certain constellations of injuries or particular circumstances have been reported.

Munchausen Syndrome by Proxy (*MSbP*). In this particular syndrome, disease symptoms in a child are alleged and/or caused deliberately—almost always by the mother (see "Case Study" above). Existing diseases may also be exacerbated. MSbP has four characteristic features:

1. The clinical picture seen in a child has been simulated or caused by a parent or other person responsible for the child.
2. The child is presented for medical investigation and treatment frequently involving extensive and often invasive medical evaluations.
3. The person presenting with the child denies any knowledge of the actual cause(s) of the clinical picture.
4. Acute symptoms improve when the child is separated from the perpetrator.

The term "Munchausen syndrome by proxy" is considered by some to be inappropriate; "pediatric condition falsification" has been suggested as a more accurate designation for this phenomenon. The motive behind MSbP is not only the repeated medical evaluation of the child but also the mother's need for care and attention. The mother appears overly concerned, seeks contact with the nursing staff and physicians, and tries to obtain medical information. Numerous deliberate acts of harm are seen, such as the administration of medication or toxins, including in particular sleeping pills and tranquilizers, ipecac, laxatives, the use of pepper, salt intoxication, water intoxication, alcohol, drugs, and household chemicals. Even without foreign substance administration, life-threatening situations may be induced in MSbP, e.g., "smothering," whereby the external airways are covered with a soft object such as a pillow until near onset of irreversible damage or death is caused. The clinical classification of this phenomenon as apparent life-threatening event (ALTE) syndrome replaces the older designation of near-missed SIDS (sudden infant death syndrome).

Misdiagnoses in the case of death and incorrect classification as SIDS occur! Close attention should be paid to discrepancies between reports given by the mother and the clinical findings. Symptoms usually abate while the child is an inpatient. Clinical findings and diagnostic results that cannot be attributed to any known disease, as well as the supposed discovery of new or rare diseases, are suggestive of MSbP. This particular syndrome has hitherto been seen as a special form of child abuse rather

than as a maternal psychiatric disorder in its own right, although a personality disorder is assumed to a greater or lesser degree.

Tin Ear Syndrome: Here, a child's head is subjected to rotational acceleration produced by a strong slap. Clinical findings include an isolated ear contusion, ipsilateral subdural hematoma, retinal hemorrhage, and cerebral edema or diffuse axonal injury as seen in shaken baby syndrome. A fatal course has been seen in some cases.

Caffey Syndrome: The appearance of chronic subdural hematomas combined with generally multiple long bone fractures.

Shaken Impact Syndrome: This involves the violent shaking of an infant accompanied by head impact, thus producing significant acceleration–deceleration forces (whiplash) as well as severe injury, in particular skull fractures.

18.6 Differential Diagnoses

Depending on medical findings, a differential diagnosis may be necessary. Abnormal bruising is suggestive of a coagulation disorder, and a patient history should be taken:

- In the case of infants: Did vitamin K administration taken place after birth?
- Has there been previous surgery with bleeding complications?
- Does the child bruise unusually easily?
- Has there ever been unusually persistent bleeding following banal superficial wounds?
- Are there any indications of accidental ingestion of anticoagulants, e.g., Marcumar tablets at home, rat poison?
- Are there any indications of malabsorption or failure to thrive?
- Is a coagulation disorder present (hemophilia, von Willebrand syndrome (most common form), immune thrombocytopenic purpura (ITP), vitamin K deficiency bleeding (neonate late-onset form), hepatopathies, other coagulopathies)?
- Are there any indications of vasculitis? Most common: Schönlein–Henoch purpura

Occasionally, multiple contusions are seen in infants and children with coagulopathies, usually in localizations typical for an impact site, but also localized to sites typical of blows or falls, as well as unusual sites (Fig. 18.19).

A spectacle hematoma always arouses the suspicion of child abuse or impact trauma, primarily a blow from a fist. There are few alternative explanations for bruising of this kind. In rare cases, when a monocle or spectacle hematoma cannot be reasonably attributed to a reported incident or an established setting, a coagulation disorder needs to be considered (Fig. 18.20).

Injury due to birth trauma is occasionally the source of misunderstandings, particularly when contusions are seen at sites typical for blows, such as the buttocks (Fig. 18.21).

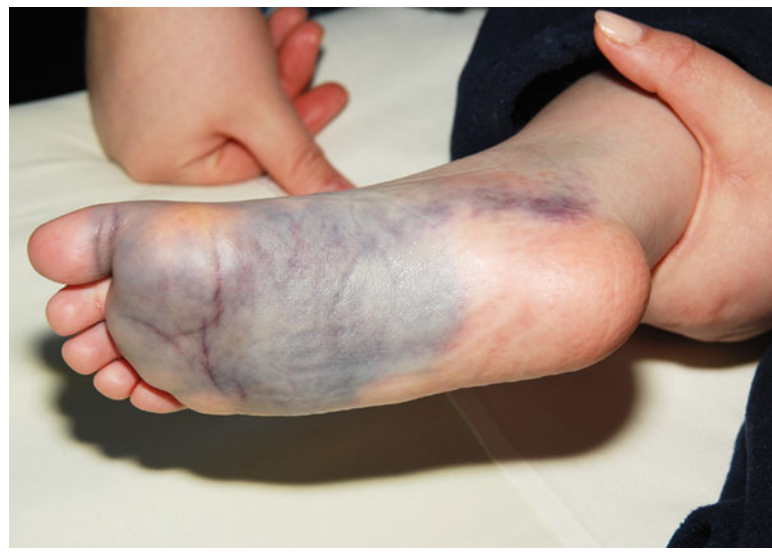

Fig. 18.19 Initial suspicion of child abuse: extensive bruising on the sole of the foot in clinically diagnosed coagulopathy

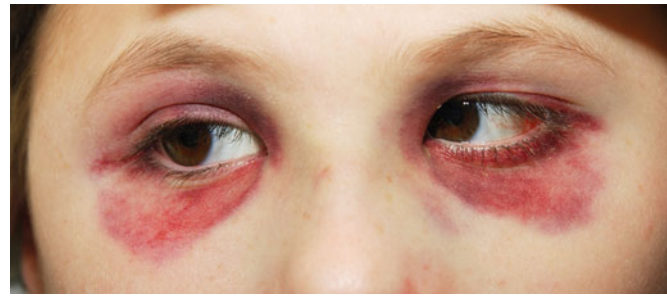

Fig. 18.20 Initial suspicion of child abuse on the basis of a spectacle hematoma due to a blow from a fist to the eyes in the setting of clinically diagnosed coagulopathy

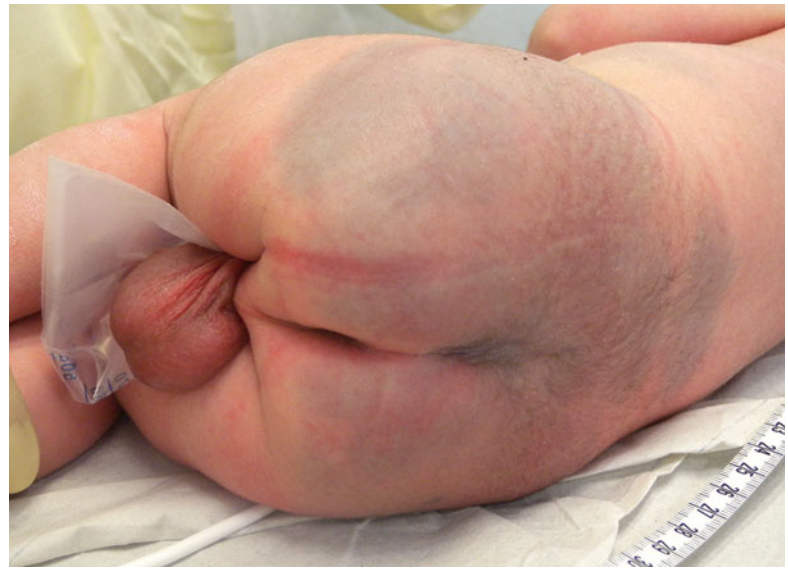

Fig. 18.21 Extensive yet symmetrical and clearly demarcated contusion on the skin of the buttocks following protracted delivery due to breech presentation, initially incorrectly interpreted as child abuse

In the same way, genetic or congenital diseases that cause increased susceptibility to fractures, in particular osteogenesis imperfecta (OI), should be considered in infants, children, or adolescents with fractures of otherwise implausible origin. Cases of mistaken classification as child abuse are known. The incidence of all four types (according to Sillence) of OI is given as between four and seven cases per 100,000 births. OI is primarily a quantitative collagen synthesis disorder caused by a point mutation on the *COL1A1* and *A2* genes, which encode for type 1 collagen (90 % of the bone matrix). However, this is accompanied by a qualitative disorder caused by a decrease in collagen triple-helical twisting, which causes reduced bone stability. OI is a predominantly autosomal dominant disorder, while recessive in type III. Any suspicion of OI warrants taking a family history: Are there any known cases of bone diseases or bone deformities in biological relatives? Questions more targeted to OI include the following: Is there an increased incidence of fractures? Blue sclerae? Dentinogenesis imperfecta? Hearing impairments? Long bone deformities? Osteoporosis? Wormian bones?

In addition, abnormal skin findings that could initially be incorrectly interpreted as bruising, e.g., naevus fusco-caeruleus, should be documented. Unusual contusions may be attributable to traditional folk medical practices, such as the Asian practice of "coining" or "spooning" (Cao gio and quat sha, respectively). Rarely, infections may cause skin changes, e.g., multiform erythema in herpes or mycoplasma infections, which could be mistaken for child abuse in the differential diagnosis.

18.7 Child Neglect

Parental neglect of a child is defined not only as denying a child appropriate care, protection, and education but also denying affection, love, and acceptance, as well as allowing a child to suffer

physical deprivation such as starvation or poor nutrition.

A distinction is made between physical neglect and psychological neglect:

Physical Neglect: This form of neglect ranges from providing poor nutrition, insufficient attention to physical needs, and refusal to or delay in seeking medical care to total abandonment. Physical signs of neglect include nonorganic failure to thrive, the effects of severe malnutrition or starvation, as well as contracting preventable diseases such as rickets or infectious diseases as a result of failure to obtain vaccinations.

Psychological Neglect: This describes insufficient attention to and fulfillment of a child's developmental needs within the boundaries of social responsibility and decency. Psychological abuse manifests itself as a child's lack of involvement and interest, withdrawn behavior or overfamiliarity, and delayed social, emotional, and speech development.

18.8 Female Genital Mutilation

According to WHO data, there are approximately 100–150 million women and girls worldwide living with the effects of female genital mutilation (FGM; also referred to a female genital cutting, FGC, and female circumcision); every year, around three million girls are added to this figure. FGM is performed for a number of reasons considered to be wholly irrational in the Western world; nonetheless, it involves deeply ingrained cultural ideas held even—and in particular—by women who have suffered genital mutilation themselves. A differentiated explanation for the various forms of FGM is lacking. The causes of FGM lie in cultural and social customs, the main causes being deeply held traditional (up to 71.3 %) and religious (up to 91.4 %) practices. The need for FGM in girls is justified or explained more specifically by the following:

- The cultural ideas and traditional myths of individual ethnic groups
- A fundamental ignorance of biological and medical facts
- The incorrect interpretation of religious guidelines
- Patriarchal structures and the accompanying suppression of women
- Mutilation as a means to ensure against loss of virginity and promiscuity in marriage or to reduce women to the level of their role as child-bearers
- The threat of social isolation and reduced chances of marriage since non-mutilated women are often considered promiscuous or "wanton"

In some areas, the procedure is seen as a ceremonial act and is accompanied by the giving of gifts and rewards. According to the literature, further reasons for FGM consist in preparing a girl for adulthood and marriage insofar as FGM can represent a precondition for marriage, or it is assumed that a man will only marry a woman if she has been "circumcised." In addition, the intervention is believed to preserve virginity, ensure fidelity, and control a woman's libido and behavior.

FGM involves the partial of complete removal of the external female genitalia. The World Health Organization (WHO) classifies FGM into four major types (WHO 2008):

- Type I, clitoridectomy: Partial or complete removal of the clitoris
- Type II, excision: Partial or complete removal of the clitoris and the labia minora, with or without removal of the labia majora
- Type III, infibulation: Partial or complete removal of the external genitalia and sewing up of the vaginal opening, leaving only a small foramen
- Type IV: All other forms of FGM or alterations to the female genitalia for nonmedical purposes that do not classify as types I–III, including the following practices:
  - Pricking, piercing, incising, or other procedures intended to alter the clitoris
  - Stretching the clitoris and labia minora
  - Cauterizing the clitoris
  - Incising the external female genitalia
  - The insertion of painful substances or herbs (usually performed by adult females on themselves for cleansing purposes or to increase the partner's pleasure. The WHO considers this also to be a form of FGM, since it can pose a serious health risk or is the result of social coercion)

Although the procedure is usually performed without anesthesia and in poor hygiene conditions (in countries such as Egypt, Kenya, or Guinea), it is increasingly undertaken in hospitals with anesthesia.

Table 18.5 African countries where FGM is practiced

	Country	Year	Prevalence (%)
Northeast Africa	Egypt	2005	95.8
	Ethiopia	2005	74.3
	Djibouti	2006	93.1
	Eritrea	2002	88.7
	Somalia	2005	97.9
	Sudan, northern region (covering ca. 80 % of the population)	2000	90.0
East Africa	Kenya	2003	32.2
	Tanzania	2004	14.6
	Uganda	2006	0.6
Northwest Africa	Burkina Faso	2005	72.5
	Guinea	2005	95.6
	Guinea Bissau	2005	44.5
	Gambia	2005	78.3
	Mali	2001	91.6
	Mauritania	2001	71.3
	Senegal	2005	28.2
	Sierra Leone	2005	94.0
Southwest Africa	Benin	2001	16.8
	Ivory Coast	2004	41.7
	Chad	2004	44.9
	Central African Republic (MICS)	2005	25.7
	Nigeria	2006	2.2
	Ghana	2005	3.8
	Cameroon	2004	1.4
	Liberia	2007	45.0
	Niger	2006	2.2
	Togo	2005	5.8

Modified from WHO (2008)

Clitoridectomy and excision comprise around 80–85 % of all cases, for example, those seen in Senegal, Togo, and the Ivory Coast, while infibulation (15–20 % of all cases) is practiced predominantly in East Africa (Somalia, Ethiopia, Eritrea, but also Sudan). FGM, which is practiced in more than 28 countries, is seen primarily in central, western, and northeastern regions of Africa (see Table 18.5 for an overview) (WHO 2008).

Cases of FGM have also been reported in India, Indonesia, Malaysia, Yemen, the United Arab Emirates, and Iraq; however, no estimates on the number of women affected in these countries are available. Ethnic origin is the main decisive factor in terms of whether where (alone at home or in a group at a certain place) and how a circumcision is performed.

Data gathered from countries where information on forms of circumcision is available show that altogether approximately 90 % of affected women have undergone type I, II, and IV circumcision and 10 % type III (infibulation). It was found that infibulation is common in Northeast African countries in particular; in addition, FGM tends to be practiced in rural areas more frequently than in urban areas.

Although literature data on the average age at which girls are affected by FGM vary, it is clear that primarily minors are affected; at a rate of 0.1–3.8 %, FGM in adult women plays only a minor role. The point in time at which FGM is performed depends strongly on the local conditions and traditions of individual ethnic groups, with significant variations between countries. In Eritrea, for example, 62 % of procedures are performed before 1 year of age, while in Guinea approximately 48 % take place between the ages of 5 and 9 years and in Egypt around 49 % between the ages of 10 and 14 years. According to a WHO report, most procedures take place between the ages of 0 and 15 years.

Irrespective of type, FGM is associated with a multitude of acute and chronic health effects and complications, the most common among these being menstrual disorders. Difficulties during childbirth or sex, as well as psychological effects such as traumatic memories or fear of gynecological examinations or sexual intercourse, are also reported.

FGM increases the risk of complications during childbirth two- to threefold. These increased risks include cesarean section, postpartum hemorrhage, increased length of stay in hospital, the need for neonatal resuscitation, stillbirth, and/or premature death of a neonate. Health risks increase according to the extent of FGM. However, FGM brings not only physical but also psychological complications with it in the form of posttraumatic stress disorders, anxiety disorders, and affective disorders.

In November 2006, senior Islamic scholars at the Al-Azhar University in Cairo issued a

توصيـــات المؤتمـر

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بسم الله الرحمن الرحيم

انعقد "مؤتمر العلماء العالمي نحو حظر انتهاك جسد المرأة" في الأول والثاني من ذي القعدة ١٤٢٧ هـ الموافق ٢٢ – ٢٠٠٦/١١/٢٣م في رحاب الأزهر، وألقي فيه عدد من البحوث، وبعد مناقشات السادة العلماء والأطباء والمتخصصين والمهتمين من مؤسسات المجتمع المدني في مصر وأوربا وأفريقيا توصل المؤتمر إلى ما يلي :

١. كرم الله الإنسان فقال تعالى: ﴿وَلَقَدْ كَرَّمْنَا بَنِيٓ ءَادَمَ﴾ فحرم الاعتداء عليه أيًا كان وضعه الاجتماعي، ذكرًا كان أم أنثى.

٢. ختان الإناث عادة قديمة ظهرت في بعض المجتمعات الإنسانية، ومارسها بعض المسلمين في عدة أقطار تقليدًا لهذه العادة دون استناد إلى نص قرآني أو حديث صحيح يحتج به.

٣. الختان الذي يمارس الآن يلحق الضرر بالمرأة جسديًا ونفسيًا، ولذا يجب الامتناع عنه امتثالاً لقيمة عليا من قيم الإسلام، وهي عدم إلحاق الضرر بالإنسان، كما قال رسول الله صلى الله عليه وسلم "لا ضرر ولا ضرار في الإسلام" بل يُعد عدوانًا يوجب العقاب.

٤. يناشد المؤتمر المسلمين بأن يكفوا عن هذه العادة، تماشيًا مع تعاليم الإسلام التي تحرم إلحاق الأذى بالإنسان بكل صوره وألوانه.

٥. كما يطالبون الهيئات الإقليمية والدولية بذل الجهد لتثقيف الناس وتعليمهم الأسس الصحية التي يجب أن يلتزموا بها إزاء المرأة، حتى يقلعوا عن هذه العادة السيئة.

٦. يُذكّر المؤتمر المؤسسات التعليمية والإعلامية بأن عليهم واجبًا محتمًا نحو بيان ضرر هذه العادة، والتركيز على آثارها السيئة في المجتمع، وذلك للإسهام في القضاء على هذه العادة.

٧. يطلب المؤتمر من الهيئات التشريعية سن قانون يُحَرِّمُ ويُجَرِّم من يمارس عادة الختان الضارة فاعلاً كان أو متسببًا فيه.

٨. كما يطلب من الهيئات والمؤسسات الدولية مد يد المساعدة بكافة أشكالها إلى الأقطار التي تُمارَس فيها هذه العادة كي تعينها على التخلص منها.

Fig. 18.22 Statement from November 2006

statement declaring that the Qur'an contains no call for or mandate to perform FGM. The statement further adds:

> Genital circumcision is a deplorable, inherited custom… there are no written grounds for this custom in the Qur'an… The female genital circumcision practiced today harms women psychologically and physically. Therefore, the practice must be stopped in support of one of the highest values of Islam, namely to do no harm to another (Fig. 18.22).

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Child Sexual Abuse

19

Case Study

At around 1 pm on a Sunday afternoon, a mother presented at an outpatient clinic with her 13-year-old daughter. The mother reported that her daughter had spent the night at the home of her older sister's friend for a birthday party. The daughter had spent the evening watching television and drinking only mineral water. She had lost all recollection from around 1 am onwards. The 13-year-old confirmed the mother's report. The daughter woke up on the sofa at around 10 am on Sunday morning with a headache and no recollection of the preceding hours. She then noticed that her clothes, including her underwear, were not on properly and that she also had pain between her legs. A gynecological examination demonstrated a small indentation in the hymenal rim with mild surrounding hemorrhage. Discrete traces of secretion were also observed. In addition to vaginal and anal smear samples, blood was taken for the detection of GHB (knockout drops) on the one hand and to rule out preexisting sexually transmitted disease on the other. On combing the patient's pubic hair, one hair remained in the comb. Further physical examinations revealed a livid blue contusion measuring 1.5 cm in diameter on the outer side of the mid-upper arm. Items of the girl's clothing were taken as forensic evidence. The examining physician advised mother and daughter to inform the police.

In routine practice, underage victims of sexual abuse rarely usually undergo gynecological examination by a forensic expert, but rather by a gynecologist. However, forensic medical experts are called upon to gather extragenital findings and to interpret findings in the context of the reported patient history and overall findings. **The absence of evidence of abuse is not evidence of the absence of abuse.** At the same time, self-inflicted injury sometimes needs to be considered in the case of older children.

19.1 Introduction

Pedophile offenders (generally males) choose their victims; the literature describes particular risk groups commonly affected:

- Emotionally neglected children
- Children whose parents are not available to them due to sickness or drug/alcohol dependence
- Mentally handicapped children

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- Children whose parents live in marital conflict
- Girls living in the same home as a stepfather and mother

Revictimization following sexual abuse is common in cases where the victim has developed low self-esteem, has a weak psychological self-defense mechanism, and, as a result of having experienced the victim role, has internalized a sense of hopelessness in terms of targeted resistance. Although offenders are predominantly male, up to 25 % are female in cases where the victim is a male minor. Offenders usually come from the victim's immediate social environment (family, hometown, clubs, schools, etc.). If the administration of alcohol, medication, or drugs is suspected, blood and urine samples need to be taken. In cases where long-term administration of foreign substances to a child is suspected, hair samples for chemical toxicological analysis should also be taken.

As with other forms of child abuse, a head-to-toe examination needs to be performed in cases of suspected child sexual abuse; optimally, this should take place as soon as possible after the abuse for the purposes of evidence collection. The preservation and documentation of evidence is subject to the same requirements as those for sexual offenses against adult victims. However, examining underage victims of violence requires a particular level of professionalism. Findings should be gathered in such a way as to avoid renewed traumatization of the victim. Where possible, victims should profit emotionally from the examination by receiving the message: "I'm fine!" The majority of sexually abused children and adolescents show largely unremarkable findings on physical examination.

Child sexual abuse is defined as the involvement and coercion of children (up to 14 years) or adolescents (14–18 years) in sexual activities that they, due to their developmental immaturity, are unable to fully comprehend and consciously consent to and which break family and social taboos.

Child sexual abuse includes sexual activity with physical contact (primarily breast and genital regions; so-called hands-on offenders), producing and sharing pornographic material, exhibitionism, as well as causing or inciting sexually related behavior (so-called hands-off offenders). As a rule, offenders are significantly older adolescent or adult individuals exploiting a relationship of dependency.

Although the law defines precise age limits, sexual abuse is not necessarily present in all cases of peer relationships between pubescent individuals/adolescents (e.g., a 13-year-old girl and a 15-year-old boy), particularly when the age difference between the sexes is minimal and the general circumstances indicate a romantic relationship or consensual sexual contact.

The prevalence of sexual abuse involving physical contact in childhood and adolescence is estimated at 10–15 % in girls and 5–10 % in boys. Up to 20 % of offenders are adolescents, of which 80–90 % are males. However, the reliability of these data is controversial, and variations both upwards and downwards are suggested.

There is no "abuse syndrome" to prove either current sexual abuse or the long-term effects of child sexual abuse.

The number of findings that provide evidence of sexual abuse is lower than commonly assumed; thus, the following is valid:

Normal findings in the anogenital region do not exclude even current penetrating child sexual abuse!

However, there are numerous congenital, dermatological, and urethral findings that may mimic sexual abuse. This is true of hematomas in the setting of coagulopathies (see Chap. 18), phytodermatitis-related changes in skin color, Mongolian spots, clothing dye, lichen sclerosus, and hemangiomas. The absence of findings despite known previous sexual abuse is explained by the greater tissue elasticity seen in children and adolescents, delayed examination, bathing or cleaning in the interim, or the type of sexual abuse.

19.2 Examining an Underage Victim of Violence

The anogenital examination of girls can be carried out in a variety of examination positions, such as in the supine or supine frog-leg posi-

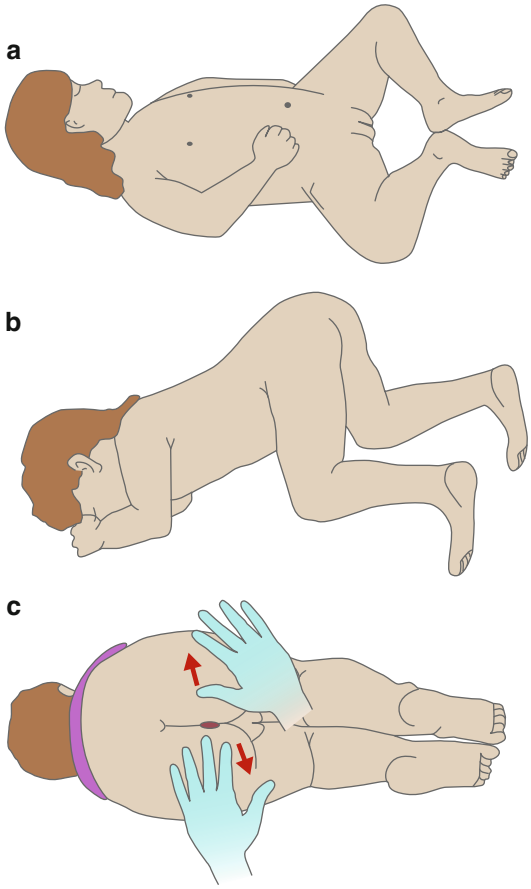

Fig. 19.1 Examination positions. (**a**) Frog-leg position. (**b**) Prone knee-chest position. (**c**) Supine lateral position (Modified according to J. Brünig; from Banaschak (2004)

tion, lateral position, and prone knee-chest position (Fig. 19.1). Examining a child while she is seated on her mother's lap is also possible. Gathering findings is made easier by a few steps aimed at labial traction and separation of the genitals. Labial traction involves taking the lower part of the labia between the thumb and forefinger and applying force downwards and outwards, while separation describes the application of mild lateral force to the labia. Particularly in the case of small lesions, colposcopy can be helpful, especially when equipped with a camera enabling simultaneous photodocumentation.

No particular examination position is required for boys. If anal abuse is suspected, a lateral supine position with buttocks separated, thus affording adequate visualization of the area, is recommended. Attention should be paid to redness, abrasions, or constriction, etc. on the penis and scrotum.

The aim of a thorough examination is to identify discrete injuries to the anogenital region, including hymenal injury. Although rare, a number of accident-related anal and/or genital or vaginal penetration injuries (impalement injuries) are seen. While straddle injuries usually cause markedly unilateral contusions, they generally do not cause injury to the hymen. When examining a child for suspected child sexual abuse, it is important to adhere to general principles, a specific examination procedure, and the recommended examination positions/techniques, while at the same time ensuring the preservation of evidence.

The general principles of examination (from Herrmann et al. (2010)) include:

- Never use coercion or verbal pressure!
- The time of the examination should be flexible according to forensic urgency.
- Anticipate possible anxieties in the child.
- Respect boundaries and allow time.
- Use age-appropriate language.
- Keep the sex of the examiner flexible, allow freedom of choice, and permit the child as much control as possible over the situation.
- Take note of behavior and anxieties.
- Emphasize positive aspects verbally.
- Praise the child for a successful examination.
- Sedation is generally not indicated, with the possible exception of severe acute bleeding injuries in the anogenital region.
- A complete head-to-toe examination is mandatory.
- Specula should not be used in prepubertal children.

Head-to-toe examination is mandatory. Specula are not used in prepubertal children, and although they are permitted in pubertal children, they are rarely helpful. No vaginal/anal palpation; gloves should be worn. In addition to photodocumentation, further documentation (statements on events, samples from clothes, adherence of foreign material, etc.) needs to be considered, as do smear and other samples for the preservation of evidence.

Insofar as a child or adolescent is questioned by a physician, care must be taken not to distort an original statement by asking leading questions.

19.3 Anogenital Examination Findings

The most important findings made on anogenital examination are shown in Table 19.1.

Although deep indentations and fissures in the hymenal rim (in particular between 3 and 9 o'clock) are not necessarily the result of penetration, they are considered a suspicious finding (see Table 19.3). Acute mucosal tears and/or lacerations in the labia are seen following (attempted) digital or penile penetration (Figs. 19.2 and 19.3).

Acute bruising of the labia, penis, scrotum, perianal tissue, or the perineum also suggests local trauma. Tear-like dissections in the skin or soft tissue between the perineum and hymen are particularly suggestive of a penetration injury. Local hematomas may also be present if trauma is recent. In the case of delayed examination, attention should be paid to possible scarring in this area (Fig. 19.4), which needs to be differentiated from a linea alba. When using the supine or frog-leg position to examine the female genital area, injuries are usually found between 9 and 3 o'clock following digital penetration and between 3 and 9 o'clock in the case of other forms of penetration, in particular with an erect penis.

Sexual abuse should be considered in this setting; however, accidental injury or local physical abuse without penetration or a sexual component needs to be considered in the differential diagnosis.

Anal Dilatation. The significance of reflex anal dilatation (RAD) is controversial. Dilatation of less than 2 cm can be considered a normal reflex reaction and is also seen in association with constipation, encopresis, and neuromuscular diseases. Anal dilatation under sedation or anesthetic is not a valid finding! Caution is also advised when interpreting anal fissures, since these are often caused by perianal infections or skin irritation of other origin.

Table 19.1 Anogenital findings consistent with sexual abuse

Criterion	Possible findings
Hymenal configuration	Annular, semilunar, fimbriated, rarely septate, cribriform, microperforate, special forms
Position of the hymenal orifice	Anterior, central, or posterior
Hymenal characteristics	Finely translucent, resting phase; fleshy, estrogen phase
Hymenal width	Regular or irregular
Appearance of the free hymenal rim	Smooth, wavy, notched, notch depth, openings
Diameter	Specify transhymenal diameter
Elasticity of the hymenal orifice	As far as can be evaluated. Caution: foreign bodies behind the hymen are suspicious for third party involvement, particularly in young girls with easily damaged and painful hymens
Injury localization	Tears, hematomas, fibrin layers, lacerations
Inspecting the perineal area	Lacerations, hematomas
Traces of secretion and other substances	Secretion characteristics; take a smear sample
Anal region: acute abuse	Perianal swelling, marginal hematomas, radial (bleeding?) fissures, dilated anus, linear skin abrasions
Anal region: chronic abuse	Thickening of the anal skin, flattened anal folds, reduced sphincter tone, anal dilatation, venous stasis, chronic fissures, wedge-shaped scarring and skin tags (not in the midline), warts; consider possibility of sexually transmitted diseases!
Consider extragenital and extra-anal findings	e.g., Signs of abuse such as hand marks, self-defense wounds, and injuries from blows

Caution: The collection of findings should be strictly separated from the interpretation of findings with relation to sexual abuse

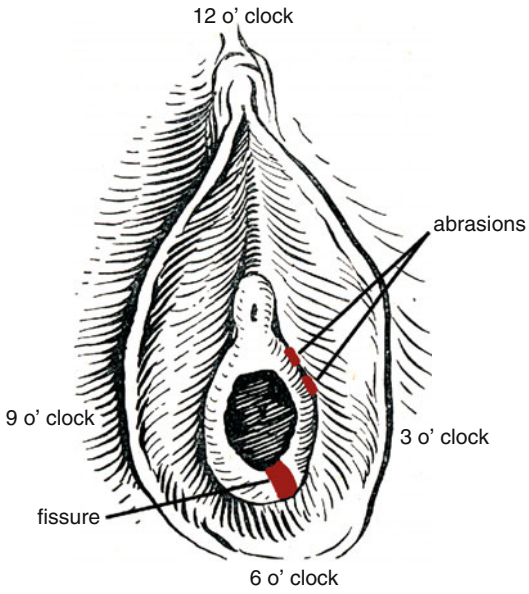

Fig. 19.2 Genital findings following digital and/or penile manipulation (Modified from Banaschak and Brinkmann (1999))

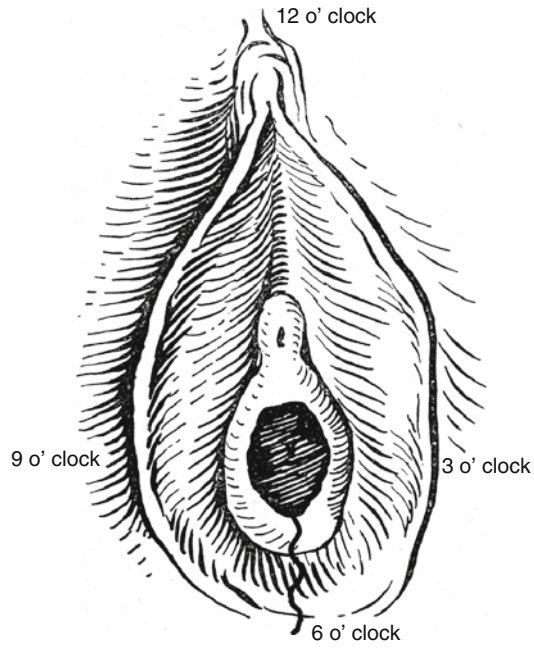

Fig. 19.4 Genital finding: scarring from the perineum to the posterior/inferior hymenal rim following penetration (Modified from Banaschak and Brinkmann (1999))

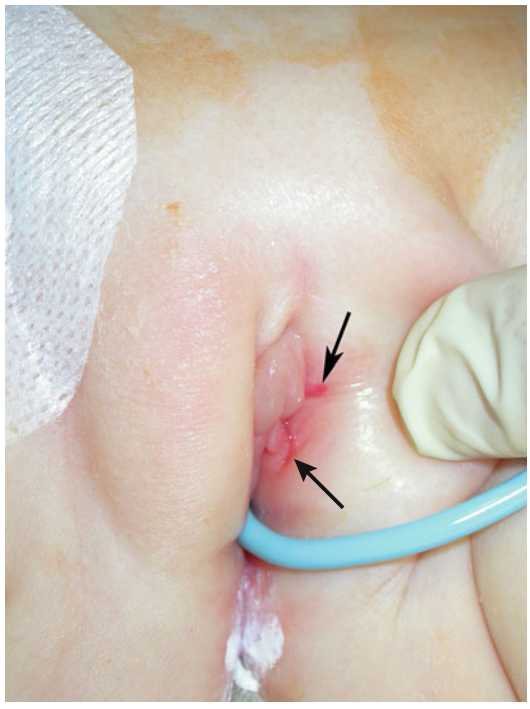

Fig. 19.3 Fresh lacerations (*arrows*) to the labia following attempted vaginal penetration

Actual anal penetration can cause deep mucosal lacerations, local edema, and severe circular-perianal hematomas that extend to the anal mucosa and perianal muscular tissue.

19.4 Sexually Transmitted Diseases and Child Sexual Abuse

The evidentiary value of sexually transmitted diseases in child sexual abuse varies, and here again, the overall picture needs to be assessed (Table 19.2).

Forensic examination requires anal, vaginal, and oral smear samples for the detection of sperm. If oral skin contact (licking, kissing, biting, etc.) is reported, the relevant areas of skin should be carefully swabbed with a moist Q-tip using a rolling motion, even when no traces are visible to the naked eye. All smear samples must be labeled like other samples with the patient's personal data, as well as the date, time, and place of sample collection.

Table 19.2 Evidentiary value of sexually transmitted diseases in child sexual abuse

Pathogen	Incubation period (detection)	Evidentiary value
HIV	6 weeks to 18 months (serum)	++
Syphilis	10–90 days (serum)	+++
Gonorrhea	2–7 days (culture)	+++
Trichomoniasis	4–20 days (microscopy/culture)	+++
Herpes simplex virus type 2 (HHSV-2)	2–24 days (e.g., blister smear)	++
HPV types 6, 11, 16, 18, e.g., condylomata acuminata	1–9 months (20 months?) (bioptic, in situ hybridization)	++
Chlamydia	Variable (culture)	++
Herpes simplex virus type 1 (HHSV-1)	2–14 days (e.g., blister smear)	+
Mycoplasma/ureaplasma	2–3 weeks? (culture)	+
Bacterial vaginosis	7–14 days	+
Candida albicans	? (microscopy, culture)	Unlikely

Modified from Herrmann et al. (2010)
Suspicion of sexual abuse is strong (+++), significant (++), or weak (+)
While all pathogens can cause intrauterine or perinatal infection in individual cases, this is doubtful or unknown for mycoplasma, ureaplasma, bacterial vaginosis, and *Candida albicans*

STD detection in a child always requires the route of infection to be established!

A number of dermatological diseases need to be distinguished from findings of sexual abuse in the differential diagnosis of abnormal skin lesions, such as nonspecific exanthemata, contact dermatitis, skin irritation due to mucosal irritants, poor hygiene, diaper dermatitis, diaper rash, seborrheic dermatitis, psoriasis, and lichen dermatoses (e.g., lichen sclerosus et atrophicus), among others. Group A ß-hemolytic streptococcal infection can cause highly acute, edematous genital or perianal inflammation suggestive of sexual abuse in the differential diagnosis, e.g., anal penetration (Fig. 19.5).

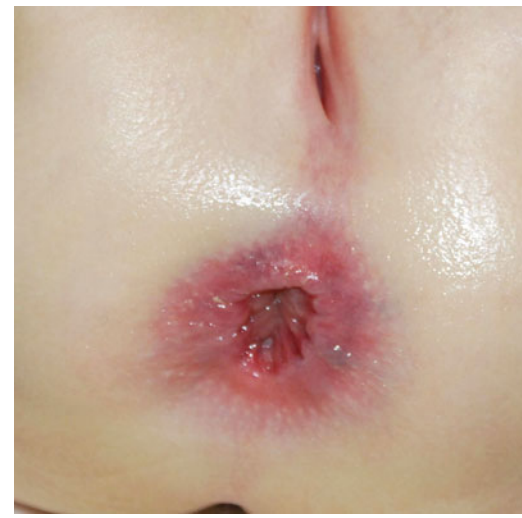

Fig. 19.5 Perianal group A ß-hemolytic streptococcal infection misdiagnosed as anal abuse in a 16-month-old female

Anogenital findings should be classified in terms of possible sexual abuse as the cause. However, classifications are often revised; a 3- to 4-stage classification is currently referred to (Table 19.3).

In the context of forensic evidence collection, all traces of evidence must undergo forensic work-up, e.g., to detect acid phosphatase, a prostatic glycoprotein. Ultraviolet light is able to provide orientation in terms of where to take swabs, e.g., the victim's skin. Caution: urine and a number of childcare products fluoresce in the same way as traces of semen.

Semen, blood, saliva, body hair, as well as other materials found on the body of the abuse victim may be able to help identify the offender by means of DNA analysis. Since the detection of semen is rarely possible in cases of child sexual abuse, recovering other traces of evidence, in particular items of clothing, is becoming increasingly important. As in child abuse, all proven injuries, however minor, must be documented photographically.

While injury findings are often absent or nonspecific, a number of injuries and indications that

Table 19.3 Four-stage classification for the overall evaluation of the probability of child sexual abuse

Category	Finding
Category I	*No indication of sexual abuse*
	Normal examination and patient history, normal behavior, no witnesses to sexual abuse
	Nonspecific findings of known or likely etiology, normal patient history, normal behavior
	Although classified as at risk of abuse, the child makes no statement on abuse; only nonspecific behavioral changes
	Physical evidence of anogenital injuries with clear and credible history of accidental trauma
Category II	*Possible sexual abuse*
	Herpes type I lesions in the anogenital region, no patient history of abuse, normal examination
	Condylomata acuminata but otherwise normal examination, no other sexually transmitted diseases, child makes no confirming statements (although condylomata in children older than 3–5 years are more likely to be sexually transmitted, sexual abuse is not present in the majority of cases)
	Although the child makes a statement, it is nonspecific and incompatible with findings, or the statement was obtained with leading questions
	Non-evidentiary findings of injury combined with significant behavioral changes, particularly sexualized behavior, no confirming statement by the child
Category III	*Probable sexual abuse*
	Spontaneous, clear, consistent, and detailed statement by the child that he/she has been sexually abused, with or without abnormal or positive somatic findings on examination
	Positive *Chlamydia trachomatis* culture from the genitals of a prepubescent child or the cervix of an adolescent, no indication of perinatal transmission
	Positive herpes simplex type 2 culture (genital or anal lesions)
	Trichomonas vaginalis infection (native smear or culture), no indication of perinatal transmission
Category IV	*Clear evidence of sexual abuse*
	Category IV anogenital findings with no credible history of accidental trauma
	Evidence of semen or sperm in or on a child's body
	Pregnancy
	Positive, confirmed cultures for *Neisseria gonorrhoeae* (vaginal, urethral, anal, or pharyngeal) or syphilis; no indication of perinatal transmission
	HIV infection where there is no possibility of perinatal transmission or transmission via blood products or contaminated needles
	Cases in which there are photos or videos of the child being abused

Modified according to Adams (1999) and Herrmann et al. (2002)

represent evidence of physical sexual abuse (hands-on offenders) may be found:

- Vaginal, oral, and/or anal smears test positive for phosphatase (prostate secretion) and contain whole spermatozoa or sperm heads. Sperm can be detected in vaginal smears for 48 h, and up to 6 days in individual cases (sometimes significantly longer in cadavers stored at cool temperature conditions). Identifying the offender's DNA is then possible.
- Pregnancy in a child.
- Fresh injuries in the anogenital region (see above; Fig. 19.6).
- If perinatal infection can be excluded, the detection of syphilis or gonorrhea represents evidence of sexual abuse (see Table 19.2).
- If infection via the mother or a transfusion can be excluded, HIV infection represents evidence of sexual abuse.
- If accident-related trauma or previous surgery on medical grounds can be excluded, a markedly dilated hymenal orifice and almost absent hymen relative to a girl's age, together with healed tears and evidence of scarring, can represent evidence of sexual abuse.
- The significance of reflex anal dilatation (RAD) is controversial. In isolation, anal dilatation does not indicate sexual abuse. Dilatation exceeding 2 cm without stool in the ampulla, on the other hand, is a suspicious finding and should be evaluated further.

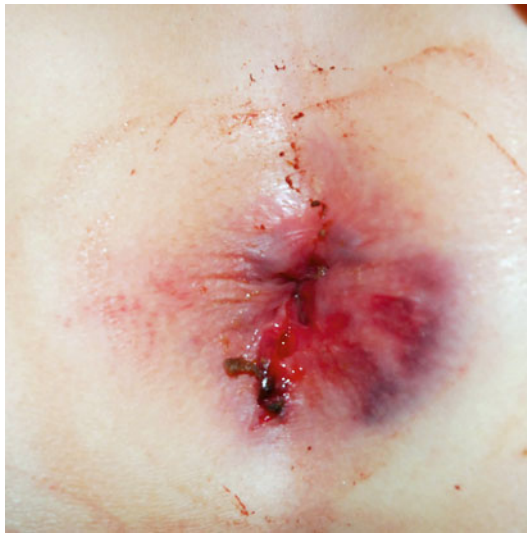

Fig. 19.6 Fresh injuries following sexual anal abuse (3 years old boy)

- If accident-related injury can be excluded, the following local injury findings provide evidence of sexual abuse: acute lacerations (tear injuries) and extensive hematomas to the labia, penis, scrotum, perianal tissue, and perineum. Similarly, acute lacerations to the posterior fourchette without hymenal involvement, perianal scarring, and scarring of the posterior fourchette or navicular fossa (differential diagnosis: linea vestibularis); acute lacerations (partial or complete) to the hymen, ecchymosis, and hematomas on the hymen (differential diagnosis: infection, coagulopathies); deep perianal lacerations extending to the external anal sphincter or beyond; healed transection of the hymen between 3 and 9 o'clock on the hymenal rim extending to or near the base; as well as a missing segment of the hymen are seen in child sexual abuse.

19.5 Behavioral Syndromes and Psychopathological Aspects of Child Sexual Abuse

The long-term psychopathological effects of (repeated) child abuse and child sexual abuse can cause secondary diseases or posttraumatic stress disorders well known in pediatric and adolescent psychiatry (see ICD-10F43.1 bzw. DSM-IV 309.81). Psychopathological symptoms include:

- Interaction abnormalities, e.g., "frozen smile" or "frozen watchfulness."
- Anxiety states may occur in situations reminiscent of abuse or the abuse context, e.g., bathing or showering.
- Reactive attachment disorder.
- Inappropriate fear of physical examination for the child's age.
- Sexualized behavior, age-inappropriate knowledge of sex, sexualized speech, sexualized behavior towards peers, and overfamiliar sexualized behavior with adults.
- Severely neglected children with deprivation syndrome may have serious sleeping, care, and nutritional disorders, as well as polydipsia.

The psychopathological long-term effects of child abuse and child molestation are grossly underestimated. The spectrum of abnormal behavioral effects includes sleeping disorders, loss of appetite, poor school performance, lying, social withdrawal, hyperactivity, regression (bedwetting, encopresis), refusal behavior, tendency towards games with a sexual theme, amnesia, hysteria, eating disorders (anorexia nervosa, bulimia nervosa, and adipositas), as well as self-injuring and self-destructive behavior. Although a forensic examination is often able to answer the question of whether recent injuries were self-inflicted or inflicted by a third party, it is unable to address the issue of the psychological or psychiatric background to self-injuring behavior following sexual abuse.

19.6 Expert Medical Appraisals in Child Sexual Abuse

Expert medical appraisals in child abuse should include judicially admissible documentary evidence of all findings, expert forensic evidence collection, and an expert appraisal that takes all available information, including reported or proven previous history, into account. Although the available guidelines, standards, and recommendations all make useful provisions, these are

not mandatory. However, expert medical appraisals in child sexual abuse cases are also made on the basis of the individual situation:

1. In the case of recent injury findings, a patient history involving an alleged incident or an incident assumed on the basis of injury pattern, as well as relevant crime scene evidence, a physical examination and expert medical appraisal should be undertaken by a forensic medical expert. Self-infliction of injuries should be considered in the differential diagnosis. Although expert forensic medical appraisals are based primarily on injury findings and the results of forensic analysis, any appraisal should include a description of the psychological status of underage victims of violence.
2. Occasionally, it is not (or no longer) possible to carry out a forensic physical examination; however, a forensic medical appraisal on the basis of available documentation and/or images of injuries is requested. Establishing whether documentation and images are of sufficient quality is crucial. In the case of expert appraisals based on records alone, greater caution is sometimes advised in the choice of formulation used in the appraisal.
3. If evidence of the alleged offense as well as any injuries that have since healed has been well documented, it may be possible to perform an expert forensic medical appraisal retrospectively. In cases where a significant period of time has elapsed since the incident, residual physical findings (scars in particular) may form the subject of an expert forensic appraisal.
4. A forensic medical "plausibility check" is also possible in cases where an incident is alleged or where possible inconsistencies or open questions remain and is based mainly on forensic experience (age, pattern of injury, offender profile, victim behavior, available evidence, etc.).
5. In cases where long periods of time have elapsed since the offense, it is generally no longer possible to establish whether child sexual abuse took place in childhood using injury findings and evidence of a crime. Insofar as psychological and psychosomatic sequelae can be attributed to sexual abuse in childhood, a psychological/psychiatric expert appraisal can be of help, whereby the questions of victim chronology since the event (primary trauma) and trauma processing also need to be addressed.

As in nonsexual child abuse, documenting child sexual abuse should be carried out using appropriate standardized examination forms where personal details, details of the authorities commissioning the examination, the examination date, the examiner, as well as a possible examination or log number, can be entered. Anatomical diagrams of the body and genitals used on the examination form are intended to support the documentation of findings (see Appendix).

Both an overview photo and a detailed photo using a scale should be made of any injuries or abnormalities on the body. Evidence should be collected using individually wrapped, contamination-free, and preferably EO-treated (i.e., DNA-free) Q-tips, which can be moistened if necessary with sterile bi-distilled water from previously unopened disposable packs. Samples should be taken only from those areas where traces of evidence are to be expected. Q-tips should then be dried in a contamination-free environment and stored. Storage times are determined in collaboration with the police or commissioning authorities and should also be specified in writing. When taking items of clothing as samples, these should be dried in a contamination-free environment and placed in paper bags (never plastic bags!).

Selected References and Further Reading

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Infanticide and Neonaticide

20

Case Study

The police received a report from an elderly woman regarding a 32-year-old woman in her neighborhood: the 32-year-old, who had always been somewhat adipose, had gained a significant amount of weight and now appeared, in the previous 2 days, to have suddenly lost a remarkable amount of weight. During a chance meeting at the bakery, the elderly woman had asked the younger woman if she were pregnant, to which the latter replied that she was not. The police searched the home of the 32-year-old, whose boyfriend—as in the past—was away on a job for 2 weeks. Following initial denial, the woman tearfully admitted to having given birth on her own in her bathroom, alleging that the child was stillborn. The infant was found wrapped in a bloodied towel and a plastic bag in a garbage can. A forensic medical investigation demonstrated that the infant must have been premature (body weight 3,150 g, length 48 cm). A hydrostatic test and a stomach–bowel test (Breslau's second life test) were both positive. The umbilical cord had been sharply severed; the placenta was not found. Although the cause of death was not apparent to the naked eye, the suspicion of suffocation with a soft object was voiced. Confronted with the autopsy results, the defendant admitted that, in her attempts to calm the neonate's crying, she had pressed it to her body for some time; at some point, the baby's crying stopped.

Infanticide and child homicide are seen in many forms and under widely varying circumstances:

- Causing the death of a neonate immediately after birth: Neonaticide (up to 8 days old; however, the time frame for neonaticide can be longer or shorter depending on national law)
- Causing the death of an infant during the first year of life (infanticide), less frequently in the second or third year of life as a result of shaken baby syndrome (SBS)
- Causing the death of a child by intoxication, in the form of either homicide or manslaughter
- Causing the death of a child in the context of an extended suicide
- Causing the death of a child by various forms of violence (gross blunt trauma, sharp force injuries, suffocation, drowning, scalds, and burns), in some cases to conceal a previous offense, in particular a previous sexual offense (i.e., killing to conceal another crime)
- Causing the death of a child as a result of physical neglect (infants and young children in particular)

Depending on the particular configuration of a case, a variety of findings and links need to be considered. In the case of neonaticide, the potential

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mother should also be examined promptly, while the search for substances possibly administered in intoxication is important. Finding an infant dead should prompt—besides classification as sudden infant death syndrome (SIDS)—the possibility of suffocation using a soft cover without trace to be considered.

20.1 Neonaticide

Potential extrauterine viability of a neonate is likely from around the 22nd to 23rd gestational week, while neonatal viability from the 32nd gestational week and a length of 35 cm can be assumed. However, diseases that threaten, or arouse justified doubt about, neonatal viability need to be excluded; these include diseases that have their onset at birth and are preexisting or acquired, as well as infectious diseases: intracranial bleeding, protracted delivery, as well as congenital deformities and malformations, in particular of the cardiovascular system.

Three important differential diagnoses that need to be considered in the case of a neonate found dead:

1. Stillbirth following intrauterine death.
2. Death from natural causes either during or immediately after birth.
3. The neonate was born alive and subsequently killed.

Neonaticide is often seen following a repressed or concealed pregnancy. Even the father of the child often claims not to have noticed the pregnancy. Neonaticide can be the result of failure to provide due care and protection, e.g., suffocation due to abandonment following aspiration of amniotic fluid, meconium, or blood or leaving a neonate to drown in a toilet, bleed to death (very rarely), become hypothermic (death due to exposure), or dehydrate (death due to dehydration). However, active cases of homicide are also seen, particularly in the form of suffocation with a soft cover, introducing foreign objects into the mouth and thereby causing airways obstruction (gagging), as well as compression trauma to the throat (strangulation or choking). The cause of death in cuts and stab wounds—which are rarely seen in infanticide—is generally death due to exsanguination, while an air embolism should be considered in the case of cut wounds to the throat involving large vessel transection.

Table 20.1 Investigating a newborn found dead and the suspected mother: questions requiring clarification

Neonate	Suspected mother
Pregnancy duration/ maturity or age	Mother's identity
Evidence of neonatal viability	Diagnosis of recent delivery
Duration of life after birth (positive hydrostatic test and/or stomach–bowel test)	Time of birth
Signs of neonatal status and form of umbilical cord clamping	Correlation between time of birth and postmortem interval
Cause of death	Statement about the birth and course of events (duration of labor, color of amniotic fluid, instrument used, method of umbilical cord transection, etc.)
Reconstruction of the act on the basis of autopsy findings	Whereabouts of the placenta and umbilical cord

Characteristic features of neonaticide are often seen in the mother's circumstances and during the pregnancy, including:

- Not older than 25 years
- Living alone or still with parents, occasionally also with the father of the child
- Unmarried (up to 80 %)
- Repressed or denied the pregnancy
- Concealed the pregnancy even from the child's father and close relatives
- Caught unprepared for the onset of labor
- Often immature, passive, and callous
- Primiparae
- No regular medical checkups during pregnancy

Delivery usually takes place in the mother's home, often while she is seated on the toilet or in bed. The neonate is usually not cared for following birth, i.e., blood and vernix caseosa are not removed.

In cases where a neonate is found dead and a mother is suspected, forensic investigations concentrate on the points given in Table 20.1.

The criteria given in Table 20.2 belong to the signs of neonatal maturity identifiable at autopsy.

In addition to signs of maturity, any signs of live birth should be identified at autopsy (Table 20.3). However, this may be impossible in cases where the newborn was suffocated with a soft cover before taking its first breath. In forensic practice, hydrostatic testing plays a crucial role here.

In the case of an unknown cause of death in newborns, placental analysis (placental cause of death: Insertio velamentosa? Chorioamnionitis? Placental infarction? Placental maturity disorder? Premature placental rupture?) must be performed in addition to an autopsy investigation.

Table 20.2 Typical signs of maturity seen in a newborn

Criterion	Threshold value/extent
Length	48 cm
Body weight	2,500 g or more
Head circumference	Approximately 34–35 cm
Umbilical cord length	Approximately 50 cm
Placental weight	Approximately 500 g
Fingernails	Exceed fingertips (Fig. 20.1)
Toenails	Exceed tips of toes
Lanugo	Remaining only on shoulders
Genitals	Testicular descent, labia majora cover labia minora
Shoulder width	12.5 cm
Hip width	9.5 cm
Calcaneal ossification center	9.5 mm
Béclard's sign (distal femoral epiphysis)	5 mm

Strangulation caused by twisting of the umbilical cord around the neck in nonhospital births is a rare natural cause of death (Fig. 20.4), as is suffocation immediately after birth due to airway obstruction by the amniotic sac, which can sometimes be difficult to remove (Fig. 20.5).

If there are grounds to suspect a woman of recently having given birth, she can be subjected to a medical examination to establish whether a pregnancy has recently taken place. A gynecological examination should clarify the following points:

- Size and consistency of the uterus
- Height of the uterus
  - Immediately postpartum: Midway between the umbilicus and symphysis
  - 1 day postpartum: One fingerbreadth above the umbilicus or at umbilicus level
  - 2 days postpartum: One or two fingerbreadths below the umbilicus
  - 3 days postpartum: Three fingerbreadths below the umbilicus
  - 7 days postpartum: Two fingerbreadths above the symphysis
  - 10 days postpartum: At the level of the symphysis

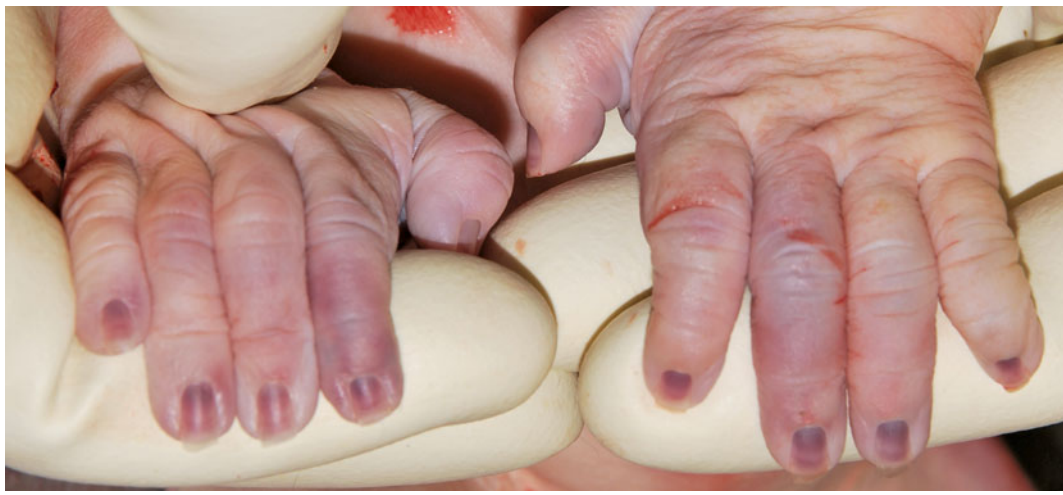

Fig. 20.1 Signs of maturity in a newborn found dead (weight, 3,050 g; body length, 51 cm): fingernails exceed fingertips

Table 20.3 Neonatal signs of live birth

Criterion	Finding
Lung	Pleural cavities fully occupied
Lung consistency	Cushion-like, crepitant to the touch
Lung surface	Light gray/red with sunken atelectasis
Hydrostatic test (caution: respiration)	Whole and parts
Throat organs + lungs + mediastinum	Hydrostatic test is positive when the lungs float and "hold" the organs
Lung only (Fig. 20.2)	Positive (possibly relevant if the first hydrostatic test with organs was inconclusive!)
Pulmonary lobes only	Caution: putrefaction or artificial respiration may cause the hydrostatic test to be false-positive; causing death prior to the first breath or fluid aspiration produces false-negative results
Stomach–bowel test (Fig. 20.3) (ligation and floatation of segments of the gastrointestinal tract)	Usually positive after pulmonary respiration due to "swallowed air"
	Usually only positive after pulmonary ventilation due to "swallowed air," thus in the case of recent birth, positive hydrostatic test and negative stomach–bowel test more likely
	Caution: airway obstruction can cause air to reach only the stomach
Duration of life as demonstrated by the stomach–bowel test	
Air only in the stomach and upper duodenum	Several minutes to maximum 30 min
Air in the duodenum, jejunum, and ileum	Approximately 6 h
Air in the entire colon	Approximately 12 h
Duration of life as demonstrated by determining the distribution of meconium	
Meconium along the length of the colon	Less than 2 days
Meconium only in the intestinal loop	2–3 days, possibly even 5 days
Presence of caput succedaneum	May be absent in small neonates, broad pelvis in the mother, and rapid birth

From Herrmann et al. (2010)

- Position of the uterus
- Width of the ectocervix and uterine cervix
- Lochial discharge
- Mammary status including mamillae
- Where necessary, sampling for DNA analysis to establish motherhood

The examination should also serve to establish the time of delivery. In cases of very recent delivery, the placenta may still be found in the uterus on examination, while the separated end of an umbilical may be found in the birth canal (Fig. 20.6). If a large number of women need to be considered in terms of identifying the child's mother, mass gene testing can be considered and results subsequently compared with the DNA of the deceased neonate.

In cases where death is caused in a neonate following an unnoticed delivery, the delivery has often taken place in a restroom or toilet ("restroom birth"). The methods of causing death that need to be considered in restroom births include:

- Suffocation with a soft cover
- Drowning in a toilet bowl
- Gross blunt trauma combined with drowning by forcing the head into a toilet (Fig. 20.7)
- Lethal gagging of the newborn with, e.g., wads of toilet paper forced to the back of the throat, resulting in the base of the tongue being pressed towards the epiglottis, thus preventing nasal respiration (see Fig. 14.11)
- Gross blunt trauma resulting in exsanguination of the newborn
- Lethal stab wounds, e.g., to the open fontanelle causing cerebral hemorrhage

The Mother's Ability to Act During or After Birth. Statements made by the mother, e.g., that

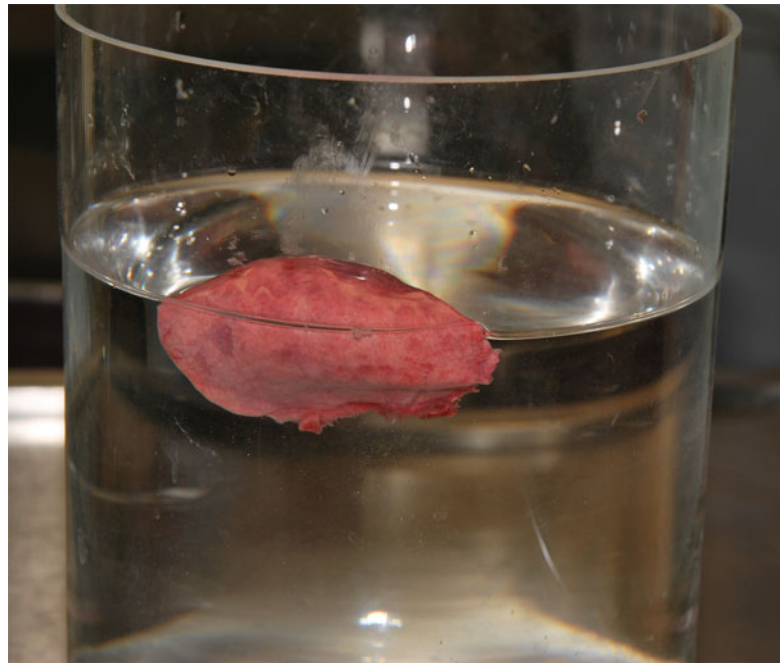

Fig. 20.2 A positive hydrostatic test represents a sign of live birth

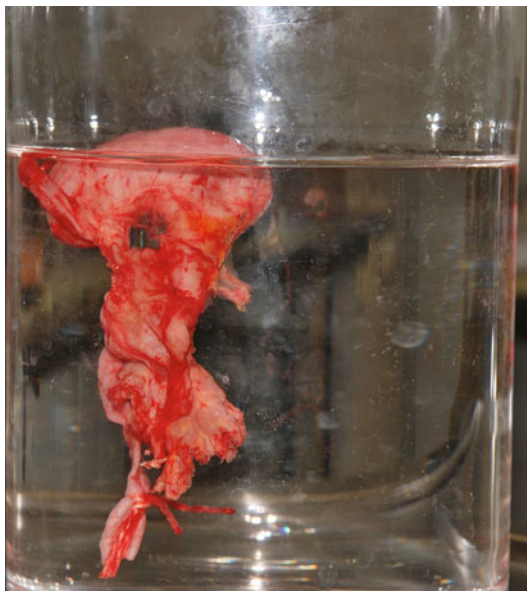

Fig. 20.3 A positive stomach–bowel test represents a sign of live birth

she fell unconscious during or after the birth and was therefore unable to care for the child, can be relevant when establishing whether a neonate was born alive. Unconsciousness in a mother either during or immediately after birth is a very rare event. Plausible explanations include eclampsia, an epileptic seizure, or very seldom severe hemorrhage. This latter event can be verified by measuring the mother's Hb value.

Precipitate Labor. It is not uncommon for a mother to report how a sudden urge to defecate resulted in the unexpected birth of a neonate in the toilet bowl. However, the height of the fall imputed here if the mother was in a squatting position would be insufficient to cause the death of the child on the one hand, while corresponding fall injuries would be found at autopsy on the other. It is sometimes also possible to verify whether a neonate was born into water in a toilet and subsequently drowned.

Umbilical Cord Rupture Resulting in Exsanguination. It is occasionally necessary to establish whether a newborn died as a result of hemorrhage following umbilical cord rupture. The umbilical cord is not easily ruptured, requiring a vertical drop of sufficient height as well as producing a transection wound compatible with a tear: protruding vessels, ragged edges, and an oblique rupture surface are consistent with a reported umbilical cord rupture, while smooth vessel edges or smooth-edged tapered

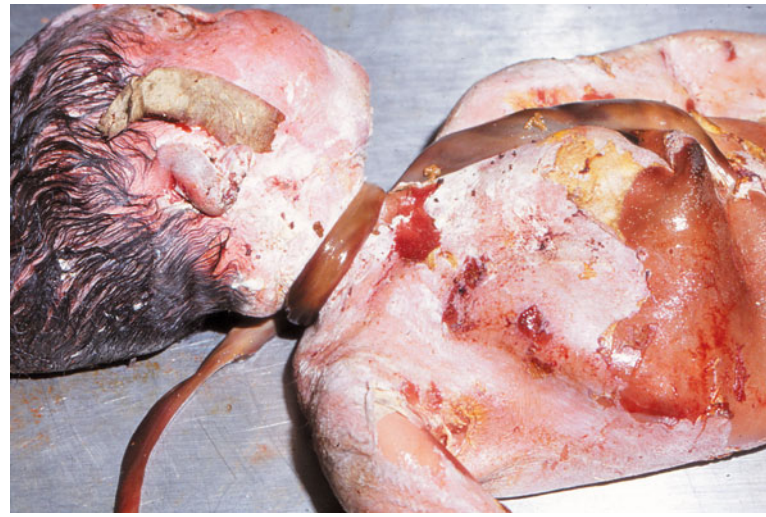

Fig. 20.4 Umbilical cord around the neck as rare natural cause of death

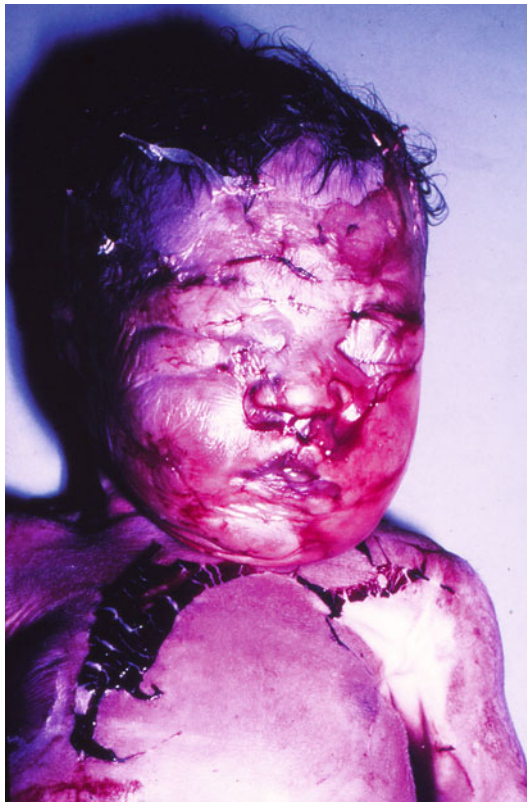

Fig. 20.5 Airway obstruction by the amniotic sac

protrusions at the end of the umbilical cord speak against a tear (Fig. 20.8).

Furthermore, exsanguination even in the case of umbilical cord rupture is unlikely, since hemorrhage of this kind generally ceases spontaneously following contraction of the umbilical artery, which displays only relatively low blood pressure, and due to postpartum distribution in the neonate's pulmonary circulation.

20.2 Shaken Baby Syndrome: Non-accidental Head Injury

The somewhat older term shaken baby syndrome (SBS) is often replaced in the more recent literature by non-accidental head injury (NAHI) or inflicted traumatic brain injury (ITBI), while some authors prefer the term inflicted childhood neurotrauma (ICN). Whichever the term used, they all describe a syndrome or constellation of injuries. There are only few studies on the incidence of nonfatal shaken baby syndrome in infancy, since milder forms of shaking may lead to intracranial injury (e.g., small hemorrhages, subdural hematoma, and retinal petechial hemorrhage) without necessarily causing death (see Chap. 18). Survivors sustain neurological damage of varying degree (e.g., cerebral atrophy, subdural hygroma, multicystic encephalopathies, cerebral seizures, mental retardation, and spastic paraparesis), the intensity of which often only becomes apparent months or years after the fact, by which time a causal connection to shaking is frequently not even considered. However, the reverse is not

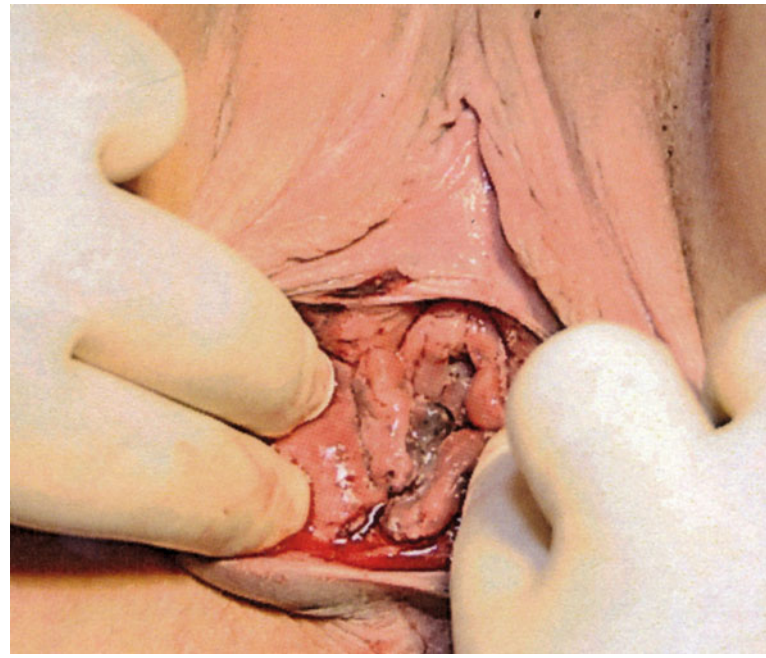

Fig. 20.6 A transected umbilical cord identified in the birth canal approximately 60 min following delivery of and death caused in a mature neonate in a hospital restroom/toilet (17-year-old patient who had denied pregnancy up to delivery)

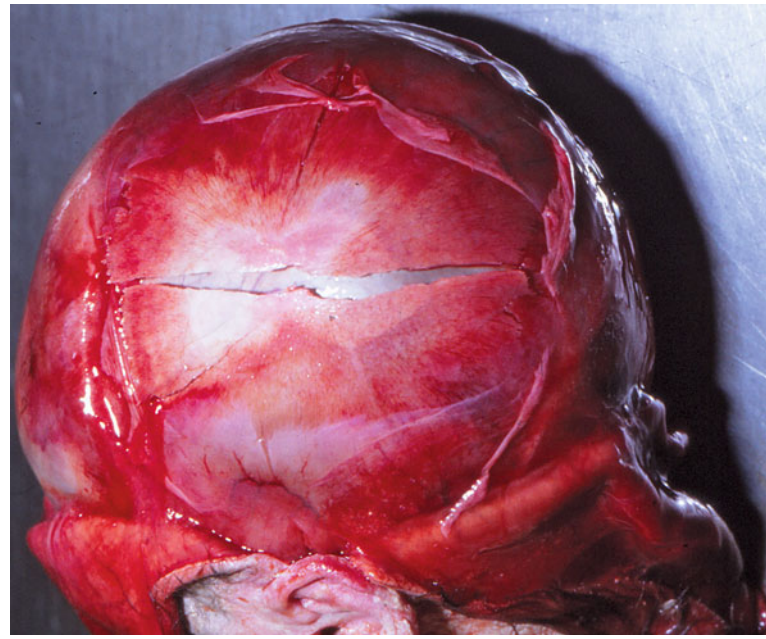

Fig. 20.7 Fractured skullcap in a neonate caused by forcing the head into a toilet

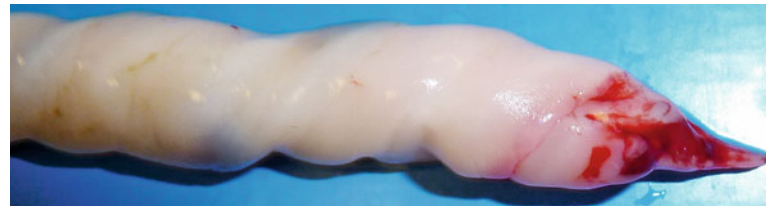

Fig. 20.8 Smooth edge of a tapered extended end of an umbilical cord without prominent vessel stumps following postpartum transection with scissors

Table 20.4 Stages in the organization of subdural hematomas

Time period following injury	Possible microscopic findings
Up to 24 h	Erythrocytes, a thin layer of fibrin between the dura and clot
48–72 h	Erythrocytes, rare fibroblasts at the interface on the side of the membrane facing the dura
4–5 days	Breakdown of erythrocytes, two- to five-cell-thick layer of fibroblasts on the side of the membrane facing the dura
5–10 days	Early capillary formation/granulation tissue of clot, some siderophages, thicker layer of fibroblasts, and, occasionally, small capillaries may be present on the side of the membrane facing the dura
10–20 days	Granulation tissue with capillary formation within the clot, fibroblast layer 1/3–1/2 as thick as the dura; siderophages; early fibroblastic membrane evident on the side of the membrane between the clot and arachnoid
3–4 weeks	Clot nearly liquefied, membrane equal to dura in thickness on the side of the membrane facing the dura; siderophages
1–3 months	Large capillaries, possibility of rebleeding, hyalinized membranes on the side facing the dura and on the side between the clot and arachnoid
Once organization is complete, only a thin yellow-gold-colored membrane adherent to the dura may remain	

According to DiMaio and Dana (2007), Dettmeyer (2011)

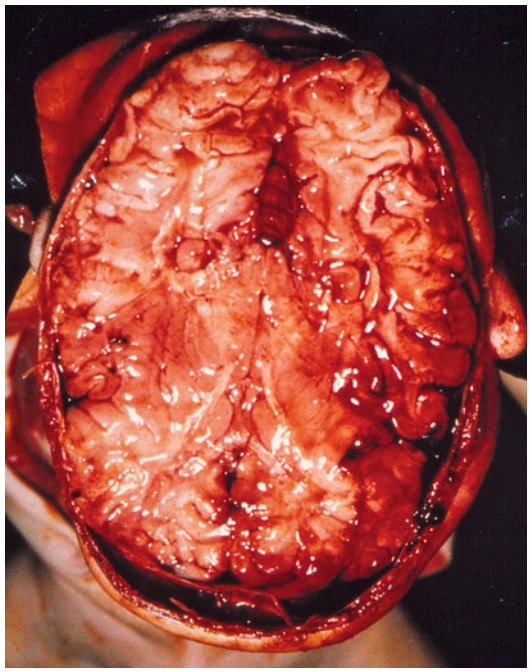

Fig. 20.9 Lethal SBS demonstrating a crescent-shaped subdural hematoma at autopsy in a 4-month-old male infant

always true, i.e., that findings, such as subdural hematoma, can be attributed to child abuse. In the case where an infant initially survives subdural hematoma only to die a number of days later, it is sometimes possible to determine the age of the subdural hematoma histologically, thus narrowing down the possible time of the act (Table 20.4).

According to current thinking, the cause of death in shaking trauma to an infant or toddler is not so much the invasive subdural hematoma as the neuronal damage caused by shaking, i.e., diffuse axonal injury (DAI) and subsequent cerebral edema, as well as shaking-related cerebral ischemia. At autopsy, a crescent-shaped subdural hematoma is usually seen (Fig. 20.9), which can also be clearly identified radiologically postmortem using magnetic resonance imaging (MRI) or computed tomography (CT). In addition, an ophthalmological examination can be carried out on the cadaver to detect retinal hemorrhage (funduscopy).

If the perpetrator has gripped the infant's chest tightly during shaking, bi- or unilateral serial rib fractures with surrounding hemorrhage may be seen, primarily in a paravertebral location (Fig. 20.10). In cases where SBS involving serial rib fractures is survived for certain length of time, corresponding callus formation can be seen at fracture level (Fig. 20.11). If an infant has been held by the upper arms during shaking, contusions in the shape of hand marks can be seen.

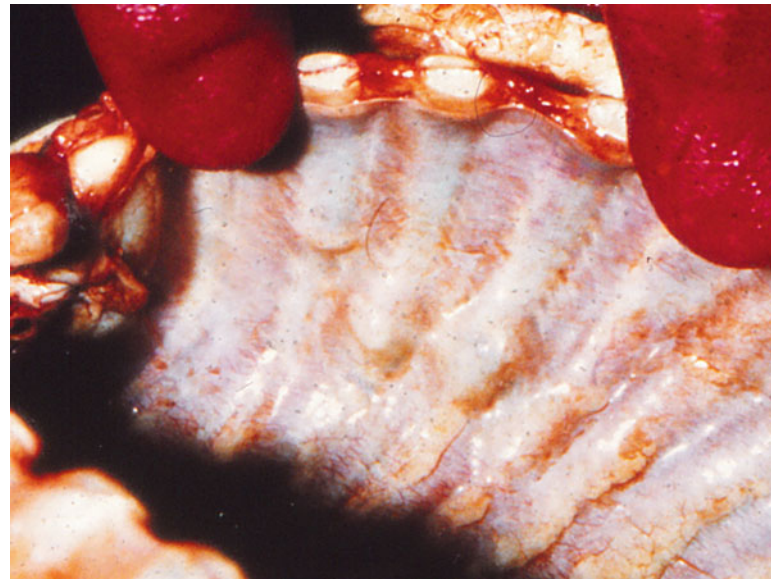

Fig. 20.10 Old serial rib fractures detected for the first time at autopsy in a 4-month-old infant following repeated SBS

Table 20.5 Stages of fracture healing (Dettmeyer 2011)

Time frame	Histological findings
1 day	Hematoma and traumatic inflammation: acute hemorrhage at the point of fracture secondary to vessel rupture, formation of a fusiform hematoma surrounding and joining the ends of the bone
1–2 days	Organization: fibrin is deposited in the hematoma, an inflammatory response with edema is seen, continuing fibrin deposition, accumulation of large numbers of polymorphonuclear cells
2–3 days	Appearance of fibroblasts, mesenchymal cells, gradual development of granulation tissue; necrosis of the bone adjacent to the fracture becomes evident; empty lacunar spaces due to death of osteocytes; clear line between dead bone (empty lacunae) and live bone
3–6 days	Provisional fibrous callus, originating from Periosteum Endosteum Havers channels Blood vessels in the bone marrow space and musculature After approximately 3 days, the devitalized bone fragments begin to be reabsorbed The periosteum is composed of an outer fibrous layer and an inner osteogenic layer: marked proliferation of the cells in the deep layer of the periosteum and the cells of the endosteum
7–14 days	Provisional bony callus: morphology of the connective tissue cells is undergoing modification. A homogeneous osteoid matrix is being deposited between the proliferating cells. Transformation of fibrous callus into provisional bony callus: connective tissue cells form ground substance and collagen fibers; fibroblasts transform into osteoblasts and produce osteoid, the organic matrix of the bone; chondroblasts are involved, and islets of cartilage develop in the fibrous stroma; bone formation, remodeling into lamellar bone (this bone forms the final callus) by means of osteoclasts and osteoblasts
2–3 weeks	Callus reaches its maximum size
3–4 weeks	Hard bony callus, bone formed from periosteal and endochondral ossification
>4 weeks	Rearrangement of callus and bony union: remodeling of the new bone from a woven appearance to mature bone; histologically ossification and new bone can be found (Fig. 20.11)

Since severe shaking can lead to subperiosteal bleeding of the clavicle, this should be sought at autopsy.

The age of fractures with callus formation can be better estimated using histological investigations (Table 20.5).

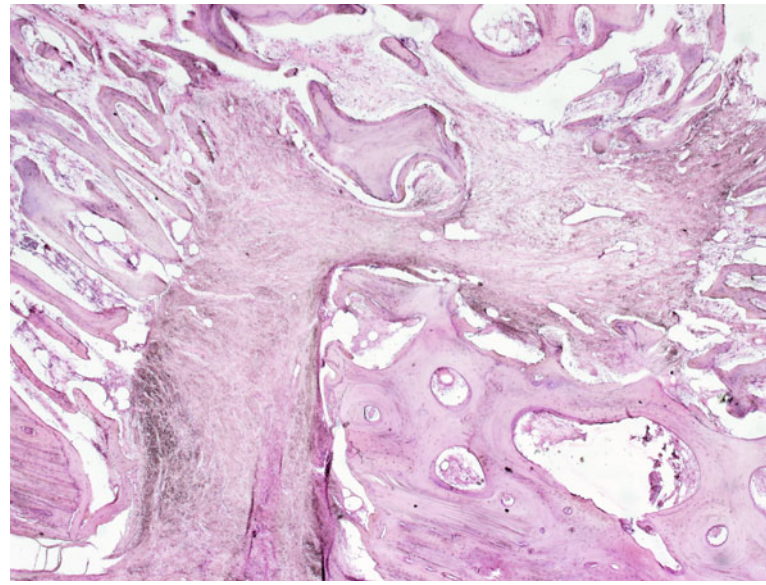

Fig. 20.11 Later in fracture healing with ossification into the fracture gap (H&E × 100)

20.3 Sudden Infant Death Syndrome (SIDS), Munchausen Syndrome by Proxy (MSbP), and Infanticide

Fatalities in infants, toddlers, and children as a result of blunt trauma or thermal injury often—but by no means always—demonstrate externally visible injuries; no injuries are detected in approximately 10 % fatalities due to child abuse.

Homicide with little or no evidence is seen particularly in the case of infants, toddlers, and children. Causes of death seen at autopsy can include previously undetectable internal bleeding (e.g., following a blow to the abdomen), intoxication, or suffocation with a soft cover.

In terms of the differential diagnosis of infant fatalities with scant evidence, infanticide needs to be differentiated from SIDS and infanticide in the context of Munchausen syndrome by proxy (MSbP), this latter particularly in the form of suffocation (Table 20.6).

20.4 Physical Neglect Resulting in Death

The birth of an unwanted child to a mother who is unable to cope, perhaps due to mental illness or addiction, is seen as a risk configuration for fatal child neglect, which can be subdivided into the following forms:

Emotional neglect (*deprivation*): Inadequate or inconsistent—and thus also insufficient—emotional care and support of a child

Physical neglect: Failure to provide sufficient care and/or obtain healthcare, resulting in failure to thrive, developmental disorders, and even psychosocial short stature

Fatal cases of physical neglect culminate in starvation or death due to dehydration. The direct cause of death seen on autopsy is often an infection, in particular pneumonia or an ascending purulent urinary tract infection with sepsis. Fatalities due to hypothermia in the context of neglect are also seen.

Physical neglect resulting in death (dying of thirst, starvation) is preceded by severe malnutrition. Symptoms of neglect seen at medical autopsy include:

- Severe emaciation or "skeletonization" (Fig. 20.12)
- Dry skin and raised skin folds (exsiccosis) (Fig. 20.13)
- Dirt and crusts in skin folds
- Severe diaper dermatitis is frequently seen (Fig. 20.14)
- Marasmus and sunken eyes
- Absence of subcutaneous fatty tissue

Table 20.6 Comparative data for the differentiation of sudden infant death syndrome (SIDS), infanticide, and Munchausen syndrome by proxy (MSbP) by suffocation

SIDS	Infanticide	MSbP
1st–12th months of life, most frequent cause of death in the first year of life	In principle, any age	Infants and toddlers; lethal course in approximately 10–15 %
Discovery following a long sleep phase, i.e., an interval between last contact and death >2 h	Acute event, rarely following long sleep phases	Hardly ever following a sleep phase
Death occurs unobserved during sleep	Death caused in the presence of at least the perpetrator	Perpetrator(s) was alone with the child
Generally infant with no clinical symptoms of disease	Infant/toddler with no relevant symptoms of disease	Perpetrator(s) often reports acute life-threatening and dramatic symptoms (screaming, rolling eyeballs, turning blue, etc.)
Autopsy and follow-up investigation unable to detect cause of death	Autopsy and follow-up investigation generally reveal the cause of death	Autopsy and follow-up investigation sometimes reveal findings comparable to SIDS, occasionally discrete suspicious petechiae in the facial skin and/or the conjunctiva in the case of suffocation with a soft cover. Live victims: symptoms remain unexplained
No perpetrator. Natural death	Perpetrator usually male	Perpetrator(s) usually female
Infant often found in the morning in a prone position	Infant found at varying time points	Even more often takes place in the late afternoon and evening
Usually no recurrences in the family	Usually no recurrences	Often several children are affected
ALTE (apparent life-threatening event) risk factors: prone position, lateral position, premature birth, obstructive sleep apnea syndrome, central hyperventilation, seizures at night, cardiac arrhythmia, infection, inborn errors of metabolism, impaired arousal, gastroesophageal reflux. Mother: postnatal depression, schizophrenic disorder, increased incidence of SIDS in the family, poor socioeconomic status	Up to 11 % of all SIDS cases believed to be infanticide; perpetrator often young (18–26) and unmarried; repressed or concealed pregnancy, living with parents or relatives, psychiatric disorders rare, act committed by stabbing, burning, battering, etc. Body disposed of in refuse or hidden	ALTE: may be result of child abuse, in particular nose bleeds, subsequent occurrence of first ALTE (3.6 vs. 0.3 months old, epileptic seizures, SIDS in siblings) In suspicious cases of ALTE: install hidden video camera in hospital
Parents show no psychological abnormalities	Psychological characteristics associated with infanticide	Although perpetrator(s) often shows psychological abnormalities, there is no conclusive psychiatric diagnosis
Infant not previously treated on an inpatient basis	Occasionally, particular circumstances	Unusually frequent and regular outpatient and/or inpatient medical consultation. Symptoms subside when child is separated from perpetrator(s)
Mother married or in a stable relationship		Aloof partnership more likely. Ostensibly concerned mother, seeks contact with hospital personnel
Previous apnea or ALTE hardly ever reported	Occasionally, “unexplained injuries” in the case history	Frequent referral to the hospital for treatment, sometimes for ALTE symptoms

Modified from and based on Noeker and Keller (2002), Häßler et al. (2007)

- Rare: (Self-) torn out hair
- Ulcers on pressure points: Buttocks, hips, heels, back of the head/knees, back
- Matted hair (lice?)
- Severe anemia
- Urine eczema as well as clothing soiled with urine and feces
- Possible (death due to) hypothermia

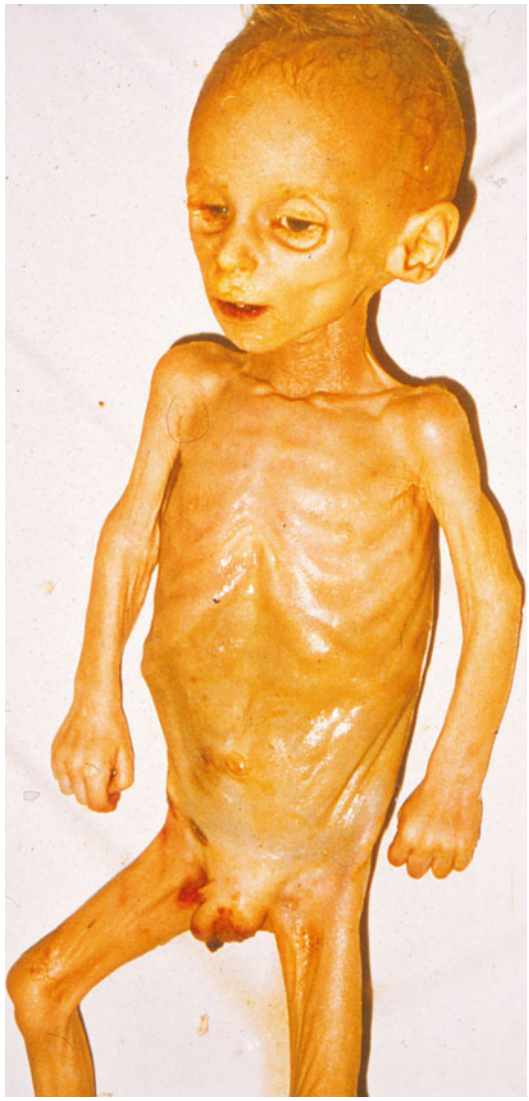

Fig. 20.12 Death due to starvation in an 8-month-old male infant weighing 3,300 g

- Signs of rachitis: Insufficient supply of vitamin D and lack of exposure to light (kept in dark rooms)
- Delayed skeletal maturation seen on radiograph with clear decalcification (including thin compact bone in the bent long bones)

Both subcutaneous fatty tissue and Bichat fat pads may be almost completely absent at autopsy, while ubiquitous edema is a sign of severe protein deficiency syndrome ("hunger edema"). Organ weight is reduced relative to the child's age. Hard fecal pellets are sometimes found in the colon. Growth in height is reduced (comparison of individual percentiles with earlier measurements). Inadequate supply of food and fluids can be identified post-mortem by biochemical findings: increased urea levels in vitreous fluid, corresponding to the dehydration pattern. Malnutrition can be classified, e.g., according to the widely used Waterlow classification (protein–energy malnutrition). In addition to final infections such as pneumonia, aspiration of stomach contents is sometimes seen, while areas of bone remodeling with increased multinuclear osteoclasts and resorption lacunae due to rachitis can be identified histologically.

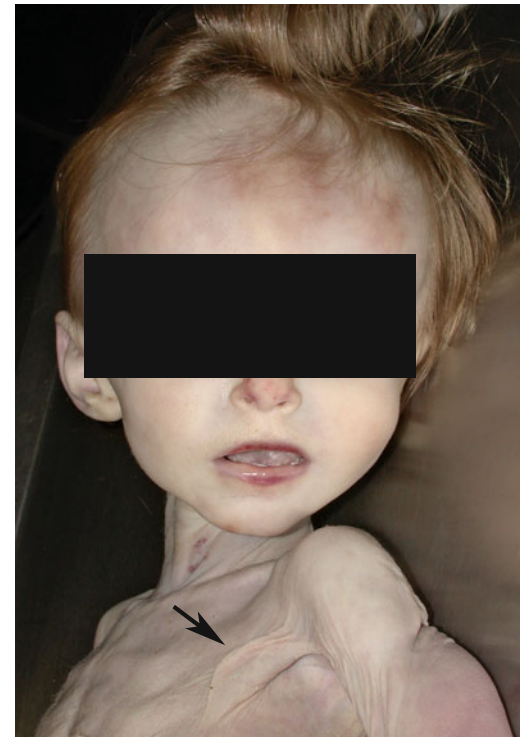

Fig. 20.13 Lethal starvation and severe exsiccosis with marked standing wrinkles (*arrow*) in an 14-month-old female

20.5 Causing Death by Gross Blunt Trauma

In addition to the described forms of lethal trauma and neglect, fatalities are seen in the context of child abuse with severe blows, primarily to the face producing intracranial injury or hemorrhage

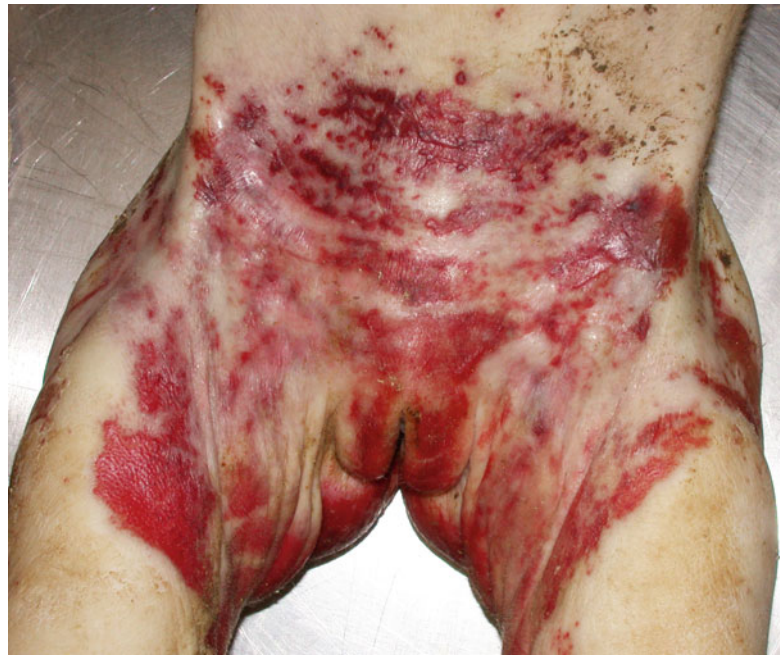

Fig. 20.14 Lethal starvation and severe diaper dermatitis in a 14-month-old female

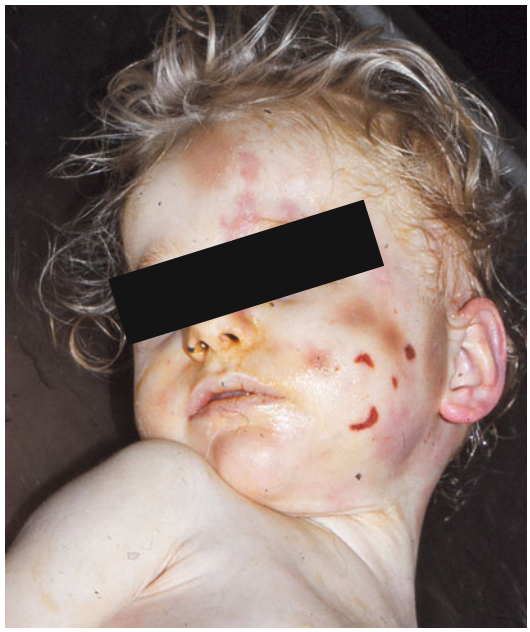

Fig. 20.15 Fatal beating in a 4-year-old girl showing numerous trauma-related hematomas (lethal extensive subdural hematoma)

(Fig. 20.15), or blunt force to the abdomen also resulting in lethal internal bleeding (see Chap. 18). Various forms of infanticide in the context of extended suicides are seen, such as drowning, shooting, hanging, fatal falls from significant heights, as well as various forms of asphyxiation.

Legislation on infanticide varies significantly. In England and Wales, the killing of a child by its mother comes under the Infanticide Act of 1938. However, in this case, protection of the child does not begin during—as in continental Europe—but rather after birth. In England and Wales, not only can causing the death of a neonate fall under the elements of infanticide but also causing the death of a child under the age of 1 year. The Infanticide Act of 1938 states:

> 1 (1) Where a woman by any wilful act or omission causes the death of her child being a child under the age of twelve months, but at a time of the act or omission the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child or by reason of the effect of lactation consequent upon the birth of the child, then, notwithstanding that the circumstances were such that but for this Act of offence would have amounted to murder, she shall be guilty of felony, to wit of infanticide and may for such offence be dealt with and punished as if she had been guilty of the offence of manslaughter of the child. (http://www.legislation.gov.uk/ukpga/Geo6/1-1/36/section/1)

Most countries make explicit provisions for considerably milder sentencing for mothers who commit neonaticide.

20.6 Suicide in Childhood

Although suicide in childhood (<14 years) is extremely rare, the possibility that a child may have been driven to suicide as a result of child (sexual) abuse should be considered in individual cases. Children who have been sexually abused are at a three- to fourfold greater risk of committing suicide in later life. Childhood suicides affect almost exclusively the 10–14-year-old age group, while suicide is a relatively common cause of death in 15–19-year-olds (teenager suicide). Suicide or attempted suicide in childhood is often seen against the background of impaired socialization and a troubled or conflict-rife family life. Adult caregivers (parents) are often mentally unstable themselves and thus unable to give a child the support he/she needs to deal with crises.

There have been reports in recent years of suicide in children prescribed with antidepressants belonging to the class of selective serotonin or serotonin–noradrenaline reuptake inhibitors (SSRI and SNRI) for the treatment of depression. These substances should be used with the utmost caution in children and never stopped abruptly.

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Traffic Medicine

21

Case Study

A 19-year-old man telephoned the police at 5.05 am on a Sunday morning to report that he had just been involved as a passenger in a motor vehicle in a road traffic accident and that the driver was seriously injured. On arrival, the police and rescue services encountered the following scene: a newish mid-class motor vehicle had left the highway to the right while negotiating a right-hand bend, the driver's door had struck a tree, and the motor vehicle had rolled over at least twice and come to rest in an upright position. The 19-year-old was kneeling beside the lifeless body of a second young man who, according to the pattern of tracks in the grass, had been dragged approximately 10 m in the direction of the highway. The second young man was the 18-year-old son of the motor vehicle owner. Unable to assist the young man, the rescue services pronounced him dead.

Although the 19-year-old showed virtually no signs of external injury, he was taken to the hospital as a precautionary measure. Once there, the police ordered a blood sample to be taken for forensic alcohol determination. This yielded a value of 0.69 %. The blood sample was also used for forensic toxicological analysis. The immunoassay was positive for cannabinoids. Using gas chromatography/mass spectroscopy, THC was measured to be 1.2 µg/l, the active metabolite 11-OH-THC 2.2 µg/l, and THC-COOH 72.8 µg/l.

A forensic autopsy was carried out on the body of the 192-cm 18-year-old the following day. Hyperextension injury to the cervical spine with damage to the cervical cord was found to be the cause of death. A blood alcohol concentration of 2.19 % was measured in femoral vein blood taken at autopsy. THC in cardiac blood was 1.4 µg/l, 11-OH-THC 2.3 µg/l, and THC-COOH 22.7 µg/l. On dissection of soft tissue on the anterior trunk, a mild band-shaped hemorrhage measuring up to 20 cm in width and extending from the right to left iliac wing and less distinctly from the left iliac wing to the right shoulder was remarkable; this lesion was interpreted as a safety belt mark.

Police inspection of the impounded motor vehicle revealed that the three-point safety belt was mounted at a typical point on the B pillar. An automotive expert found micro-tears in the driver and passenger safety belts, indicating that they had been worn during the accident. Also of note was the fact that the headrest on the passenger seat was in its lowest position.

The public prosecutor's office ordered a clinical forensic examination to be carried out on the 19-year-old man. The examination took

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place in hospital the following day, i.e., 2 days after the accident. Although no serious injury had been identified, the patient had remained in hospital for several days for observation. Examination of the body surface revealed extensive yet largely band-shaped bluish-purplish hematomas extending horizontally across the lower abdomen and transversely from the left shoulder to right flank. The forensic pathologist identified these as possible driver-side safety belt marks.

In the context of the overall examination results, the 19-year-old was questioned by the police as a suspect. He reported that he and his friend had gone together to a disco that Saturday night; the 18-year-old man had driven his mother's motor vehicle. While at the disco, both young men had drunk alcohol and smoked hashish, whereby the younger man had consumed significantly more alcohol than he. After leaving the disco, the 18-year-old had asked him whether he could drive, since he (the younger man) had had too much to drink. Feeling sober himself, he had agreed to this. His subsequent misjudgement of a curve had caused the accident. Immediately after the accident, he rescued the lifeless passenger from the motor vehicle, which he feared might explode—like one sees in the films. After moving him to a "safe distance" from the motor vehicle, he could detect neither breathing nor a pulse in his friend. It was at this point that it became clear to him that he could get into "real trouble"; for this reason, prior to telephoning the police, he decided to give his friend as the driver, particularly since the motor vehicle belonged to the friend.

Traffic medicine is an interdisciplinary field embracing medicine, psychology, accident research, and automobile construction. The goal of traffic medicine is to apply scientific medical knowledge for the purposes of increasing traffic safety. Traffic accident analysis involves recording injuries in order to increase the passive safety of persons involved in accidents. Investigating the level of human error involved in an accident yields insights into how active safety can be increased.

Performing autopsy examinations on traffic accident victims remains the principle basis on which to determine the type and extent of injuries, as well as the cause of death. Autopsies form an important part of accident reconstruction, which in turn can have criminal legal, civil legal, and insurance consequences. Moreover, the forensic medical examination of surviving accident victims can also make an important contribution to reconstructing the circumstances of an accident. Autopsy results or clinical examinations can be complemented by radiological diagnosis and/or imaging techniques.

The severest and most frequent traffic accidents involve passenger motor vehicles. Thus, the various accident constellations involving passenger motor vehicles are discussed here in detail. Although rarer, accidents involving buses or trucks are no less serious. Collisions between cyclists or with pedestrians can also have fatal outcomes.

Another important task of forensic medicine in a traffic medicine context is to assess fitness to drive (driving safety) in the case of mental or physical impairment due to foreign substances or physical defects, as well as to assess driving ability.

21.1 Traffic Accidents

The number of traffic accidents in Germany has risen steadily since statistical records first began after the Second World War, a phenomenon attributable to the continuous increase in the volume of traffic. Other countries report similar figures. Following an intermediary peak, the annual number of traffic deaths in Germany has been decreasing continuously since the 1990s, due in large part to active and passive safety concepts in automobile construction.

Definition: **A traffic accident is an injurious or fatal event caused primarily by blunt force impact occurring while participating in road, rail, sea, or air traffic.**

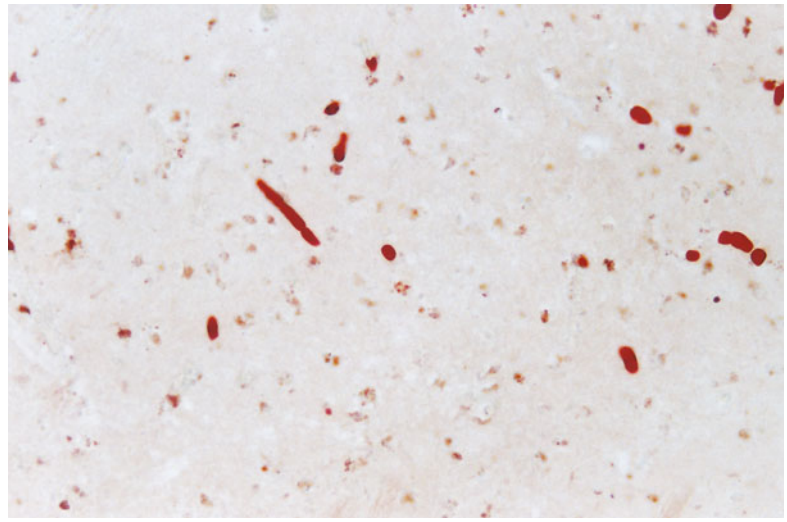

Fig. 21.1 Fat embolism in the brain: traffic accident involving polytrauma survived for approximately 9 h (Sudan III ×400) (Dettmeyer 2011)

Causes of Accidents. In the search for accident causes, a distinction is made between external and internal causes. External causes include traffic routing, highway and weather conditions, and motor vehicle conditions. The evaluation of external causes of accidents falls to the technical expert (automobile expert). Internal (or subjective) causes of an accident may be attributable to psychophysically impaired performance due to fatigue or foreign substance use, among others. Increased risk-taking behavior while driving or failing to apply a safety belt can also be due to foreign substance use or represent a manifestation of the premorbid personality.

Force of Impact. A collision that generates momentum forms the basis of the blunt force that impacts individuals involved in an accident. A velocity change (Δv) is created in the form of acceleration or deceleration; inelastic impact results in deformation and exceeding the elastic limit of material or tissue results in destruction and lesions.

Change in Velocity. Since Δv is the essential parameter in momentum transfer, determining collision speed takes high priority.

Causes of Death. Death following a traffic accident usually occurs as a direct result of blunt force impact. The term polytrauma is used in the clinical setting and refers to multiple injuries incurred simultaneously in various body regions, whereby at least one injury, or the combination of several injuries, is life-threatening.

Late deaths in which the accident represents an indirect cause of death are also of relevance in forensic medicine. Fat embolism, e.g., in the brain and kidneys (Figs. 21.1 and 21.2), may occur in the first few hours following bone fracture or soft tissue destruction. Pneumonia resulting from accident-related immobilization would also be considered an indirect cause of death. Pulmonary thromboembolism following venous thrombosis, e.g., due to accident-related damage to the venous network, can develop days or even years after the event.

21.1.1 Reconstructing Traffic Accidents

Technical and medical reconstructions of a traffic accident should be conducted hand in hand. Conclusions on the dynamics and kinetics of an accident are drawn from the pattern of damage seen on both man and machine, ultimately permitting the event to be reconstructed.

The Task of the Physician. Any physician dealing with accident victims should be familiar with findings relevant to accident reconstruction and be able to document these accurately. In the setting of trauma surgery, the argument that lifesaving measures take priority is often used to support the inadequate documentation of findings. Treating physicians frequently need to provide expert

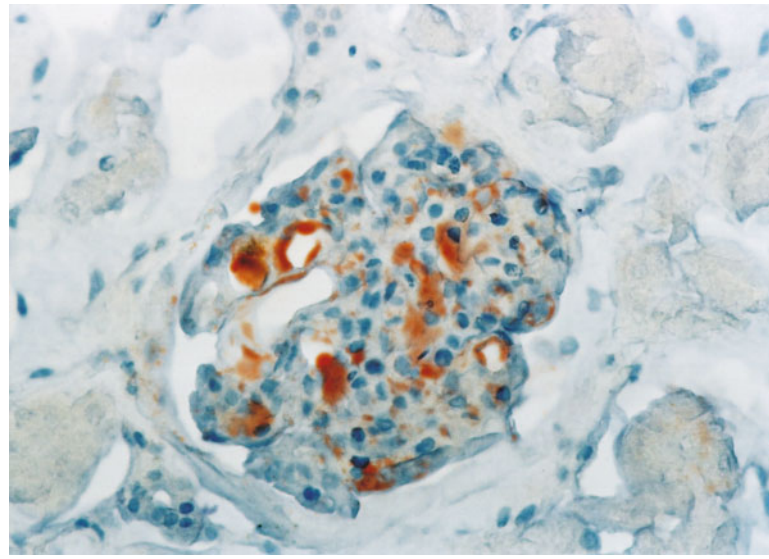

Fig. 21.2 Fat embolism in the glomerular loops of both kidneys: traffic accident involving polytrauma survived for approximately 9 h (Sudan III ×400) (Dettmeyer 2011)

testimony in court proceedings on whether a pattern of injuries is consistent with the particular circumstances of an accident. It is often impossible to make statements of this nature on the basis of the available documentation. In the majority of cases, a few digital photographs of findings on presentation would suffice to clarify contentious issues.

The Task of Forensic Medicine. The autopsy examination of an accident victim (forensic or insurance-medicine autopsy) begins with a highly detailed external examination involving a precise description of all skin lesions, their dimensions, and their distance from the soles of the feet. Layer-by-layer dissection is performed, starting at the corium–adipose tissue border. If necessary, the skin is dissected away from the entire body surface. In a final step, all bone fractures are visualized. In the course of the mandatory opening of all three body cavities, intracranial hemorrhage, brain injury, and injuries to the chest and abdominal organs are identified. The examination is usually documented by means of real-time dictation, complemented by photographic documentation of the individual stages of dissection or by drawings. Postmortem X-rays or whole-body CT scans obtained prior to autopsy may yield information relevant to autopsy planning and provide helpful documentation, particularly of bone injuries.

Examining or appraising living accident victims is another important task of forensic medicine. It was experience with precisely those critical cases that could not be resolved using conventional medical documentation that led to forensic medical expertise being called upon at an earlier stage. Thus, a forensic medical examination, including thorough documentation of all findings, is performed in the first few days following an accident (see Chap. 17). An expert appraisal can take all medical records and, in particular, available radiological data (see Chap. 24) into consideration. The most frequent lines of inquiry in an investigation relate to the position of the victim in the accident vehicle and the reconstruction of accidents involving pedestrians.

21.1.2 Isolated-Vehicle and Vehicle-to-Vehicle Accidents

Vehicle-to-vehicle accidents and vehicle collisions with other moving or static objects can be divided into the following collision types (frequency rates are intended as guidelines):

Frontal impact: 60 %, of which approximately 15 % have complete overlap, 15 % right side overlap, and 30–50 % left side overlap.

Side impact: 20 %.

Rear impact: 10 %, rollover and frontal underrun. Besides the type of collision, the pattern of injuries caused depends on various factors, such as passenger compartment response, belts, airbags, and properties of the material impacted by the body.

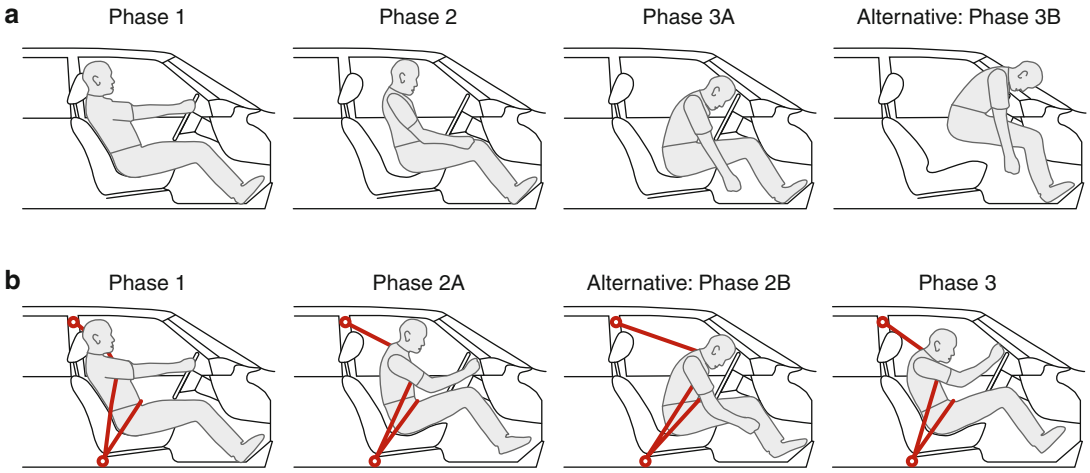

Fig. 21.3 Motor vehicle frontal impact: driver dynamics. (From Wehner (2007)). (**a**) Unrestrained (*top row*). *Phase 1*, bracing injuries are caused to the upper extremities; *Phase 2*, injuries are caused to lower extremities; *Phase 3A*, head impacts the steering wheel; and *Phase 3B*, head impacts the windshield. (**b**) Restrained (*bottom row*). *Phase 1* and Phase 2*A*, no injuries caused; *Phase 2B*, belt is not fastened sufficiently tightly, hence impact with the steering wheel; *Phase 3*, end position

Frontal Impact Collision. The vehicle occupants are accelerated in a forward direction (Fig. 21.3). Unless restrained, occupants will sustain blunt force trauma as they impact interior vehicle fittings.

Injuries to the Driver. Typical injuries to the motor vehicle driver in frontal impact accidents are shown in Table 21.1. To these can be added the formation of a safety belt mark, the shape of which may be able to indicate the position of the driver or passenger (Fig. 21.4), as well as impact injuries, such as knee injuries due to impacting the dashboard (Fig. 21.5).

Important: **Wearing a safety belt can prevent fatal injury**, **particularly at collision speeds of up to around 45 km/h**, **in isolated cases up to 100 km/h**.

Body-restraining safety belts and airbags form a central element of passive safety in modern motor vehicles. Failure to wear a safety belt, particularly at low collision speeds, can cause significant injuries as a result of airbag activation. Injuries caused in this way may be more severe compared with failure to wear a safety belt in a similar accident in an older model of car without airbags.

Injuries to Other Vehicle Occupants. It is common for the unrestrained front-seat passenger in particular to impact their head against the windshield. Other injuries are characterized by internal vehicle fittings and possibly also airbags. Injuries to internal organs caused in response to inertia generally demonstrate the same degree of severity in all vehicle occupants. In addition, other injuries to rear passengers depend on how loaded the vehicle is; skull and chest injuries are common, as are lower extremity fractures.

Side Impact Collisions. The material used to build the sides of motor vehicles has low deformation properties. In a collision where one motor vehicle impacts the side of another motor vehicle, the impacting vehicle penetrates deep into the passenger compartment, and the upper body's deceleration distance is extremely short. This often results in massive injury to internal organs. Moreover, the abrupt sideways movement of the head causes cervical spine injury. Fitting motor vehicles with airbag systems has brought significant advantages for vehicle occupants involved in side impact accidents (side airbags).

Rear Impact Collisions. Minor rear impact collisions are common, occurring primarily when a moving vehicle collides with a stationary vehicle at traffic lights, at the side of the road, or at the tail end of a traffic jam. Whiplash injury is the typical outcome here and is frequently the subject of civil litigation involving compensation claims.

Table 21.1 Typical injuries to motor vehicle drivers involved in frontal collisions

Injuries	Cause
Hematomas, abrasions, and crush/laceration injuries to the knee, patella fracture, femur and tibial fractures proximal to the knee	Knee impacts the dashboard
Hip dislocation, pelvic fractures	Femoral compression
Ankle and midfoot fractures	Reflexive slamming of the foot on the pedals or foot plate
Upper and lower arm fractures	Bracing the steering wheel
Sternum and rib fractures	Impacting the steering wheel; airbag activation
Cardiac and pulmonary contusions, pulmonary rupture, liver and spleen lacerations	Chest and abdominal compression. Direct and indirect forces (organ inertia)
Hemorrhage from the pulmonary hilum or mesentery	Intrathoracic and intra-abdominal whiplash movements
Crush/laceration wounds to the lower jaw and chin	Contact with the steering wheel
Fine cuts and abrasions to the face	Face impacting the windshield, shattering of the windshield
Cervical spine dislocation and fracture, particularly of the 6th and 7th cervical vertebral bodies	Headrest absent or incorrectly adjusted: body thrown backwards causing whiplash effect to the head and neck
Band-shaped areas of drying or hematoma extending horizontally across the lower abdomen and usually from the right lower abdomen to the left shoulder	Safety belt mark: blunt force impact due to the pressure of the belt and additional tangential movement of the skin

Severe Rear Impact Collision. This type of collision results in force being transferred to the trunk via the backrest. This subjects the head firstly to a shearing motion backwards involving forced reclination and secondly to flexion (whiplash phenomenon). Injuries include pelvic ring fractures, spinal injuries, and aortic rupture. Airbag systems are able to reduce injuries even in rear impact collisions, i.e., those caused in the secondary phase.

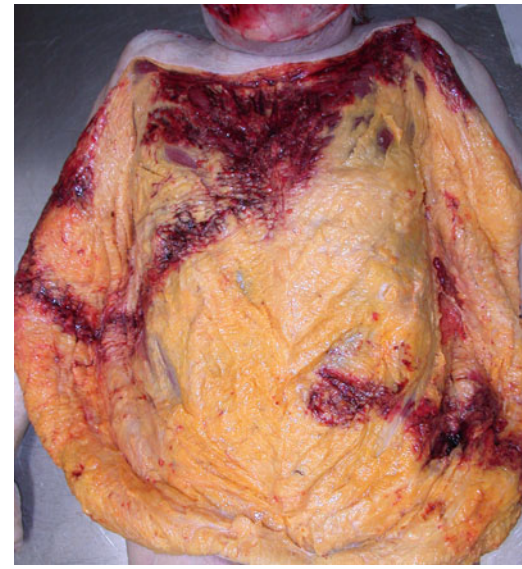

Fig. 21.4 On dissection of subcutaneous soft tissue, a safety belt mark in the form of a linear hemorrhage extending transversally from the upper left to lower right side and consistent with the driver's position is seen

Combined Collision Types. Only very few accidents can be classified as strictly and exclusively one of the abovementioned collision types. In addition to overlapping, slightly different collision directions can cause force vector deviation in all four directions. Thus, combinations of collision types and attendant injuries are often seen. Accidents involving multiple collisions serve to complicate matters further.

Reconstructing the Position of Vehicle Occupants. In the aftermath of motor vehicle accidents involving injuries or fatalities, forensic pathologists and technical experts are frequently tasked with reconstructing occupants' seating positions. The following injuries indicate that an individual was the driver of a vehicle involved in an accident:

- Safety belt marks extending, in most vehicles, from the left shoulder to the right flank
- Steering wheel injuries to the face
- Fractures to hands and lower arms caused by bracing
- Pedal injuries to the skin of the right ankle region and midfoot dislocation

Cervical Acceleration–Deceleration (CAD) Injury (Whiplash Injury). CAD injury is considered a posttraumatic condition. Victims

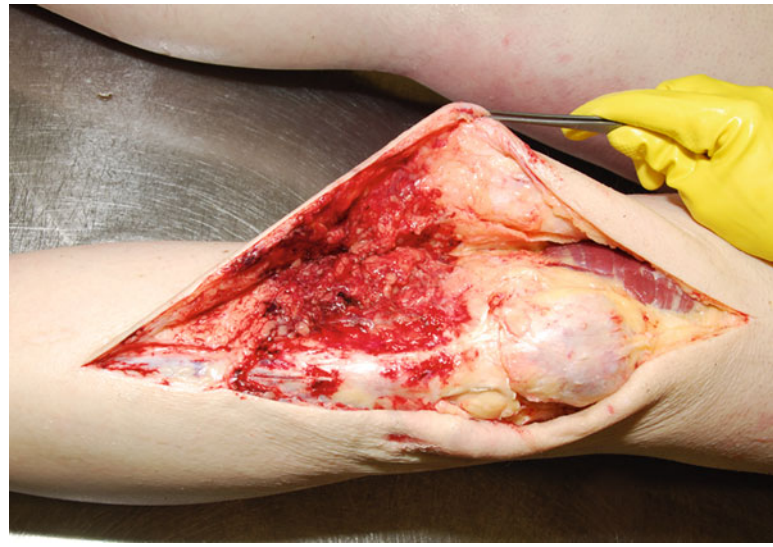

Fig. 21.5 The knee impacts the dashboard causing lower leg dislocation towards the femur

report headaches; shoulder, arm, and back pain; difficulty in swallowing; dizziness; as well as impaired sleep and concentration (Table 21.3). Clearly, these symptoms are not caused by hyperextension, but rather by the translational and rotational acceleration caused by rear impact and the secondary tilting of the head forwards (Fig. 21.6). Thus, traction, pressure, and shearing forces all come into play. Some authors set a minimum Δv of 10 km/h as a prerequisite for whiplash injury to occur, while others reject this rigid value, attributing instead greater importance to the absence of a brace position and reduced muscle tone. Morphological correlates are increasingly required to support diagnoses in civil litigation; these can be obtained almost only using imaging techniques:

- Mechanical impairment of the cervical nerve roots
- Damage to nerve endings in the neck muscles
- Tears or ruptures to the yellow ligaments, the anterior longitudinal ligament, or the capsular ligaments of the facet joints
- Injuries to the alar ligaments
- Hematomas (not caused by direct sequelae of trauma!)

In addition, attempts are being made to develop a severity classification for whiplash injury on the basis of reported symptoms (Table 21.2). According to a review of a number of studies, symptoms in the acute phase and chronic symptoms are reported with varying frequency (Table 21.3).

21.1.3 Pedestrian–Motor Vehicle Accidents

In physical terms, a motor vehicle colliding with a pedestrian represents a nonelastic impact with a highly unequal distribution of mass. In the majority of cases, the pedestrian is struck by the front of the vehicle; however, there are cases of motor vehicles, trucks, and buses striking pedestrians while reversing. From a forensic medical perspective, examining the injured or deceased pedestrian should answer the following questions:

- Direction of impact (from the front, rear, or side)
- Position at the time of impact (standing, lying, sitting, kneeling, walking)
- Number of cars that collided with the pedestrian
- Sequence of individual injuries and, where appropriate, involvement of multiple accident vehicles
- Capacity of a particular vehicle to cause all or some of the injuries identified
- Acute impairment experienced by the pedestrian at the time of the accident/death due to foreign substances (forensic toxicological analysis and blood alcohol determination) or,

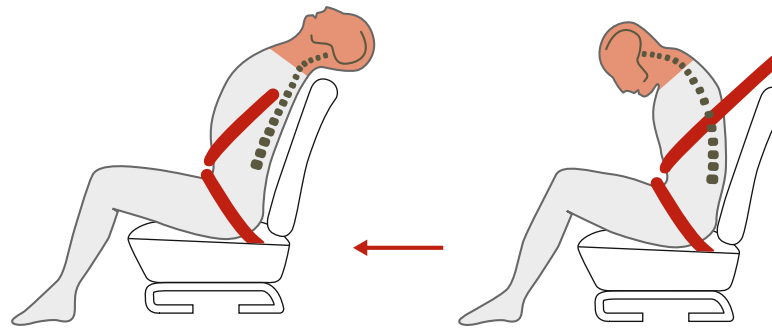

Fig. 21.6 Translational and rotational acceleration of the head and neck in cervical acceleration–deceleration injury (whiplash injury)

Table 21.2 Cervical acceleration–deceleration injury severity classification

Symptoms	Severity grade I	Severity grade II	Severity grade III
Almost pain-free interval	Common (12–16 h)	Rare (4–8 h)	Absent
Pain on swallowing, pain in the base of the mouth or rectus muscles of the neck	Rare (lasting 3–4 days)	Common (lasting 3–4 days)	?
Total postural insufficiency in muscles supporting the head	Absent	Absent as immediate phenomenon, later onset occasionally seen	Always present as an immediate phenomenon
"Stiff neck" or painful restriction of movement in the head and neck, palpable on manual inspection	Common, usually as a secondary symptom	Often present, usually as a primary phenomenon, becoming rarer after an interval	Always present, lasts longer than 2 months
Paravertebral pain between the shoulder blades	Occasional (in around 15 %)	Common (in around 30 %)	?
Primary paresthesia of the hands, occasionally also of the lower arms	Rare	More common, but usually without motor paralysis	?
Positive injury characteristics on cervical X-rays:			
1. Primary	Absent	Absent	Present
2. Secondary (after 3–6 weeks)	Absent	Occasionally present	Present
Prostration, confinement to bed	Often absent (usually only 2–3 days)	Usually present (around 10–14 days)	Always present (4–6 weeks)
Duration of accident-related incapacity to work	1–3 weeks (sometimes absent)	2–4 weeks	Over 6 weeks

According to Erdmann (1973)

e.g., cardiovascular disease leading to acute cerebral insult or myocardial infarction

An accident that involves a pedestrian being struck while walking or standing can be divided into three phases of impact: primary impact, secondary impacts, and tertiary impacts. It should be noted that the term secondary impact is sometimes used to describe contact with the ground rather than the second contact between the body and the car. Depending on the construction of the vehicle (e.g., truck with a straight, high front) or height (e.g., child) and position of the pedestrian (e.g., crouching), the secondary impact phase may not occur, and the pedestrian may be directly thrown to the ground. If the pedestrian is in a supine position after being thrown forwards, they may additionally be run over by the motor vehicle or its wheels (Fig. 21.7). Successive vehicles or their wheels may also run over the victim.

The Primary Impact Phase. Initial impact usually occurs at the level of the bumper, which, on a

Table 21.3 Frequency with which acute or chronic symptoms are reported following collision-related cervical acceleration–deceleration injury

Reported symptoms	Acute (%)	Chronic (%)
Headaches	48–86	33–86
Neck pain	66–100	21–96
Shoulder/arm pain	42–71	12–71
Back pain	39–43	33–43
Motor deficits	10–50	3
Sensory deficits/paralysis	15–100	15–45
Reflex deficits	13	0
Ocular symptoms/impaired vision	8–43	0–48
Ear symptoms/impaired hearing	0–38	14–52
Difficulty in swallowing	9–30	2–10
Nausea/vomiting	14	0
Dizziness	0–76	0–29
Impaired sleep	13–86	5–76
Fatigue	56–76	71–76
Irritability	21–33	18–43
Depression	43	12
Anxiety	44–67	6–67
Impaired concentration	26–62	29–57
Impaired memory	15–48	24–57

From Di Stefano (1999)

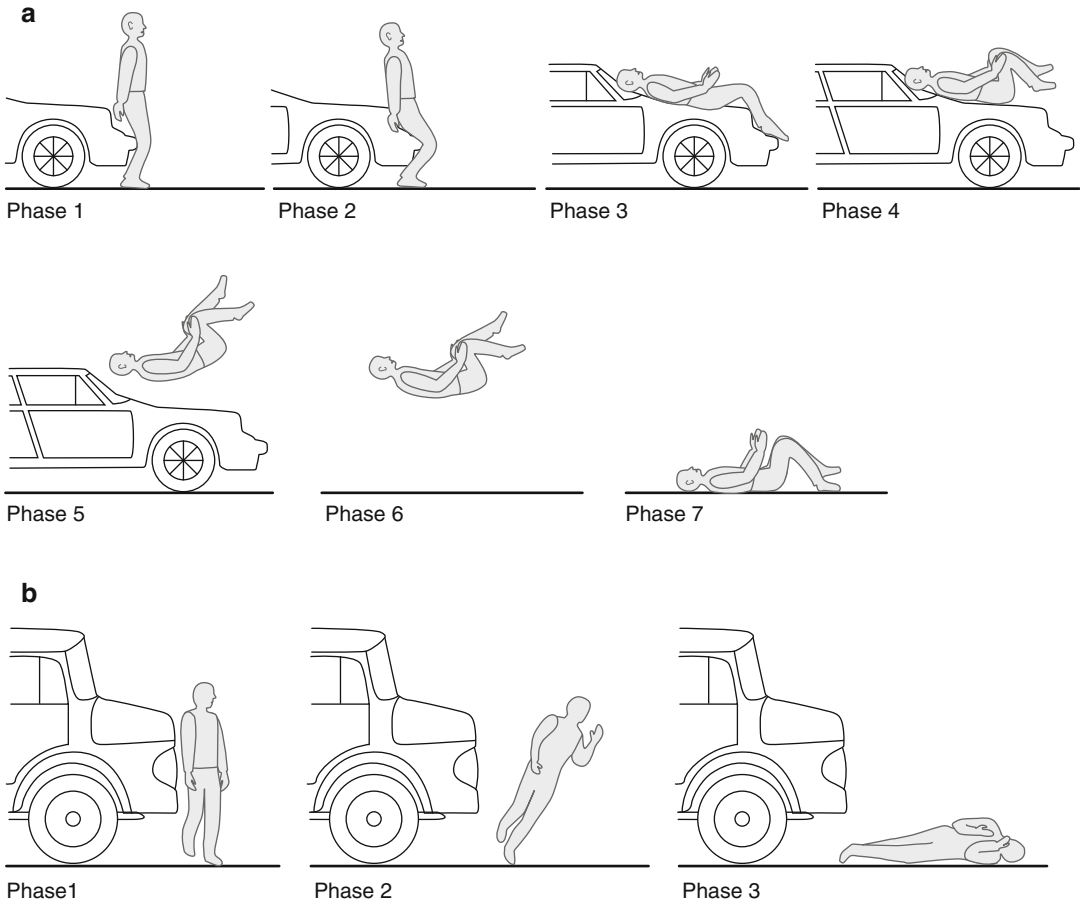

Fig. 21.7 Motor vehicle–pedestrian and truck–pedestrian collisions (From Wehner (2007)). (**a**) *Phases 1–7*: impact below the pedestrian's center of gravity. *Phase 1*, primary impact; *Phases 2–4*, secondary impacts; and *Phases 5–7*, tertiary impacts. (**b**) Impact above the pedestrian's center of gravity. *Phase 1*, impact; *Phases 2 and 3*, pedestrian is slammed down

motor vehicle, is level with the pedestrian's lower leg. However, the bumper height measured on a stationary vehicle is subject to variation: a braking maneuver causes the front of the vehicle to "dip," thereby lowering the height of the bumper. On the other hand, if the vehicle is accelerating at the moment of impact, its front will be raised, thereby shifting the point of impact upwards. The point of impact in a truck–pedestrian collision is higher, often in the femoral region. If a truck has a particularly flat front, a distinguishable point of primary impact may be absent.

Findings on the Injured/Deceased Individual. Tire marks or damage to fabric may be visible on clothing (trouser legs). Corresponding abrasions or areas of dry skin and hemorrhage in subcutaneous soft tissue and underlying muscles on the lower leg may be seen. If the force of impact is sufficient, a typical impact injury to bone occurs: a wedge-shaped fracture (Figs. 21.8 and 21.9), characterized by a triangular-shaped wedge whose apex points in the direction of force. This makes it possible to reconstruct the position of the pedestrian at the time of impact. In terms of a legal appraisal, it is relevant, for example, whether from the driver's view the pedestrian crossing the road was hit on the right side of the body (in right-hand traffic: the pedestrian has to travel a greater distance, meaning there is more likelihood they will be seen by the vehicle driver) or on the left side of the body (in right-hand traffic: the pedestrian may walk out unexpectedly from between parked cars). Entirely different circumstances may account for a pedestrian being struck from the front or from behind.

Assessing Shoe Soles. Particular importance is attached in the reconstruction of accidents to abrasions on shoe soles. Firstly, they indicate that the pedestrian was in an upright position at the time of collision. This position makes it possible to draw conclusions about the direction of impact. Abrasive damage to only one of the two shoes indicates that this shoe was worn on the weight-bearing leg; hence, a dynamic process (walking) was taking place. On the other hand, in the context of an even more dynamic process (running), shoe sole abrasions may be absent if both legs were off the ground at the time of collision.

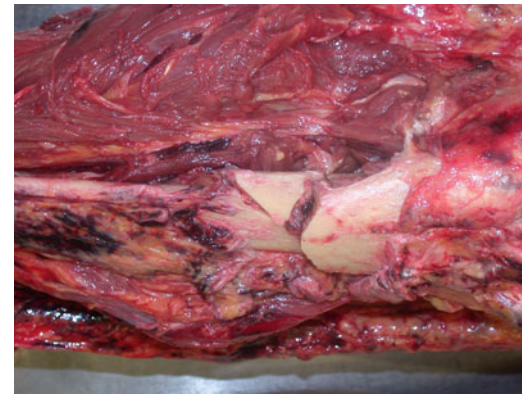

Fig. 21.8 Wedge fracture caused by a motor vehicle–pedestrian collision

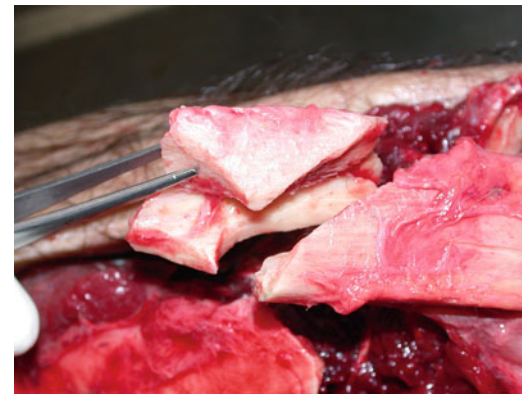

Fig. 21.9 A wedge fracture is exposed following a motor vehicle–pedestrian collision

The Secondary Impact Phase. This phase is characterized by the pedestrian's body rising up over the hood, rotating, and impacting and sliding over the hood, windshield, and A pillar. This causes injury to the pelvis and trunk in the first instance. Depending on the direction and intensity of impact, one sees textile abrasions, skin abrasions, blunt chest injuries (rib fractures, which possibly puncture the lung causing pneumothorax, and pulmonary or cardiac contusion), blunt abdominal trauma (organ contusion or rupture), and chest or lumbar spine fractures. Depending on the speed of the vehicle, the head may impact the upper or lower portion of the windshield or its upper frame, causing crush/laceration injuries, fractures to the facial bones or skull, and cerebral contusion. In addition, glass

fragment injuries occur if the windshield shatters during this phase (Fig. 21.10).

If a vehicle is travelling at speed, it is conceivable that the head will not impact the front of the vehicle. In such cases, the fact that the trunk is "caught" on the upper frame of the windshield can cause hyperextension or deceleration trauma to the cervical spine and head/neck joints, with attendant damage to the cervical cord. In extreme cases involving very high collision speeds, the pedestrian may "fly over" the vehicle following initial impact, in which case no secondary impacts occur.

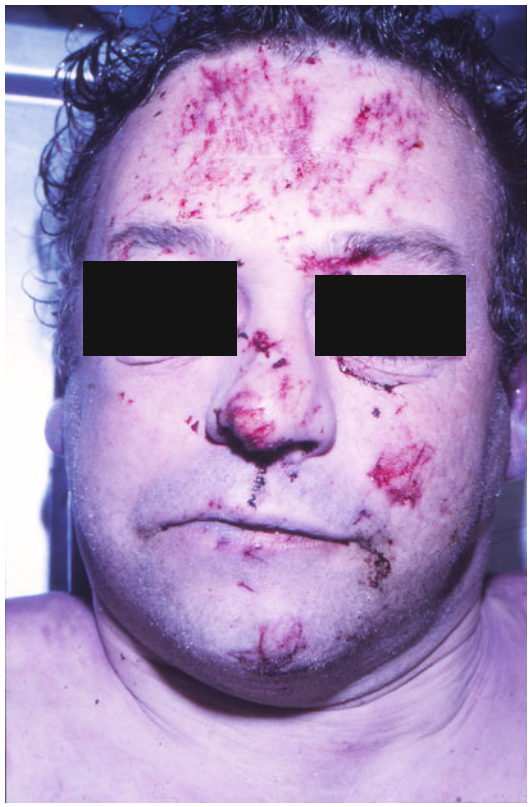

Fig. 21.10 Glass fragment injuries due to a shattered windshield

The Tertiary Impact Phase. As a result of the secondary impact phase, the body of the pedestrian is accelerated in the direction of travel. The vehicle's braking maneuver causes the pedestrian to be thrown in front of the vehicle, i.e., in its direction of travel. Depending on pedestrian dynamics, vehicle speed, and the point of impact on the vehicle, the pedestrian may be thrown slightly sidewards or thrown completely sidewards without being moved in the direction of the vehicle; this latter scenario, however, results in rotation and possible impact with side portions of the vehicle (A or B pillars; Fig. 21.11). The tertiary impact phase ends with the pedestrian landing and sliding on the ground. This can result in extensive skin abrasions and avulsion, additional facial injuries, serial rib fractures, as well as injury to the knees and backs of hands.

Pedestrians Run Over by Vehicles or Vehicle Wheels. The pedestrian who is already in a lying position, or who comes to lie following the tertiary impact phase, may be run over by the wheels

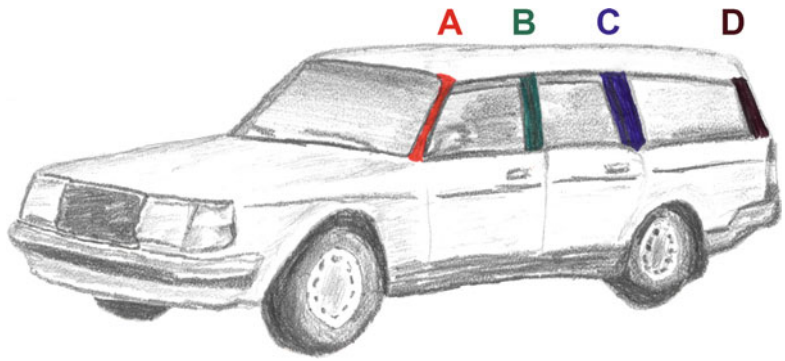

Fig. 21.11 In automobile construction, the *A pillar* connects the roof of the vehicle with the windshield and front of the vehicle. The *B pillar* connects the floor of the vehicle with its roof at the midpoint of the passenger compartment. The B pillar is often absent in coupés and cabriolets. The *C pillar* connects the roof of the vehicle with the fenders (rear side panels) at the tail end of the vehicle. The *D pillar* is the rear, fourth pillar on station wagons, vans, and small buses

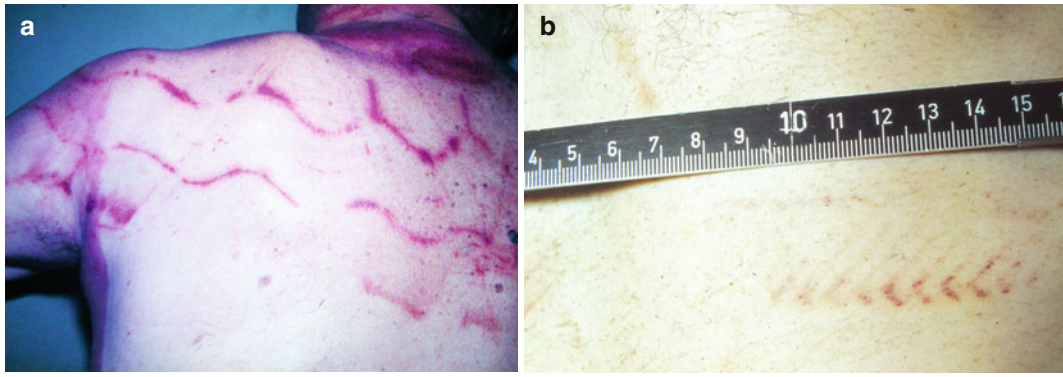

Fig. 21.12 (**a**) Tire tread mark in the skin produced when the victim was run over by the wheel of a truck (**b**) A partial tire tread mark after being run over by a wheel

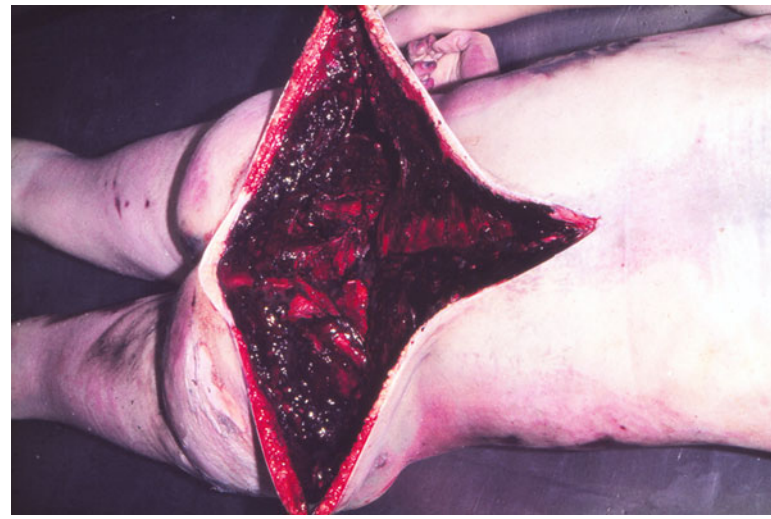

Fig. 21.13 Extensive décollement caused when the victim was run over by a vehicle wheel

of the vehicle (involving one or more wheels) or by the vehicle itself (no wheels involved). If the pedestrian is thrown forwards, they may additionally be run over by the primary colliding vehicle or its wheels. There is also the danger that the pedestrian may be run over by successive vehicles or their wheels.

Injuries. The clothing or skin of pedestrians run over by vehicle wheels typically show tire tread marks (Fig. 21.12). Stretch lacerations are seen on the skin, and on dissection, décollements are found: large wound cavities caused by the detachment of skin from subcutaneous soft tissue or detachment within subcutaneous soft tissue (Fig. 21.13).

Décollements are usually located on the side of the trunk or extremities struck by the wheel. Serial rib fractures as well as pelvic and spinal fractures are seen on the trunk, alongside other fractures to the extremities. The head is usually run over at the point of smallest diameter, i.e., in a transverse direction. Biparietal compression causes transverse fractures to the base of the skull and cranial vault. Stretch lacerations behind the ears are often visible externally (Fig. 21.14).

In order for a pedestrian to be run over by only the vehicle and not its wheels, they need to be lying in a longitudinal position or at a slight angle to the long axis of the vehicle, be of short stature (children), or the vehicle needs to be extremely wide (truck or bus). The injuries caused in this way depend on the height of the underbody and its components. Combinations whereby a pedestrian

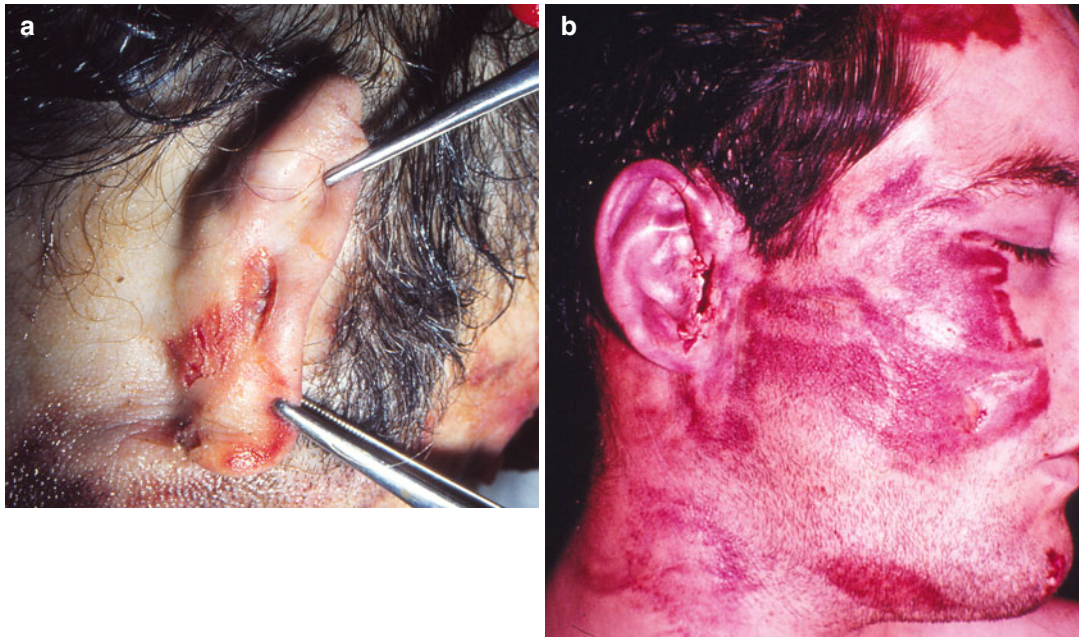

Fig. 21.14 (**a**) Stretch lacerations to the skin behind the ear. (**b**) Extensive abrasions to the face following biparietal compression caused when the victim was run over by the wheel of a vehicle

is run over by both a vehicle and its wheels are frequently seen.

"Dragging" is yet another phenomenon seen in traffic accidents: the victim that has been run over either by a vehicle or only its wheels is dragged along by the vehicle. In order for this to happen, the underbody must be sufficiently high and the victim possibly needs to be caught on underbody components. When this occurs, severe extensive abrasions as well as thermal injury to the skin are seen.

Children Run Over by Vehicles or Vehicle Wheels. A child that has been run over by a vehicle or its wheels may demonstrate no external signs of injury or fracture whatsoever. In such cases, one should work on the assumption that severe internal bleeding in the chest or abdominal cavities is present.

21.1.4 Two-Wheeled Vehicle–Motor Vehicle Accidents

Although collision mechanisms are similar for motorized and non-motorized two-wheeled vehicles, collision speeds can vary considerably. Frontal collisions between two-wheeled vehicles and motor vehicles are rare. Constellations in which the front of the motor vehicle strikes the side of a two-wheeled vehicle or a two-wheeled vehicle drives into the side of a motor vehicle are more common. Following impact, the rider of the two-wheeled vehicle is accelerated sideways, which, together with the preexisting frontal speed (own direction of travel), produces a rotational motion. Constellations such as these make the reconstruction of two-wheeled vehicle–motor vehicle accidents highly complex.

Frontal Collision Between a Motor Vehicle and the Side of a Two-Wheeled Vehicle. The rider of the two-wheeled vehicle is thrown onto the hood and possibly also the windshield of the motor vehicle. The body's first point of impact is usually the pelvic region against the frame of the windshield, resulting in pelvic, hip, femoral neck, and femoral shaft fractures. As the body is flung across the vehicle, injury to chest and abdominal organs may be caused, as well as pelvic and spinal fractures. Depending on whether or not the rider is wearing a helmet, the head impacting the

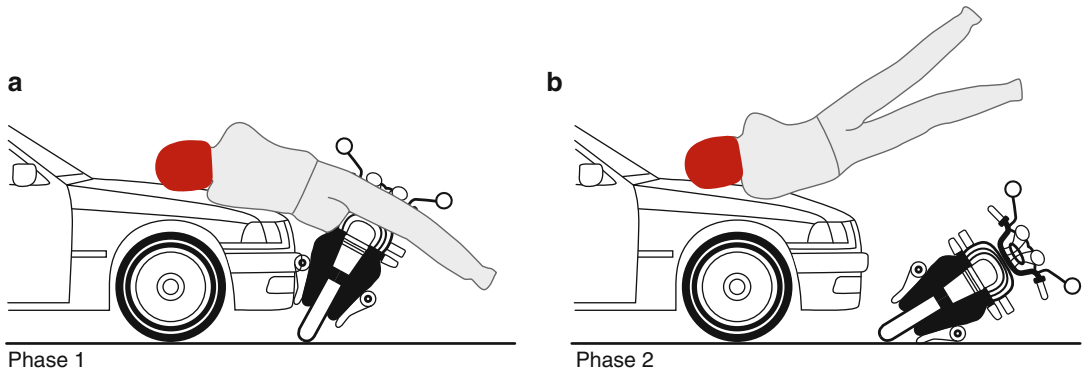

Fig. 21.15 (**a**, **b**) *Phase 1* and *Phase 2* of a side collision between a motorcycle and the front of a motor vehicle (From Wehner (2007)

vehicle is likely to cause severe craniocerebral trauma. As the body is flung away from the vehicle, typical injuries similar to those seen in pedestrians ensue (Fig. 21.15).

Frontal Collision Between a Two-Wheeled Vehicle and the Side of a Motor Vehicle. When a two-wheeled vehicle impacts the side of the passenger compartment, the primary point of collision is between the head or upper body and the roof frame. The rider's knees usually strike the doors or side windows. Typical sequelae of blunt force impact are seen at these sites. Metacarpal fractures caused by attempts to brace the handlebars are occasionally observed. Secondary fall injuries occur once the motor vehicle has passed and the rider lands opposite the site of impact.

The event becomes even more complex and diverse in the case of a side collision at the level of the hood. Depending on the proportions of the two vehicles, their speeds, and the precise point of impact, it is possible that the rider will not come into contact with the motor vehicle at all, or only with his lower leg, sustaining mainly fall injuries caused by landing on the ground after the hood has passed. If the motor vehicle is travelling at speed and the point of impact is located close to the passenger compartment, the rider's side may impact the windshield and its frame. This can result in direct and indirect injury to the head (Fig. 21.16a–d).

The Question of Helmet Use in the Expert Appraisal. A question frequently asked in the aftermath of accidents involving motorcyclists is whether a helmet was worn and, if so, whether it was worn correctly. In principle, the absence of craniocerebral trauma suggests that a helmet was worn. Skin abrasions and hematomas to the anterior and lateral neck and base of the mouth caused by the chinstrap may offer further evidence that a helmet was worn and fastened correctly.

Besides traffic accidents involving the most commonly used vehicles, i.e., passenger motor vehicles, trucks, and two-wheeled vehicles (including bicycles), injuries are also seen in the context of accidents involving other means of transport, e.g., rail vehicles, boots, ships, ferries, aircraft, helicopters, gondolas, cable cars, chairlifts, paragliders, hang gliders, gas and hot-air balloons, as well as means of transport used in motorized sports and recreational sports. The reader is referred to the relevant literature for a more detailed discussion of the injuries seen in these contexts.

21.2 Fitness to Drive and Suitability to Drive

An individual's ability to drive safely can be impaired by: alcohol consumption, the use of drugs or medication, fatigue, mental illness, and physical disease. A conceptual distinction is made between:

Suitability to drive: An individual's sustained ability to drive a vehicle in traffic safely irrespective of current situation parameters

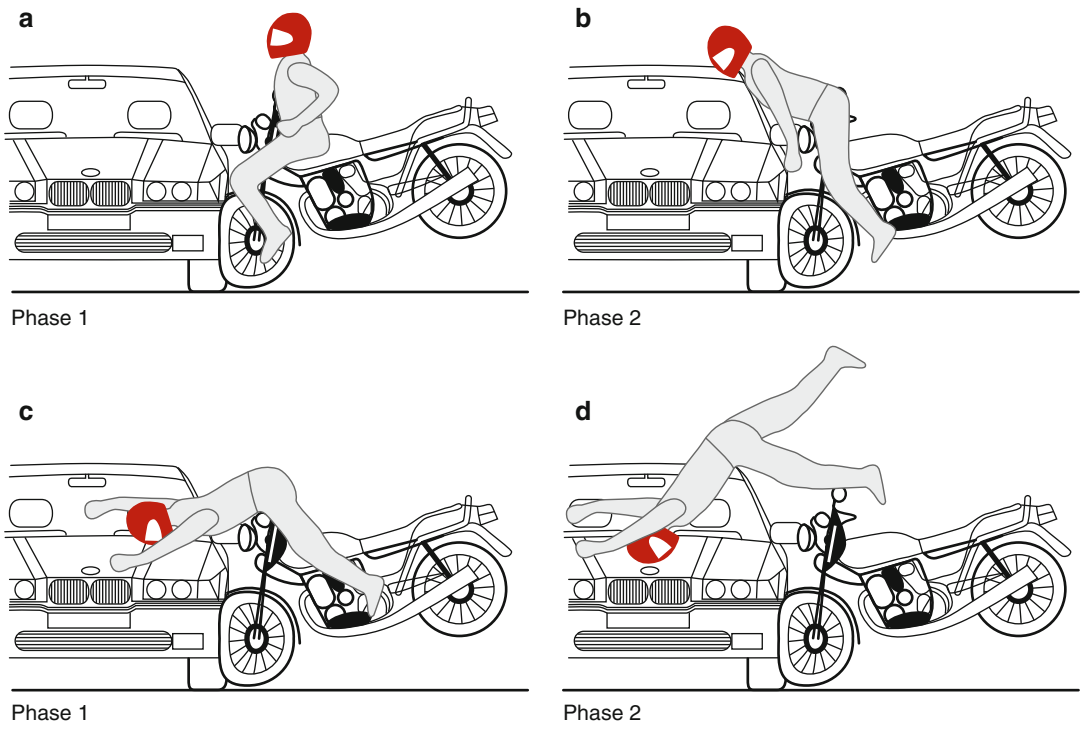

Fig. 21.16 Frontal collision between a two-wheeled vehicle and the side of a motor vehicle (From Wehner (2007)). (**a**, **b**) Collision at the level of the passenger compartment. (**c**, **d**) Collision at the level of the hood

Fitness to drive: An individual's effective and actual fitness to drive a vehicle safely at a particular time and in a particular situation

An absence of the capacity to drive a vehicle safely equates to either "unfitness to drive" or "unsuitability to drive." While fitness to drive relates to an individual's ability to control a vehicle safely at a given moment in time, suitability to drive refers to an individual's sustained and longer-term capacity to drive a vehicle safely. Fitness to drive can alter rapidly and may be impaired by external factors (e.g., foreign substances or fatigue); thus, it is reversible. Suitability to drive is more temporally stable and is contingent on personality traits and in some cases chronic disease.

In a given situation, unsuitability to drive would induce unfitness to drive or at least make it highly likely.

The term "unsafe to drive" describes a vehicle driver whose overall driving performance is impaired—particularly as a result of disinhibition or mental and physical (psychophysical) performance deficits—to the extent that he is no longer capable of safely controlling a vehicle in traffic over a long distance or in the event of challenging traffic situations suddenly arising. Table 21.4 lists the groups of medications most commonly referred to in association with potential unfitness to drive. The effects of alcohol, drugs, and medication are discussed in greater detail in Chaps. 29 and 30.

Table 21.4 Commonly prescribed drugs with the potential to impair fitness to drive

Psychotropic drugs	Insulin
Sulfonylureas	Analgesics
Antihypertensives	Muscle relaxants
Tranquilizers	Antiepileptic drugs
Antihistamines	Sleep-inducing drugs
Anesthetics	Local anesthetics

Fatigue-Induced Unfitness to Drive. Not only does fatigue represent the most common non-foreign-substance-related cause of unfitness

Table 21.5 Early and late symptoms of fatigue

Early symptoms	Late symptoms
Eyelids become heavy	Sensation of driving too fast
Convergence insufficiency	Fantasies
Double vision	Desire to sleep
Ocular foreign body sensation	Sudden loss of tone in the neck muscles
Dry mouth	Being suddenly startled (accompanied by sweating) by alternations in the driving situation
Frequent yawning	Being suddenly startled (accompanied by heart palpitations) by alternations in the driving situation
Feeling cold or warm	Sudden transitory absences
Impaired sensory perception when changing gear or engaging the clutch	Sudden transitory absences with eyes open, followed by being startled

to drive, it can also serve to compound the effects of foreign substance-related impairment. It is generally assumed that a driver is able to recognize the signs of fatigue at an early stage. A distinction is made between early and late symptoms of fatigue (Table 21.5).

21.2.1 Unsuitability to Drive Due to Disease

Guidelines issued by the German Federal Department of Transportation for the appraisal of fitness to drive factor in physical and mental deficits that often significantly impair or even negate an individual's suitability to drive. Table 21.6 summarizes the most relevant diseases.

A detailed patient history and diagnostic work-up is sometimes necessary to assess suitability to drive. Patients with addictive disorders, epilepsy, type 1 or 2 diabetes mellitus, and syncope of unknown etiology are relatively frequently affected. In the presence of a disease that can affect suitability to drive, the question of whether or not a patient can be successfully treated such that (resumed) participation in traffic is possible is of particular relevance in their assessment. Medication capable of affecting fitness or suitability to drive plays a significant role in this context. This is particularly true for psychotropic or neuroleptic drugs that may cause greater impairment at the start of therapy. Particular attention needs to be paid to patients with addictive disorders due to the significant impairment seen on use of, or withdrawal from, addictive substances; on the other hand, opioid substitutes such as methadone sometimes result in a restoration of suitability to drive. This also applies to patients with other diseases that can be stabilized with appropriate medication, such as epilepsy, thus resulting in fitness to drive. From a medical point of view, due compliance is expected of the patient, and regular monitoring, e.g., to measure blood medication levels, may be warranted. If, as part of outpatient treatment, a patient is sedated (e.g., using midazolam) to the extent that their ability to participate in traffic is significantly impaired for an extended period of time, the physician is obliged not only to inform the patient of this but also to ensure that the patient does not leave the premises prematurely. The issue of suitability to drive in the elderly is increasingly a topic of discussion; again, the reader is referred to the relevant literature for a more in-depth discussion of this particular issue.

21.2.2 Unsuitability to Drive Due to Character Deficits

Cases of serious misconduct in traffic (e.g., overtaking at 100 km in a built-up area) arouse suspicion of disinhibition and increased risk-taking behavior due to alcohol or other foreign substances. If no ethanol or other substance is detected in blood taken shortly after the incident, disease should be considered. Where this latter possibility can also be excluded, it is assumed that the individual possesses fundamental character deficits that appear to render them unsuitable to drive a vehicle, a suspicion heightened in particular by repeat offenses.

Table 21.6 Diseases and disease groups affecting suitability to drive

Group	Disease	Effect/comments
Visual	Visual impairments	Insofar as these cannot be compensated using visual aids
Auditory/inner ear	Impaired hearing	Only in the presence of other sensory organ impairments or intellectual deficits
Motor disabilities	Various	Basic motor skills required for driving a vehicle are absent/deficits cannot be compensated using relevant aids (e.g., steering aid, manual pedals)
Cardiovascular diseases	Cardiac arrhythmias	Risk of sudden loss of consciousness
	Hypertension	Risk of sudden heart failure or cerebral insult, retinal hemorrhage and impaired vision, kidney damage
	Hypotension	Rapid fatigue, risk of seizure-like loss of consciousness
	CHD	Risk of myocardial infarction, arrhythmias, angina pectoris, sudden cardiac death
	Cardiac insufficiency	Risk of collapse
	Peripheral AOD	Loss of control and strength in arms or legs
	Circulation-related impairments to brain activity	Transitory ischemic attacks (TIA), loss of performance due to microangiopathies; risk of stroke recurrence
Metabolic diseases	Diabetes mellitus	Risk of unstable metabolic states, greater susceptibility to exhaustion, generalized slowing down, impaired vigilance; late complications: retinal damage, diabetic neuropathy, kidney damage
Chronic renal diseases	Various	In particular, dialysis treatment: reduced performance and ability to react, unstable metabolic state, risk of electrolyte imbalance, heart failure, impaired vigilance and vision
Status post organ transplantation	Various	Drug effects, functional disorders, pyschoactive impairment
Lungs and respiration	Severe chronic obstructive pulmonary disease (COPD)	Circulatory effects associated with sudden syncope
	Sleep apnea syndrome	Daytime fatigue, microsleep episodes
Nervous system	Status post spinal cord injury	Subject to the severity of motor and sensory deficits
	Neuromuscular periphery	Sudden-onset inability to act due to periodic paralysis, impaired performance due to myatrophy
	Parkinson's disease, pyramidal and cerebellar disorders	Generalized slowing down, disintegration of motor, organic psychosyndromes
	Status post brain injury or surgery, congenital brain damage, or brain damage acquired in early childhood	Risk of organic brain psychosyndromes, seizures, personality changes
	Epilepsy	Sudden changes in vigilance
Mental disorders/psychiatric diseases	Organic brain disorders	Sudden loss of consciousness, failure to grasp reality
	Dementia	Generalized slowing down, impaired memory, and other cognitive functions
	Affective disorders	Depression, generalized slowing down, lack of drive, mania, impaired ability to adjust to novel situations
	Psychoses	Impaired sense of reality, generally impaired concentration and performance
Alcohol	Abuse	Impaired reactions, mood changes
	Dependence	Additional psychomotor impairments
Narcotic substances and medications	Dependence (addiction) and acute intoxication	Severe physical and mental damage accompanied by self-overestimation, apathy, irritability, dedifferentiation, and loss of personality
	Long-term drug therapy	Risk of generalized slowing down and impaired concentration, cardiac arrhythmias, hemorrhage, dizziness, states of collapse

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Forensic DNA Analysis

22

Case Study

A maid went to the police in a distraught state claiming that she had been raped by one of her private employers in his home 6 days previously. She had not returned to the residence since then and was too afraid to keep her regular appointment the following day. Once her complaint had been recorded, the decision was taken not to perform a gynecological examination or take a smear sample due to the comparatively long 6-day interval. The police accompanied the victim to the home at which the incident was alleged to have taken place. The occupant they met there denied all charges. The residence appeared untidy. The victim indicated the coffee table in the living room, where crumpled paper towels lay, and stated: "He wiped his penis with those afterwards."

The paper towels were collected as evidence, and saliva samples were taken from the suspect and the victim. On forensic examination at a forensic institute, irregular light-yellow shiny smears, which were apparent macroscopically on the white paper towels, reflected UV light. One sample tested positive for acid phosphatase. Microscopic examination of a Baecchi-stained smear preparation showed abundant intact sperm and sperm heads. Forensic DNA analysis (15 STR loci and amelogenin) yielded a mixed stain, wholly attributable to the suspect and the victim as proportional stain donors. Following differential lysis, the complete STR profile of the suspect could be seen in the male fraction.

In the light of these examination results, the suspect claimed via his legal representative that he had used the paper towels to masturbate and then left them on the table. On the day of the alleged offense, the maid had demanded more money; when he refused, she had cried and raised her voice. During this time, she had blown her nose in the paper towels lying on the table. In order to calm her, the suspect had then touched her shoulders, upon which she cried: "You touched me you pig, now I'm going to the police" and left the residence.

Against the background of this statement, a renewed forensic examination was called for. To this end, a total of nine smear preparations from varying points on the paper towels were analyzed. Sperm was detected in all nine samples. Moreover, predominantly large intact epithelial cells were found and only scant cell debris. This analysis was complemented by further STR analysis of all nine points on the towels, all of which showed the same mixed stain as initial tests. The forensic report stated that this constellation of findings contradicted the suspect's statement, since

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the large intact epithelial cells could be classified more as vaginal epithelial cells, whereas cell debris would have been predominant in the case of nasal mucus. At trial, the defendant then alleged the incident to have been an instance of consensual sexual intercourse. The court found this plea unconvincing and sentenced the defendant.

Classic haemogenetics involving the analysis of erythrocyte membrane antigens as well as ABO and Rhesus blood group testing no longer plays an important role in modern forensic practice. Thus a brief review is sufficient for the purpose of this chapter. In 1901, Karl Landsteiner (1868–1943) discovered the ABO blood group system. Thanks to Paul Uhlenhuth (1870–1957), it also became possible to differentiate human from animal blood (species-specific protein differentiation). Together, these methods laid the foundations for techniques of attributing biological evidence to a suspect as well as for paternity testing. Other blood group systems of erythrocyte membranes were later discovered, as well as other plasma protein polymorphisms. Attribute systems needed to show:

- A polymorphism
- High genetic stability (i.e., low mutation rates)
- Reproducible detectability
- Determinable hereditary transmission

It was possible to apply the well-known Mendelian laws of inheritance:

- Law of uniformity
- Law of segregation
- Law of independent assortment

In this regard, it was (and still is) also important to bear in mind that forensically relevant genetic traits remain constant for a lifetime.

Erythrocyte membrane antigens were sufficient for analysis purposes, in particular the ABO blood group system, which enables a distinction between the four blood groups A, B, AB, and O depending on agglutination test results. Establishing paternity by means of ABO blood-typing, however, did not become a legally accepted method until 1930.

Isoagglutinins, i.e., antibodies present in serum against nongenetically fixed or against erythrocyte antigens not present in the individual, were also used. Several subgroups were identified within blood group A, and these can also be used where necessary. Since the ABO blood-typing system follows a dominant/recessive inheritance pattern, it is possible to make diagnostic exclusions in paternity testing, possibly also when identifying trace evidence. ABO blood types are detectable not only in erythrocyte membranes but also in most organs and body fluids (saliva, sperm, and urine). Individuals demonstrating ABO blood types in secretions are referred to as secretors, those who do not as nonsecretors. Secretor status is also genetically fixed and inherited according to Mendelian laws.

The Rhesus system represented a further identifying attribute: by injecting blood from Rhesus monkeys, Landsteiner and Wiener were able to produce an immune serum that agglutinated the erythrocytes of approximately 85 % of humans (Rhesus-positive), while the other 15 % were Rhesus-negative. The identification of other structural antigens followed. These were named C, D, and E according to the Fisher nomenclature commonly used today as an extension of the ABO system, together with their allele pairs C/c, D/d, and E/e, whereby d, rather than being directly detectable, can only be detected by the absence of D. D is the classic Rhesus trait. Today, the Rhesus system is of greater relevance clinically than forensically. For example, in the case of transfusion incidents or when a Rhesus-negative mother becomes immunized by a Rhesus-positive embryo or fetus—the antibodies produced can cause intrauterine fetal death (hemolytic disease of the newborn) in a subsequent pregnancy. Other significant milestones in the field of serology included the discovery of further blood groups (MNSs, Kell, and Duffy, among others), serum groups (Hp, Gm, Gc, Tf, Pi, and complement factors, among others), enzyme groups (SEP, PGM, AK, ADA, GPT, EsD, GLO), and HLA systems.

22.1 DNA Analysis

The practices of investigating erythrocyte membrane blood group systems and analyzing plasma protein polymorphisms, polymorphisms of intracellular isoenzymes, and HLA antigens commonly performed in the past to classify biological trace evidence for the purposes of identification or to establish paternity have largely been abandoned in favor of more individualized investigations using DNA analysis. The use of repetitive sequences unique to the individual (minisatellite DNA) was pioneered by Alec Jeffreys in 1985. Today, *polymerase chain reaction* (PCR) forms an integral part of DNA analysis. It enables a theoretically unlimited number of amplifications of specific DNA sequences to be produced. The primer pair used determines the boundaries of the segment to be amplified.

The remarkable thing about forensic DNA analysis is that no coding sequences of human DNA, i.e., no genes, are investigated. Instead, hypervariable regions (HV) of DNA that show the greatest interindividual differences and yet are subject to a certain regularity are the focus of interest. Another unique characteristic of forensic DNA analysis relates to sample quality and quantity. In the case of genetic investigation (or parentage testing), well-preserved specimen materials (blood or saliva samples) are usually available, whereas in the investigation of biological traces and the identification of deceased persons, the following problems often arise:

- Scant (critical) quantities of DNA
- Poorly preserved DNA
- PCR-inhibitory substances

Problems such as these are encountered in forensic practice due to the increasingly elaborate methods of DNA extraction, purification, and amplification available today. Thus, it is quite possible that, initially, biological trace evidence collected in connection with a crime cannot be analyzed successfully, whereas subsequent analysis many years later with improved or modified methods may yield positive results.

In the case of poorly preserved DNA, attempts are made to achieve successful duplications by shortening the primer and segments to be amplified. If only scant DNA is available, the number of PCR cycles can be increased. However, this is possible only to a limited extent, since the risk of artifacts or contamination rises exponentially as the number of cycles increases.

Contamination Control. Protecting against contamination is an important aspect of forensic DNA analysis. This leads to yet another essential aspect: in conventional analysis, for example, of genes, only the presence or absence of particular gene variants in hetero- or homozygosity is registered. There is no way of checking whether the (successfully) analyzed DNA in actual fact originates from the cells (supposedly) investigated or originates instead from a member of the laboratory personnel (contamination). Since the explicit goal of forensic DNA analysis is to link an individual to a sample (DNA typing), the results of any DNA analysis need to be compared with DNA profiles of personnel or other persons involved in the collection of biological trace evidence (contamination control).

Methods. The investigation of autosomal *short tandem repeats* (STRs) is at the center of modern forensic DNA analysis. In special cases, gonosomal STRs (X-STRs and Y-STRs) are investigated. For poorly preserved specimens, *single-nucleotide polymorphisms* (SNPs) are available. *Deletion/insertion polymorphisms* (DIPs or indels) represent an intermediate type between STRs and SNPs. Mitochondrial DNA (mtDNA) hypervariable region sequencing has become established as a further option in the case of scant volumes of DNA or poorly preserved cellular material.

22.1.1 STR Analysis

This method involves the analysis of short repeating sequences of DNA made up of identical short base-pair sequences, each of between two and seven base pairs. Tetrameric STRs, i.e., those made up of four base pairs, are most commonly used in forensic DNA analysis. Each chromosome has several STR regions, known as STR loci. To ensure independent inheritance, those

Table 22.1 STR loci in the German Federal Office of Criminal Investigation database

Locus	SE33	D21S11	VWA	TH01	FIBRA	D3S1358	D8S1179	D18S51
Chromosome	6	21	12	11	4	3	8	18
Major alleles	8–37	24–38	11–24	3–13.3	17–51.2	12–19	8–19	7–27
Major repeat motif	AAAG	TCTA (+TCTG)	TCTA (+TCTG)	AATG	CTTT	TCTA (+TCTG)	TCTA (+TCTG)	AGAA
Example	18/19	31.2/32.2	17/19	6/9.3	19/23	17/18	14/14	15/15

Initially, only the first five loci were incorporated in the database, with D3S1358, D8S1179, and D18S51 being added in 2002. Each locus is given an actual allele combination in the bottom line; together these constitute an STR profile. D8S1179 and D18S51 are homozygous, while D21S11 and TH01 have intermediate alleles

STR loci located on different chromosomes are preferentially selected. The actual selection is generally based on criteria of practicability and convention. In addition, the locus should have sufficient variability and be easily combinable with all amplified and analyzed STR loci in a multiplex kit. To date, the DNA database of the German Federal Office of Criminal Investigation uses eight STR loci as standard in Germany (Table 22.1). However, depending on the information sought, other STRs can be investigated.

As part of European standardization, the legal groundwork was laid in 2009 for the eight loci used in Germany to be complemented by five new loci as well as three further optional loci already used in other EU countries. This move forms the basis for the creation of a pan-European DNA database.

22.1.2 DNA Databases

Often incorrectly referred to as a "gene database," the DNA database of the German Federal Office of Criminal Investigation is a collection of STR profiles of individuals found guilty of serious offenses. In addition, the database stores the STR profiles from a wide variety of crime scenes. The profiles are regularly compared with one another. When two profiles match, the program gives a "match" notification. With the help of this information, it has already been possible to successfully convict numerous perpetrators. However, even a match between two crime scenes—without a match to a person-specific profile—can be helpful: establishing a link between two crime scenes in different areas may provide new avenues of investigation.

Technical Background. In designing the primer used for the various STR loci, it is important to ensure that the primer-binding sites flank the repeat structure and show as little variability as possible. Modern multiplex kits, for example, can analyze 16 STR loci simultaneously. This can be achieved by marking the primers with four different stains, such that the STR loci with different stain combinations have different lengths, allowing their amplification products to be easily distinguished from one another. Amplicon detection is performed using methods such as capillary electrophoresis and a laser detection system capable of distinguishing between color channels.

The two regions flanking the repeating segment of the amplicons have a constant length. Therefore, a particular amplicon length delivers an exact value for the number of repeats in the STR (Table 22.2). For the tetrameric STR loci, the possible segment lengths vary by multiples of four base pairs. The length is determined by comparison with a so-called allelic ladder. Autosomal STR loci are found on both corresponding chromosomes. In the majority of cases, the number of repeats on the two chromosomes differs (heterozygosity), such that for one person two alleles are detected for each STR locus. However, it is possible that only one allele occurs at the investigated locus, in which case one speaks of homozygosity, i.e., the number of repeats on both chromosomes is (coincidentally) identical. Sometimes, three alleles can be present in the same person at one STR locus, as in the case of trisomy, for example, D21S11, located on chromosome 21, is the locus most commonly affected (trisomy 21). There may also be technical reasons for a third allele, as in the so-called triallelic pattern.

Table 22.2 The TH01 (TC11) STR locus: possible alleles, their amplicon length, and percentage distribution, based here, for example, on a central European population (*n*, 7373)

Amplicon length (bp)	Allele	Percentage (%)
154	5	0.27
158	6	22.34
162	7	16.04
166	8	11.31
169	8.3	<0.01
170	9	16.55
173	9.3	31.74
174	10	1.72
177	10.3	<0.01
178	11	<0.01
182	12	<0.01

The TH01 (TC11) locus provides an excellent example of the possible alleles and their distribution (Table 22.2).

Mixed Stains. Three or more alleles occurring at multiple STR loci investigated are an indication that DNA from more than one individual has been analyzed. In biological trace evidence analysis, these individuals are referred to as "stain donors" and the result a "mixed stain." A possible scenario could be that a knife, for example, was used by several individuals and that trace material collected from the handle of the knife contains the cells of two or more individuals. However, the issue of contamination should always be considered, and it is important to exclude the possibility that DNA from laboratory personnel, or from other samples, has become mixed with the samples for investigation. For this reason, the standards of protection against contamination are extremely high in forensic DNA laboratories. Moreover, the possibility that contamination occurred outside the laboratory, for example, during evidence collection, also needs to be taken into account.

22.1.3 Probability of Identity

The investigator's aim in analyzing DNA is to ascertain how high the probability of identity is between, e.g., trace DNA evidence from a crime scene and the DNA of a suspect. The first step comprises investigating all STR loci analyzed; if both trace evidence and suspect show the same alleles at all loci, it is possible that the suspect is the stain donor.

Excluding Identity. If the suspect and trace evidence differ in one of the STR loci analyzed in only one allele, the suspect could theoretically be excluded as the stain donor. However, mutations can occur, with the result that blood and saliva, for example, from the same individual may differ in one allele. "Null alleles" represent a possible further problem: if, for example, an evidentiary sample and a saliva sample are analyzed in two different laboratories using different primers (different kits), an allele may be absent in the one analysis due to incompatibility in the primer-binding region, mimicking homozygosity, whereas analysis at the other laboratory, or using a different kit, detects both alleles. In cases of so-called exclusion, variations are typically seen in two or three alleles. According to parentage testing guidelines for paternity testing in Germany, at least four exclusion configurations on different chromosomes from the analysis of 15 STR loci are required to exclude paternity, since mutations during meiosis can play a significant role.

22.1.4 Calculating Probability of Identity

The first step is to establish how often a particular STR profile identical to both a sample of trace evidence and a suspect occurs in the general population. To this end, firstly, the *single probabilities* need to be calculated for the individual STR loci analyzed: the probability of the first allele is multiplied by that of the second allele. If, for example, the allele combination 6/9.3 is found for locus TC11, a value of 22.34 % can be derived for allele 6 and 31.7 % for 9.3 (Table 22.2). Multiplying these two values yields 7.1 %. However, since it is not possible to determine which allele (6 or 9.3) comes first and which second, the calculated value needs to be corrected by a factor of 2. Thus this yields a probability of 14.2 % for the allele combination 6/9.3.

For alleles with values below 1 %, the possibility that the value is affected by the sample size

needs to be considered. Therefore, a conservative approach is taken: 1 % is generally assumed as the lowest possible value when *calculating the single probability*. This would thus yield a probability of 3.21 % (16.04 % × 1 % × 2) for the allele combination 7/10.3. (Table 22.2).

In the case of homozygosity, the value of the relevant allele is squared, yielding the single probability for the STR locus; correcting by a factor of 2 is unnecessary.

The same procedure is followed for all STR loci investigated. Finally, the single probabilities of the individual, independently inherited STRs can be multiplied with one another. Depending on the total number of STR loci investigated and the single probabilities calculated, it is relatively easy to achieve a high probability of identity. Thus, statements like "In purely mathematical terms, this STR profile occurs in one out of 1 billion people" are not uncommon. However, this does not guarantee conclusive identification. Even if the probability of identity becomes absurdly great or if, vice versa, it is stated that a particular STR profile occurs only once in a particular number of people which vastly exceed the world population, this does not exclude the possibility that there is (or was) someone living somewhere in the world with exactly the same pattern but who can be excluded as the stain donor.

If no exclusion is found in the analysis of trace evidence or when determining identity, a statement on the *probability of identity* is expected. A "probability bordering on certainty" (generally accepted from 99.99 %) is usually required. If a given STR profile occurs for example in one in 1 million people, the probability of identity is calculated by dividing this million by a million plus the individual affected, i.e., 1,000,000 divided by 1,000,001. If this sequence is detected in trace evidence from a crime scene or in a suspect, the probability of identity is calculated to be 99.9999 %.

In the case of mixed stains, the probability with which a suspect is one of the stain donors can be calculated; prerequisites of this include a complete STR profile of the suspect, as low an incidence of single alleles as possible, and a low overall number of stain donors.

Case Study

Following a burglary at a jewelry shop in northern Germany in 2000, a trace of blood was collected from the crime scene. Using a multiplex kit, 11 STR loci could be successfully analyzed. The results of the (then) eight loci in the DNA database (see Table 22.1) were sent to the German Federal Office of Criminal Investigation. A "match notification" followed shortly: in the five (old) loci, the sequence matched that of an individual from southern Germany (the person had been entered in the system before the database was extended to eight loci), who had a previous conviction for a similar offense. In purely mathematical terms, the sequence occurs in 1 of 1,254,000 people, equivalent to a probability of identity of 99.99992 %.

On police interrogation, the new suspect protested his innocence. He was also able to prove with the help of a time-clock printout that he was working on a night shift approximately 600 km away from the crime scene at the alleged time of the crime.

A saliva sample was taken from the suspect, and additional STR loci to the five loci originally in the database were investigated; three of these yielded exclusions when compared to the trace evidence from the scene of the crime.

As the example above shows, a DNA database can yield matches based not on the identity of the person in question but rather on an incidental match in profile of two separate individuals stored in the database. The larger the number of records in the database, the greater the risk of incidental matches of this kind. The solution to this problem lies in increasing the number of STR loci, as was carried out in Germany in 2002. Given the large volume of records following integration on a European level, a substantial addition of between five and eight loci seems unavoidable. Detecting an incidental match is

generally possible by analyzing further STR loci on the samples being compared.

To calculate probability of identity from "database matches," this preselection can be taken into consideration. The underlying concept here is that when making a query, each individual stored in the database is, at the outset at least, treated equally as a suspect, even if he/she—as shown in the above example—could not have been at the scene of the crime. Thus the calculated probability of the STR profile firstly needs to be corrected by the number of individual profiles in the database. If one assumes, for example, that 100,000 individuals are stored in the database, an occurrence of 1:1,254,440 is reduced (when applied to the present case) to only 1:12.5 and thus a probability of identity of 92.6 %.

22.1.5 Gonosomal STR Loci

The use of *gonosomal markers* (sex chromosome markers) in *paternity testing* is particularly recommended in deficiency paternity testing, i.e., when the alleged father is absent or deceased. There are several STR loci on both gonosomes, which are referred to as X-/Y-STRs or X-/Y--chromosomal markers. Since 95 % of Y chromosomes do not recombine, Y-STRs are not inherited independently of one another. Two X chromosomes (in female individuals) recombine; however, coupling groups need to be taken into consideration in the calculation.

Important: **Y-STR analysis always identifies haplotypes**, **i.e.**, **simple alleles**. **These are as a general rule transmitted from father to son**, **such that half brothers of the same father**, **the father's brother**, **and even the brother's sons all have identical haplotypes**; **this is referred to as a paternal line of inheritance**.

Y-STR haplotypes can also help in the investigation of population affinity. X-STRs in men are detected as haplotypes, whereas in women two alleles are obtained. The individual probabilities of X- or Y-STRs must not be multiplied. The probability of identity for a specific Y-STR haplotype can be determined by searching an online database. An online database for X-STRs is currently under development.

22.1.6 Mitochondrial DNA (mtDNA) Analysis

When DNA analysis using conventional STR loci is unable to yield satisfactory results, mtDNA provides useful information in the case of:

- Markedly degraded human genomic DNA.
- Insufficient volumes of DNA.
- Identifying, for example, urine samples (long postmortem interval, bacterial contamination!) primarily in the case of specimen manipulation, perhaps in the context of doping analysis.
- Only individual hairs or bone parts are available following very long postmortem intervals.

Mitochondrial DNA (mtDNA), which functions independently of nuclear DNA, is extranuclear and autonomously replicating DNA. mtDNA is a circular double-stranded molecule with around 16 kbp (thousand base pairs) essentially containing packed information primarily for respiratory chain enzymes. There are between a few hundred and a few thousand copies of human mtDNA per cell. Only a small connected portion is noncoding and is found in the so-called D-loop, the control region, where replication begins and ends. The D-loop is around 1,100 bp (base pairs) long and contains the hypervariable regions HV1, HV2, and the smaller HV3.

Since several mtDNA molecules are found in each mitochondrion and each cell contains several mitochondria, there can be (depending on cell type) up to a thousand copies of mtDNA in one cell—and only one copy of nuclear DNA. This makes forensic mtDNA analysis particularly suitable in the case of low-volume cell or DNA samples, as well as poorly preserved DNA.

mtDNA contains no STRs. Sequencing HV1, HV2, and more rarely HV3 is the method of choice. Results are not given as a complete sequence, but instead only differences with respect to a reference sequence [Cambridge Reference Sequence (CRS), or Anderson sequence] are noted. A *base substitution* compared to the reference sequence is called a *substitution*. There are two types of substitution:

Transition: A purine is replaced with a purine (adenine for guanine or vice versa) or a pyrimidine

Table 22.3 Differences between two sample specimens (P24 and P24) and the Anderson sequence (CRS)

	HV1				HV2				
Haplotype	16166	16184	16223	16325	73	146	195	263	315.1
CRS	A	C	C	T	A	T	T	A	#
P24	D	T	T	C	G	C	A	G	C
P25	D	T	T	C	G	C	A	G	C

with a pyrimidine (cytosine for thymine or vice versa).

Transversion: A purine replaces a pyrimidine or vice versa, occurs more rarely.

If one or more bases are absent from the sequence in relation to the CRS, these are referred to as a *deletions*. If, however, there are bases in the sequence which are not described in the CRS, these are referred to as *insertions*.

The example specimens P24 and P25 in Table 22.3 each show the same differences in relation to the reference sequence at the same nine positions. It should be borne in mind here that the CRS has a particular feature in relation to most individuals in that it has an A at position 16166 and "lacks" a base at position 315.1. Thus differences at these two positions compared to the CRS contribute little to identity typing. The other seven differences provide more information, although ultimately they are not person-specific but can instead be assigned to a haplotype group (maternal inheritance line). Moreover, mtDNA has a significantly higher mutation rate compared to nuclear DNA. Thus, highly specific differences beyond haplotype groups can be seen.

Probability of Identity. The constellation shown in Table 22.3 suggests in the first instance that the two specimens possibly originate from the same individual. Probability of identity can only be calculated by making specific queries to large databases, such as the database at the Institute of Forensic Medicine in Innsbruck.

Maternal Inheritance. An important genetic feature of mtDNA is that it is only transmitted through the maternal line. This feature can be used in population genetics to determine migration dynamics and ethnic origin. For the purposes of forensic DNA analysis, this offers the advantage that, when identifying human remains, reference samples can be obtained from all relatives in the maternal line of a missed person. However, siblings cannot be distinguished from one another in trace evidence analysis. Strictly speaking, forensic mtDNA analysis is not suitable for identity typing, but rather for attributing an individual to a maternal line of inheritance. Therefore, it is very useful in family tree analysis, human tissue sample identification, and anthropological research.

Spontaneous Mutations. The spontaneous mutations that occur more frequently in mtDNA than in nuclear DNA can contribute to identity typing within an inheritance line. Conversely, a single or even two differences in an mtDNA profile do not represent an exclusion of identity.

22.1.7 Single-Nucleotide Polymorphisms

Single-nucleotide polymorphisms (SNP), biallelic genetic markers, are single base-pair variations in noncoding autosomal regions; in contrast to STRs, their fragment length is not important. Each locus typically has only two different alleles, which are correspondingly either homo- or heterozygous. To determine identity with sufficient reliability, at least 50 different SNPs need to be analyzed (so-called minisequencing). The statistical significance of 50 SNPs in relation to probability of identity is equivalent to that of approximately 12–15 STRs. Since SNP analysis uses very short amplicons, it was conceived for use in highly degraded DNA. However, the method has not enjoyed the level of success originally anticipated for it, nor has it brought the expected extent of desired advantages over STR typing in critical samples.

22.1.8 Biallelic Deletion/Insertion Polymorphisms (DIPs)

DIP (or indel) analysis is a comparatively new method conceived for the forensic analysis of

degraded DNA and mixed stains. Its simplicity of analysis and evaluation is comparable to that of STR loci, but amplicon lengths are below 150 bp. Thus DIPs assume a position between STRs and SNPs. A single DIP locus is less reliable in terms of identity typing than an STR locus. Using the PCR method, one commercial kit mentioned here by way of example is able to analyze 30 DIPs distributed over 19 autosomes. To what extent DIPs will become firmly established in forensic DNA analysis remains to be seen.

22.2 Applications

Applications of DNA analysis in daily practice are illustrated here with examples. While trace evidence collection and quality are important, great attention must also be paid to the interpretation of DNA analysis results.

Case Study

In a case of homicide, the victim received multiple kicks to the face and died of blood aspiration (asphyxia). At the scene of death, a large pool of blood was found next to the victim's head, as well as traces of blood and footprints suggestive of a shoe sole with a herringbone pattern. Several days later, a pair of sneakers with a herringbone patterned sole were found at the bottom of a laundry bag in the home of a suspect. Crusted reddish-brown material could be seen embedded in the grooves of the sole. Dried reddish-brown stains were visible on the upper part of the shoe. Preliminary blood tests (Combur test) were positive. Smear samples were taken from the shoe soles, and all evidentiary traces, including the shoe uppers, were cut out. Analysis of these samples showed identical STR profiles to that of the suspect in each instance. Via his legal representative, the suspect stated that he had never denied having been at the scene of the crime; however, the victim had already been dead on his arrival. Due to the poor light, he had stepped in the pool of blood close to the victim's head. This had made the soles of his shoes wet and spattered the uppers. No photodocumentation had been made of the bloodstain pattern on the shoes. It was not possible to make further inferences from the results of DNA analysis.

Since they often host trace evidence, items of clothing such as shoes, textiles, or textile fibers frequently require analysis. Prior to DNA analysis, the macro- and microscopic investigation and documentation of findings, as well as performing preliminary tests before removing samples for DNA analysis, are essential. Initially, sperm or sperm heads can be detected microscopically; samples for DNA analysis are then removed in a targeted manner (Fig. 22.1).

22.2.1 Perpetrator Identification

Case Study

A 28-year-old male had previously been given a custodial sentence for burglary. Following an altercation with work colleagues, one of the latter had stated his intention to "get his own back on him." At the next opportunity, as they left a (smokers') bar, the colleague surreptitiously removed one of the 28-year-old's cigarette butts from the ashtray with a folded piece of paper. He placed the butt in an unused envelope. That same night, the colleague smashed a jewelry shop window, stole a pendant, and let the cigarette butt fall from the envelope to the ground in front of the window. Ten weeks later, the 28-year-old received a police summons. The cigarette butt had been collected during investigations at the scene of the crime, and subsequent forensic DNA analysis had been successful. The STR profile had been entered in the German Federal Office of Criminal Investigation database and produced a match.

Since he had returned to the home where he lived alone immediately after leaving the bar, the suspect was unable to provide an alibi for the time of the offense. All witnesses confirmed that he had left the bar at 1.30 a.m. There were two "ear witnesses" to the shop window being smashed; they had heard clattering at 2:45 a.m. but could see no one at the scene 2–3 min later.

The court considered the "conclusive DNA evidence" and "relevant previous conviction" to be compelling and found the 28-year-old guilty as charged. He was given a custodial sentence and his parole was revoked. The convicted man's legal representative appealed against the court's decision.

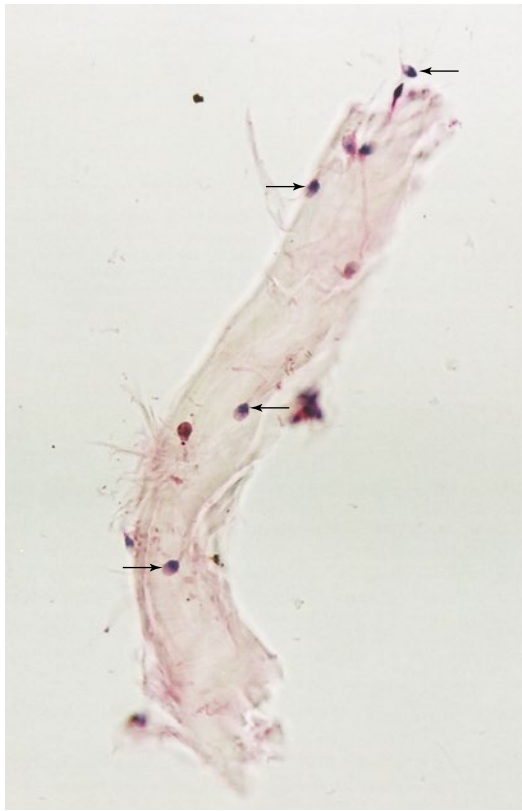

Fig. 22.1 A single minute textile fiber with adherent sperms heads (*arrows*) (×40)

Perpetrator identification is based on the principle that a perpetrator leaves material containing his/her own DNA at the scene of the crime, on the instrument used to commit a crime, or on the victim. DNA in the following media in particular should be considered:

- Blood
- Saliva
- Sperm
- Desquamated epithelial cells
- Hair

These media can be found in walls, floors, objects, etc., at the scene of a crime. Perpetrators often leave an object with their DNA behind. Leaving a cigarette butt at or near the scene of a crime is a relatively common phenomenon, possibly resulting from the perpetrator's state of agitation before, during, or after the crime. Thus, cigarette butts collected at the scene of a crime have led to numerous convictions.

Samples are taken from items of trace evidence, DNA is isolated, and an STR profile generated. This profile is then compared with the profile obtained from a saliva sample taken from the suspect. If the two samples match, a probability of identity is calculated. If there is no suspect to compare the STR profile with, the profile is sent in the first instance to the central DNA database (in Germany, to the Federal Office of Criminal Investigation, the EU authorities, or Interpol). If the profile matches that of someone stored in the database, the two profiles need to be compared once again, possibly including other loci not stored in the database. Depending on how far in the past the relevant individual's profile was entered to the database, a new saliva sample may need to be taken from that individual in order to investigate further STR loci that had previously not yet been analyzed.

Important: Forensic DNA analysis is able to reveal that a particular profile is present in trace evidence from a crime scene as well as in an individual; however, this does not mean conclusively that the DNA originates from that person. Having said that, the probability with which the DNA does originate from that person can be calculated.

If DNA does indeed originate from a particular person, forensic DNA analysis is not able to elucidate how it came to be at the crime scene. Conversely, profile variations found in forensic DNA analysis are able to conclusively exclude the possibility that DNA originates from a particular person; however, this does not mean that the individual in question was not at the crime scene.

Blood Samples. Taking blood samples from a suspect for comparison purposes goes back to the times of serology. Extracting DNA from leukocytes in blood is a promising method, and one that was necessary to obtain sufficient DNA in pre-PCR times.

Saliva Samples. Today, DNA for comparison purposes is obtained almost exclusively from saliva samples. However, "buccal swab" is a more appropriate term: the cotton wool tip of a sterile swab is rubbed along the inside of the subject's cheek. It is important to store the swab in dry conditions. Special cardboard boxes have been developed for this purpose. Placing the swab in a conventional airtight tube immediately after taking the sample entails the risk of significant damage to the DNA through putrefaction and autolysis.

Problematic Trace Evidence. Despite advances in forensic DNA analysis, there are still cases where STR typing is not possible; essentially three factors can be responsible for this:

- Insufficient DNA
- Poorly preserved DNA
- PCR-inhibitory substances

These problems can be solved using the following approaches:

Insufficient DNA: To all intents and purposes, a single fragment of DNA should be sufficient to perform successful PCR amplification. This is in principle true in the case of a single cell containing nuclear DNA. Practice shows, however, that a single cell, or the DNA contained therein, is not sufficient for successful STR typing. In borderline cases, increasing the number of PCR cycles by two or four can solve the problem, albeit at the increased risk of contamination and artifacts. Where attempts at STR typing are unsuccessful, the problem of quantity can be addressed by sequencing the D-loop in mtDNA.

Poorly Preserved DNA: For PCR to be successful, a sufficient quantity of the DNA segment to be amplified and primer-binding region need to be present intact; however, despite sufficient initial quantities of DNA, this may not be the case if multiple strand breaks are present. One option here is to use shortened primers, which are available for most STR loci. Even if STR typing fails, the option to use an alternative method still remains. Due to significantly shorter amplificate lengths in SNP analysis compared to STR analysis, the former holds the promise of certain advantages, particularly in the case of degraded DNA. In practice, however, these purported advantages could not be verified as yet. mtDNA may show greater stability compared to nuclear DNA due to its ring structure. But it is perhaps also its quantitative advantage that makes the sequencing of mtDNA hypervariable regions suitable for degraded DNA samples. DIPs have recently come to represent a further option.

PCR-Inhibitory Substances: Some types of trace evidence contain PCR-inhibitory substances, such as humic substances, enzymes, and putrefaction products. It is assumed that these types of inhibitory substances either cause or contribute to problems in the DNA analysis of fecal samples. It may be possible to detect PCR inhibition using real-time PCR: a certain quantity of a known DNA sequence and appropriate primers is added as an internal control to the sample to be analyzed and amplified along with the sample. The increase in PCR products in this internal control is measured for each cycle. If the expected rate of increase is not observed, PCR inhibition must be occurring. The most effective way to address PCR-inhibitory substances is by using special purification methods. However, one should take care here to avoid significant losses of DNA.

Morphology and Preliminary Testing. The reliability of forensic DNA analysis on its own is limited. This makes it important to be able to integrate the results of subsequent analysis to the sum of all findings. Thus, the investigator's objective is to establish which cells, tissues, or secretions the DNA originates from and where exactly these traces of evidence were found at the

crime scene, on the victim, or on the alleged weapon, for example.

Photodocumentation. Good photodocumentation, including both overview and detailed images with a scale of reference, is an important prerequisite for the subsequent correlation of results. Additional 3D documentation using photogrammetry or laser scanners can be produced for large spaces or, alternatively, measured drawings of the crime scene can be made.

Bloodstain Pattern Analysis. Assuming the blood in question is human blood, an exact description of bloodstain patterns can play a significant role in the subsequent reconstruction of a crime and course of events. A basic distinction is made between the following types of bloodstain patterns (Fig. 22.2):

- Drip pattern
  - Projected blood pattern
  - Arterial spurting pattern
- Transfer pattern
- Impact pattern
- Wipe pattern

Projected patterns may be caused by blood being flung from a blood-bearing object or as a result of blows to a bleeding wound. The height from which blood falls, its angle of impact, and the properties of the surface it lands on all determine bloodstain morphology. Blood impacting a surface at a 90° angle makes a circular stain; the greater the height of drop, the larger the extent of satellite spatter. As the angle of impact narrows, the bloodstain becomes increasingly oval or elongated, sometimes to the extent of resembling an exclamation mark.

Preliminary Testing. This helps establish the origin of trace evidence found at a crime scene or on a sample. The question asked here is whether it is really "worthwhile" taking samples for DNA analysis from a particular point. Preliminary tests need to be sensitive, sometimes at the cost of specificity. A variety of methods are used for preliminary testing (Table 22.4).

22.2.2 Microscopic Investigations

Cells are primarily specified, and their state evaluated using smear preparations (Table 22.5). Although microscopy usually precedes DNA analysis, it can also be performed afterwards for the purposes of interpreting the results of DNA analysis.

Blood-Type Determination. Ouchterlony radial immunodiffusion is well suited to blood

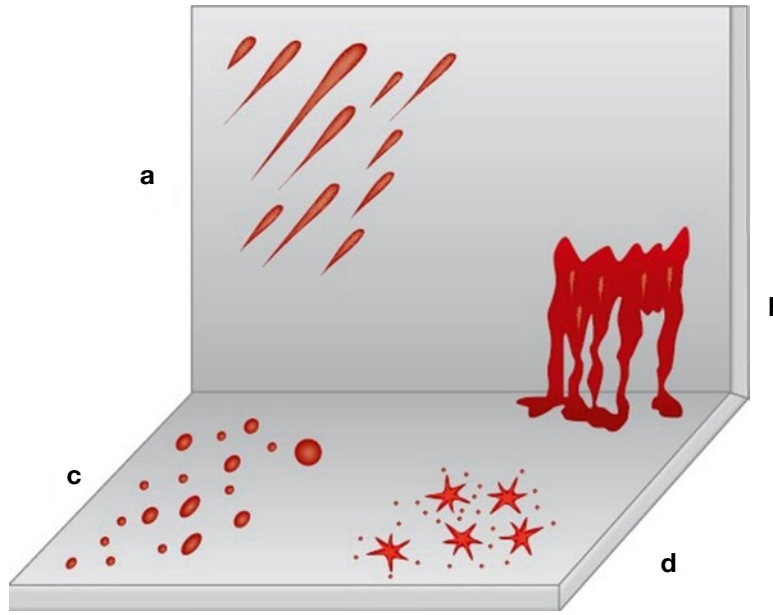

Fig. 22.2 (**a**–**d**) Various bloodstain patterns. (**a**) Projected blood pattern (slanted projection). (**b**) Transfer pattern. (**c**) Drip pattern from a low height. (**d**) Drip pattern from a large height

Table 22.4 Preliminary tests used on trace evidence

To detect	Method
Blood	*Addition of hydrogen peroxide*: bubble formation *Luminol test*: chemiluminescence (glows in the dark) *Sangur or Combur test*: test strip discoloration
Human blood	For example, the *Hexagon OBTI human blood test*: immunological detection
Sperm	*Acid phosphatase test*: test folder, color change (immunological detection) *PSA* (*prostate-specific antigen*) *test*: immunological detection
Saliva	*Amylase test*: applied to a starch-based gel; negative iodine–starch reaction following removal

Table 22.5 Microscopy in trace evidence analysis

Cells/substrate	Microscopic finding
Blood (general)	Erythrocytes eucocytes
Abortus blood	Erythrocytes, chorionic villi, meconium corpuscles, lanugo hair, vernix cells
Female genital secretions/smear	Vaginal epithelial cells: large epithelial cells, stain brown with Lugol's
Sperm	Sperm or isolate sperm heads
Saliva	Predominantly small epithelial cells
Feces	Small epithelial cells and in particular cell debris, food remnants such as plant fibers

determination. It uses an antigen–antibody reaction, whereby antibodies are directed against the type protein. Modern blood determination methods are based on the amplification of type-specific fragments of nuclear DNA or mtDNA.

Sex Differentiation. The usual method for differentiating sex involves molecular genetic investigation of the amelogenin gene, which is present on both gonosomes in varying forms. Females are homozygous for the shorter variants, males heterozygous.

Collecting Trace Evidence in Sexual Offenses. Sexual assault or rape of either a homo- or heterosexual nature produce complex patterns of evidence—the options for taking samples of trace evidence are correspondingly manifold (Table 22.6).

Table 22.6 Possible trace evidence in sexual offenses

Trace from	Action	Detects
Vaginal swab	Unprotected vaginal intercourse	Sperm and perpetrator's DNA
	Vaginal intercourse using a condom	Possibly microscopic lycopodium spores and/or starch granules (condom coating material) (Fig. 22.3)
Anal swab	Anal intercourse	Sperm and perpetrator's DNA
Buccal swab	Oral intercourse	Sperm and perpetrator's DNA
Skin swab	Biting, kissing, licking	Perpetrator's DNA
	ejaculation	sperm and perpetrator's DNA
Penile swab	Penetration of a body cavity of the victim	Victim's DNA
Paper towels	The perpetrator or victim wipe themselves following intercourse	Sperm, perpetrator's or victim's sperm
Victim's clothing	Traces of sperm, saliva, or desquamated cells from the perpetrator	Sperm, perpetrator's DNA
Perpetrator's clothing	Traces of saliva, vaginal secretion, or desquamated cells from the victim	Victim's DNA
Swabs from objects, furniture, floors	Traces of various secretions or desquamated cells from the perpetrator or the victim	Perpetrator's or victim's DNA, sperm

Following alleged rape, vaginal, anal, and oral swabs are recommended. Specimen containers should be dry-stored immediately. A smear preparation can possibly be made prior to drying using a fresh slide, which should also be dried. In the laboratory, a small specimen is taken from the dried swab for preliminary testing for sperm, e.g., acid phosphatase test. If a smear preparation has not already been made, the swab is moistened with sterile, isotonic saline solution and spread

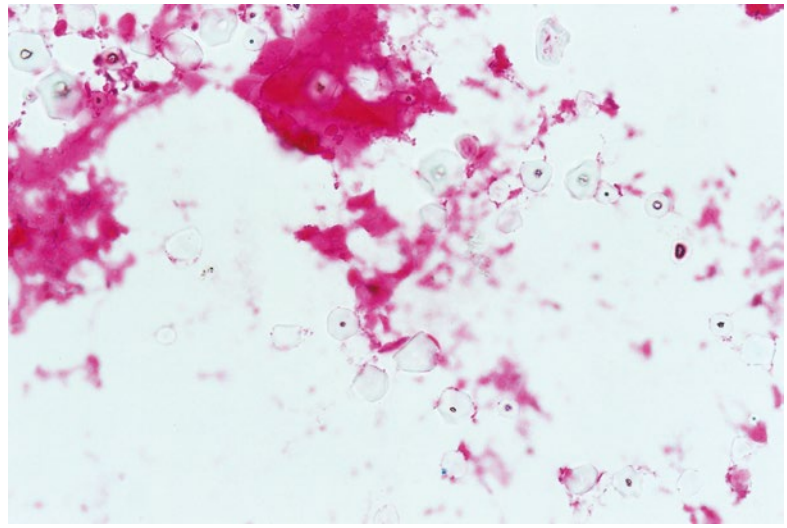

Fig. 22.3 Vaginal smear with starch granules after condom use, autolytically changed cells and agglutinated erythrocytes (Papanicolaou × 200)

on a fresh slide. Smear preparations are then viewed microscopically, either natively or following staining, e.g., Baecchi. The main objective here is to detect sperm, which may be present either as intact sperm or as isolated sperm heads. Depending on the results of analysis and the particular circumstances of a case, DNA analysis is the next step. Thus, even if microscopy is negative, DNA analysis may nevertheless be helpful, for example, if a perpetrator has undergone vasectomy (sterilization), which renders sperm detection impossible.

When analyzing items of clothing or paper towels, a UV lamp can be helpful in the detection of possible traces of sperm.

The detection time for sperm in a vaginal swab from a (living) victim is limited—a maximum detection time of 24 h is generally assumed. However, sperm detection has been reported after 48 and even 72 h in some cases. In cases of positive microscopic sperm detection from a vaginal swab, a mixed stain can be expected on DNA analysis. Comparing STR profiles from a saliva sample from the victim and from the suspect can help in stain differentiation. However, in the absence of a suspect, the mixed-stain result cannot be entered in the DNA database. *Differential lysis* was developed to address this problem. This method takes advantage of the fact that sperm heads are more difficult to lyse than epithelial cells. In an initial gentle step, epithelial cells are lysed and their DNA released (female fraction). Following centrifugation, the still intact sperm heads are separated and lysed using dithiothreitol (DTT) to disrupt the sulfur bonds of the sperm head cell membrane (male fraction). The perpetrator's STR profile can be identified in the male fraction obtained from differential lysis. The female fraction usually remains a mixed stain, since other nucleated cells (epithelial or inflammatory) in the perpetrator's ejaculate would have also been lysed in the first step. However, the male fraction is usually significantly smaller, as made clear by the peak areas in Fig. 22.4.

If microscopy findings for sperm from a vaginal swab are negative, analyzing Y-STRs can provide a way of detecting a male small-fraction mixed-stain donor and of identity typing.

22.2.3 Identifying Deceased Persons

Case Study

After renting an apartment in a high-rise block, a 60-year-old man of foreign nationality was hardly ever seen by his neighbors. Approximately 9 months later, tenants began to complain of an unpleasant odor. The man's apartment was concluded to be

the source of the odor. When the man failed to respond to ringing and knocking at his door over a period of several days, the apartment superintendent contacted the police. The lock and key service then gained access to the apartment in the presence of the police. A severely decomposed body was discovered lying in a bed.

An autopsy examination failed to establish a cause of death. Forensic osteological analysis showed the man to be advanced in years. A dental record was drawn up.

The police were unable to locate either relatives or a treating physician or dentist. Forensic DNA analysis was ordered to establish the man's identity. A toothbrush found in a toothbrush tumbler in the bathroom of the apartment was sent in as comparison material. The STR profile for the body matched those for the toothbrush in all 16 loci analyzed. Identity was subsequently established and the town bore the costs of cremation.

About 2 months later, the meanwhile 61-year-old man appeared at the apartment superintendent's office to ask why his key did not fit the lock of his apartment. He had been abroad for an extended period of time and had let a school friend stay in his apartment temporarily in his absence.

Alongside dental records, comparative X-ray analysis, and fingerprints, forensic DNA is further method of identifying an individual. The availability of comparative material or data obtained from the deceased while still alive is an essential prerequisite of these methods. STR profiles are only available in Germany for people who have "qualified" for the DNA database. Fingerprints are generally only available if identity screening has taken place. X-rays are only taken in the case of medical indication. Dental records, on the other hand, are compiled for all visits to the dentist.

If it is possible to compile an STR profile from human remains, a query can be made in the relevant DNA database. In this respect, forensic DNA analysis may be helpful even at the early stage of the identification process (collecting evidence of identity). If evidence relating to a specific person has been obtained from other sources, an STR profile needs to be made from this for the purposes of comparison. Material suitable for analysis includes, for example, saliva-stained stamps or implements such as toothbrushes or razors used by the person while alive. However, comparative materials should be selected with caution, as the case study above illustrates.

Alternatively, DNA profiles of relatives may be used. In this context, the profiles of parents or children are the most conclusive, since a common allele must be present for each STR locus. Even sibling profiles are poorly suited. If siblings are the only available next of kin, mtDNA D-loop sequencing may be useful on the basis of maternal inheritance and the subsequent match between siblings, as may Y-STR male lineage testing.

In principle, it is possible to obtain material containing DNA from all the tissue types of the body, depending naturally on the state of preservation. A buccal smear or blood sample can be taken—as in living subjects—from a recently deceased individual. Between one and three drops of blood dried on filter paper are sufficient. Obtaining samples becomes more difficult as decomposition advances. As a precaution even in the case of incomplete skeletonization, samples should be taken from the compact long bones, as well as (preferably intact) teeth. It is difficult to estimate how successful forensic DNA analysis on bodies showing varying degrees of decomposition will be. Early mummification often proves to be an advantage, while marked adipocere formation is more likely to be a disadvantage. Another phenomenon often observed is that samples taken from bodies that have lain in earth for only a few years are often more difficult to analyze than skeletons that have lain in earth for decades. It is assumed that PCR-inhibitory substances, which reduce over time, are responsible for this.

Bone samples or teeth are ground in a mixer mill prior to lysis and extraction. There are several STR analysis kits commercially available.

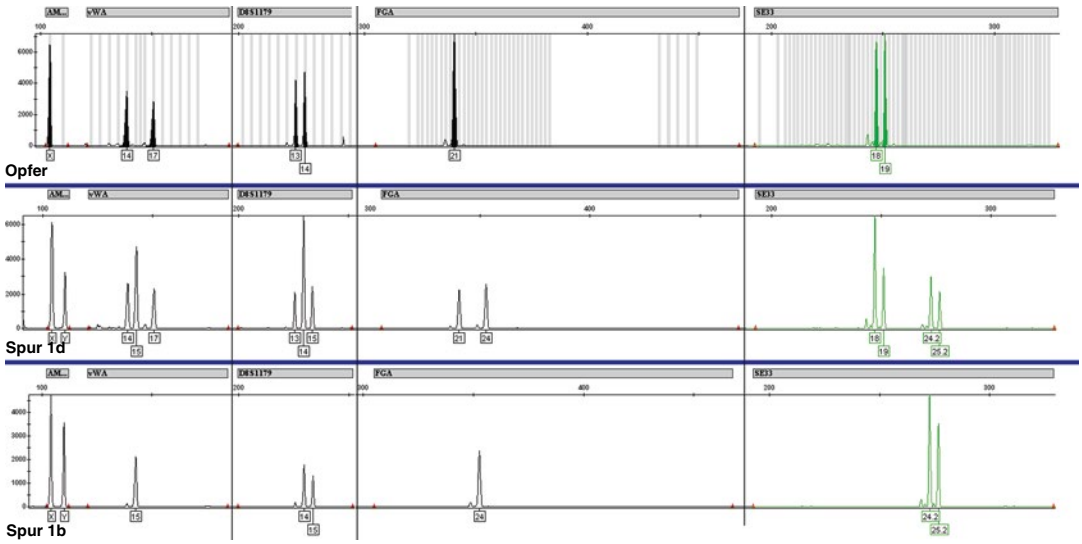

Fig. 22.4 STR analysis following differential lysis. The electropherograms of four selected STR loci and the amelogenin from a saliva sample from the victim (*Victim*), the female fraction (*Trace 1d*) and the male fraction (*Trace 1b*) following differential lysis of a vaginal swab. Allelic ladder peaks (*gray background bands*) are shown for the victim; no allelic ladders are shown for the other two samples. It is clearly evident that Trace 1d is a mixed stain composed of respective alleles from the victim and Trace 1b. Within amelogenin, the victim has a peak only at *X*, Trace 1b shows virtually identically sized peaks for *X* and *Y*, and Trace 1d a larger peak at *X* and a smaller peak at *Y*

If attempts at STR typing prove unsuccessful, the option—as with problematic trace evidence—to apply various purification methods, mtDNA sequencing, or SNP analysis is available.

22.2.4 Parentage Testing

The goal of parentage testing is usually to establish or exclude paternity. Ideally, samples from all three parties of the "paternity trio" are available, i.e., the child–mother–"putative father." Although serological testing is essentially still an accepted method, STR analysis is virtually the only testing method required today. According to current standards, a minimum of 15 independently inherited STR loci need to be analyzed. Surreptitious paternity tests are not admissible in Germany. The German Genetic Diagnostics Act (*GenDG*) regulating genetic testing in humans came into force on 1st February 2010. Section 17 of the Act (genetic testing to determine parentage) states that the person whose genetic sample is to be tested must be informed about and give their consent to the investigation prior to testing.

Although test reliability is limited if the profile of one parent is lacking, it is usually possible to definitively exclude paternity or maternity.

Saliva samples from the subjects involved are usually suitable for testing purposes. To prevent attempts at fraudulent testing, a person's identity should be carefully checked against an identity document at the time the sample is taken. Additionally, photographs and fingerprints should be taken.

It is assumed in the interpretation of analysis results that for each STR locus the child must have inherited one allele from the mother and one allele from the father. If the child has an allele at one locus that is not accounted for by either the mother or the father, this is considered an exclusion. However, an exclusion at one or even two loci does not exclude paternity (or more rarely maternity), since mutations (particularly meiotic mutations) may be responsible for this. Parentage is excluded if exclusions for at least three of the loci analyzed are found. It is not uncommon for two "putative fathers" to be under consideration and require testing (Table 22.7).

Table 22.7 Configuration of parentage testing with two putative fathers

STR locus	Mother (M)	Child (C)	Putative father 1 (PF1)	Putative father 2 (PF2)
D21S11	30/32.2	29/32.2	29/29	30/31.2
D3S1358	14/18	14/16	15/16	16/19
VWA	14/18	18/18	14/18	18/19
FGA	23/26	23/24	21/24	23/25
TC11	6/8	6/9.3	9.3/9.3	7/9.3
D16S539	12/14	12/14	9/12	9/9
CSF1PO	12/12	12/12	12/12	9/12
D13S317	12/13	12/12	12/12	8/9
D7S820	7/9	7/11	8/11	11/12
TPOX	8/8	8/8	8/8	8/10
D8S1179	13/14	13/14	14/15	13/14
D18S51	14/14	12/14	12/17	16/16

The child shows one allele from putative father 1 for each locus. A total of five exclusions are seen for putative father 2 at loci D21S11, FGA, D16S539, D13S317, and D18S51

Probability of Paternity. If there are no exclusions between the putative father and child, the commissioning court requires a statement to be made on the probability of paternity. The allele inherited by the child from the father is decisive in this calculation. Moreover, whether the putative father is hetero- or homozygous for the locus in question is significant. The frequency of the allele (*f*) is included in the *paternity index formula* (see the first two formulas below). If it is not possible to determine which allele the child has inherited from the father and which from the mother (child and mother have the same alleles), formulas (3) or (4) should be used. The individual paternity indices for each STR locus analyzed are then multiplied together.

Probability (*p*-) values of 99.9 % and above indicate that "paternity is practically proven." If 15 STR loci are analyzed, *p*-values of over 99.9999 % can be achieved.

Paternity Index Formula

In the case of a so-called paternity trio, the algorithm is reduced to the following formulas to calculate the paternity index (PI) for an STR locus:

(1) If putative father (Pf) and child (C) are heterozygous:

$$\frac{1}{2f(Pf)}$$

(2) If Pf is homozygous:

$$\frac{1}{f(Pf)}$$

(3) If C and mother (M) have the same alleles and Pf is heterozygous:

$$\frac{1}{(2f(C)_A + 2f(C_A))}$$

(4) If C and M have the same alleles and Pf is homozygous:

$$\frac{1}{(f(C_A) + f(C_B))}$$

In the example presented in Table 22.7, the following formulas should be used for the respective loci; the paternity indices calculated are given in parentheses after each locus:

Formula (1): D3S1358 (2.1), VWA (2.3), FGA (3.76), D7S820 (2.16), D18S51 (3.8)

Formula (2): D21S11 (4.68), TC11 (3.15), D13S317 (3.5), CSF1PO (2.78), TPOX (1.71)

Formula (3): D16S539 (1.38), D8S1179 (0.93)

Formula (4): Not to be used for any loci in this example

Multiplying all PIs together yields a combined PI (PI_{Ges}) of 48971. Dividing this value by (n + 1) gives a probability of paternity of 99.9979581 %.

Deficiency Paternity Testing. Testing becomes significantly more complex if samples from the

putative father are not available and samples from other blood relatives of the putative father need to be used instead.

Important: Deficiency paternity testing in the case of an absent or deceased putative father increases in complexity according to the genetic distance of the blood relative involved from the putative father.

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Forensic Osteology

23

Case Study

A walker found a human skull lacking the lower jaw in a forested area. The skull lay propped on a pile of leaves. It was very light in color, had a washed-out appearance, and showed no residual soft tissue. The walker informed the police. One of the two police officers called to the scene was familiar with the area and was immediately convinced that the skull dated back to the Second World War, when several aircraft had crashed in the forest.

Forensic medical investigations revealed that the skull belonged to a young adult man. Only three untreated teeth were still present, the other teeth having been lost post-mortem. In terms of injuries, two penetrating shots and two through-and-through shots from a small-caliber weapon from three different directions (twice from the left, once from the front, and once from the right) were identified. The projectiles were no longer present. The police officer familiar with the area was quick to provide an explanation: towards the end of the Second World War, a number of summary executions using pistols had been carried out. Since, from a forensic medical perspective, it was not possible to rule out a forensically relevant postmortem interval, and since an unnatural death had quite obviously occurred, the public prosecutor's office called for forensic DNA testing to be carried out. It was possible to create an STR profile from one of the remaining teeth, which was then registered in the database of the Federal Office of Criminal Investigation. Although a match was found, this was not to a person-specific profile but rather to a torso that had been found wrapped in plastic bags at a highway service station in another part of the country approximately 1.5 years previously. At that time, the torso had undergone only moderate putrefaction, and a postmortem interval of approximately 1 week had been estimated.

Forensic osteology is by far the oldest and most popular branch of forensic anthropology and forms at the same time the largest area of overlap between physical anthropology and forensic medicine. The term "forensic osteology" is usually reserved for the investigation and assessment of discovered bones, which can range from (almost) completely skeletonized bodies and complete or partial skeletons to individual bones or mere bone fragments.

Dental investigations for identification purposes (dental formula) and age determination also belong to the field of forensic osteology. This gives rise to areas of overlap and collaboration with "forensic odontostomatology," (see Sect. 6.4) a well-established specialist field of dentistry and forensic age determination in living individuals.

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Forensic anthropology can be viewed as an interdisciplinary specialist field. In addition to numerous related sciences, such as geology, archaeology, or zoology, other auxiliary sciences like biostatistics and nonscientific disciplines like forensic technology play an important role.

Although forensic anthropology's range of tasks may have changed since the late twentieth century and early twenty-first century due to social and geopolitical changes, as well as rapid advances in modern technology, a basic canon of tasks and applications still remains at the core of forensic osteology, based on the classic triad of methods: morphology, osteometry, and biometry.

If one compares conditions and developments on an international scale, significant differences in terms of training and areas of activity are apparent. Forensic anthropology in North America traditionally focuses on forensic osteology, which is mainly concerned with determining human specificity, postmortem interval (PMI), identity, and analyzing traces of injury. A broad spectrum of skills is required to this end. For example, expertise and skill in archaeological methods is required in the recovery of skeletons or skeleton fragments. The PMI yields valuable information in terms of identity and, in the case of homicide, the time of a criminal offense. PMI determination involves combining osteological, medical, geological, and physical knowledge. Historical, partly sociocultural, and ethnological knowledge is required to recognize and interpret "findings," i.e., artifacts dating back to the temporal horizon of death. Understanding the significance of these findings presupposes in turn a knowledge of law and criminology since, according to current conventions, bones with a PMI of over 50 years represent historical bones and, as such, are no longer justiciable.

Forensic Anthropology in Europe. Forensic anthropology in Europe, in particular Germany, is not restricted to osteology alone, but includes instead the examination of living subjects. This is associated with the prevailing notion of biological anthropology in Germany, which takes the individual's external appearance as well as the constitutional doctrine into account.

A frequent task of forensic anthropology is to identify individuals on pictorial documentation, such as photographic or film material obtained from traffic speed measurements, bank raids, or a wide variety of surveillance cameras. Pictorial documentation can also form the basis for age estimations, e.g., establishing whether pornographic material shows minors.

Age estimation in living individuals is a separate area of activity tasked with establishing whether or not specific legally relevant age limits, e.g., in criminal or asylum law, have been reached.

Parentage testing formerly represented a classic area of forensic anthropological activity in Germany; however, due to modern forensic DNA testing (see Chap. 22), it is now only required in cases where no (more) molecular biological material can be obtained from one of the parties involved, but for whom pictorial documentation is available.

23.1 Discovering Bones

Bones are most commonly discovered during construction work, as well as by children at play or walkers (with dogs). Investigations are only initiated if uncovered bones are reported to the investigating authorities (the police or public prosecutor's office in Germany). Whether or not the bones are of human origin is the first question the investigating body puts to the forensic osteological expert. If a nonhuman origin can be established, the investigating authorities generally deem further lines of inquiry unnecessary. Possible exceptions include the contravention of a country's animal protection laws.

If uncovered bones are found to be of human origin, it is necessary to determine the PMI, detect possible traces of injury on the bones, and identify the deceased.

It is important in the assessment of traces of physical injury to differentiate between pre-, peri-, and postmortem injuries; establishing the mechanism of injury is also relevant. A stepwise procedure is followed that involves drawing conclusions about the type of trauma (e.g., sharp or blunt force) and the mechanism (e.g., blow or stab wound), as well as determining a—or even identifying the specific—weapon or instrument used.

Determining sex, age (age at death), height, and (ethnic or geographical) origin yields what

are considered classical indications of identity. This information represents a "biological" profile, a criterion that helps reduce the list of possible missing persons or victims that the human remains under investigation could originate from.

If one or more potential matches to the profile in question are found among missing person profiles, the next step in the identification process is to verify or establish identity. The antemortem material or information available, as well as the parameters that can be obtained post-mortem or in view of the degree of decomposition, determines which methods are applied.

If these approaches initially fail to yield a potential victim, attempts can be made by means of facial soft tissue reconstruction to make information relating to the likely appearance of the deceased person available to the public. The aim here is to create a type of portrait photo of the deceased individual that can be published in the media, thereby increasing the chances of identification.

The main task of forensic anthropology in the case of large-scale disasters or mass graves is victim identification. One distinctive feature here is that a large number of victims need to be examined and identified on the one hand, while on the other the potential victims in many large-scale disasters are known (e.g., aircraft passenger lists or missing person profiles). The sheer number of victims can represent a logistical and organizational challenge in terms of examining and comparing data and therefore calls for very particular organizational approaches.

23.2 Human Specificity

In the vast majority of cases, most notably those where the state of preservation is good and complete bones or large bone fragments are available, the study of species-specific skeletal characteristics can be of great assistance.

Complete or largely well-preserved nonhuman mammalian skulls are generally not mistaken for human skulls. The characteristic shape of dentition offers important clues. The morphological differences between the three dental types illustrate this well in that they have developed as a result of pressure conditions and the manner of feeding, leading, for example, to the characteristic lamellar masticatory apparatus seen in herbivores or the typical pointed dental cusps seen in carnivores.

Diagnosing human specificity from completely preserved bones is generally straightforward even in the postcranium. However, a number of special characteristics are seen regionally. For example, the hand bones of grizzly bears are regularly mistaken for human bones. In Germany, the deer tibia is by far the bone most frequently mistaken for a human bone (Fig. 23.1).

Fig. 23.1 *Top*, deer tibia, *bottom*, human tibia, right side in both, frontal view. Both bones were found separately outdoors on the forest floor; both showed scavenging defects, primarily rodent bites. The deer tibia additionally demonstrated moss growth. The human tibia was subsequently marked with the No. 11 during examination. At first glance, both bones show considerable similarity. However, the deer tibia has a more pronounced tibial tuberosity and a flatter and more compact shaft and is oval in cross section in the midshaft. The midsection of the human tibial diaphysis, on the other hand, has a virtually triangular cross section.

The situation becomes more problematic when only bone fragments are available for investigation. Diagnosing human specificity from portions of distal extremities is particularly challenging due to the similarities between humans and other mammalian species. In general, nonhuman mammal bones are compacter and heavier compared to bones of the same size. The bones of forest animals are often darker in color. The size ratio between the bone cortex (compact bone) and the medullary space often provides valuable insight.

Case Study

A dog ran away from its owner in a clearing in the forest and began to dig furiously at a particular spot. Toys, remnants of stuffed animals, and bones appeared. The dog's owner suspected a child to be buried at the spot and immediately used his cell phone to inform the police. A wide area around the spot was cordoned off. Officials from the criminal police and evidence collection unit carefully examined the scene of discovery, where they found several dark hairs and only small bones, in addition to heavily softened tissue. They believed the dog owner's suspicion to be founded and worked on the assumption that they were dealing with the illegal disposal of an infant's body. A forensic pathologist was summoned.

The forensic pathologist undertook the recovery of the body. The body and toys were (or had been) wrapped in a child's blanket. The bones were still held together to some extent by heavily decomposed soft tissue that had undergone partial adipocere transformation. An initial striking feature was the number of dark hairs, which appeared to be too abundant and too thick to be human hair. As soon as the remnants of decomposed soft tissue had been provisionally removed from the first bones, the forensic pathologist was able to conclude that the bones were not of human origin. Once all the bones and skull were displayed, he concluded that the skeleton was that of a dog.

The discovery of small bones often leads lay people to suspect a child's bones, skeleton, or body. This suspicion is often heightened if—as in the case described above—an association can be made between accompanying artifacts, such as toys or a blanket, and a child. The fact that the skull is absent, or at least initially not visible, completes this "standard scenario," given that a nonhuman skull is readily distinguishable as such (see above).

It is precisely for this reason that, every year, the police receive numerous notifications relating to the discovery of children's bodies; on the whole, these discoveries are incorrectly verified by police officers and only identified as animal cadavers once a forensic pathologist or forensic anthropologist has been called in. Cadavers are canine in the majority of cases, with the remains of domestic pigs, sheep, or cats representing rarer finds.

A child's bones are not only smaller than an adult's bones—they also show an important difference in terms of growth plates, referred to as epiphyseal plates in the long bones (see Sect. 23.5.3 and Table 23.7). The problem is, however, that it is not possible to identify the epiphyseal plates as such from a child's bones, since the epiphyseal caps become detached and get lost. This produces bones that are unique in appearance and with a shape that is difficult to attribute to a particular species and which additionally show unusual markings on the articular surface (Fig. 23.2). Therefore, the bones or skeleton of a human child is less likely to be recognized as human bones.

In cases where it is not possible to reach a conclusive macromorphological diagnosis, additional investigations using morphological or metric histology on undecalcified ground bone sections are possible. Human bone remains show randomly distributed round, markedly polygonal, and virtually

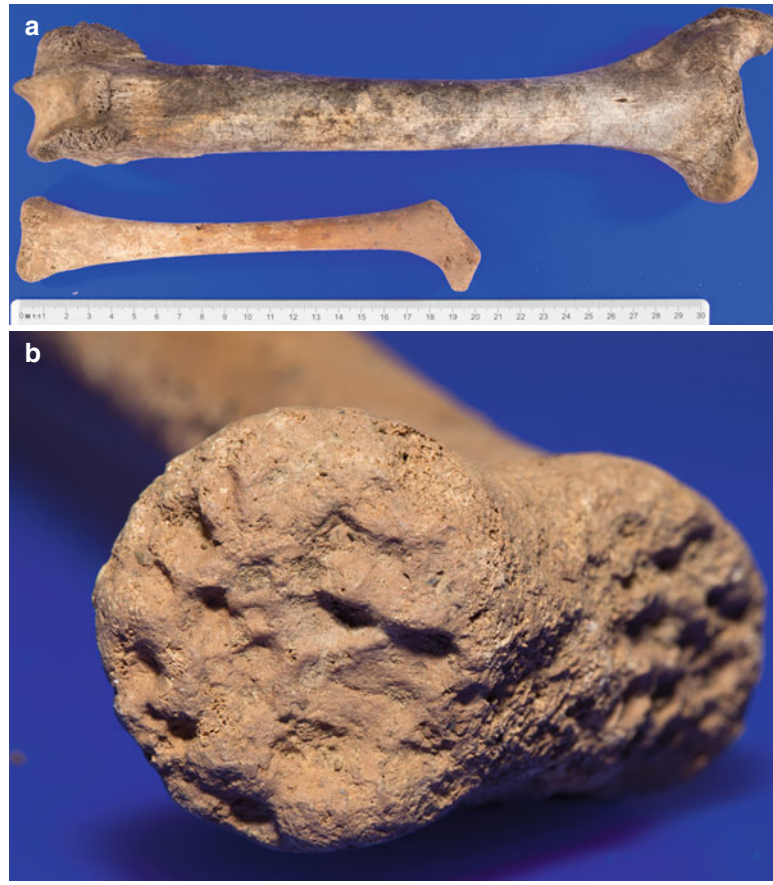

Fig. 23.2 (**a**) The femur of an approximately 4-year-old child. The epiphyseal caps are absent (*bottom*). The right femur of a deer, readily distinguishable as nonhuman simply from its shape (*top*). (**b**) View of a portion of a human child's femur (**a**, *bottom*) incorrectly classified as the proximal articular surface. In reality, this is the surfaces of the epiphyseal plates pointing towards the diaphysis and showing characteristic surface structure

equally sized osteons and Haversian canals, whereas numerous domestic species often show a plexiform, occasionally linear arrangement of osteons of varying sizes (Table 23.1 and Fig. 23.3).

Optical polarization of thin ground sections of compact bone represents a further option for demonstrating human specificity. Two polarizing filters are introduced into the microscope's beam path, one between the light source and the sample, the other between the sample and the eye, and rotated in such as way that only a very limited direction of light appears in the eyepiece. Human osteons interrupt the polarized light and produce characteristic cross-shaped effacements, the centers of which are located in Haversian canals (Fig. 23.4). Since these so-called Brewster crosses occur only in ground bone slides of human compact bone, they are well suited to determining human specificity.

Using Molecular Biology to Determine Human Specificity. Forensic DNA testing (see Chap. 22) offers several options for obtaining information on the human specificity of bone finds. However, isolating DNA from bone is a basic challenge. Depending on the conditions in which a bone has lain, DNA may be heavily degraded, or the specimen may contain many PCR-inhibitory substances.

The most obvious solution to this drawback would be an attempt to amplify STR loci (see Sect. 22.1.1). If this can be achieved—and contamination can be ruled out at the same time—human specificity is deemed to be demonstrated. However, if amplification attempts fail, it is impossible to say whether the bone is not of human origin or whether the DNA is too poorly preserved.

The method already proposed in 1999 by Bataille et al. offers a good solution: mitochondrial

Table 23.1 Histological distinction between human and animal bone tissue on the basis of Haversian canals

Haversian canals	Average diameter (µm)	Average number per visual field	Haversian canals in overview magnification
Human neonate	54.5	2.3	Medium-sized to very large, increasing in size towards the center, round to oval shape
Human, 6 months	60.5	1.7	–
Human, 12 months	71.6	1.6	–
Human, 18 months	56.8	1.7	–
Human, 41 years	52.9	1.7	–
Human, 70 years	70.0	1.5	–
Horse	30.0	2.7	Small- to medium-sized, predominantly medium-sized, regularly shaped
Cattle	47.9	1.4	Predominantly medium-sized but also large, regularly shaped
Goat	21.2	2.4	Predominantly medium-sized but also large, becoming smaller towards the center
Sheep	18.2	3.6	Predominantly medium-sized but also large, irregular structure
Pig	32.8	2.1	Predominantly medium-sized but also large, becoming smaller towards the center
Dog	21.2	3.0	Predominantly very small but also medium-sized, regularly shaped
Rabbit	12.6	8.0	Very small, round to oval
Cat	20.3	2.8	Predominantly very small but also medium-sized, irregular structure
Chicken	14.0	7.0	Very small, round
Goose	15.7	14.4	Medium-sized, irregularly shaped

Rämsch and Zerndt 1963; according to Dürwald 1987
Very small <10 µm, *small* 11–20 µm, *medium-sized* 21–40 µm, *large* 41–80 µm, *very large* >80 µm

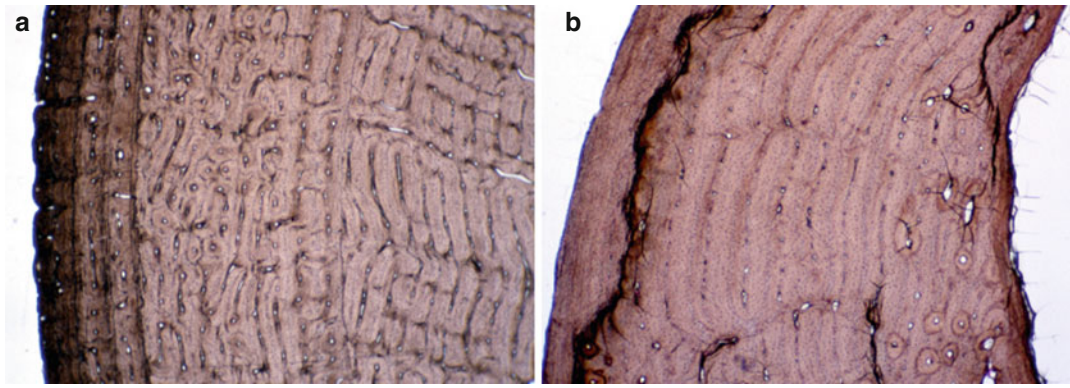

Fig. 23.3 Ground bone sections of mammal bone for species differentiation. Undecalcified ground sections of the compact bone tissue of long tubular bones of different mammals: (**a**) sheep, (**b**) dog, (**c**) pig, (**d**) human (Kossa × 4) (Figures kindly provided by Dr. F. Ramsthaler)

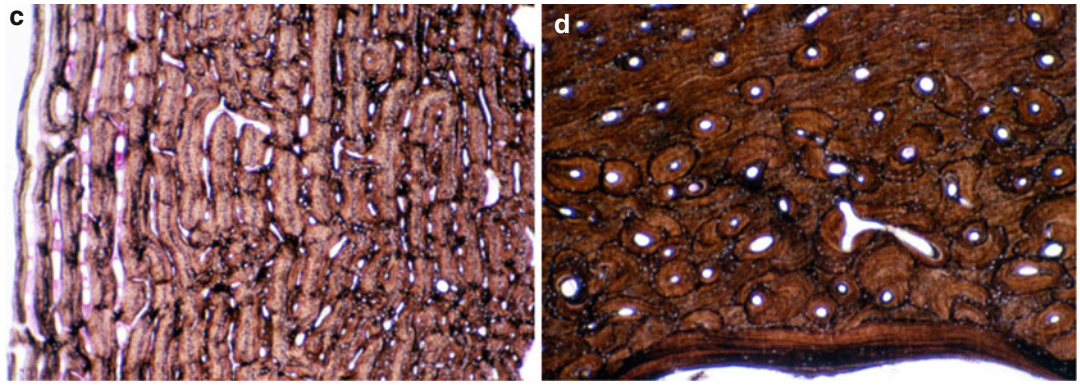

Fig. 23.3 (continued)

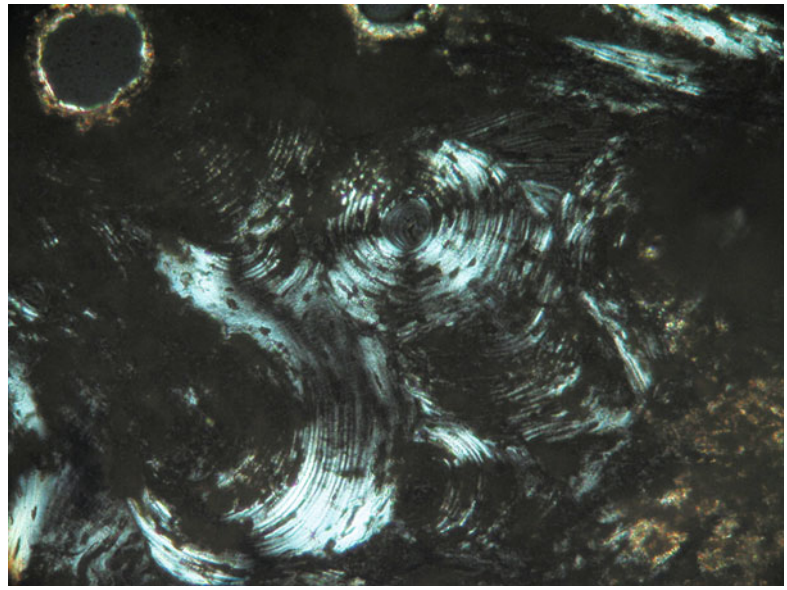

Fig. 23.4 The Brewster cross: cross-shaped effacements at the center of Haversian canals are seen by visualizing long compact bone under polarized light. This effect is best seen in the osteon located at the center of the image. This phenomenon, known as the Brewster cross, is only seen in human long bones

DNA (see Sect. 22.1.6), which is found in cells in higher copy numbers compared with nuclear DNA, is investigated. In addition, the ring-shaped mitochondrial DNA molecule is more stable than nuclear DNA. Attempts are made to co-amplify a 259-bp-long segment of the human D-loop (HV1 region) and a 309-bp-long segment of the cytochrome-b gene, which all mammals (including humans) possess. Human samples demonstrate two bands in the gel, nonhuman mammal sample only one (cytochrome-b gene) (Fig. 23.5). If no amplification product is obtained, one can assume that the condition of the DNA in the sample was too poor, and no conclusions can be drawn on human specificity.

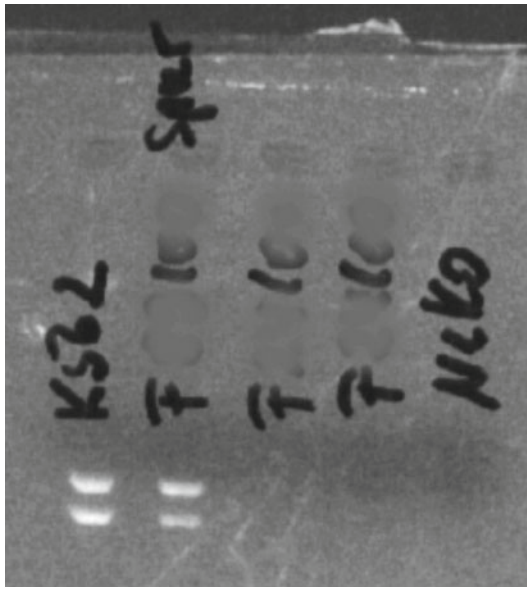

Fig. 23.5 Polyacrylamide gel following co-amplification of a 259-bp-long segment of the human D-loop and a 309-bp-long segment of the cytochrome-b gene (Method according to Bataille et al. (1999)). The (human) positive control is plotted in the *first row*. The *upper* of the two bands represents the cytochrome-b gene segment, the *lower* the human segment from the D-loop. The bone sample plotted in the *second row* is human. The samples in *rows 3* and *4*, as well as the negative control in *row 5*, have no distinguishable amplification product. A nonhuman mammal would show only one band—the *upper* of the two bands

In special cases where more precise identification of the nonhuman mammal species is relevant, mitochondrial DNA offers further loci with species specificity.

23.3 Postmortem Interval

Case Study

The installation of floodlights was planned for a tennis court built 20 years previously and belonging to a tennis club. Club members carried out a significant amount of the preliminary works themselves. As part of these works, they dug a narrow canal for cables near the concrete foundations of the fence at the top of one of the courts. In the process, they discovered some bones. An orthopedist (and club member) who was called to the scene confirmed the initial suspicion that the bones were of human origin. The club members continued to dig, upon which they discovered more bones. At this point, the decision was made to inform the police.

An investigation was opened on the suspicion that a body could have been buried at that point 20 years ago when the fence foundations had been poured.

The bones were systematically uncovered by a forensic pathologist and an anthropologist. A complete and relatively well-preserved human skeleton emerged. The teeth, which showed distinctive signs of wear, showed no signs of dental treatment. Anthropological analysis indicated a middle-aged man measuring approximately 180 cm in height. Radiocarbon dating yielded a postmortem interval of around 1,850 years.

Although there is no statute of limitations on homicide in many jurisdictions around the world, only a PMI of up to 50 years has been considered forensically relevant to date. Bringing a perpetrator to justice becomes unrealistic at longer PMIs. As life expectancy increases, it is not inconceivable that the forensically relevant PMI will be extended by 5 or 10 years in the future. As yet, there is no investigation technique that is able to measure PMI within the time period of the first 50 years or so sufficiently reliably. The established radionuclide methods, such as 14C determination, for example, can be used in forensic cases to only a limited extent due to their long half-lives (5,730 years for 14C).

Decomposition. Decomposition refers to bone changes caused by ambient conditions during the course of the PMI. The study of decomposition processes is known as taphonomy. However, the fact that these discernible changes depend on ambient conditions represents a major drawback and often makes conclusions regarding PMI challenging.

Ambient conditions are often difficult to evaluate. A body lying in the open air in central Europe during the summer months can skeletonize completely within a few weeks. Soft tissue remnants may be present even after decades if extensive natural mummifications have occurred due to the hot, dry weather conditions associated with the summer months.

Decomposition processes are easier to evaluate in a soil environment. However, it could be shown that two skeletons buried in the same cemetery and with identical PMIs demonstrated quantitatively and qualitatively different decomposition processes. Thus, in principle, only cautious conclusions in terms of PMI are possible.

However, decomposition findings on bone that have not been seen hitherto at PMIs of less than 50 years in soil environment have emerged. In terms of external appearance, these findings include:

- No remaining macroscopic evidence of adipocere.
- Deep incisions on external layers of compact bone.
- Extensive bone surface defects (Fig. 23.6).
- Intense blackish-brown film of microorganisms.
- Frayed outer lamellar systems.
- Cortical detachment.
- Tissue torsion.
- Presence of brushite.
- Bone can be broken manually.

The following findings cannot be made in freshly cut cross sections at a PMI of less than 50 years in soil:

- Absence of adipocere
- Brushite in the medullary cavity
- Reduced or absent UV fluorescence

If a bone demonstrates one or more of these findings—and there is no indication that the bone has been exposed to outdoor conditions (Fig. 23.7)—a PMI of less than 50 years can be ruled out. This is not possible, for example, if mass adipocere filling of the medullary cavity is detected in fresh cross sections (Fig. 23.8).

Numerous complex chemical and physical techniques have shown no advantage over macromorphology. One is currently forced to conclude that only *radionuclide*-based methods are suited to determining PMI independently of other effects. The wide range of radioisotope

Fig. 23.6 Decomposition in a soil environment. Ventral view of the proximal and mid-third of both femora after 150–200 years in acidic soil conditions. Only superficial compact bone layers are extensively absent on the right femur, while deep extensive compact bone defects and a deep incision can be seen on the left

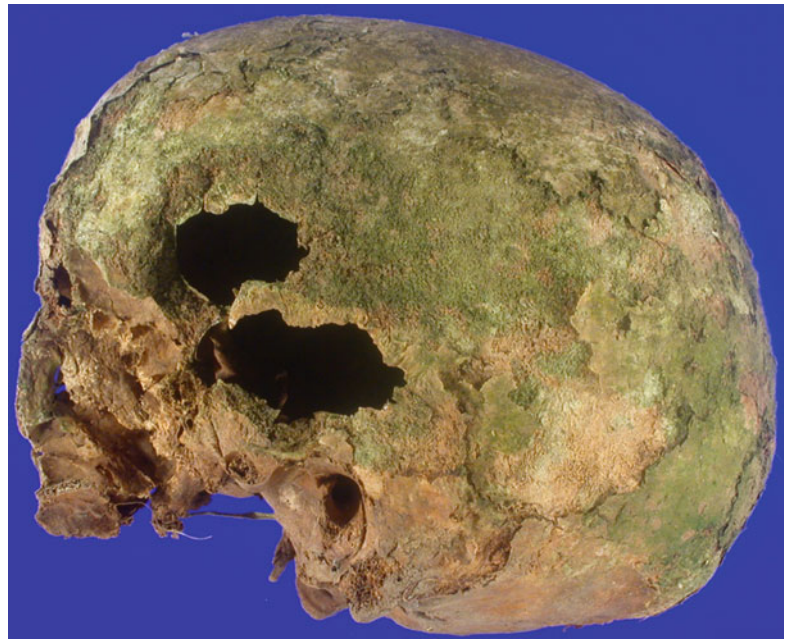

Fig. 23.7 Signs of exposure to outdoor conditions. Left sagittal view of a human skull showing significant changes due to decomposition. Green algae growth suggests long-term exposure to outdoor conditions, thereby rendering conclusions on postmortem interval almost impossible

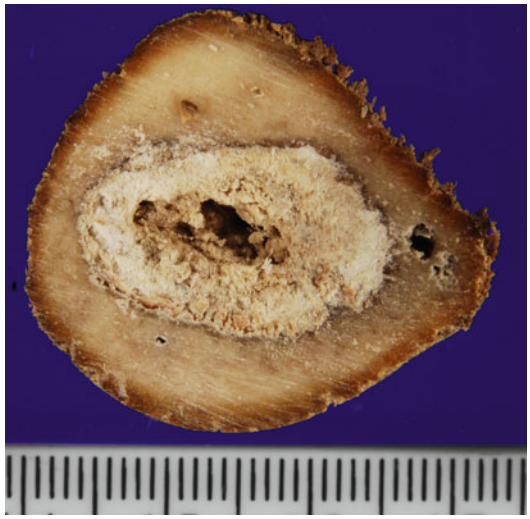

Fig. 23.8 Mass adipocere filling of the medullary cavity. Fresh cross section through a femur. Adipocere fills the medullary cavity. Although this finding speaks against a postmortem interval (PMI) of more than 50 years, PMI is unlikely to be short-term. In general, very particular conditions involving an oxygen-free environment or submersion in water are responsible for findings of this kind. The actual PMI in this case was narrowed down to between 50 and 70 years

ratios can offer additional information on the origin of an individual or where they spent the majority of their life.

In addition to investigating tissue, appropriate attention needs to be paid to the scene of discovery and accompanying findings; remnants of clothing, coins, newspapers, tools, and weapons, among others, can help to narrow down the temporal horizon. A coffin mark (Fig. 23.9) or objects that could represent grave furnishings can help to distinguish a normal burial from the illegal disposal of a body. Furthermore, land register archives should be checked to establish whether the scene of discovery has ever been associated with, for example, a cemetery or a medical institution. Other possibilities include the transferal of earth from old or existing cemeteries to the scene of discovery.

23.4 Forensic Osteological Identification

Since forensic osteology deals with human remains that are skeletonized or at least partially skeletonized, physical integrity is either wholly or partly lost. Depending on ambient conditions, it is possible to find a complete skeleton in situ, i.e., in a correct anatomical position, in soil, for example. On the other hand, bodies exposed to

Fig. 23.9 A coffin mark is found at the edge of the opened grave (exhumation after 5 years). The mark did not originate from the case in question (an almost intact casket was recovered), but rather from an earlier grave allocation dating back 35 years

outdoor conditions or buried in shallow graves are susceptible to animal scavenging. The first question to be answered when bones, or largely skeletonized human remains, are found randomly distributed at one spot or spread over a wider area is whether the bones originate from one or more than one individual. Multiple occurrences of the same bones represent the simplest indicator of more than one individual. For example, the discovery of three left-sided femoral bones indicates that the human remains originate from at least three individuals. An alternative constellation: placing all uncovered bones on a table results in an incomplete human skeleton and no bone occurs more than once. Indications may nevertheless point to more than one individual if the different bones are inconsistent with one another in terms of shape and size and therefore cannot necessarily be attributed to the same skeleton. For example, one might have a right and a left humerus, but one is much finer and shorter compared to the other and to the rest of the skeleton. In this setting, however, one would have to consider possible pathological changes, such as arm paralysis. On the other hand, if there are other finer bones from different body regions, it can be assumed that one is dealing with at least two individuals.

As with better-preserved bodies (Chap. 6), the identification of unknown deceased individuals is a two-step procedure in forensic osteology. Firstly, as much information as possible is gathered on the individual. This is commonly referred to as a "biological profile," which, once created, can be compared with missing persons lists. A certain tolerance is allowed in this process. For example, if a height of 180 cm (see Sect. 23.5.2) is calculated for the unidentified individual determined as male, most formulas would use a possible error of approximately ±5 cm; that is to say, individuals on missing persons lists measuring 175–185 cm in height would be taken into consideration. A further factor of uncertainty lies in the fact that antemortem data may be incorrect. Often, the height given in the first identity card (obligatory from the age of 14, 16, or 18 years, depending on the country) is not remeasured, and the same height is repeatedly used in subsequent identity documents. Since growth in height in men can continue up to the age of 21 years (see Sect. 23.5.3, Table 23.7), height values given in official documents, and hence also in missing persons lists, are often too low.

In cases where it is possible to ascertain missing persons who, when the necessary tolerance is applied, match the biological profile of the unidentified deceased, it must first be established which antemortem information on the missing person is available. It is on the basis of this vital criterion that the method of identification is selected (see Sect. 23.7). Whether the available methods can then be used depends in turn on

whether the required data can be obtained from the skeletal remains. If, for example, antemortem dental records or a clinical X-ray of the head is available but the head of the human remains to be identified is lacking, neither forensic odontostomatology (see Sects. 6.4 and 23.7.2) nor comparative X-ray analysis will be helpful in establishing identification.

23.5 Indicators of Identity: The Biological Profile

Determining the sex, height, age, and origin of skeletons or bones provides an important indication of identity and offers parameters that can be compared with missing persons.

23.5.1 Sex

The morphological sex determination of skeletons is performed by evaluating sexually dimorphic skeletal traits morphognostically or morphometrically. Particularly the pelvis and skull show this sexual dimorphism. Thus, the overall size and roughness of muscle attachment sites in the pelvis, as well as all other skeletal parts, are seen as sexually dimorphic traits, whereby male trait carriers are generally described as larger, heavier, and less regular. Individual pelvic traits are summarized in Table 23.2.

Greater differentiation is seen in skull traits used to discriminate sex (Table 23.3).

In particular, skulls with only male or only female traits are very rare (Fig. 23.10). Traits used for morphognostic assessment are classified into a scale according to degree of prominence, ranging from hyperfeminine, feminine, or indifferent to masculine and hypermasculine. The diagnosis "female," "male," or "indifferent" is reached by making an overall assessment of all traits assessed. This overview shows a population-dependent variability based primarily on physical activity or strain. An investigator familiar with medieval skeletons, for example, is more likely to incorrectly classify an overly high number of modern skulls (forensic background) as female.

Table 23.2 Sexually dimorphic traits on the pelvis (selection)

Trait	Female	Male
Subpubic angle	Obtuse	Acute
Pelvic inlet shape	Elliptic	Heart-shaped
Obturator foramen	Triangular	Oval

Table 23.3 Sexually dimorphic traits on the skull (selection)

Trait	Female	Male
Decline of frontal bone	Vertical	Sloping-fleeing
Glabella	Poorly defined	Pronounced
Superciliary arch	Poorly visible or not visible	Bulging
Frontal and parietal eminences	Prominent	Poorly developed if at all
Mastoid process	Small	Large, voluminous
Nuchal plane	Flat, smooth	Rugose, high muscle attachment crests
External occipital protuberance	Poorly defined	Beak-like projection

Conversely, an investigator regularly active in forensic osteology might diagnose an overly high number of male individuals among skulls found in a medieval burial field.

In order to address claims of subjectivity and a lack of scientific approach in the application of morphological methods, morphometric methods have been developed. Of these, discriminant function analysis is the best established method for the determination of sex. This approach involves recording length and distance measurements in supposedly sexually dimorphic samples and using these to develop discriminant functions that permit the sex of the person in question to be determined. A further option for making trait evaluation more objective is to quantify traditional morphognostic traits. By applying a wide variety of methods, attempts have been made to use morphological sex traits of the skull to express in numbers descriptions such as rounded or angular orbital cavity, sharp or rounded orbital rim, and voluminous mastoid process or small mastoid process in numbers or at least to record them using standardized techniques.

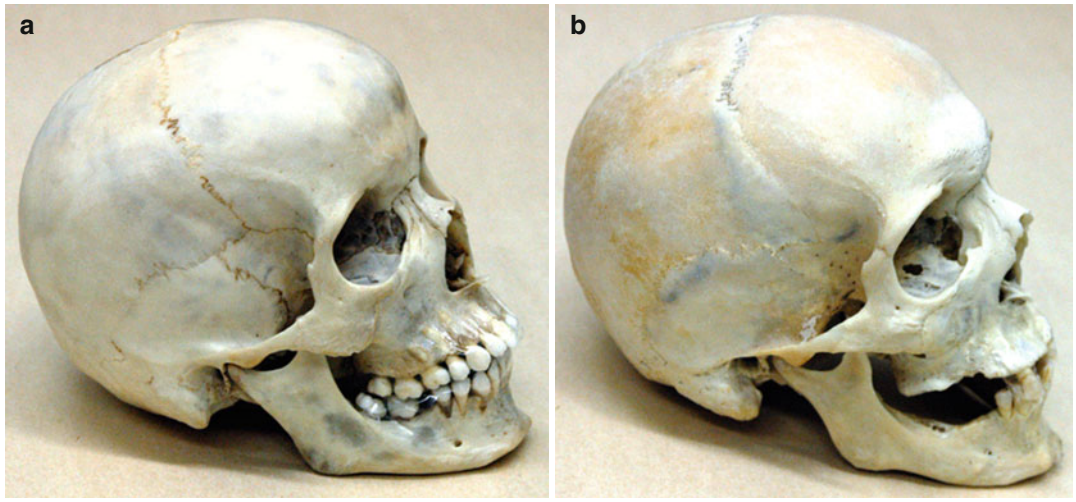

Fig. 23.10 (**a**, **b**) Skull-based sex determination. A "hyperfeminine" skull showing only female traits is seen on the left (**a**), a "hypermasculine" skull on the right (**b**). These images differ from the standard planes used in order to show as many traits as possible at one time (see also Table 23.2) (Images kindly provided by PD Dr. Karl-Heinz Schiwy-Bochat, Cologne)

Important: The accepted rule when determining sex from the entire skeleton is that less-pronounced or smaller traits are indicative of a female individual. The frontal and parietal eminences, which are more readily visible or indeed visible at all on the female skull compared to the male skull, are the only exception to this rule.

Areas of overlap in terms of degree of prominence in both morphognostically and morphometrically recorded traits can vary in size to such an extent that sex cannot be sufficiently reliably determined for forensic purposes. This is particularly the case with children, since many sex traits are not fully developed until after puberty. Altogether, by using the established morphological methods, sex can be correctly determined in approximately only 85–90 % of cases, not least due to secular trends, the relevance of population affiliation, and skeleton incompleteness.

23.5.2 Body Height

The fact that long extremity bones stand in linear relation to overall body length is used to estimate body height. There are numerous formulas based on the mathematical model of linear regression which, by determining primarily the length of intact or fragmented long bones, permit body height to be reconstructed. Several authors stress that, when choosing a regression formula, not only secular acceleration needs to be taken into consideration, but also the fact that formulas of this kind tend to be highly population- and sex-related, thereby fundamentally limiting their application to only those populations and skeleton collectives on the basis of which the regression formulas were developed. Since these calculations are estimations, giving the margin of error or a statistical confidence interval (up to a few centimeters for combined methods) is recommended. Tables 23.4 and 23.5 summarize the main regression formulas for women and men, respectively.

23.5.3 Age (Age at Death)

Anthropology and forensic medicine have at their disposal numerous empirical investigations to determine the age of an unidentified person. On the basis of these investigations, the phenomenon of human aging is correlated with the subsequent appearance of traces on the skeleton. Numerous noninvasive (macroscopy, dental status, overall

Table 23.4 Regression formulas for calculating body height from long bone measurements in women

Formula according to	Long bones	Factor	Constant	Confidence interval
Bach (1965)	Humerus	2.121	98.38	3.9
	Radius	1.925	116.89	4.5
	Femur	1.313	106.69	4.1
	Tibia	1.745	95.91	3.9
Pearson (1899)	Humerus	2.754	71.475	3.5
	Radius	3.343	81.224	4.1
	Femur	1.945	72.844	3.3
	Tibia	2.352	74.774	3.4
Rother (1971)	Humerus	1.70	96.85	3.92
	Radius	2.21	102.17	4.30
	Femur	1.11	102.63	4.03
	Tibia	1.53	96.44	4.10
Trotter (1977)	Humerus	3.36	57.97	4.45
	Radius	4.74	54.93	4.24
	Femur	2.47	54.10	3.72
	Tibia	2.90	61.53	3.66

Body height is obtained by measuring the long bone in centimeters, multiplying this value by the factor, and adding the constant. The one-sided confidence interval is given in the last column

Table 23.5 Regression formulas for calculating body height from long bone measurements in men

Formula according to	Long bones	Factor	Constant	Confidence interval
Breitinger (1938)	Humerus	2.715	83.21	4.9
	Radius	2.968	97.00	5.4
	Femur	1.645	94.31	4.8
	Tibia	1.988	95.59	4.7
Pearson (1899)	Humerus	2.894	70.641	3.3
	Radius	3.271	85.925	4.0
	Femur	1.880	81.306	3.3
	Tibia	2.376	78.664	3.5
Rother (1971)	Humerus	2.83	69.40	4.81
	Radius	3.66	74.24	4.49
	Femur	2.31	56.58	4.04
	Tibia	2.95	50.94	3.27
Trotter (1977)	Humerus	3.08	70.45	4.05
	Radius	3.78	79.01	4.32
	Femur	2.38	61.41	3.27
	Tibia	2.52	78.62	3.37

Body height is obtained by measuring the long bone in centimeters, multiplying this value by the factor, and adding the constant. The one-sided confidence interval is given in the last column

appearance, X-rays) and invasive (chemical and histological analysis of teeth or compact bone in long bones) methods are available to this end. Individual aging, lifestyle, and living conditions at the respective place of domicile create discrepancies between chronological and biological age.

Assessing age at death in adults (i.e., once epiphyseal plate ossification is complete) is a particularly challenging task in forensic osteology. There is no one answer to the question of whether combining more than one method is helpful. There is growing evidence that different methods

Table 23.6 Tooth eruption ages

Teeth	Age
71, 81	3–6 months
51, 61, 72, 82	5–9 months
52, 62	8–11 months
54, 64, 74, 84	10–14 months
53, 63, 73, 83	12–18 months
55, 65, 75, 85	16–24 months
16, 26, 36, 46	4.5–6 years
31, 41	6–6.5 years
11, 21	6.5–7 years
32, 42	7–7.5 years
12, 22	7.5–8 years
14, 24	9–10 years
15, 25, 33, 34, 35, 43, 44, 45	9.5–10.5 years
13, 17, 23, 27, 37, 47	10–11 years

The age ranges shown here do not represent absolute extremes

Table 23.7 Age ranges for complete epiphyseal fusion for both sexes given in an approximately chronological order

Epiphyseal plate	Age range, female	Age range, male
Distal humerus, proximal radius, and proximal ulna	14–17	14–18
Acetabulum	15–18	15–18
Distal tibia and distal fibula	15–18	17–19
Distal femur, proximal tibia, proximal fibula	15–19	17–20
Proximal femur	15–19	18–21
Calcaneus, lower-extremity phalanges	15–20	15–20
Distal ulna and distal radius	16–19	17–20
Lateral scapula	16–22	16–22
Upper-extremity phalanges	16–20	16–20
Ischial tuberosity	17–20	21–24
Proximal humerus	18–22	20–25
Medial scapula	19–21	20–24
Iliac crest	21–24	21–24
Medial clavicula	21–23	22–24

The values given here denote the age range in years within which complete fusion of the respective epiphyseal plates is seen at the earliest and at the latest

or method combinations in the different age groups yield the best results.

The stage of dental development represents the most important criterion for age estimations in children and up until growth is complete, followed by epiphyseal plate ossification. For this reason, the minimum and maximum ages at which the individual teeth erupt (Table 23.6) and epiphyseal ossification takes place (Table 23.7) have been determined.

Sex differences need to be borne in mind in the assessment of epiphyseal fusion. Assuming all evaluable parameters have been taken into consideration, highly accurate age determination with an error margin of only a few months is often possible in adolescents.

In adults, exogenous factors become ever more relevant with increasing age. Macroscopic assessment can include dental abrasion and intravital tooth loss, surface changes to the symphysis, skull suture obliteration (endo- and ectocranial), or degenerative processes to the musculoskeletal system.

A number of readily applicable methods are based on the fact that dental roots become increasingly transparent with age. The Lamendin method, for example, involves illuminating a single-rooted tooth using a light box, measuring the length of the translucent zone from the root apex, and comparing this length to whole-root length. Age can then be calculated from this ratio.

More complex approaches to ascertaining the chronological age of an individual involve determining the degree of aspartic acid racemization and counting growth layers in dental cement. However, the estimate accuracy determined in evaluation studies of these two methods could not be achieved either in reevaluation studies or in practical case work.

The current wisdom is that all known methods, when combined, are able to provide important information on age, whereby estimations to within ±5 years are possible when technical methods are used, and even greater accuracy is possible in younger individuals. Possible pathological processes should be assessed with a particularly critical eye.

23.5.4 Origin

Determining origin can be very helpful in ascertaining or excluding whole demographic groups

and hence also particular individuals in the identification process. Subdivision into three large demographic populations is possible primarily on the basis of skull morphology: Negroid, Caucasoid, and Mongoloid. The essential differences here are seen in the facial bones and the shape of the cranial vault. However, it is not always possible to classify a specific skull unequivocally, and the ability to differentiate between skulls will certainly be reduced in the future by increasing globalization and migration. Morphometry, i.e., measuring distances between given measuring points on the skull and creating indexes for these, can be used as an alternative to morphognostic analysis. A commercial program available in the USA (FORDISC®) suggests ethnic classification and probabilities on the basis of the measured distances entered. A distinction is made between the following ethnic groups in the USA: White, Black, Amerind, Hispanic, Japanese, Chinese, and Vietnamese.

Dental Treatment. Significant national and international differences are seen in terms of dental treatment. Therefore, it is possible to draw conclusions about the country where dental treatment took place, and hence also where the deceased originates from, on the basis of certain types of dental treatment.

Radionuclides. Global concentrations of various radionuclides in food, drinking water, and air vary widely. As a result, there are regional differences in radionuclide uptake in bone. Numerous projects are currently engaged in radionuclide mapping; the aim here is to create a radionuclide profile of discovered bones that would indicate where on the planet an individual resided for the majority of his/her life.

23.5.5 Healed Injuries

Healed injuries are the first indication of possible violent events in the course of an individual's life. Inferences about particular weapons, for instance, can suggest certain geographical regions or (earlier) periods in time. Furthermore, healed injuries can point to possibly impaired or unusual gait (e.g., a limp), which may prompt recollection of the individual in question.

23.6 Population Dependence and Reevaluation

A population dependence, which can sometimes be considerable, needs to be taken into account when classifying morphological as well as osteometric parameters or when applying metric parameters in mathematical estimation models.

Constant adjustments in forensic anthropological knowledge are essential against the background of ever-changing populations in a globalized world where modified nutritional habits, lower physical activity, and continual currents of migration all have an impact. The composition of populations (global intermixture of ethnic groups) is not the only effect to be seen in this process—for example, average heights, body proportions, external bone structure (extent of muscle attachment), and average life expectancy are also factors affected.

The consequence of the above is that known estimation formulas, cutoff values, and evaluation systems need to be constantly reevaluated through the prism of recent collectives representative of the current population and, where necessary, adjusted. However, there is only limited availability of modern reference material.

There appears to be no alternative approach to the problem other than to use data obtained from systematic recordings of traits seen on virtual bones at postmortem CT. Naturally, whether or not established morphological or osteometric parameters can be identified on virtual bones unrestrictedly needs to be critically assessed.

The next logical and necessary step in this approach is to set up a European database similar to the "forensic database" established in North America, to which unequivocally classified forensic osteological cases with known personal data are added. In addition to the sex and origin of victims, all parameters identified on the remains of each case are entered in this forensic

database. The application software *FORDISC*® widely used in North America makes use of the database to compile individual statistics using the parameters that can be obtained from a current case. This results in an individual statistic for each case, including information on the probability of gender and demographic group affiliation. A European database based on postmortem CT data sets is currently planned for FORDISC.

23.7 Establishing Identity

The most important methods for establishing identity in forensic osteology include forensic DNA analysis (see Chap. 22), forensic odontostomatology (see Sect. 6.4), comparative X-ray analysis (Sect. 6.5), and skull–photo comparison (see Sect. 6.6).

23.7.1 Forensic DNA Analysis of Bones

Special isolation methods are required to obtain DNA from bone tissue for the purposes of forensic DNA analysis. The chances of isolating DNA that is still susceptible to amplification depend on the bone's state of preservation and the corresponding degree of DNA degradation. Specific environments or fire-damaged bones, for instance, can pose significant challenges. It is nevertheless possible to create an STR profile in many cases. This profile may already be helpful at the biological-profile creation stage: the STR profile is compared with the Federal Office of Criminal Investigation's DNA database; if the victim's profile is in the database, a match is made and, following further verification, may lead directly to establishing identity. Alternatively, STR profiles may be found among missing person profiles and can be directly compared. The most important application of forensic DNA testing, however, is during the identification phase in cases where an antemortem STR profile matching the ascertained biological profile is obtained from a putative victim, e.g., using cells taken from a toothbrush, and compared with the postmortem profile obtained from bone. If no items belonging to the putative victim are available, a comparison can be made with close relatives.

23.7.2 Dental Status

Recording dental status is a fundamental technique used in forensic odontostomatology. A record is made of missing and present teeth, as well as their particular characteristics, using a recognized nomenclature. Dental treatment and the surfaces affected are described in detail for each tooth. Postmortem dental status is useful to investigating authorities in that it can be used for comparison with possible previous findings, i.e., a missing person's antemortem dental records.

Forensic odontostomatology holds its position of prominence in the identification process by virtue of the fact that a dental record is compiled every time someone visits the dentist, meaning that antemortem data for comparison purposes are available for most people. This assumption was verified during the tsunami disaster of 2006 when most German victims were identified using odontostomatologic methods.

23.7.3 Comparative X-Ray Analysis

Comparative X-ray analysis represents a method that has been successfully applied to identity determination. It shares a similar background to odontostomatology in that many people have been X-rayed for medical indications in the course of their lives and these images may be available as antemortem reference material. When generating postmortem comparison X-rays of bones, attempts are made to reconstruct the beam path as precisely as possible. Anteroposterior skull X-rays or dental X-rays (orthopantomogram, digital intraoral X-rays) are particularly well suited to this end. CT data sets can also be used for postmortem comparison:

virtual X-ray images with a freely selectable beam path are reconstructed from CT data by comparing these directly with antemortem radiographs using maximum intensity projection techniques.

23.7.4 Skull–Photo Comparison and Forensic Facial Reconstruction

Since facial photographs are available for many people, *skull–photo comparison* offers one option for establishing identity. This approach is based on attempting to "fit" a skull onto a portrait photo at the correct angle, thereby creating a superimposition. As part of this process, spacers are placed at defined points on the skull to represent average soft tissue thicknesses, which should subsequently correspond to the soft tissue margins on the facial photograph.

Forensic facial reconstruction is based on the same principle. In addition to the classic manual approach of applying soft tissue by hand to a cast taken of a skull according to the spacers denoting average soft tissue thickness, methods using graphics and, more recently, digital techniques have also been developed.

23.8 Traces of Injury

Injuries incurred around the time of death (perimortem injuries) are generally considered relevant in terms of cause of death. They need to be differentiated from injuries incurred—and survived—during life (antemortem injuries). Lesions produced after death form the largest group of injuries. Experiences gained in paleopathology and forensic medicine complement one another well.

23.8.1 Postmortem Changes

Postmortem changes occur as a result of intentional and non-intentional removal by either animals or humans, the process of recovering bones, e.g., dig marks, as well as a multitude of soil and surface conditions during the PMI.

Fig. 23.11 Postmortem lesions (dig marks). Ventral view of a proximal femur recovered from a medieval burial field. The lesions were caused by an excavation tool during recovery. The light-colored and rough fracture surfaces are readily apparent

The most important differential diagnostic criterion in postmortem changes is the color of cut or fractured surfaces, which are usually distinctly lighter compared with other bone surfaces (Fig. 23.11). In addition, the absence of signs of decomposition on cut or fractured surfaces, despite visible decomposition on other bone tissue, suggests a postmortem origin.

As long as bone still possesses fresh biomechanical properties at the time of injury, early postmortem bone lesions may fail to demonstrate any of the morphological criteria typical of postmortem origin. In some cases, however, once the circumstances have been reconstructed, origin can be classified only as postmortem, such as postmortem defects caused by animal scavenging (Fig. 23.12).

23.8.2 Antemortem Changes

In order to diagnose antemortem bone injury, signs of healing and bone remodeling need to be present, such as callus formation following long bone fractures (Fig. 23.13). However, indirect bone changes are also seen following soft tissue injury: hematoma clearance can produce marks on the surface of the bone via vascularization. Reactive osteogenesis can also occur due to inflammation and soft tissue destruction.

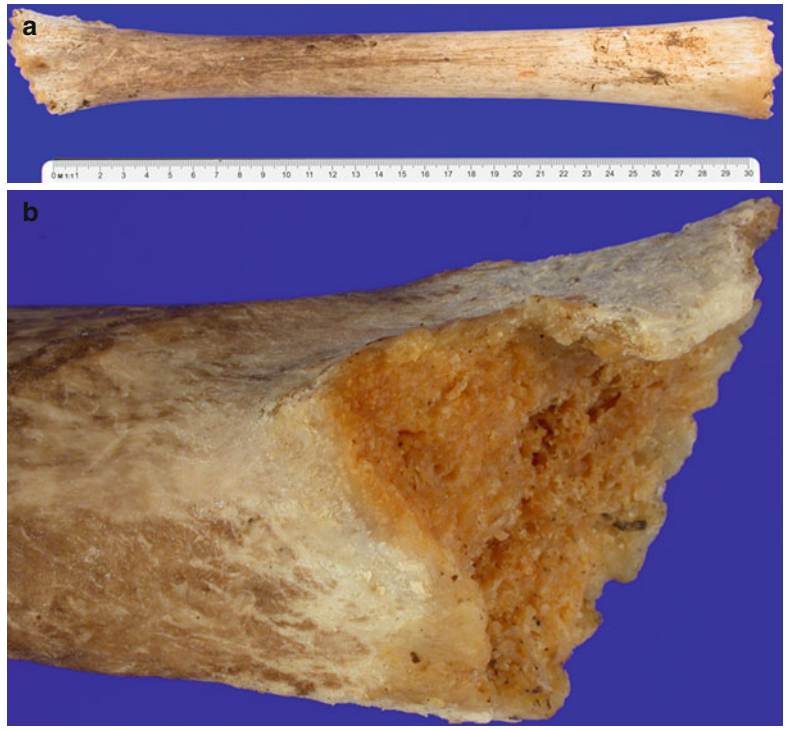

Fig. 23.12 (**a**, **b**) Defects caused by wild boar scavenging. (**a**) Dorsal view of a left human femur, the epiphysis is absent; rough yet rounded-off borders of the adjacent epiphysis due to scavenging are seen. (**b**) On the close-up ventral view of the proximal end, the exposed medulla is fatty, and light-colored soft tissue remnants are seen on the bone surface. An incomplete skeleton was found in the forest; all long bones recovered demonstrated defects of this kind, which are typical of wild boar scavenging. Identity was established using dental records. The 26-year-old man had hanged himself in the forest approximately 4 weeks prior to discovery (mid June)

Fig. 23.13 Antemortem injury. Ventral view of a left femur. This long bone fractured at the transition from the mid to distal third of the shaft healed in malalignment on all levels with distinct callus formation. The bone is a medieval find demonstrating a defect which, thanks to modern trauma surgery treatment, would not be seen today

23.8.3 Perimortem Changes

In principle, all traces of injury that cannot be identified as either ante- or postmortem need to be classified as perimortem injuries. From a forensic osteological perspective, the possibility that perimortem injuries occurred in close temporal relation to death cannot be ruled out. Depending on the localization and severity of injury, it is important to consider whether it is a possible cause of death or associated with death in some other way. Cut and fractured surfaces of

perimortem origin generally demonstrate the same coloring as other bone surfaces; signs of decomposition are also comparable.

If a perimortem origin of a bony injury cannot be excluded, the underlying mechanism of injury needs to be analyzed (Sect. 8.1). The first step is to determine the type of trauma inflicted on the bone. Overlaps in types of injury, as well as multiple injuries, may be seen (see Table 8.3). However, findings on the body surface and in soft tissue are not available—the forensic osteologist only has bone findings at his disposal.

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Forensic Radiology

24

Case Study

A clinical forensic examination of the victim of a stabbing attack that had taken place the previous evening was ordered for early in the morning. The examination was intended to establish the number, severity, and lethality of wounds, as well as to identify possible active or passive defense wounds.

The examination took place approximately 12 h following the incident. All wounds had been treated surgically, some expanded intraoperatively, others brought together. In addition, a laparotomy had been performed. As a result, any evaluation of stab or incised wounds was no longer possible. The surgeon gave a report on the injuries that had been present and how he had treated each fissure. Only one stab wound to the left side of the chest had penetrated more deeply and injured the left hepatic lobe. However, it was not possible to reconstruct the full pattern of injuries. The coroner asked his surgical colleague whether the original findings had been photographed, to which the latter responded that this was not the case since the memory card for the camera had been missing.

On looking though the file, it became apparent that no explorative laparothoracotomy had been performed on admission to the hospital, but that a CT scan of the chest and upper abdomen had been performed instead. A copy was made of the DICOM data. Following 3D reconstruction using volume rendering, a total of six stab/incised wounds could be visualized and the stab wounds measured. Using the oblique mode, it was possible to reconstruct the wound track originating at the left side of the chest and extending to the liver. Ultimately, the CT data set was able to provide answers to all medicolegal questions.

The term "forensic radiology" is relatively new, and there is still controversy among experts as to whether it is a field of forensic medicine in its own right. X-rays were used in forensic medicine as early as in the year following their discovery: in 1896, Arthur Schuster (Lancashire, England) localized four projectiles in the head of a 22-year-old woman, who died of her injuries a few days later. Her husband was found guilty of her murder. In the same year, radiographs were used in England and the USA as evidence in court. Today, the spectrum of diagnostic radiology has broadened significantly. Since the term "radiology" refers in actual fact only to the radiation-related process, there is discussion as to whether "radiology" should be renamed as "medical imaging." Where this the case, the term "forensic imaging" would be used in forensic medicine.

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The basic principle of forensic radiology is an interdisciplinary collaboration between forensic medicine and radiology. Its field of application includes both ante- and postmortem diagnosis. Table 24.1 provides an overview of imaging techniques used in forensic medicine.

Table 24.1 Imaging techniques and examples of their application in forensic medicine

Technique	Possible applications
X-rays	Postmortem diagnosis, as an adjuvant to autopsy Body identification Injury evaluation in living subjects Forensic age estimation in living and deceased subjects
Computer tomography	Postmortem diagnosis, as an adjuvant to autopsy Injury evaluation in living subjects: only if already available in a clinical context Forensic age estimation in living subjects (medial clavicular epiphysis): in criminal proceedings and to exclude underage status in asylum procedures
Magnetic resonance imaging	Postmortem diagnosis, as an adjuvant to autopsy Soft tissue injury evaluation in living subjects, particularly following alleged neck trauma
Ultrasound	Post-mortem, primarily as an adjuvant to autopsy, e.g., extent of hematomas To examine hematomas in living subjects Forensic age estimation in living subjects (still experimental)
(Digital) photography	To document the entire spectrum of morphological findings in forensic medicine
3D surface scanning	Three-dimensional imaging of the surface of human bodies, parts of the body, or objects (weapons) for digital reconstruction, e.g., matching weapons or bite wounds In combination with other 3D data sets (MSCT or MRI)
Photogrammetry, 3D laser scanning/ photography	Three-dimensional surface imaging of large objects or spaces For the purposes of reconstruction in combination with 3D data sets mentioned above

Postmortem imaging encompasses all imaging techniques suited to postmortem assessment of findings, documentation, and diagnosis, as well as to establishing the cause of death in particular. Techniques can be applied before, during, and after autopsy. For more detailed information on photography and surface scanning techniques, the reader is referred to the relevant specialist literature.

24.1 Postmortem X-Rays

Radiography remains the most important tool in postmortem imaging.

Important: **Radiographs are able to document findings lost due to dissection at autopsy, and radiological diagnosis is able to assist in the approach to dissection at autopsy**.

Thus, a pneumothorax imaged prior to autopsy will influence the dissection approach (pneumothorax probe), while at the same time providing documentation of the pneumothorax (Fig. 24.1).

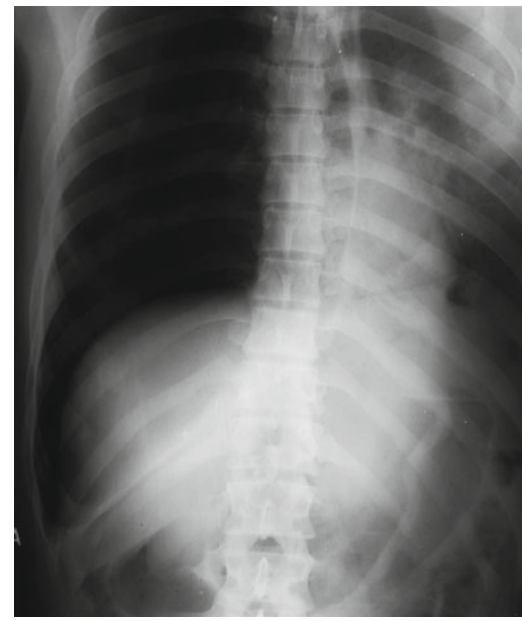


Fig. 24.1 Tension pneumothorax. Postmortem anteroposterior X-ray of the chest and upper abdomen. On the right, a tension pneumothorax is seen with a cardiac silhouette displaced to the left

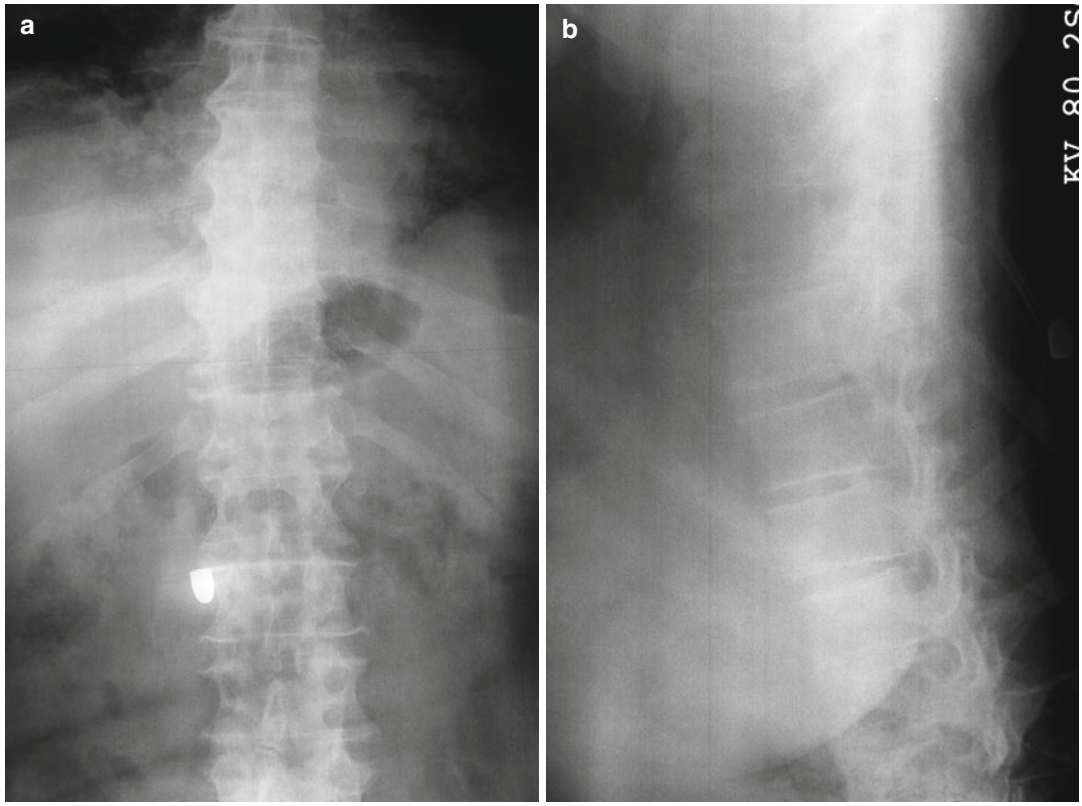


Fig. 24.2 (**a**, **b**) Visualization of a projectile following an abdominal gunshot wound. (**a**) On suspicion of an abdominal gunshot wound, only an anteroposterior image was obtained prior to autopsy. On this image, the projectile appeared to lie directly adjacent to the second lumbar -vertebra. On opening the abdomen, the projectile could not be located. (**b**) A lateral view was then taken of the lumbar spine: the projectile, visible in the mid portion on the right-hand side of the image, was found in the strongly developed autochthonous back muscles

Gunshot Wounds. If one or more projectiles are lodged in the body, locating these is an important task at autopsy. Two-plane X-rays can provide valuable guidance here and are vital on occasion (Figs. 24.2 and 24.3).

Particularly in the case of multiple gunshot wounds caused by different weapons or shotgun shells, orientation using X-rays or CT taken prior to autopsy is often indispensable (Figs. 24.4 and 24.5).

Bone Fractures. Old healed bone fractures in particular may be overseen at autopsy. Even recent bone fractures in infants and young children often remain undetected due to tissue elasticity and the absence of crepitation. It is also for this reason that full-body X-rays of infants' and young children's bodies are recommended prior to autopsy. Examples of fresh and healed bone fractures in infants are shown in Figs. 24.6 and 24.7.

24.2 Postmortem Computer Tomography

Postmortem computer tomography (CT) permits the entire body to be visualized in rapid-succession cross-sectional slices. The radiation protection necessary in living subjects is irrelevant in deceased subjects. Multi-slice CT (MSCT) additionally permits reconstruction using 3D data sets (Fig. 24.5a).

As early as the turn of the millennium, the Berner Institute of Forensic Medicine with its

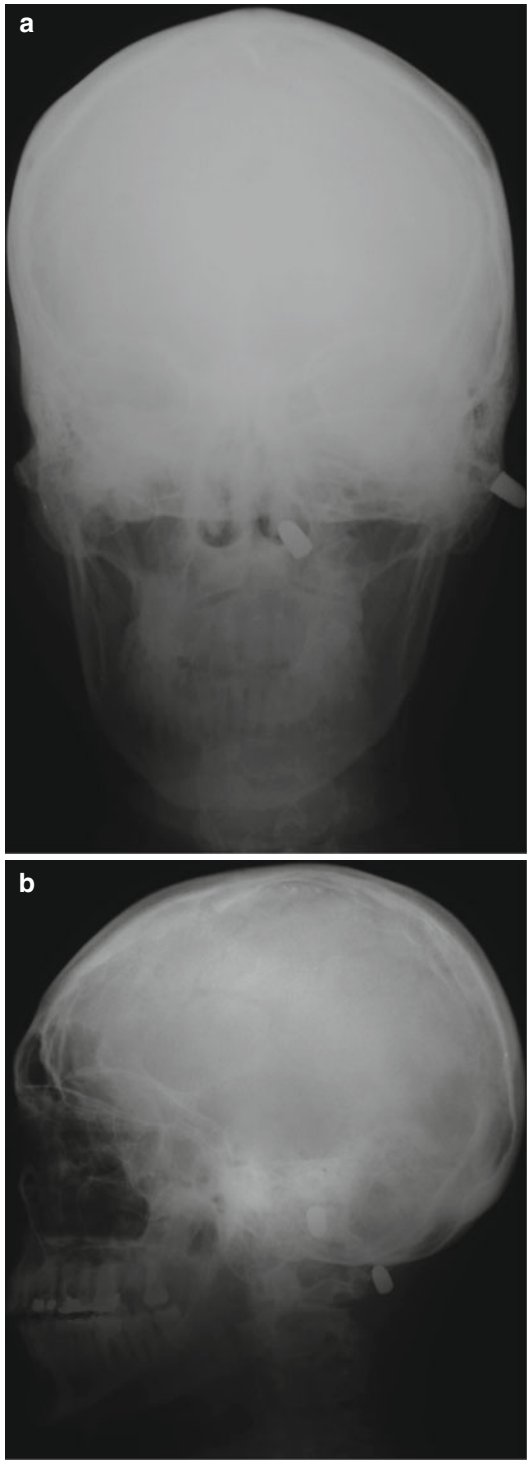


Fig. 24.3 (**a**, **b**) Two lodged bullets following gunshot wounds to the head. Accurate localization of the bullets could only be achieved with images in two planes

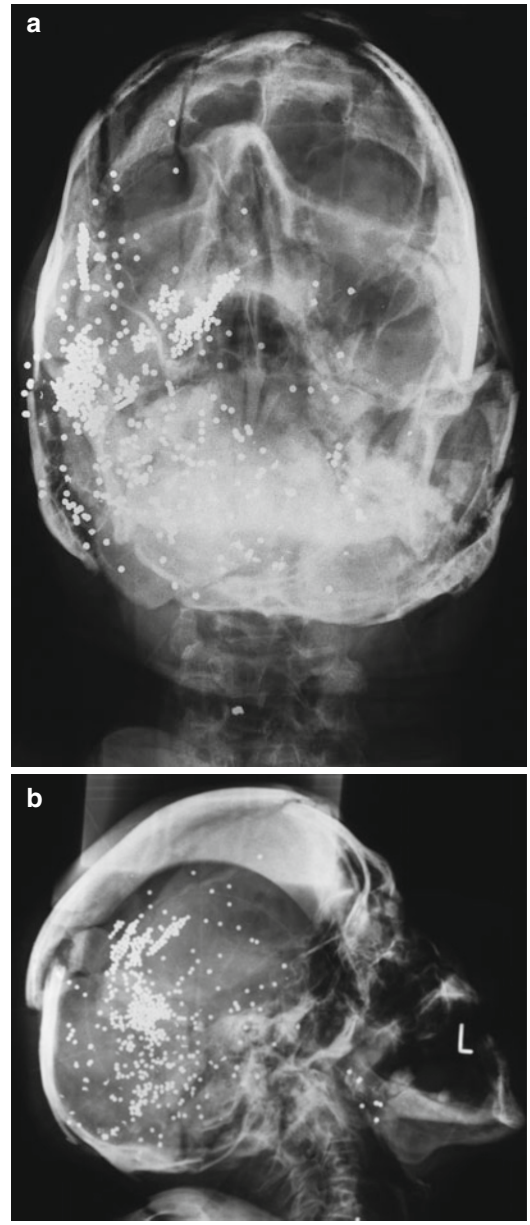


Fig. 24.4 (**a**, **b**) Suicidal shotgun shell wound. Suicide with a shotgun, one shot behind the right ear, blast wounds to the cerebral and facial skull bones

working group Virtopsy (www.virtopsy.com) was pioneering postmortem CT. As part of their project, two independent teams examined deceased subjects. The first team (a radiologist

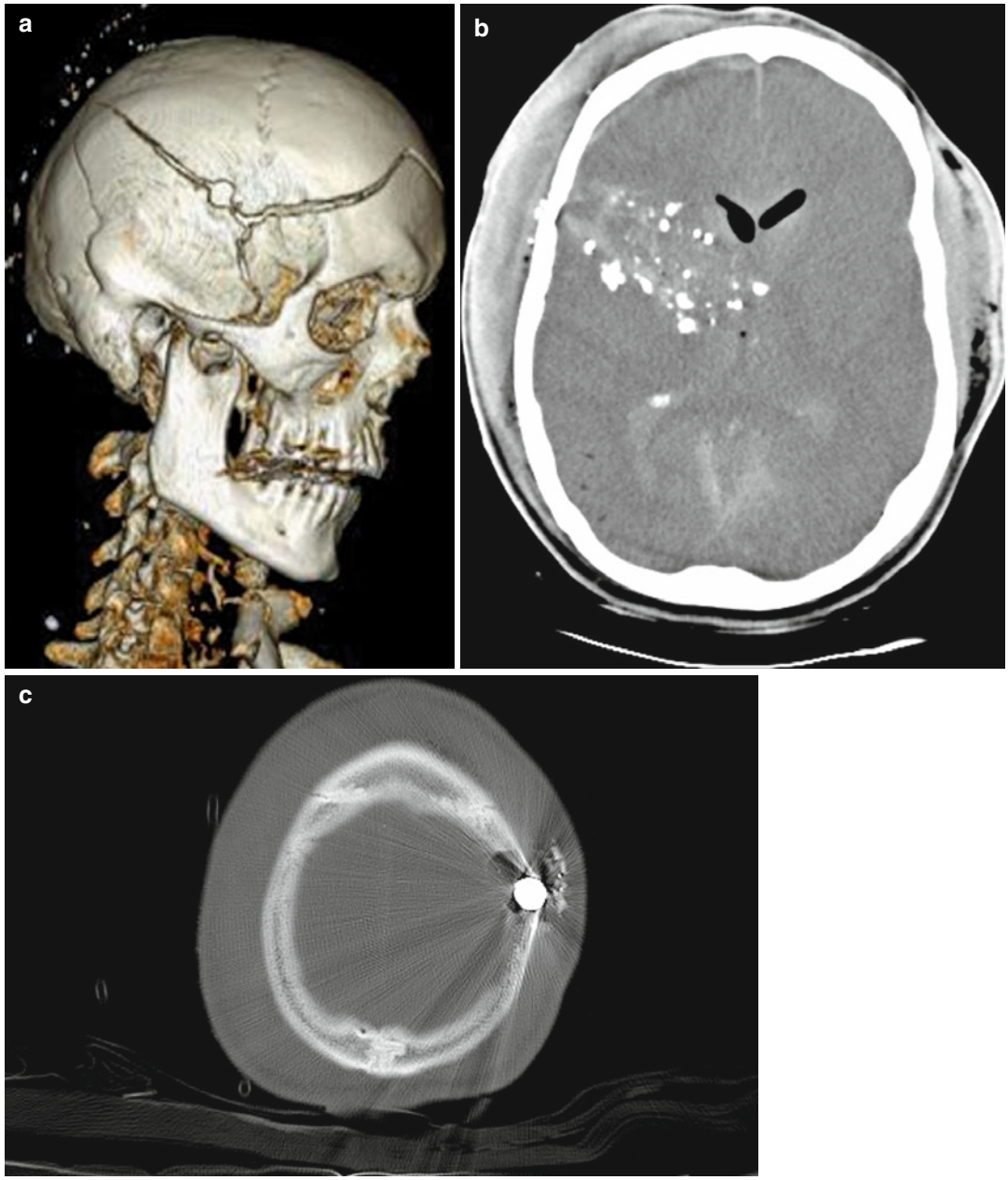


Fig. 24.5 (**a**–**c**) Postmortem-CT: A bullet lodged in the cranium with (**a**) cranial bone fractures originating from the point of gunshot entry (3D data reconstruction), (**b**) spread of bony particles from the point of entry to cerebral tissue, and (**c**) the final position of the projectile

and a forensic pathologist) had only postmortem CT data as their disposal, complemented where necessary by MRI and 3D surface scans. The second team depended exclusively on autopsy findings (including radiological data in a second phase of the project). The two teams' diagnoses were compared after examinations. The results of the project showed that both procedures have

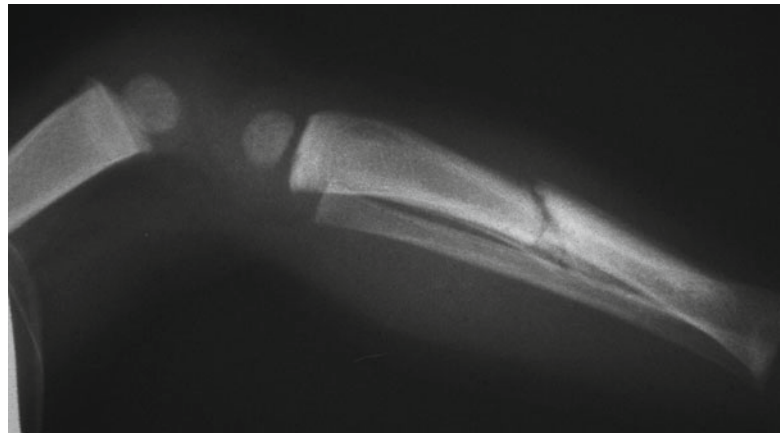


Fig. 24.6 Wedge fracture. Lower leg of a 6-month-old infant with a so-called wedge fracture in the mid-tibial shaft. No increase in lower leg mobility was seen due to the intact fibula

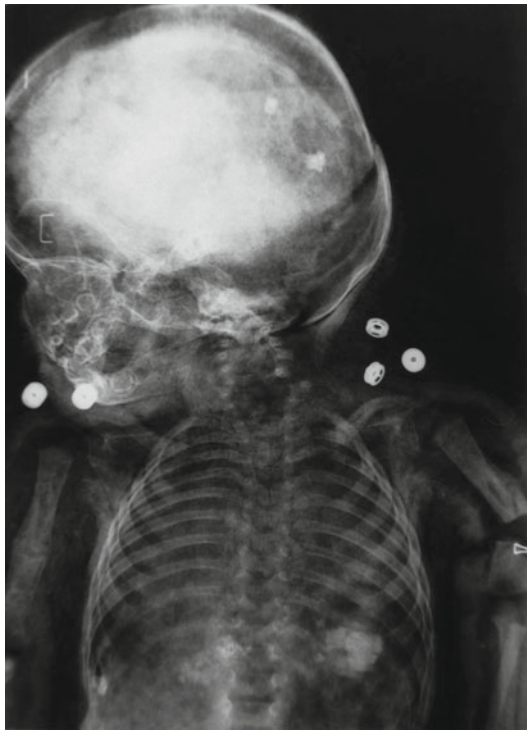


Fig. 24.7 Battered child syndrome. The body of a 9-month-old infant. The left humerus shows a fresh fracture, the right a healed fracture in the midshaft

their strengths and weaknesses and that diagnostic quality is only increased when one procedure is complemented by the other. The initial belief that virtual autopsy could replace real autopsy examinations was quickly abandoned. Figures 24.8 and 24.9 highlight how challenging the interpretation of postmortem CT findings can be.

3D Reconstruction. *Radiopaque structures*, especially bones, are *particularly suited* to 3D visualization using surface reconstruction. Thanks to "windowing," only those voxels with Hounsfield unit values within the given minimum and maximum value ranges are displayed. In this way, "interference" from soft tissue structures can be "hidden."

3D visualization of soft tissue and internal organs using (native) CT scanning is challenging, if not impossible. One solution to this problem is the use of contrast medium, which is distributed by attaching the body to a heart–lung machine.

3D bone visualization using postmortem CT data sets enables a 3D representation of bone injuries (Figs. 24.5a and 24.10). Also, these "virtual" bones can be used for anthropological investigations and osteometric analysis. Moreover, it is sometimes helpful to perform CT investigations of individual parts of the body or even an isolated skull (Fig. 24.11).

24.3 Postmortem Magnetic Resonance Tomography

Postmortem MRI offers significantly better soft tissue visualization than CT; however, it is less well suited to displaying bones. Although it is

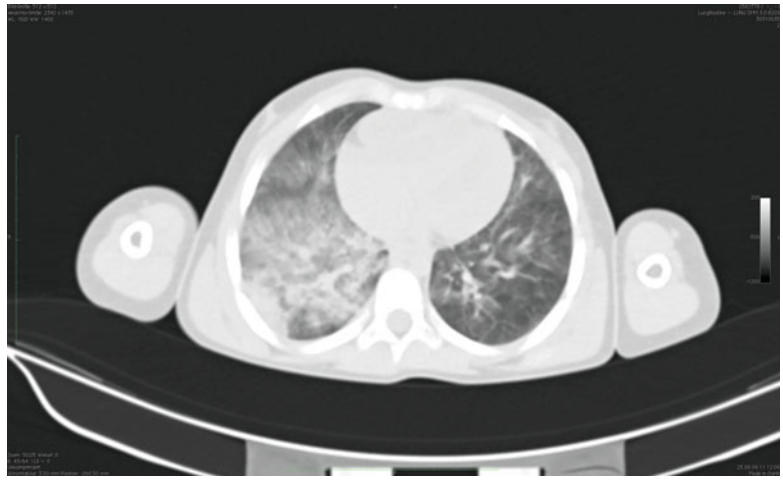


Fig. 24.8 Postmortem computer tomography: suspected pneumonia. The 6-year-old girl's case history pointed to a swimming-pool drowning. Attempts at cardiopulmonary resuscitation were unsuccessful. No signs of drowning were seen at autopsy, and no cause of death could be identified macroscopically. An opacity in the right lung, seen on postmortem CT scan performed prior to autopsy, was classified as pneumonia, mainly due to the fact that it was unilateral. In the light of negative forensic toxicological analysis including blood alcohol determination, the cause of death was concluded to be immersion syndrome and the changes to the right lung seen radiologically the result of resuscitation

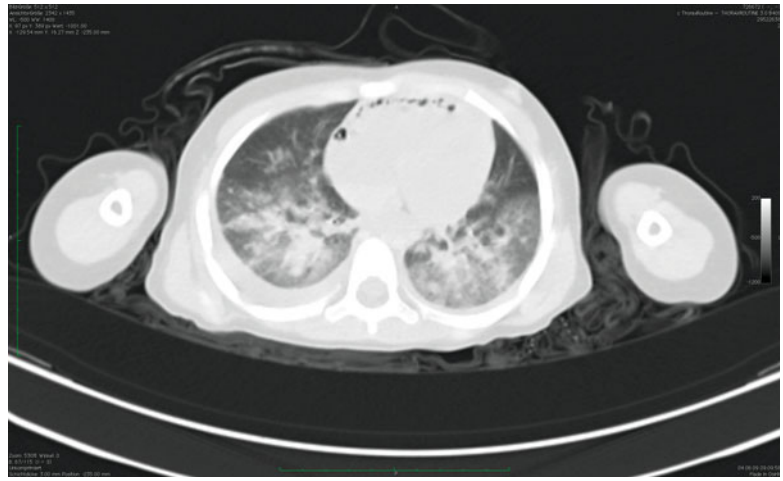


Fig. 24.9 Postmortem computer tomography: pneumonia as an unexpected finding. Macroscopically, autopsy was unable to establish the cause of death in a 3.5-year-old girl. Symmetric dorsal opacities seen on both lungs on postmortem CT were interpreted as postmortem changes ("internal livor mortis"). Microscopically, extensive bilateral bronchopneumonia was found to be the cause of death

often possible to visualize hematomas, small hematomas in subcutaneous fatty tissue that are often relevant in forensic medicine often escape detection due to the limited resolution of MRI. In practice, postmortem MRI is used only as an adjunct to MSCT.

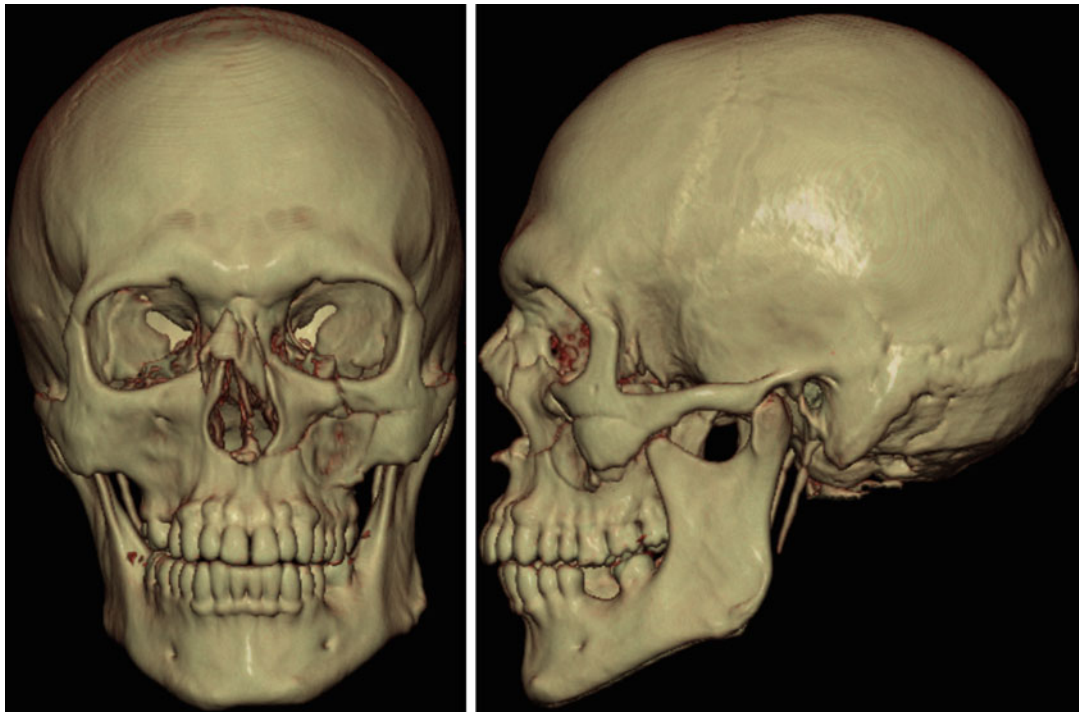


Fig. 24.10 3D representation of a skull using postmortem CT data sets. A 16-slice computed tomography scanner was used with a slice thickness of 0.625 mm. Depending on the workstation used, 3D reconstruction and visualization is referred to as "volume rendering" or "surface rendering." Nasal bone and midface fractures are clearly visible

24.4 Imaging in Clinical Forensic Medicine

X-rays—as well as CT data to an increasing extent—obtained on the basis of clinical indications for diagnostic purposes are usually used for forensic assessment. This takes place either immediately at the time of clinical forensic examination in the clinic or at a later point in time if a subsequent assessment needs to be made solely on the basis of the investigation file and medical records.

Radiography in Clinical Forensic Medicine. A "whole-body X-ray" of an infant or young child in the case of suspected child abuse and for the purposes of detecting or excluding both recent and old injuries caused by (previous) trauma represents the only purely forensic indication for radiography. Cases of this kind need to be decided on an individual basis, whereby the well-being of the child is weighed up against radiation protection. If the decision is taken to proceed with X-rays, these will be ordered in the clinical setting.

In cases of acute injury to children seen in the context of suspected abuse, X-rays are only indicated clinically if they are necessary to therapy.

Computer Tomography in Clinical Forensic Medicine. The possibilities offered by CT in clinical forensic medicine depend to a great extent on the availability of data sets obtained on the basis of clinical indications. Given its level of radiation exposure, it is inacceptable to use CT to document findings in children, adolescents, and young adults purely on forensic grounds. From a forensic imaging perspective, the case study given at the beginning of this chapter represents a justified exception, since relatively high-resolution CT images were obtained on the basis of a clinical indication which then affected the therapeutic approach.

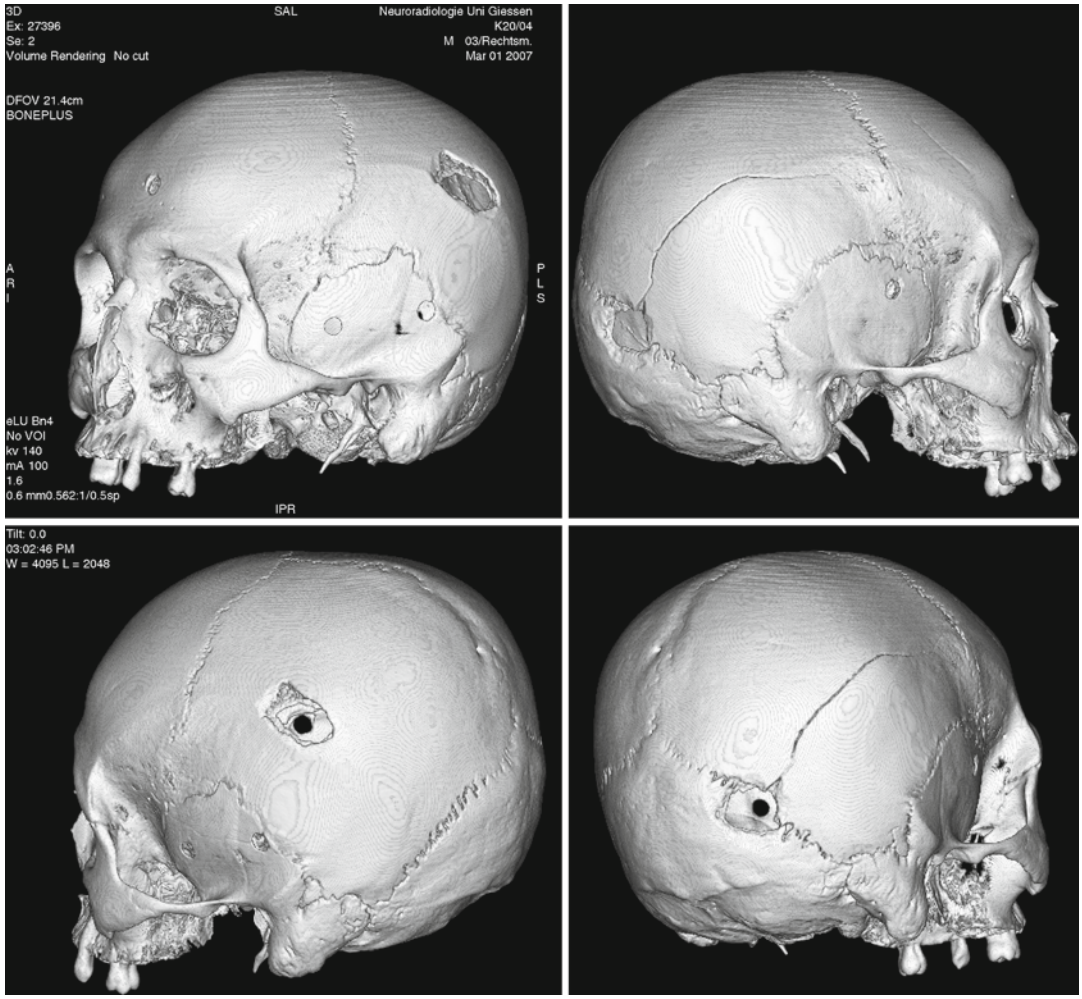


Fig. 24.11 Postmortem CT to evaluate gunshot wounds. Isolated-skull images showing gunshot wounds were obtained using MSCT. The upper images show four gunshot entry wounds (2× left temporal, 1× frontal and 1× right temporal) and two gunshot exit wounds (left parietal and right parieto-occipital). On the freely rotatable virtual skull in the lower images, gunshot exit wounds are aligned with the corresponding gunshot entry wounds

Figure 24.12 shows early postoperative findings and Fig. 24.13 a reconstruction of the severest of the six stab/incised wounds.

Using high-resolution MSCT data sets is not the only method that contributes to clinical medicolegal reconstructions—sequential images, as used in neuroradiology to visualize intracranial hemorrhage or brain contusions, can also be reconstructed three-dimensionally, although some distortion artifacts are unavoidable. Nevertheless, images of this kind are suitable for gaining an overview of the extent of injuries and for presentation in court (Fig. 24.14).

Magnetic Resonance Imaging in Clinical Forensic Medicine. Magnetic resonance imaging (MRI) offers two advantages over CT and conventional X-rays: firstly, it permits better visualization of soft tissue structures, and secondly, it bears no risk of radiation exposure. MRI may be used, for example, in the setting of alleged neck trauma. In general, it is possible to visualize hematomas in subcutaneous fatty tissue and muscles, as well as measure their depth and extent. Since radiation protection is unnecessary, an investigation can be carried out purely on the basis of a forensic indication, i.e., as part of a

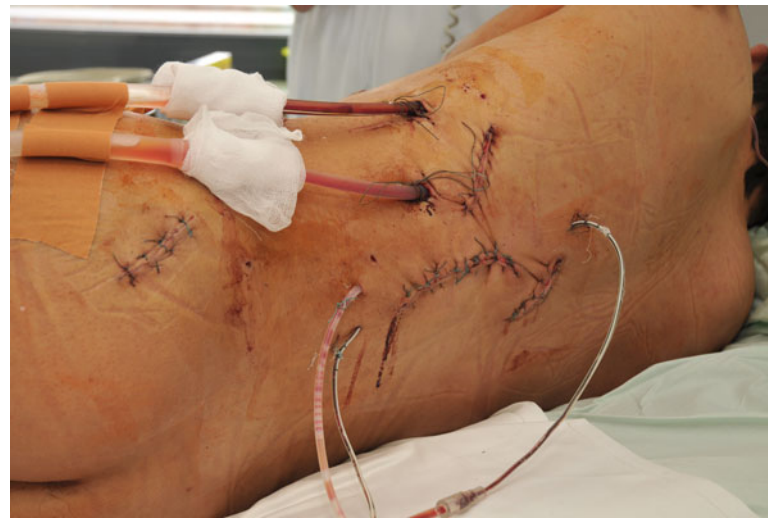


Fig. 24.12 Postoperative findings following six stab/incised wounds. The patient's status at the time of forensic examination 12 h after the incident (see "Case Study" above) did not permit an assessment of the original six stab/incised wounds to be made

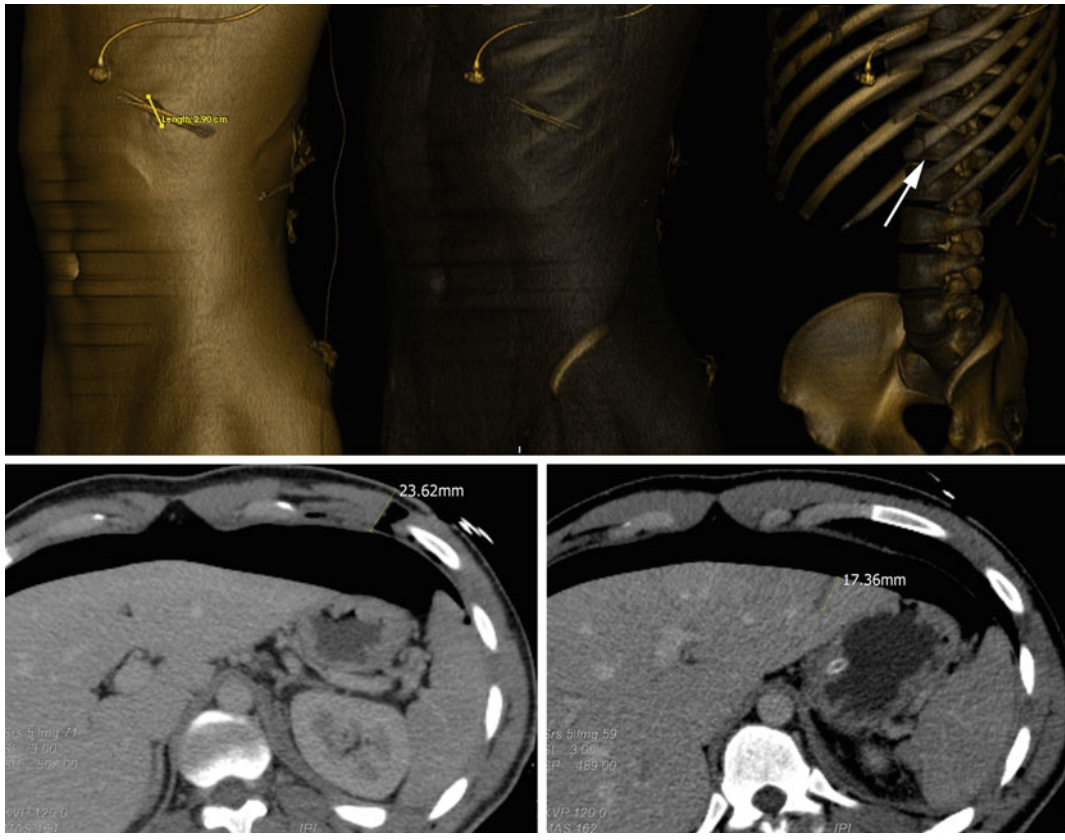


Fig. 24.13 CT visualization of a stab wound and wound track. The upper images show the stab entry wound to the left side of the chest in volume-rendering mode. The window on the left-hand side is the widest, displaying the skin surface in order to evaluate wound morphology and size. In a slightly narrower window (*center*), the stab entry wound can be localized to the sixth intercostal space. Sharp dissection of the seventh rib is clearly visible in the narrowest window (bone window) on the right-hand side (*arrow*). The lower images are cross-sectional images produced from the same data set, which following 3D reconstruction enable free selection of the sectional plane. The image on the left shows the point of skin incision at the level of the seventh intercostal space, as well as subcutaneous trapped air (beginning of the wound track) somewhat more medially. The right-hand image shows a more cranial plane where the wound track advances to the left hepatic lobe. Wound track "displacement" is due to positional artifacts in CT scanning

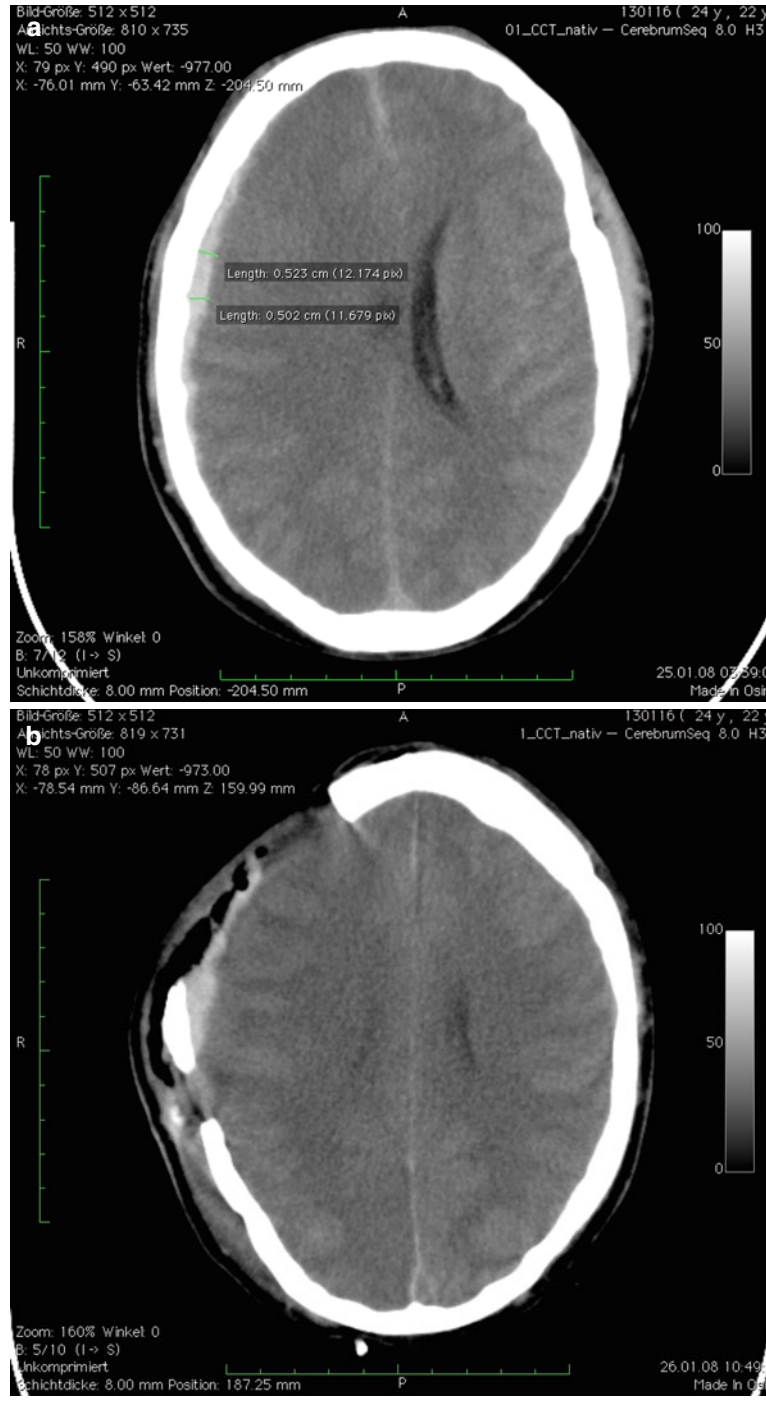
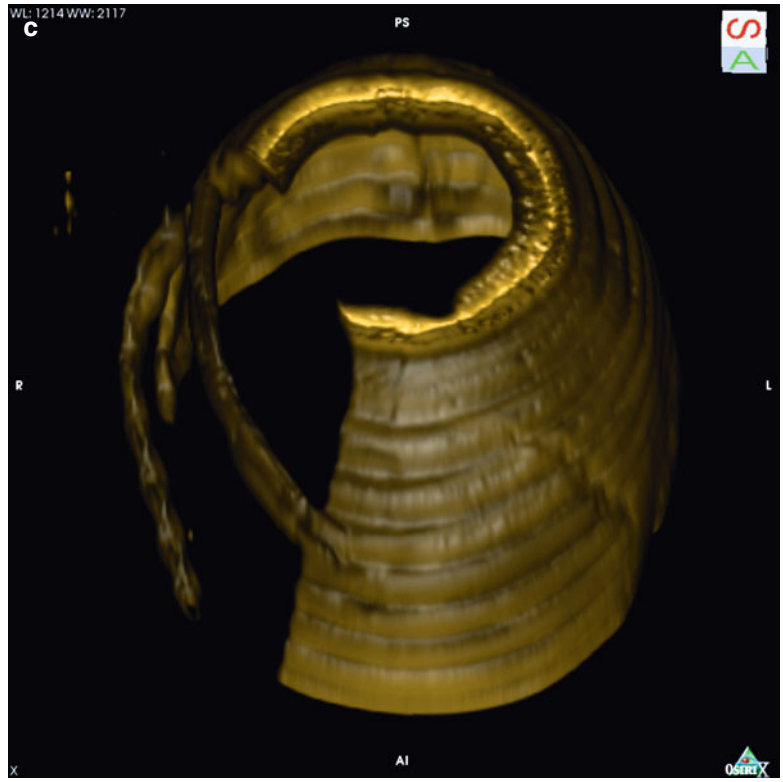


Fig. 24.14 (**a**–**c**) Subdural hematoma and status following trepanation. A 24-year-old man was evidently subjected to hours of abuse involving punches and kicks to the head. (**a**) A sectional image from the diagnostic findings on admission of a right temporo-parietal subdural hematoma. Midline brain shift and significant contralateral soft tissue swelling can be seen. (**b**) The patient's status immediately following trepanation. This data set comprising only ten images was used to generate image (**c**) using volume rendering. The cranial vault defect caused by trepanation is well demonstrated in this mid-frontoparietal view. The virtual object can be rotated freely on the computer

specific line of criminal inquiry. However, since in the absence of a clinical indication the costs of an MRI examination need to be borne by the investigating authorities, use of the method is currently limited.

Ultrasonography in Clinical Forensic Medicine. In addition to representing a cost-effective and radiation-free method, ultrasonography also yields immediately available results. The method enables, for example, good visualization of superficial

Fig. 24.14 (continued)



hematomas. However, its resolution is poor and precise measurements are not possible due to the dynamic nature of the method. Nevertheless, ultrasonography is able to provide approximate information relating to the extent of subcutaneous injuries.

Radiographic Evaluation and "Body Packing." "Body packers," swallowers, or mules are those people who smuggle illicit drugs internationally. These drugs are concealed for transport in capsules, condoms, balloons, plastic bags, or fingers of latex gloves in various anatomical cavities or body orifices (Fig. 24.15). The mouth, rectum, gastrointestinal tract, ear, vagina, and foreskin have been reported to be sites for drug concealment. The drug containers endanger carrier because of the risk of leakage or rupture with subsequent absorption. Acute poisoning due to a leakage of illegal drugs concealed in body cavities is called "body-packer syndrome." Though management of these patients is controversial, radiographs of the abdomen have become an essential part of their work-up.

24.5 Identification

This section underlines the particular relevance of X-ray analysis in the identification process. Postmortem X-rays of fresh and decomposed bodies can yield findings which may not have been visible or securable at autopsy and which can assist the identification process (Fig. 24.16).

Using maximum intensity projections (MIPs), X-rays can be generated from CT data sets with the advantage that the beam projection can be freely selected and thus adapted to antemortem X-rays for the purposes of direct comparison. One disadvantage lies in the relatively low resolution of X-rays generated from CT data sets.

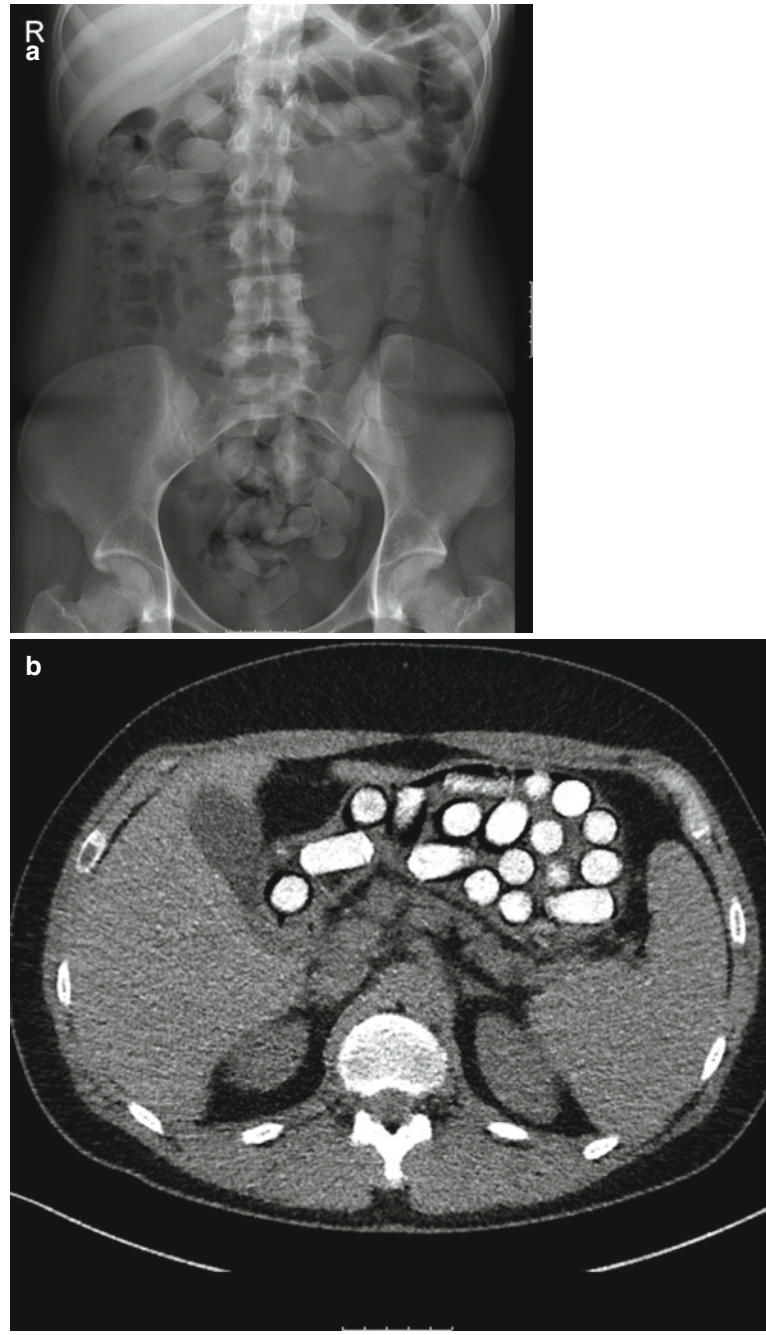


Fig. 24.15 (**a**, **b**) Body-packer syndrome. (**a**) A plain abdominal X-ray shows multiple foreign bodies in the gastrointestinal tract. (**b**) Computed tomography of the abdomen demonstrates multiple packets in the colon (Figures kindly provided by Prof. Antoch, Düsseldorf, Germany)

Using high-resolution CT, e.g., flat-panel CT (not authorized for use in living patients due to radiation exposure), it is possible to reconstruct postmortem X-rays that can be used for comparison with antemortem dental spot films (Fig. 24.17).

24.6 Forensic Radiological Age Estimation

Radiology plays an important role in forensic age estimation in living subjects undergoing criminal proceedings. In addition to the required

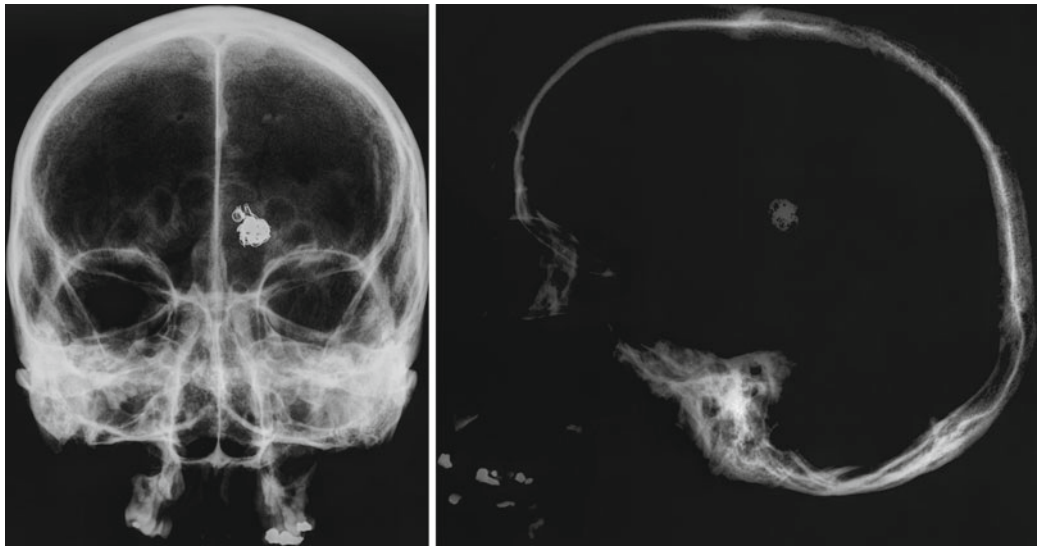


Fig. 24.16 Signs of a neuroradiological intervention as an indication of identity. The body of this unknown male was severely decomposed. The already isolated head was X-rayed in two planes and was found to be status post coiling of a left-handed vessel in the circle of Willis. With this information, it was possible to investigate a missing 65-year-old male. Primary opening of the skull may well have caused destruction of the finding due to putrefactive changes to the brain. Dental records were used to establish the man's identity

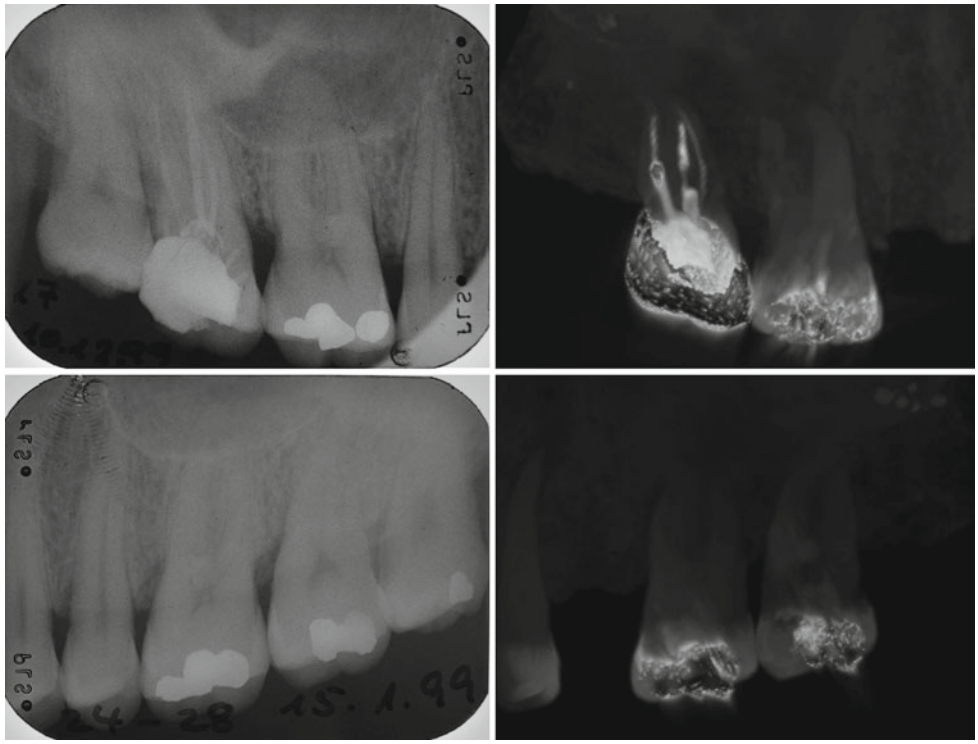


Fig. 24.17 Comparative X-ray analysis of antemortem dental spot films and postmortem CT. Images on the left show the dental spot films made 10 years prior to the discovery of the body; the upper image is the first quadrant and the lower the second. The upper jaw region of the skull was scanned using flat-panel CT and the two images on the right reconstructed using maximum intensity projections (MIPs). Positive identification could be made despite significant jaw modifications and tooth loss over the intervening decade

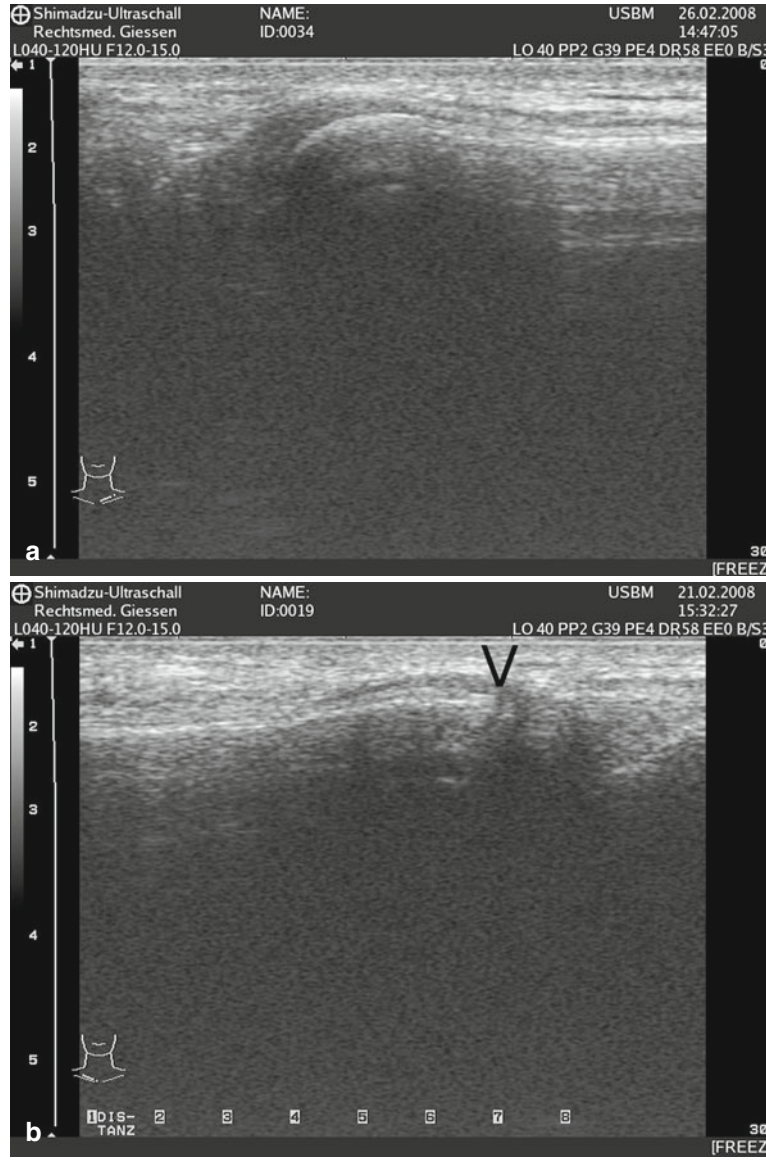


Fig. 24.18 (**a**, **b**) Ultrasound examination of the medial clavicular epiphysis. (**a**) A fully ossified left medial clavicle. (**b**) A right clavicle with a distinctly visible medial epiphyseal plate

clinical/physical examinations, an X-ray examination of skeletal development of the left-hand wrist, an orthopantogram (dental development), and, in the case of complete skeletal development, an X-ray or preferably a CT examination of the clavicular bone to evaluate medial clavicular epiphyseal ossification can be used. In the light of radiation protection and possible applications outside the context of criminal proceedings, attempts are made to substitute these methods with radiation-free methods, such as MRI or ultrasound (Fig. 24.18).

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Special Case Constellations in Natural, Unexplained, and Unnatural Deaths

25

Case Study

A 32-year-old woman who lived alone failed to appear at work on a Monday morning. It transpired that she had already missed a date on the previous day. Concerned colleagues informed the woman's parents, who had a key to their daughter's apartment. On inspection of the apartment with the police, the woman's body was discovered lying in a supine position on the floor. A chair had been knocked over in the living room, and there were signs on the table that the victim had received a guest or guests. However, food remains on one of the plates had already started to show signs of mold. Windows and doors were all intact and locked shut. Further investigations revealed that the 32-year-old woman had had a visitor for breakfast on Saturday morning. The visitor reported that the woman had complained of headache, nausea, and vomiting, which she attributed to gastric flu. Before the visitor left the apartment, the woman had said that if her symptoms did not improve, she would go to the doctor on Monday. No injuries were found at either the police or the forensic examination of the body. Extensive subarachnoid hemorrhage arising from a ruptured basal artery aneurysm was found at autopsy.

In addition to deaths due to trauma (homicide, culpable homicide, negligent homicide, bodily harm resulting in death, suicide, and accidents), forensic practice deals with unexpected and unexplained fatalities. In general, long before any forensic examination takes place, emergency physicians and practice physicians have been summoned, have certified the death, moved the body, and on the basis of their own appraisal ensured that the police are informed. In differential diagnostic terms, all cases of sudden unexpected death prompt the question of whether the death was violent.

Important: **Sudden unexpected death in adults is defined according to the International Classification of Diseases (ICD-10) of the World Health Organization (WHO) as death that occurs within 24 h of symptom onset**.

Unexpected and (initially) unexplained deaths can be characterized by a multitude of different situations and circumstances of death in terms of location, triggering factors causal to death, and underlying diseases. Notable among these are:

- Deaths in custody, e.g., police custody or custody in prison/psychiatric clinic
- Deaths in private homes or hotels
- Deaths in the bath/bathroom/bathtub/shower
- Deaths under particular circumstances, e.g., during sport or sexual activity or while on the toilet
- Fatal occupational accidents

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- Autoerotic activity resulting in death
- Discovery of multiple bodies
- Sudden death in the setting of chronic disease (diabetes mellitus, epilepsy, alcoholism, or drug dependence)

Further classification produces collectives associated with sudden death, e.g., while driving, in the sauna, or red-light district, and during pregnancy.

Individuals taken into police custody against their will are often affected, as are inmates in prisons and patients committed to psychiatric clinics by the state. In other cases, individuals are found suddenly and unexpectedly dead in their homes with no indication of any criminal circumstances surrounding the death.

25.1 Deaths in Police Custody

Any individual taken into police custody should be fit to be held in custody, which requires medical clarification in the case of doubt. Particularly in heavily inebriated individuals, craniocerebral trauma can be overlooked, and the person's condition incorrectly attributed to intoxication alone. The regulations governing custody generally make no provision for medical checks to be carried out on individuals in custody cells. Fatalities—seen predominantly among men aged between 30 and 40 years—affect individuals with cardiovascular disease, alcohol-related disease, and lung disease, as well as individuals in whom craniocerebral trauma has not been recognized. Intoxications (alcohol, drugs) carry a risk for aspiration. Suicide in police custody is also seen, generally in the form of hanging or sometimes wrist-cutting. Deaths while in police custody as a result of prohibited restraint techniques—positional asphyxia—warrant particular attention and are dealt with in a separate chapter (see Chap. 14).

25.2 Deaths in Inpatient Psychiatric Institutions

Due to the nature of their underlying disease (depression, schizophrenia, personality disorders, etc.), there is an increased incidence of suicide amongst psychiatric patients, in particular those aged between 20 and 40 years. Even inpatient monitoring is not infallible in terms of always recognizing suicidal tendencies. Hanging is the most common form of suicide, followed by poisoning, jumping from a height, and rarely self-immolation. Patients who poison themselves are often found to have previously collected prescription medications secretly. Natural deaths are also seen in psychiatric institutions (myocardial infarction and pulmonary embolism), as well as rare adverse drug reactions (e.g., clozapine-induced myocarditis).

25.3 Deaths in Prison

Fatalities in penal institutions are seen primarily in the context of suicide (hanging, wrist-cutting, and poisoning). Belts, bedsheets, and electrical cables are used for hanging. Young men aged between 20 and 30 years who have been placed in solitary confinement at the start of their first period of incarceration are most commonly affected. Fatal poisonings in penal institutions prove that drugs are in circulation in such places; moreover, drug-dependent inmates continue to receive methadone as part of drug substitution therapy.

25.4 Deaths in Private Homes

Bodies found at home are done so incidentally, often after long postmortem intervals and generally showing advanced postmortem changes. Suspicion is aroused either by putrefaction odors, an unemptied mailbox, or failure to keep an appointment. Individuals living in social isolation are more commonly affected (elderly people, alcoholics, and drug users), and the home is often in a squalid condition (uncleaned, with multiple empty alcohol bottles and possibly bloodied towels from tending fall-related lacerations). The condition of the body is often such that unequivocal identification is not possible, and in many cases, no medical history relating to preexisting disease is available. If no plausible cause of death can be found, the police need to be informed.

The investigation should then concentrate on whether any indication of a forced entry, recent visitors, or physical conflict, among other things, can be found in the home.

25.5 Deaths in the Bathroom

The majority of deaths in the bathroom involve accidents or suicide; homicide accounts for around 5 % of deaths and natural causes for around 30 %. CO poisoning, medication intoxication, as well as electrocution are also seen and need to be considered.

Important: **Any indication of CO intoxication or electrocution should always prompt a technical expert to be called upon**.

In the case of a filled bathtub, attention should be paid to water temperature and water level, as well as to whether the respiratory openings were found to be above or below the surface of the water. Was there a foam cone at the mouth and nostrils? Were there any objects in the bathtub (hairdryer)? Electrical burns may be absent in the case of electrocution in the bathtub! Occasionally, individuals who commit suicide enter a (filled) bathtub either fully or partially clothed. Partially undressed individuals found in a more or less seated position on the toilet have usually suffered a natural death (pulmonary embolism, myocardial infarction, or ruptured aortic aneurysm).

25.6 Deaths During Sports and Sexual Activity ("Mors in Actu")

Fatalities occurring during physical activity in a sports context are generally natural deaths, predominantly caused by cardiovascular diseases such as coronary heart disease due to coronary arteriosclerosis, myocarditis, cardiomyopathies, cardiac valve defects, and myocardial hypertrophy (hypertension). Deaths in fitness studios should arouse suspicion of anabolic use (doping). Death during sexual activity predominantly affects men aged 50 years or older, particularly in the setting of coronary heart disease. These deaths tend to occur outside the matrimonial home (girlfriend, red-light district) and in a variety of locations (hotel, brothel, car, outdoors). Accidental deaths resulting from sadomasochistic practices are also seen, as well as autoerotic accidents (bondage, self-induced hypoxia; see Fig. 14.7). Fatalities in young women during sexual activity should arouse suspicion of a ruptured basal artery aneurysm.

25.7 Pregnancy-Related Deaths

Pregnancy-related deaths are relatively rare. Fulminant pulmonary embolism or intoxication should be considered during pregnancy and amniotic fluid embolism or ruptured uterus during childbirth. In the postpartum setting, unrecognized secondary atonic bleeding raises the question of a treatment error. Cases of fatal exsanguination following ruptured ectopic pregnancy are rare (Fig. 25.1), as are suicides due to postpartum depression.

Depending on the prevailing national legislation, illegal abortions also contribute to maternal mortality rates, particularly in those countries where abortions are prohibited. However, illegal terminations are an extremely rare phenomenon in countries that have adopted a more liberalized approach to this issue.

Illegal Abortions. One of the principal reasons behind the statutory liberalization of abortion was, historically, the realization that this represented the only way to prevent fatal illegal abortions. For this reason, experience with deaths due to illegal abortions is gained primarily in countries where abortion is to all intents and purposes prohibited, with the exception of cases of rape or where the pregnancy poses a threat to the mother's life.

Illegal abortions, often carried out by lay people, involve introducing objects into the uterine cavity using instruments with varying degrees of mechanical rigidity (forceps, knitting needles, hairpins, wire, pencils, animal bones, etc.). Catheters are also used. Pain caused by the procedure can range from tolerable to intense. Abortion can prove more complicated in nulliparous than in parous women.

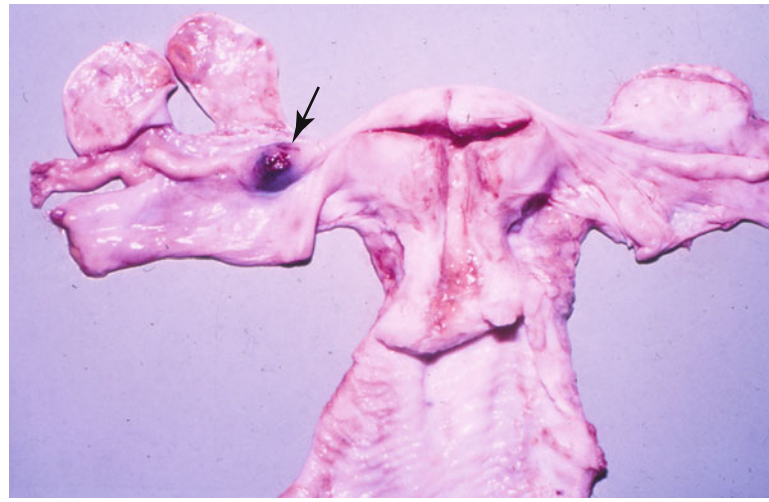


Fig. 25.1 Acute bleeding into the abdominal cavity following ruptured ectopic pregnancy (*arrow*): pericentral distension of the fallopian tube with hemorrhage beneath the serosa and small rupture sites

Alongside the purely mechanical method of abortion, there is also the injection of fluids using, for instance, an enema syringe or a bicycle pump. The injected fluid usually comprises water mixed with various possible additives (e.g., soap, vinegar, tobacco infusion, corrosive sublimate, potassium permanganate, or detergent).

Attempts at abortion are also made by ingesting abortifacient agents, such as alkaloids, glycosides, tobacco decoction (also used locally in enema form), strychnine, as well as numerous plant and herb extracts reputed to have abortifacient effects (ranging from arnica and rosemary to cinnamon), although these effects have rarely been actually proven. Animal toxins and metal compounds are rarely used. In cases where previous medical knowledge is involved, the use of abortifacient drugs, most notably hormones, should be considered.

Lethal complications of an illegal abortion include hemorrhage (Fig. 25.2), (infected) remnants of the pregnancy remaining in the uterine cavity, mechanical injury, infections involving thrombosis and thromboembolism, air embolisms (primarily due to injection into the uterine cavity that causes lifting of the placenta and opening of vessels in the inner uterine wall), fluid and extract embolisms (a soapy solution reaching the blood stream is referred to as "soap intoxication" or a "soap abortion"), chemical burns, as well as various types of intoxication.

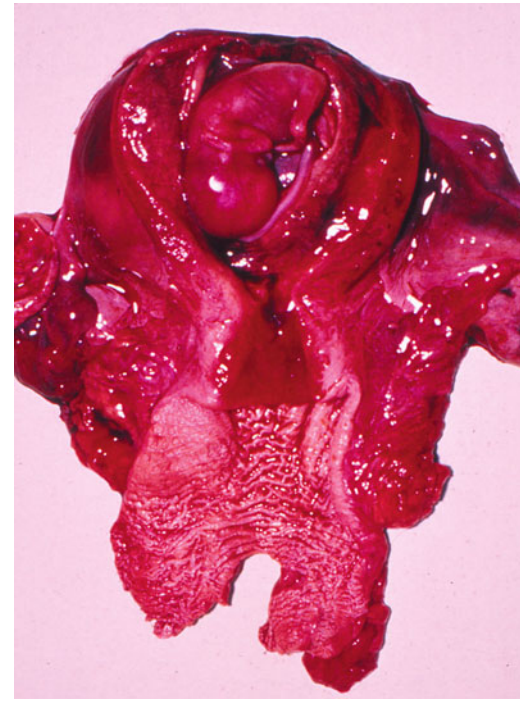


Fig. 25.2 Fatal exsanguination in a pregnant woman following self-inflicted mechanical manipulation extending to the uterine cavity and placental abruption

25.8 Fatal Occupational Accidents

Workplaces harbor all manner of hazards that should be protected against by the accident prevention regulations issued, for example, by

accident insurance institutions. Contravening these regulations can represent a criminal offense. Possible causes of accidents include electricity, malfunctioning of technical systems or machines, falling objects, or toxic substances. In order to be recognized as an occupational accident, a link needs to exist between the accident and the insured activity. Depending on the prevailing legal situation, accidents while travelling directly to and from work are also considered occupational accidents. Accidents in and around silos (putrefaction gases, e.g., in tower silos) are encountered in the agricultural sector, while intoxications that follow an apoplectic course are seen in the chemical industry. Ultimately, work-related physical strain can also trigger sudden death, for instance, in the case of preexisting cardiovascular disease. A scenario such as this gives rise to special challenges in terms of expert appraisals from an insurance medicine perspective, such as the question of whether life was significantly shortened by work-related effects, despite the fact that the severity of disease could have caused sudden death to occur under any comparable strain outside the workplace. Previous occupational exposure to asbestos fibers should be considered in all deaths involving pleural mesothelioma (peritoneal mesothelioma being rarer).

25.9 Discovering Multiple Bodies

The sudden death of several individuals in the same place (home, workplace, or in public) generally points to unnatural deaths, possibly as a result of accidents involving two or more bodies, homicides, a combination of homicide and (attempted) suicide (possibly involving a survivor), terror attacks, and extended suicide. The site at which two bodies are found is almost always the site at which the two individuals in question died; transporting two or more bodies and depositing them at another site is extremely rare. Occasionally, bodies are disposed of (buried), mutilated (to render them unrecognizable), or dismembered.

Disposal, Mutilation, and Dismemberment of Bodies. The distinction commonly applied in the past between defensive and offensive body dismemberment has largely been discarded in favor of a classification that takes both the motivational component and the findings into consideration (Table 25.1).

Table 25.1 Types of body dismemberment

Type	Term	Finding
I	Defensive mutilation	Dismemberment to make transportation easier and identification more difficult; decapitation and severing of large joints are common (often after a certain time interval following death)
II	Aggressive mutilation	Caused by an act of rage involving random dismemberment and mutilation of the body
IIIa	Offensive mutilation	Motivated to cause death with the intention of performing sexual acts on the body or parts of the body following dismemberment
IIIb	Offensive mutilation	Performing sexual acts while inflicting injury until death is caused and continuing thereafter to satisfy sexual-sadistic impulses
IV	Necromantic mutilation	Body parts are removed from the body as trophies, symbols, or for fetishistic reasons

According to Rajs et al. (1998)

In the case of defensive mutilations, body parts need to be classified anatomically; in complex cases, involving, for instance, several mutilated bodies, molecular genetic analysis is needed to distinguish between the body parts of the different victims. Bodies are usually dismembered by means of transection and detachment at the joints, rarely by, for instance, sawing through long bones. The instruments used include saws, axes, and knives, which can leave corresponding marks (saw marks, incisions, indentations, and grooves that match the instrument). The following is a list of methods used to conceal bodies or body parts, as well as methods used to prevent their identification:

- Concealment.
- Burial.
- Dumping in water.
- Bricking up or embedding in concrete.

- Feeding to animals.
- Packaging in plastic bags, which can have a preserving effect on a putrefied body and cause adipocere formation due to lack of oxygen. Where this is the case, forensically relevant findings can sometimes still be made after many years.
- Burning of body parts, although (near) total elimination requires temperatures of around 1,000 °C at burning times of at least 1–2 h; sintered bone remnants may remain even after complete incineration.

There are several ways of preventing dactyloscopic or visual identification of a body, including removal of fingers, hands, the head, or facial skin, as well as facial mutilation, e.g., by means of burning or acids. Cannibalism is sometimes seen in the context of chiefly type III body dismemberment.

25.10 Deaths in Alcoholics and Drug Users

Fatalities in heavily inebriated individuals and drug users can result either directly or indirectly from intoxication. Corresponding case histories are seen in the majority of alcohol deaths; parallel consumption of other drugs that may contribute to the cause of death is rare. The average age of actual drug-death victims is between 30 and 35 years, while alcohol deaths are generally seen in an older age group. An increase in the number of methadone intoxications has been seen since the development of methadone substitution programs for drug addicts. In some urban centers, the number of methadone deaths exceeds the number of heroin deaths, a phenomenon attributable not least to the iatrogenically created black market for methadone. Thus, specifications set out in substitution guidelines, for example, need to be followed particularly closely ("take-home" dose, continuous physical examination, etc.) when treating drug addicts with methadone.

Lethal Alcohol Intoxication. As a general rule, blood alcohol concentrations need to reach or exceed 3.5 % for intoxication to follow a fatal outcome; however, lower values may prove fatal in individuals not accustomed to alcohol consumption, while higher concentrations of over 5 % can be survived. The rapid surge of alcohol associated with binge or competitive drinking as well as "coma drinking" can contribute to death, the cause of death being acute cardiovascular and respiratory depression. Intoxication-induced loss of reflexes can result in asphyxia following aspiration of vomited gastric contents. Large chyme particles or the act of eating may lead to "bolus death," for instance, if the larynx becomes obstructed by a piece of sausage. Inebriated individuals may also fall from significant heights, fall into water and drown, or succumb to fatal hypothermia. Findings consistent with long-term alcohol consumption are often made at autopsy, including:

- Fatty changes in liver, hepatic steatosis, liver fibrosis, and/or portal liver cirrhosis, possibly decompensated and accompanied by jaundice and ascites
- Cerebral cortex atrophy
- Acute esophageal variceal hemorrhage accompanied by tar-like blood (due to the oxidation of hemoglobin to hematin) in the gastrointestinal tract
- Acute necrotizing or chronic fibrosing pancreatitis, possibly accompanied by pancreatic pseudocysts and older tryptic fatty tissue necrosis
- Alcohol-toxic cardiomyopathy
- Infections, most notably lobar pneumonia, more rarely recurrent florid pulmonary tuberculosis

Where no plausible cause of death is found and a blood alcohol concentration alone cannot account for death, ethylene glycol ingestion (see Fig. 30.1) as well as alcoholic ketoacidosis need to be considered in the setting of alcoholism.

Drug Deaths. Depending on the duration of drug addiction, drug-death victims may demonstrate highly revealing findings at autopsy:

- Puncture marks or needle marks, usually on the flexor side of the forearms or elbows.
- Scarring on subcutaneous veins gives rise to more unusual injection sites: Between toes or fingers, at the base of the tongue or back of the penis, in the groin area, and neck.

- Foam cone at the mouth and nostrils, due to toxic pulmonary edema.
- Narrow pupils are more likely in the early postmortem period.
- The overall picture in advanced drug dependence is one of poor general and nutritional status and neglect.

Important: **The term "drug death" encompasses all deaths with a causal link to the abusive consumption of narcotics or substances used as substitution agents (methadone!), including suicides and accidents**.

The (generally intravenous) injection of narcotics is often followed by a long phase of unconsciousness (in the case of heroin: miosis, coma, lethal respiratory depression) or a prolonged agonal phase. In such cases, fellow drug users present at the time may be charged with failure to provide assistance. The life-threatening potential of an individual's condition can be misjudged on occasion. Often, medical assistance is not summoned for fear of discovery. It is not unknown for fellow drug users to transport a body to a different location ("dumping").

Marked cerebral and pulmonary edemas are often seen at autopsy (see Fig. 30.13a, b), possibly accompanied by foamy contents in the respiratory tract and occasionally a foam cone at the mouth and nostrils. Acute or chronic hepatitis is often demonstrated on microscopy in intravenous drug users, as well as cases of pulmonary granulomatosis of varying severity ("junky pneumopathy"; see Fig. 30.12).

25.11 Sudden Unexpected Natural Deaths

Around 10–15 % of all deaths occur suddenly and unexpectedly. While in many cases of death the decedent's previous history provides a plausible explanation for death in terms of a known disease, particularly in cases of sudden unexpected death, the previous history often fails to provide a clear indication of cause. Depending on the acuteness of death, either lethal cardiovascular diseases (approximately 50 %) or chronic progressive diseases that remain largely clinically asymptomatic until death are primarily responsible. Diseases of the respiratory tract account for some sudden deaths (around 15 %), as do gastrointestinal diseases (around 10 %), diseases of the central nervous system (around 10 %), and acute lethal infections.

25.11.1 Coronary Sclerosis and Myocardial Infarction

Sudden unexpected deaths are frequently seen in forensic autopsy practice. In such cases, pathological findings in the heart can often explain the acuteness of death. In addition to ruptured myocardial infarctions, these pathological changes include rare diseases such as primary heart tumor (atrial myxoma, rhabdomyosarcoma) or pericardial tamponade in the case of a dissecting aortic aneurysm, in rare cases with compression of an arteria coronaria and without pericardial tamponade, so-called bloodless aortic dissection. Pathological changes also include:

- Acute coronary insufficiency in the case of stenosing coronary sclerosis
- Myocardial infarction
- All forms of myocarditis
- Cardiomyopathies of varying etiology
- Hereditary anomalies of coronary artery development
- Lesions of the cardiac conduction system
- Primary cardiac tumors

Histological and/or immunohistochemical findings of varying severity can be expected in all of the abovementioned pathological changes to the heart, confirming on the one hand the macroscopically suspected diagnosis and only then enabling the crucial differential diagnosis on the other. Primary cardiac tumors cause sudden death extremely rarely. Sudden cardiac death (SCD) is among the most frequent causes of death, and a significant number of sudden deaths, particularly among young people, are due to genetic heart disorders involving both structural and arrhythmogenic abnormalities.

The morphological correlate of coronary heart disease (CHD) is coronary artery stenosis which, depending on localization, collateral circulation,

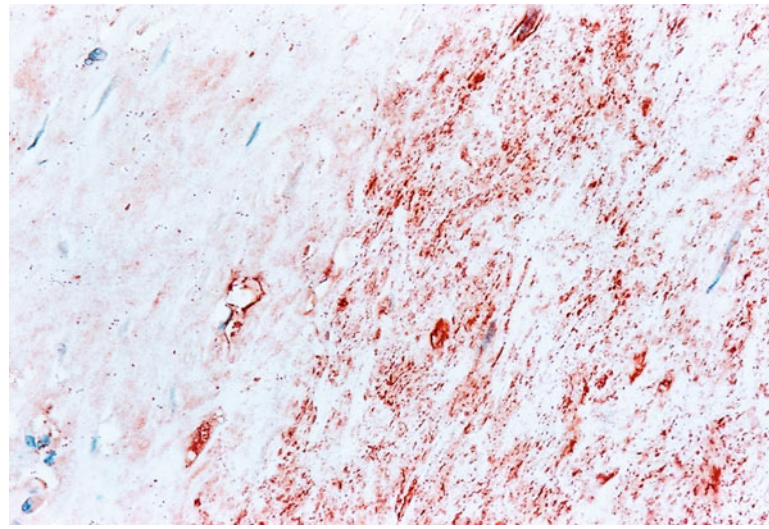


Fig. 25.3 Fresh myocardial infarction seen immunohistochemically with the early necrosis marker C5b-$9_{(m)}$ (×400)

and physical strain—yet irrespective of severity—can cause arrhythmogenic cardiac death. However, since even severe coronary sclerosis can be present for long periods of time without causing death, lethal acute coronary insufficiency is a diagnosis of exclusion. Coronary artery samples need to be obtained with great care in order to detect morphological lesions caused by an acute event: hemorrhaged or edematous atherosclerotic plaques, obturating coronary thrombosis, or fresh plaque breakdown. Myocardial infarctions are characterized by a hemorrhagic rim, while yellow necrosis can be seen following a survival time of 6–8 h. Fresh myocardial necroses can be observed histologically and immunohistochemically (Fig. 25.3).

Older myocardial infarctions are characterized by whitish-gray infarction scars. Disseminated so-called myocardial scarring (small scars <1 cm due to coronary insufficiency) also denotes myocardial damage, as does myocardial hypertrophy, as part of which the critical heart weight of 500 g is exceeded. Infarction-induced myocardial rupture leads to fatal hemopericardium following acute blood loss (200–300 ml) into the pericardium (clinical cardiac tamponade). Aneurysms of the cardiac wall that form after survived myocardial infarction can thrombose (involving an increased risk of embolism), cause heart failure, and rupture. Sometimes it is necessary to estimate the age of a myocardial infarction approximately. Microscopic analysis of the myocardium or of the border and central zones of myocardial infarction can be helpful to this end (Table 25.2).

25.11.2 Coronary Anomalies

In addition to coronary muscle bridges (Fig. 25.4), rare primary arteritis (coronary arteritis), and coronary artery dissections (e.g., in the postinterventional setting following coronary angiography), congenital anomalies in coronary artery structure (e.g., a coronary artery arising from the pulmonary artery, Bland–White–Garland syndrome) can cause sudden death. Coronary anomalies in which the left coronary artery arises from the right coronary sinus may remain asymptomatic until adolescence. Sudden death occurs if a coronary artery follows an intertruncal course accompanied by compression-induced ostial occlusion.

25.11.3 Valvular Disease and Endocarditis

Postinflammatory cardiac valve stenosis, in particular conditions following aortic valve endocarditis, as well as mitral valve prolapse can cause cardiac arrhythmias. Bacterial endocarditis, on

Table 25.2 Chronology of microscopic findings in myocardial infarction

Time	Microscopic findings
From 15 min	Measuring distances between horizontal striae in myocardial fibers in unstained sections: several myocardial sections are compared using an eyepiece micrometer on a phase contrast microscope; hyperextension is evidence of myocardial infarction (Hort 1965)
Up to 30 min	Electron microscopic changes to the mitochondria with swelling and dissolution of the cristae mitochondriales (Büchner and Onishi 1968)
30–60 min	Edema of the myocardial fibers; decrease in glycogen; in animal studies immunohistochemical loss of myoglobin and early detection of fibrinogen (Xiaohong et al. 2002); in cases of ischemia of at least 30 min, contraction bands can be seen on chromotrope aniline blue staining (CAB) as an expression of collapse of the myofibril apparatus (Amberg 1995)
From 60 min	Positive tartaric acid cresyl violet inclusion staining: preserved musculature, bluish violet to reddish violet; damaged musculature, pale blue to sky blue (Holczabek 1973)
2–3 h	Early homogeneous eosin red hyalinized myocardial fibers in peripheral areas of myocardial infarction (Janssen 1977); the stain according to Lie: dark red indicates ischemic myocardial fibers (Tausch 1974) Unfixed tissue sections: fluorochromization with acridine orange can demonstrate damaged myocardium by means of bright green fluorescence (Korb and Knorr 1962)
3–4 h	First agglutinated sarcolemma tubes, discrete fatty degeneration of the myocardial fibers; possible hemorrhagic demarcation of the infarction with hyperemic edges (can also be present at an earlier stage), early fading of cardiomyocyte cell nuclei
4–5 h	Immunohistochemical visualization of the infarct area with the early necrosis markers fibronectin and C5b-9(m), fibrinogen is also positive, visible loss of desmin and myoglobin
4–7 h	Necrosis in the infarct area, early peripheral leukocyte reactions, gradual general eosinophilia of the myocardial fibers and shrinkage of the heart muscle cells in the infarct area, nuclear staining possible (Janssen 1977)
9 h	Pronounced necrosis in the infarct area, strong leukocyte reaction, now also in the infarct area, nuclear staining of cardiomyocytes no longer possible, cell nuclei of the interstitial connective tissue can be stained for somewhat longer
18–24 h	Pronounced necrosis, further leukocyte penetration of the infarct area
5–6 days	Continued leukocyte penetration of the infarct area, abscess-like dissolutions are possible with myocytolysis and rupture of the heart chamber wall (Janssen 1977)
2–3 weeks	More pronounced peripheral granulation tissue with branched capillary blood vessels, fibrocytes, fibroblasts, lymphocytes, few plasma cells, macrophages, possibly siderophages, scant granulocytes
5 weeks to 2–3 months	Collagen fiber or scar tissue with endothelially coated capillary blood vessels of varying density (Mallory et al. 1939), siderophages still possible, loose infiltration with lymphocytes, few plasma cells, scant granulocytes
3–6 months	Scar tissue with fewer cells, fewer capillary blood vessels, scant siderophages
6–12 months	Scar tissue with few cells (DiMaio and Dana 2007), dystrophic calcification with basophilic calcium salt deposits is possible later

Dettmeyer (2011)

the other hand, is rarely seen as a cause of death, including drug deaths in Europe. Congenital defects such as atrial or ventricular septal defects (ASD and VSD, respectively), however, usually become clinically manifest and are diagnosed promptly or are of little hemodynamic relevance, with the result that they play no role as a cause of death in forensic medical practice.

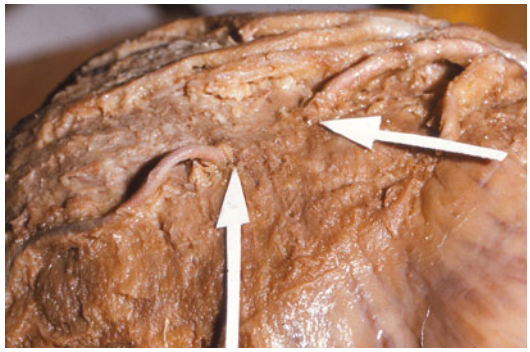


Fig. 25.4 A coronary muscle bridge (*arrows*) as the cause of recurrent ischemia in the downstream myocardial supply area

Table 25.3 Conventional histological diagnosis of myocarditis according to the Dallas criteria

First myocardial biopsy	Findings
Active myocarditis	Myocytolysis, lymphomonocytic interstitial inflammatory infiltrate in the myocardium, interstitial edema
Borderline myocarditis	Sparse accumulation of lymphocytes, subsequent control biopsy
Control biopsy	**Findings**
Persistent myocarditis	Unchanged evidence of myocarditis
Healing myocarditis	Decrease in lymphomonocytic infiltration
Healed myocarditis	No myocytolysis, no necrosis, no increase in lymphomonocytic cells

Aretz (1987)

25.11.4 Myocarditis

Viral myocarditis, the most common form of myocarditis and often lacking clinical symptoms, can cause acute death. Affected individuals appeared either healthy or had suffered flu-like symptoms in the weeks leading up to death. Excess physical exertion in the setting of myocarditis seems to increase the risk of sudden death. It is not uncommon to find at most moderately increased leukocyte infiltration of the myocardium, while in some cases virus detection can be performed using molecular genetic analysis of myocardial samples (enteroviruses, Epstein–Barr virus, adenoviruses, etc.). Myocarditis is initially diagnosed using conventional histology according to the Dallas criteria (Table 25.3).

Immunohistochemical and molecular pathological diagnosis, which can be helpful in the case of inconclusive findings from conventional histological diagnosis due to the method's relatively high interobserver variability, requires that a number of prerequisites be fulfilled (Table 25.4).

The immunohistochemical diagnosis of myocarditis is initially based on the qualification and quantification of interstitial leukocytes, T-lymphocytes, and macrophages within the scope of empirically determined standard values for adults. For the expression of pro-inflammatory endothelial markers and noncellular molecules, a semiquantitative evaluation (0, +, ++, +++) is recommended, also against the background of empirically determined levels of expression, e.g., for MHC class I and II molecules and other pro-inflammatory markers. In addition, the significance of such markers is partially proven in animal experiments, especially in connection with Group B coxsackievirus infection. Enterovirus detection in the myocardium is currently considered a pathological finding. On closer scrutiny, only very few studies involving detection of the enterovirus genome in control groups were shown to have methodological limitations in molecular genetic virus detection.

25.11.5 Cardiomyopathies

Cardiomyopathies can be of widely varying etiology. Alongside inflammatory cardiomyopathy (DCMi) included in the newer classification (frequently seen macroscopically as dilated cardiomyopathy), and which represents chronic myocarditis, there are various forms of congenital cardiomyopathy: hypertrophic cardiomyopathy frequently associated with asymmetric left ventricular hypertrophy and in the form of subvalvular aortic stenosis, arrhythmogenic right ventricular cardiomyopathy (ARVC), and the recently described left ventricular non-compaction cardiomyopathy (LVNC), as well as other forms.

Table 25.4 Recommended methods of conventional histological, immunohistochemical, and molecular genetic analysis for the diagnosis of myocarditis

Method	Important points requiring attention
Fixative	Neutrally buffered formaldehyde (pH control) or an acceptable alternative fixative
Duration of fixation	Up to 36–48 h
Routine histology	Hemalaun–eosin staining of representative samples of all internal organs
Conventional histology of the myocardium	Hemalaun–eosin staining, additionally Mallory, LFB, and EvG staining; taking at least eight myocardial samples from defined locations is recommended for postmortem diagnosis
Immunohistochemistry	Qualification and quantification of interstitial leukocytes, T-lymphocytes, and macrophages (markers: e.g., LCA, CD45R0, CD68, CD3): count 20 high-power fields (hpf) at ×400 or per mm^2, then determine average value
Immunohistochemistry	Detection and semiquantitative evaluation of pro-inflammatory, e.g., endothelial, proteins, or molecules [e.g., MHC class I and II, selectin, cytokine, necrosis marker-like fibronectin, and C5b-9(m), ICAM-1, etc.]
Molecular genetic pathogen identification	The eight myocardial samples with more pronounced lymphomonocyte infiltrates are preferred: PCR and rt-PCR on DNA and RNA viruses [particularly enteroviruses (EV), coxsackieviruses, belonging in particular to Group B (CVB; especially CVB3), adenoviruses (AV), Epstein–Barr virus (EBV), parvovirus B19 (PVB19), herpes simplex viruses, especially type 6 (HHSV-6), cytomegaloviruses (CMV), etc.]

Dettmeyer (2011)

The term cardiomyopathy includes various diseases of the heart muscle, with a distinction being made between primary and secondary cardiomyopathies. The expert consensus panel has proposed the following definition:

> Cardiomyopathies are a heterogenous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that are frequently genetic. Cardiomyopathies are either confined to the heart or are part of generalized systemic disorders, often leading to cardiovascular death or progressive heart failure-related disability.

Cardiomyopathy is a disease of the heart muscle that has not been caused by coronary artery disease, mechanical stress through congenital heart disease, a coronary anomaly, or hypertension in the greater and lesser circulation.

Primary cardiomyopathy types are classified according to the revised version of the 1996 American Heart Association (AHA) definitions. In addition, the European Society of Cardiology Working Group produced a statement on myocardial and pericardial types. Hypertrophic cardiomyopathy and other forms of cardiomyopathy that may become symptomatic in infants have a genetic cause. A modified classification of cardiomyopathy is given in Table 25.5 (please refer to the relevant literature for additional details).

The microscopic criteria for the histological and immunohistochemical diagnosis of chronic myocarditis and DCMi are given in Table 25.6.

25.11.6 Hypertension and Cor Pulmonale

It is not uncommon for undetected hypertension of long standing—generally associated with myocardial hypertrophy (hypertensive heart disease)—to cause acute lethal cardiac arrhythmia. Sudden deaths are also seen in the setting of both acute cor pulmonale (pulmonary embolism; Fig. 25.5) and chronic cor pulmonale (e.g., silicosis, asbestosis, pronounced junky pneumopathy, as well as other lung diseases).

25.11.7 Vascular Causes of Sudden Death

Acute rupture of arteriosclerotic (abdominal) aortic aneurysms is relatively common. Occasionally, dissecting aneurysms are seen against

Table 25.5 Modified classification of cardiomyopathy types

Genetic forms	Mixed forms	Acquired forms
Hypertrophic cardiomyopathy (HCM), e.g., as idiopathic, hypertrophic subaortic stenosis (IHSS)	Dilative cardiomyopathy (DCM)	Inflammatory cardiomyopathy (DCMi)
Arrhythmogenic right ventricular cardiomyopathy (ARVC)	Restrictive cardiomyopathy	Stress-induced cardiomyopathy Takotsubo cardiomyopathy
Glycogen reservoir diseases		Periportal cardiomyopathy
Transition defects		Tachycardia-induced cardiomyopathy
Mitochondrial cardiomyopathy		Acquired in children with insulin-dependent diabetic mothers
Ion channel defects		Thyrogenic cardiomyopathy
Isolated non-compaction cardiomyopathy (NCCM)		Drug-induced forms, e.g., cocaine cardiomyopathy

There are other rare forms of acquired cardiomyopathy

Table 25.6 Histological and immunohistochemical diagnosis of inflammatory dilated cardiomyopathy (DCMi)

Histology	Immunohistochemistry/molecular pathology
Focal, interstitial edema, fiber structures sometimes showing a somewhat irregular appearance	Often only a moderate increase in leukocytes, T-lymphocytes, and macrophages
Interstitial fibrosis, caliber deviation of the cardiomyocytes, differences in nuclei size	Somewhat increased expression of MHC class I and II molecules
Pronounced perivascular fibrosis (EvG stains)	Sign of progressive restructuring with expression of tenascin at the margin of microscopically small zones of fibrosis
Myocardial single-cell necrosis	Focal loss of desmin detectability
Single, empty sarcolemma tubes	Myocardial single-cell necrosis, seldom group necrosis (fibronectin, C5b-$9_{(m)}$)
Microscopically small zones of scarring possible	Expression of additional pro-inflammatory markers
Circumscribed endocardial fibroses	Molecular-pathologic detection (PCR, rt-PCR), possibly of viral genome typically with small numbers of viral copies

Dettmeyer (2011)
The differentiation between chronic myocarditis and inflammatory cardiomyopathy has been discussed in the literature (Kühl et al. 1992)

the background of well-defined underlying diseases [e.g., Marfan syndrome and idiopathic cystic medial necrosis (Erdheim–Gsell)], ascending aorta involvement and extension of the dissection to the pericardium and lethal cardiac tamponade. Dissection-induced compression and coronary artery luminal stenosis with acute lethal myocardial ischemia in the absence of massive hemorrhage (bloodless aortic dissection) is rare. Aneurysms in other locations, most notably basilar artery aneurysms (Fig. 25.6) and coronary artery aneurysms, may also cause sudden death that can only be explained at autopsy.

Basilar artery aneurysms—located most commonly at the level of branch points in the circle of Willis; approximately 2–3 % of the population is believed to be affected—cause subarachnoid hemorrhage. Lethal undetected arteritides (aortitis, coronaritis, or plasma cell-rich syphilitic aortitis) are rare. It is occasionally necessary to provide an expert appraisal on whether a basilar artery aneurysm ruptured as a result of trauma (a road accident, blows to the head, or a fall). The expert appraisal criteria should include, among others:

- Short time between trauma and rupture
- Presence of pontine symptoms between trauma and rupture
- Aneurysm size (> or <5 mm)
- Presence of other aneurysms
- Detection of a "daughter aneurysm" in the wall of the "main aneurysm"

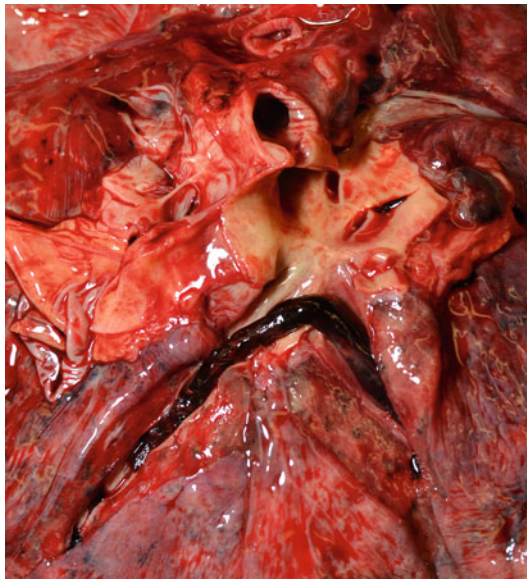


Fig. 25.5 Fresh non-wall-adherent pulmonary embolism in the setting of pronounced dilation of the right atrium and right ventricle

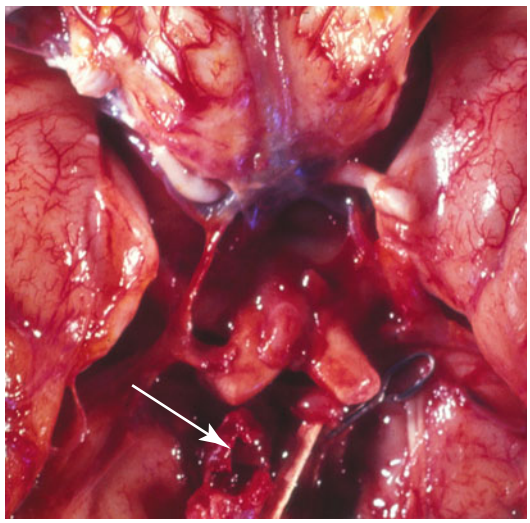


Fig. 25.6 Ruptured basilar artery aneurysm (*arrow*)

- Age and sex at the time of rupture
- Histological findings in the aneurysm wall and surrounding tissue (fibrosis? siderophages?)
- Status following previous surgery and/or clipping

It should be borne in mind that aneurysms can rupture as a result of even minimal trauma or normal physical strain.

25.11.8 Respiratory Tract and Pulmonary Embolisms

Immunocompromised individuals in particular (the elderly, sick, homeless, or alcoholics) are prone to developing acute pneumonia, either in the form of acute purulent bronchopneumonia or as lobar pneumonia, although tuberculous pneumonia is also seen. By causing inflammatory vascular wall erosion, tuberculosis can lead to acute hemoptysis accompanied by massive blood aspiration. In the case of immobilization-induced pneumonia, the cause of immobilization (generally confinement to bed) requires clarification: posttraumatic (e.g., fracture of the femoral neck accompanied by hypostatic pneumonia due a constantly recumbent position), organic disease (e.g., retention pneumonia due to obturating bronchial cancer), or severe intoxication (intoxication-induced aspiration pneumonia?). Acute hemoptysis of other etiologies can also cause death, in particular metastasized malignancies involving erosion of pulmonary blood vessel walls. An acute asthma attack can also cause sudden death.

Dominant among pulmonary embolisms are pulmonary thromboembolisms, which can be either acute or chronically recurrent and sometimes wall-adherent obturating thromboembolisms. Thromboembolisms arise primarily in deep leg and pelvic veins; obstruction of more than 50 % of the pulmonary arterial bed results in acute fatal cor pulmonale. Posttraumatic pulmonary thromboembolism is an unnatural cause of death that can be causally linked to trauma in the (sometimes distant) past. For other embolisms, see Table 7.4).

Case Study

A 57-year-old cyclist was struck by a car after the car driver failed to grant right of way and suffered an open left lower leg fracture. After several months of inpatient treatment, the cyclist regained mobility. In May of the following year, the man went missing while on a cycling tour. His body was discovered next to his undamaged

bicycle at the side of the road during a police search. Although the forensic pathologist could find no external injuries on external examination, he noticed a slight increase in circumference in the left lower and upper leg compared with the right leg. At autopsy, fresh fulminant pulmonary thromboembolism was found in deep vein thrombosis only in the left leg, where it extended to the calf veins at the level of the previous lower leg fracture. The deceased's wife and two children thereupon claimed a widow's and partial orphan's pension.

25.11.9 Diseases of the Central Nervous System

Intracerebral Hemorrhage. Causes of sudden death can include intracerebral hemorrhage—usually in the form of hypertensive intracerebral hemorrhage—and acute stroke. Both events can lead to fall-related injuries during the agonal phase. The expert assessor is sometimes called upon to establish whether a stroke caused a fall (natural death) or whether a fall caused intracerebral bleeding (unnatural death).

Meningitis. Alongside brain tumors, other causes of death include fulminant forms of meningitis (Fig. 25.7), most commonly purulent leptomeningitis. These forms can cause sudden death. Particularly in the case of sudden death in young individuals, fulminant meningococcemia with bilateral hemorrhage into the adrenal glands should be considered (Waterhouse–Friderichsen syndrome; Fig. 25.8). In rare cases, the macroscopic picture at autopsy is one of purulent meningitis, only for subsequent histological investigations to reveal that lymphomatous meningitis was already present and that sudden death represented the first manifestation of underlying disease (Hodgkin or non-Hodgkin lymphoma).

Epilepsy. Sudden deaths in epilepsy are known; a bite injury to the tongue can be an indication of this at autopsy (Fig. 25.9). Other seizure-related injuries are not necessarily present. Sudden unexpected death in epilepsy (SUDEP) is only considered once all other causes of death have been ruled out. Postmortem blood analysis for the detection of antiepileptic drugs is recommended. Small dermal injuries resulting from the epileptic seizure are sometimes seen, severe injuries only rarely.

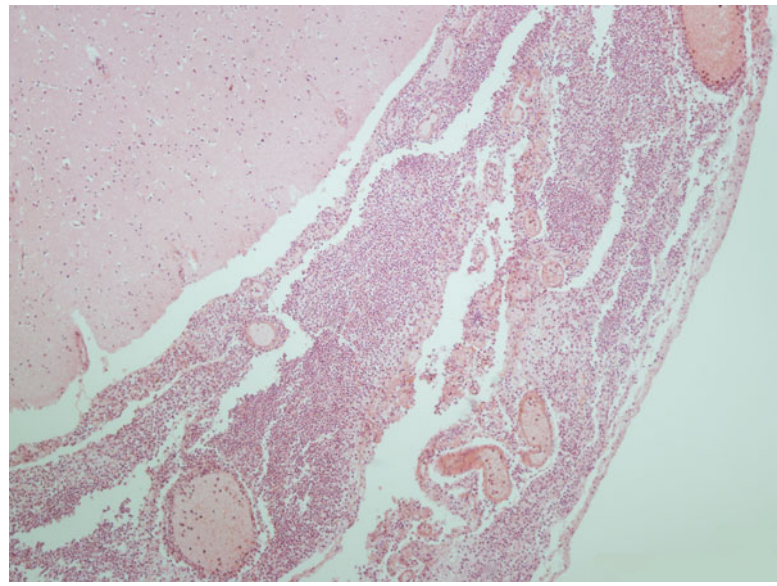


Fig. 25.7 Extensive purulent meningococcal meningitis

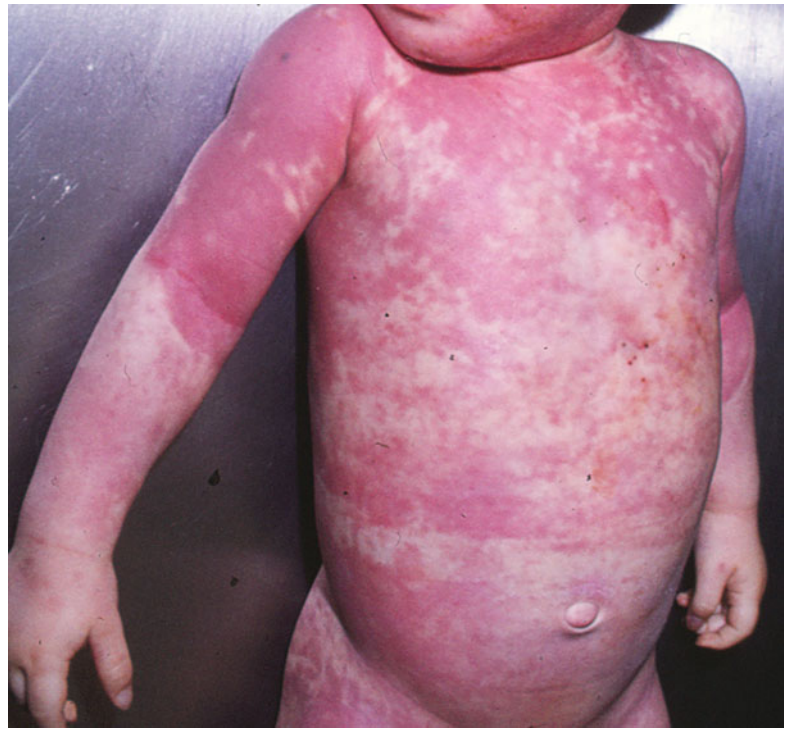


Fig. 25.8 A 7 months old girl with Waterhouse–Friderichsen syndrome: diffuse marbled red skin

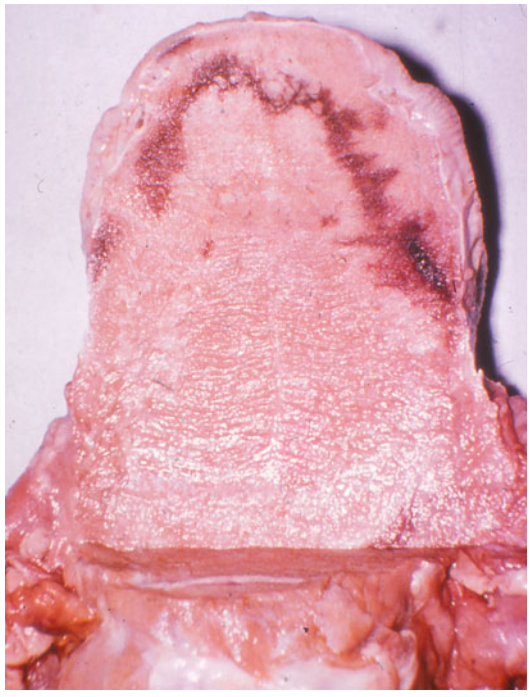


Fig. 25.9 Death due to epileptic seizure with a fresh bite injury to the tongue and detection of an antiepileptic drug in postmortem blood

25.11.10 Gastrointestinal Tract

Bleeding into the upper gastrointestinal tract, notably esophageal variceal hemorrhage in portal cirrhosis or bleeding due to ulcerous erosion (peptic ulcer, duodenal ulcer), should initially be suspected as the cause of a sudden death (Fig. 25.10).

In cases such as these, traces of blood around the body do not necessarily suggest injury due to physical confrontation; coffee ground-like material is suggestive of bleeding from an ulcer. In alcoholics, there may be multiple hematomas in localizations typical of falls and impact trauma, particularly in the case of impaired coagulation due to alcoholic portal cirrhosis.

Important: **In the case of suspected esophageal variceal bleeding**, **the ligated esophagus should be exenterated in continuity with the stomach**. **This permits better visualization of rupture sites on subepithelial veins** (**see** Fig. 4.7).

Acute hemorrhagic necrotizing pancreatitis is rarely the cause of sudden death; in most cases, hospital admission is prompt due to acute pain

comparable to peritonitis (perforated appendix, perforated ulcer, perforated diverticulum); however, purulent peritonitis can cause death within hours.

Infections. Severe infections can lead to fulminant fatal sepsis (bilateral hemorrhage into the adrenal glands, signs of disseminated intravascular coagulation, as well as petechiae on the skin and mucous/serous membranes). Although pathogen detection requires the appropriate microbiological and viral diagnostic steps, these should nevertheless be taken. In some cases, infections may need to be reported in accordance with infection prevention laws. Sudden infection-related deaths in individuals returning from distant parts of the world, e.g., infected with tropical malaria (Fig. 25.11a, b), are rare in Europe.

25.11.11 Diseases of the Endocrine Organs

Diabetes, *Hyperglycemia*, *and Diabetic Coma*. Undetected metabolic decompensation in diabetes leading to death in diabetic coma is of particular relevance. Autopsy reveals nonspecific macroscopic findings (cerebral edema, possibly

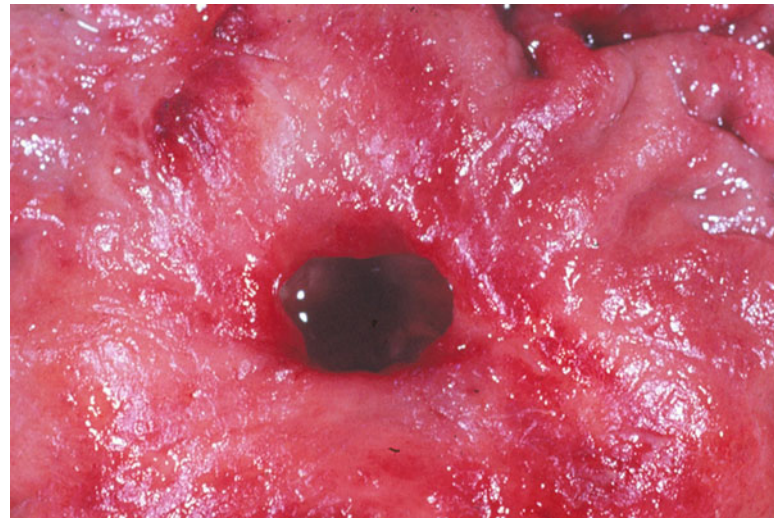


Fig. 25.10 Deep pyloric peptic ulcer. Cause of death: fatal hemorrhage

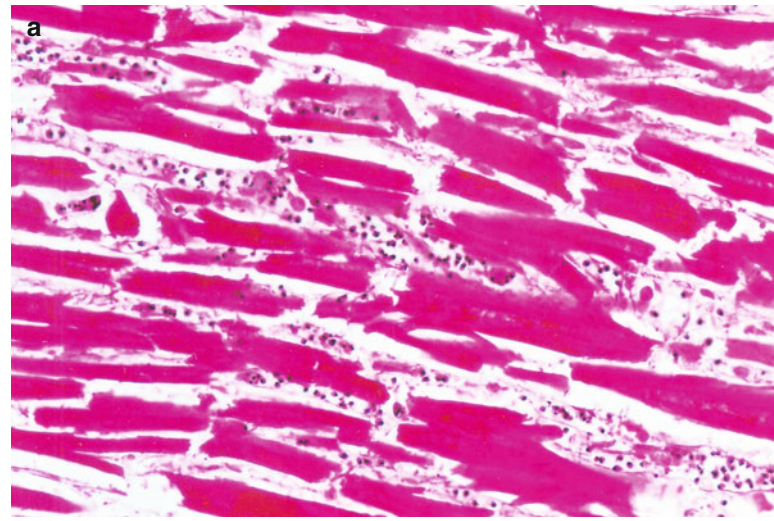
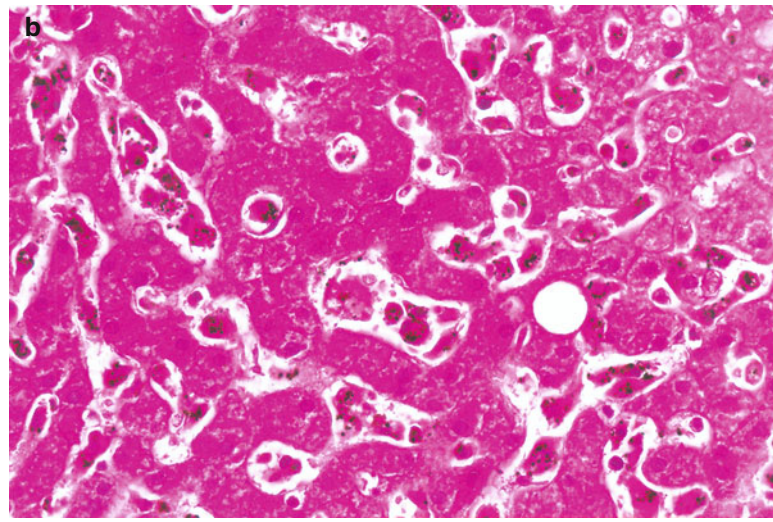


Fig. 25.11 (**a, b**) Abundant malaria pigment in intramyocardial capillary blood vessels (**a**) and in liver sinusoids (**b**) (H&E × 200; ×400)

Fig. 25.11 (continued)



early hypostatic pneumonia); firm red kidneys with a finely granular surface are seen in the case of diabetic nephropathy (Kimmelstiel–Wilson syndrome). Postmortem biochemical analysis to determine glucose levels and HbA1c is essential, while histological findings are helpful (Table 25.7).

If the combined values of glucose and lactate in cerebrospinal fluid add up to more than 362 mg/dl, a lethal diabetic coma is considered probable, assuming all other causes of death have been ruled out. An upper limit value of 410 mg/dl in vitreous humor has been reported in the literature. Both values are believed to remain relatively stable for up to 10 days post-mortem under cold-storage conditions. HbA-1c determination can be used to estimate antemortem glucose levels, whereby an increased value is an indication of preexisting hyperglycemia of long standing.

Hypoglycemia. Acute hypoglycemia can occur in association with infections, in the case of inadvertent ingestion of sulfonylureas, or as a result of injecting insulin but failing to have sufficient food intake (insulin overdose). Again, determining glucose and lactate in cerebrospinal fluid and vitreous humor is highly useful for diagnosis here. Hypoglycemia is also seen in death by starvation and dehydration (see Chap. 16).

Adrenal Glands. Sudden unexpected deaths are seen in the context of undetected pheochromocytomas [catecholamine (adrenaline/noradrenaline)-producing tumors] of the adrenal medulla, occasionally in undetected Addison's disease.

Table 25.7 Diagnostic methods in suspected lethal diabetic coma

Investigation	Findings in long-standing diabetes and diabetic coma
Cranial vault, subcutaneous fatty tissue	Xanthochromia
Lungs	Early hemorrhagic pneumonia possible
Urine	Glycosuria diagnosis for orientation at autopsy
Kidneys (histology)	Diabetic nephropathy Armanni–Ebstein cells = vacuolated renal tubular epithelial cells at the corticomedullary junction on light-optical microscopy
Pancreas (histology)	Unspecific; acute coma in young individuals is possibly due to pancreatitis; insulitis
Cerebrospinal fluid	Combined values of glucose and lactate in diabetic coma: 500–600 mg/dl; high HbA1c values (>12.1 %)
Vitreous humor	High combined glucose and lactate values
Liver	Microvesicular hepatic steatosis is common

Rarer causes of sudden death include certain metabolic diseases, such as pronounced cardiovascular amyloidosis (Fig. 25.12).

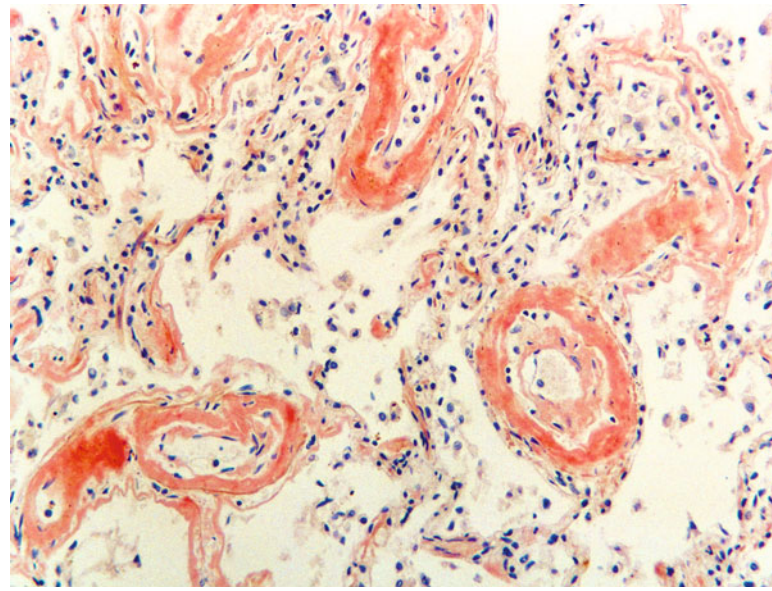


Fig. 25.12 Acute collapse in an 82-year-old female: cardiovascular amyloidosis with massive amyloid deposits in intrapulmonary vascular walls (Congo red staining)

25.12 Sudden Infant Death Syndrome (SIDS)

In Europe, sudden infant death syndrome (SIDS) is the most frequent cause of death in the first year of life, and of unknown etiology, the incidence of SIDS in Germany is 0.5/1,000 (1.2–1.8/1,000 30 years ago). The maximum incidence is seen between the ages of 2 and 4 months, with a second peak at 6/7 months.

Definition

Sudden Infant Death Syndrome: "**Sudden death of any infant or young child which is unexpected by history, and in which a thorough postmortem examination fails to demonstrate an adequate cause of death**" (Beckwith 1970).

The 1994 Stavanger definition specifies: "**Sudden death in infancy unexplained after review of the clinical history, examination of the circumstances of death and postmortem examination**" (Rognum 2001).

SIDS represents neither a diagnosis nor a cause of death, but rather a phenomenon to which unexplained deaths can be attributed. The following applies:

Important: **Only once all diagnostic possibilities—including autopsy and subsequent morphological, toxicological, molecular pathological, and postmortem biochemical analyses—have been exhausted and no plausible cause of death has been found can a death be attributed to SIDS**.

Death Scene. The infant is most commonly found lifeless in its bed or stroller after a prolonged period of sleep. This phenomenon is seen more frequently during the cold season in North Europe. The infant's respiratory openings may be partially covered by a pillow or bedcover, and vomitus is a frequent finding. Some infants have perspired (damp/clammy clothing), and measuring body temperature in the case of recent death can reveal significantly increased temperatures. At a ratio of around 60:40, male infants are more frequently affected than female infants.

Autopsy Findings. While unable to account for death, macroscopic findings at autopsy include:

- Distribution of livor mortis consistent with a prone position
- Cyanosis of the lips and fingers
- Partial hemorrhagic pulmonary edema
- Foamy contents in the upper respiratory tract and at respiratory openings
- Frequently, abundant subserous petechiae, particularly beneath the thymic capsule (Fig. 25.13), as well as within the thymic tissue, in the subpleural and subepicardial regions

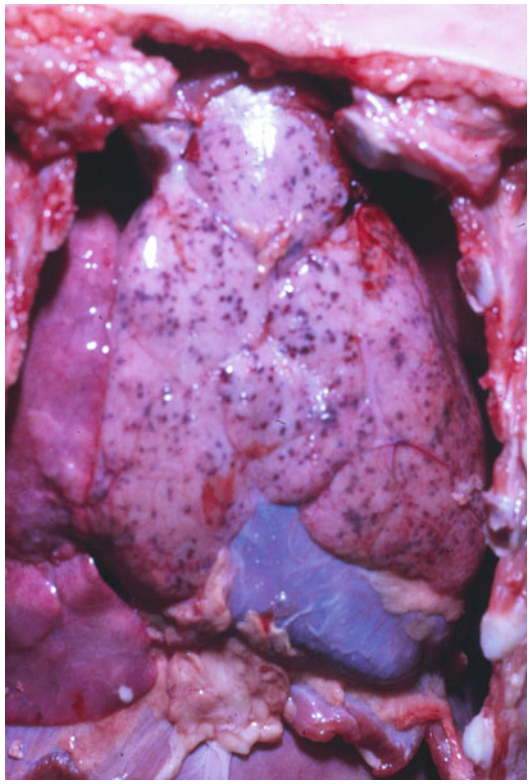


Fig. 25.13 Numerous petechiae beneath the thymic capsule in a case of alleged sudden infant death syndrome

- Occasionally, uni- or bilateral mucopurulent otitis media
- No internal organ abnormalities
- No indication of external gross blunt force trauma immediately prior to death

Even in cases where autopsy findings are unable to establish whether a death was natural or unnatural death, the following is worthy of note:

Important: **In all cases of suspected SIDS, a differentiation needs to be made where possible between SIDS and homicide with little or no evidence, most notably asphyxia by means of airway occlusion using a soft cover. Cases of recurrent SIDS within one family, which in reality proved to be infanticide, are known.**

Homicide by asphyxiation with a soft cover, which is then incorrectly interpreted as a case of SIDS, is most commonly seen in association with Munchausen syndrome by proxy (MSbP). The International Society for the Prevention of Sudden Infant Death (ISPID) has formulated a differentiation of deaths classified as SIDS (Table 25.8).

Table 25.8 The International Society for the Prevention of Sudden Infant Death classification of SIDS

Group	Characteristics
SIDS in its narrowest sense	Autopsy and clinical findings reveal no cause of death
Borderline SIDS	Preexisting congenital diseases or clinical symptoms and/or autopsy findings offer no adequate explanation of cause of death
Non-SIDS	Cause of death adequately explained by clinical information and autopsy findings
Suspected SIDS	Cases in which no autopsy was performed

Studies carried out in recent decades have been able to identify—and thus to a certain extent avoid—risk factors for SIDS, which has led in turn to a significant reduction in SIDS cases. Premature neonates with a birth weight <2,000 g (small for date) and respiratory distress syndrome or bronchopulmonary dysplasia, manifesting as postnatal disorders such as obstructive and central apnea and infections, are at particular risk for SIDS. The children of very young mothers (<19 years), older mothers (>40 years), multiparous mothers, drug-dependent mothers, and mothers who smoke are more commonly affected. Other risk factors include failure to breastfeed, the prone position for sleeping, as well as exposing an infant to excessive heat (excessively warm covers/room temperature and sleeping in the parents' bed).

The etiology of SIDS is undoubtedly multifaceted. Special specimen techniques often yield evidence of nasopharyngeal infection. Immunohistochemical and molecular pathological analyses of the myocardium make it possible to conclude that, in a proportion of cases, myocarditis undetectable using conventional histology could come into consideration as the cause of death as well as primary cardiomyopathies. There are also indications pointing to interstitial lymphomonocytic viral pneumonia. Occasionally, viral sialoadenitis (Fig. 25.14) is demonstrated in what was initially believed to be a case of SIDS.

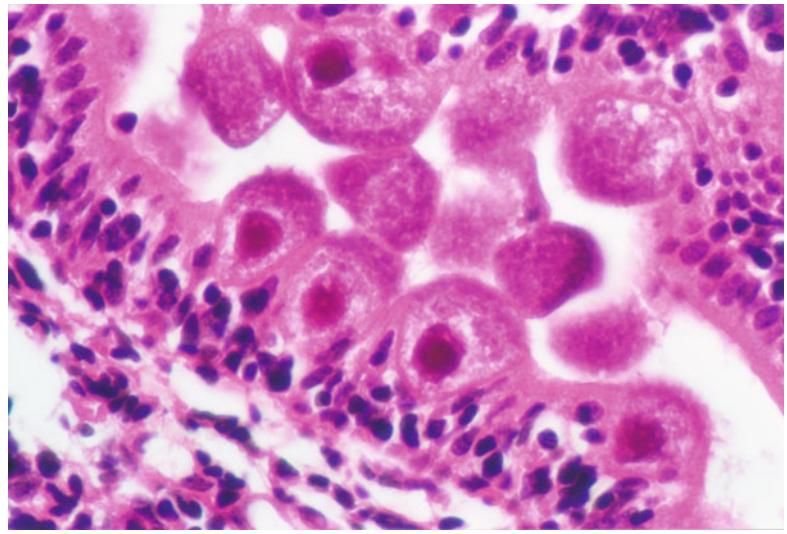


Fig. 25.14 Inclusions typical for cytomegaly in the epithelium of the parotid gland with concomitant focal viral myocarditis (H&E×400) (Photo courtesy of Prof. M. Risse, Gießen, Germany)

Selected References and Further Reading

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Torture

26

Evaluating violence against individuals and identifying evidence of violence on individuals form a traditional part of forensic medicine. However, surprisingly, forensic diagnosis is seen primarily as the assessment of specific cases (homicide, manslaughter, child abuse, etc.) on behalf of the organs of state authority bodies (police, state prosecutor's office, and courts). Even more surprising still is the fact that, particularly when viewed internationally, the basic task of forensic medicine is apparently to determine facts to the detriment of victims of violence committed by precisely those agents (police, soldiers, secret services, plant security forces, self-appointed militias, private security agents, as well as other official entities) who (claim to) represent the state, possibly in the knowledge of, in the presence of, or with the support of physicians.

Against this background, there have been reports of forensic pathologists who, for the purposes of concealing evidence of torture, have collaborated with torturers or with authorities whose agents practice torture. It was circumstances such as these that prompted Amnesty International 1996 to address the issue of physician participation in and false appraisal of torture (Amnesty International 1996).

The spectrum of allegations against the abovementioned official agencies or groups range—depending on one's perspective—from inhumane detention in homes, psychiatric clinics, police custody, and prisons to the illegal fixation of detainees and inmates and severe cases of abuse and torture.

Graessner and Wenk-Ansohn (2000) list the following as indications of government involvement in torture:

- Keeping and using equipment, e.g., for electric shock torture
- Interrogations carried out in special rooms, e.g., tiled rooms with water drainage
- Special interrogation methods, e.g., "good guy, bad guy"
- Attempts to leave little or no trace of torture, e.g., blows with sand sacks
- Using blindfolds to prevent torturers from being recognized
- Attempts at disorientation to the point where the torture victim believes the situation to be unreal
- Certificates from forensic pathologists who regularly deny torture, or the presence of physicians and psychologists during torture
- Writing on walls, e.g., "God is absent and the prophet on holiday"
- A preponderance of psychological torture methods
- Release once a confession has been made and signed

Attempts to make a conceptual distinction between abuse and torture tend to produce a relatively broad gray zone in which abuse sometimes starts to take on characteristics of torture.

Both abuse and torture can leave visible marks or injuries on the body and cause severe and permanent psychological harm. Injuries occasionally enable inferences to be made about the type of trauma (beatings, electric shock torture, etc.),

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thereby putting any evaluation under the remit of forensic medicine. Psychologists and psychiatrists are able to diagnose psychological harm, including simple and complex posttraumatic stress disorders. Despite the existence of criminal legal provisions relating to abuse and torture (homicide, manslaughter, bodily harm, etc.) in countries where acts of this kind are committed, perpetrators are rarely brought to justice precisely because the act is on behalf of the respective criminal prosecution system and can depend on the support of both politicians and the court system.

International norms have been drawn up with the aim of outlawing abuse and torture carried out by state bodies or their agents, and monitoring institutions have been set up.

26.1 Norms and Institutions

The Universal Declaration of Human Rights (UDHR) was adopted by the United Nations General Assembly on 10 December 1948 and includes the following article (Article 5):

> No one shall be subjected to torture or to cruel, inhuman or degrading treatment or punishment.

In 2000, Muslim member nations of the Organization of the Islamic Conference (Organization of Islamic Cooperation) officially resolved to support the Cairo Declaration on Human Rights in Islam, proclaiming that people have the "freedom and right to a dignified life in accordance with the Islamic Shari'ah." Article 1(a) of the Cairo Declaration on Human Rights in Islam states:

> 1(a) All human beings form one family whose members are united by their subordination to Allah and descent from Adam. All men are equal in terms of basic human dignity and basic obligations and responsibilities, without any discrimination on the basis of race, colour, language, belief, sex, religion, political affiliation, social status or other considerations. The true religion is the guarantee for enhancing such dignity along the path to human integrity.

On 26 June 1987, a UN convention against torture came into force (Convention against Torture and Other Cruel, Inhuman or Degrading Treatment or Punishment). Article 1 of the convention defines torture as follows:

> **Article 1**. Any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted on a person for such purposes as obtaining from him or a third person, information or a confession, punishing him for an act he or a third person has committed or is suspected of having committed, or intimidating or coercing him or a third person, or for any reason based on discrimination of any kind, when such pain or suffering is inflicted by or at the instigation of or with the consent or acquiescence of a public official or other person acting in an official capacity. It does not include pain or suffering arising only from, inherent in or incidental to lawful sanctions.

The Optional Protocol to the Convention against Torture and other Cruel, Inhuman or Degrading Treatment or Punishment (OPCAT), adopted by the General Assembly on 18 December 2002 and in force since 22 June 2006, provides for the establishment of "a system of regular visits undertaken by independent international and national bodies to places where people are deprived of their liberty, in order to prevent torture and other cruel, inhuman or degrading treatment or punishment," to be overseen by a Subcommittee on Prevention of Torture and Other Cruel, Inhuman or Degrading Treatment or Punishment.

The UN Committee against Torture (CAT) is a body set up to monitor implementation of the Convention against Torture. It consists of ten members who meet as a rule twice a year in Geneva. UN member states are obliged to submit a report every 4 years stating what they have done to implement the UN Convention against Torture in their country and what measures have been taken. These reports are reviewed by the Committee against Torture, which then give its opinion and, where appropriate, makes proposals.

The European Committee for the Prevention of Torture and Inhuman or Degrading Treatment or Punishment (CPT) is an institution belonging to the Council of Europe. Its powers are set out in the European Convention for the Prevention of Torture and Inhuman or Degrading Treatment or Punishment.

CPT members are independent and impartial experts from various sectors, including attorneys and physicians. CPT delegations periodically visit detention facilities in contracting states (approximately every 4 years), but ad hoc visits are also possible. The results and findings of visits are documented in a report, which visited states can comment on.

The UN Working Group on Arbitrary Detention (WGAD) was set up by the UN Human Rights Commission and is a Special Procedure of the UN Human Rights Council. Since 1991, the Working Group has been investigating situations involving people arbitrarily, i.e., with no legal basis, deprived of their freedom.

In August 1999, a manual that came to be known as the Istanbul Protocol was submitted to the UN High Commission for Human Rights providing a systematic outline of potential means to identify evidence of torture. The Istanbul Protocol contains details on how to perform thorough physical examinations on torture victims, taking all body areas into account and including skin, face (eyes, ears, nose, jaw, oropharynx and neck, oral cavity and teeth), chest, abdomen, musculoskeletal system, genitourinary system, as well as the central and peripheral nervous systems. The manual also describes the following individual forms of torture in detail:

1. Beatings and other forms of blunt trauma
   (a) Skin damage
   (b) Fractures
   (c) Head trauma
   (d) Chest and abdominal trauma
2. Beatings to the feet
3. Suspension
4. Other positional torture
5. Electric shock torture
6. Dental torture
7. Asphyxiation
8. Sexual torture including rape

It is important to point out here that the above describes the effects of only the commonest forms of torture. Each injury or pattern of injury identified needs to be compared for consistency with the reported history or alleged torture. Essentially, the following formulations contained in the Istanbul Protocol are used in the evaluation process:

I. Not consistent: The lesion could not have been caused by the trauma described.
II. Consistent with: The lesion could have been caused by the trauma described, but it is nonspecific, and there are many other possible causes.
III. Highly consistent: The lesion could have been caused by the trauma described, and there are few other possible causes.
IV. Typical of: This is an appearance that is usually found with this type of trauma, but there are other possible causes.
V. Diagnostic of: This appearance could not have been caused in any way other than that described.

As far as we know, the International Rehabilitation Council for Torture Victims (IRCT) and the American University Washington College of Law set up the first 3-year project entitled "Use of forensic evidence in the fight against torture" in the USA in April 2009 with the collaboration of European institutions. Insofar as forensic pathologists have been (or will be) referred to in their capacity as experts, it should be noted that the following is valid to date on a European level:

- No standardized speciality of forensic medicine has been established.
- European political authorities have evidently failed to recognize the importance of forensic expertise for the monitoring of adherence to the convention against torture.
- Moreover, the international importance of forensic medial expertise has not been sufficiently understood (see Chap. 1).

In 2011 and despite international regulations, Amnesty International identified cases of torture and abuse in 101 countries. Torture and abuse do not always leave visible traces. Only physicians are qualified to make objective, neutral, and above all impartial diagnoses in abuse and torture victims. By virtue of their routine activities and experience, forensic pathologists are eminently well suited to the task of examining and identifying injuries in victims of (state) violence, assuming they are awarded a status permitting them

proper powers of appraisal in their capacity either as an expert in the country in question or as a member of, e.g., an investigating commission. This status also includes:

- Assistance for (forensic) medical experts from the relevant state bodies in the context of previous commitments undertaken according to international law
- Unlimited rights to visit individuals held in state custody
- Unrestricted access to state facilities (police custody, prisons, psychiatric clinics, etc.) on behalf of inter-, intra-, or supranational institutions and organizations, including nongovernmental organizations
- Unhindered and confidential communication with individuals held in state custody (e.g., free choice of an impartial interpreter)
- Unhindered and confidential communication with officials of the relevant (state) institutions (e.g., prison officers)
- Freedom to carry out medically appropriate examinations or diagnoses in individuals held in state custody (confidentiality and freedom to document findings, including photodocumentation, and record case history details)
- Unrestricted freedom to instruct the transfer of any prisoner held in state custody whose health is particularly severely impaired to a hospital or clinic, where appropriate medical care can be provided if medically indicated
- Freedom to carry out on-site monitoring of improvements deemed necessary at specified or agreed time intervals

It is the task of policy makers in the respective countries to prohibit the practice of human rights violations by state bodies and to bring those responsible to justice. Where policy makers fulfill this mission, (forensic medical) experts also need to be given a guarantee that they will have a sufficiently loud voice in transparent court proceedings, without fear of repression. Torture seen in the context of genocide, crimes against humanity, and war crimes may be referred to the International Criminal Court (ICC) in The Hague, Netherlands, an international instrument designed to punish crimes of this nature. The Rome statute setting up the ICC came into force on 1 July 2002. The court relies on experts active in a variety of fields and has compiled a database of experts by calling upon qualified individuals to apply for inclusion. The main fields of relevance include:

- Forensic expertise
- Ballistic expertise
- Military expertise
- Police expertise
- Political or geopolitical expertise (specific to the various situations)
- Judicial expertise (specific to the various situations)
- Historical expertise (specific to the various situations)
- Linguistic expertise
- Financial expertise
- Psychological expertise

Torture is seen not only against a background of genocide, war, and crimes against humanity where relatively large numbers of people fall victim to violence but also in individuals, relatively small groups of people, as well as persecuted minorities. Since the concept of "torture" is not precisely defined, Article 1 of the UN Convention against Torture mentioned above is generally used for orientation.

Victims of abuse and torture in (but not exclusively) state custody are subjected to various procedures, whereby a distinction is made between physical and psychological forms of torture.

26.2 Physical Torture

A wide range of torture methods are used to cause primarily physical pain and may leave evidence of injury, although this is not necessarily always the case. Methods of this kind include:

- Blunt trauma: Beatings with fists and objects (punches, kicks, slaps, whipping, beating with wires/truncheons, or falling down).
- Deliberately striking both ears simultaneously.
- Blows to the soles of the feet (*falanga*, *falaka*, *bastinado*).
- Forced walking over salt or glass fragments, also seen, e.g., following beatings to the soles of the feet.

- Electric shock torture, e.g., using electrical equipment on sensitive areas of the body such as genitals, breasts, mouth, head, and anus (possibly producing electrical marks or permanent scarring).
- Crushing one or both testicles.
- Crushing other body areas.
- Inflicting burns at various sites on the body, e.g., stubbing out cigarettes and cigars on a victim's skin, producing evidence of round or roundish oval scars; inflicting burns with heated instruments, e.g., a hot iron or hot plastic; and inflicting burns with scalding liquid or a caustic substance.
- Force-feeding with fluids containing pepper or high-concentration saline solution, which usually causes severe emesis but which can also have more serious harmful effects.
- Providing insufficient food (leaving a victim hungry and thirsty; see Chap. 16).
- Providing contaminated food.
- Pharmacological torture using toxic doses of sedatives, neuroleptics, paralytics, etc., e.g., forced injection of overdoses of psychotropic drugs and other medications that damage the central nervous system (hair analysis may be able to detect long-term exposure).
- Injection of addictive narcotics e.g., heroin (detected using chemical-toxicological analysis of urine and hair).
- Positional torture: Suspension (e.g., being suspended by tied wrists with no contact between the feet and the ground over many hours, sometimes producing permanent tie marks), stretching limbs apart, prolonged constraint of movement, forced positioning.
- Other forms of suspension by hands and feet.
- Tearing out head, pubic, or facial hair.
- Joint dislocation.
- Prolonged standing in freezing temperatures wearing clothing soaked in cold water, in snowy conditions, or in underclothes (frostbite on the feet or lower extremities).
- Inducing forced posture in small cages over extended periods of time.
- Using high-pressure (cold) water jets.
- Piercing finger tips with sharp (bamboo) sticks and nipples with wire (scar detection at these sites).
- Inflicting gunshot wounds.
- Being forced to stand for extended periods in painful positions.
- Forced abnormal posture, e.g., inducing a bent position by tying the penis and neck together.
- Continual daily beatings (blunt, sharp, and semi-sharp force, which may leave corresponding injuries).
- Sleep deprivation.
- Incarceration with snakes and scorpions, exposure in an unclothed state to mosquito-infested areas (bite/sting injuries on the body).
- Stuffing the mouth with objects such as used hygiene articles, cleaning rags, and dirty socks.
- Crush injuries, such as smashing fingers or using a heavy roller to injure the thighs or back. In cases where extremities have been amputated (toes, fingers), X-ray examination at the level of amputation can indicate whether an accidental event occurred or whether the alleged amputation by a third party indeed took place.
- Forced surgical amputation of limbs or removal of organs.
- Tooth breaking or extraction without anesthesia.
- Inducing asphyxia, including wet and dry methods, drowning ("water boarding"), smothering, choking, or the use of chemicals.
- Sexual torture or sexual violence: Violence to genitals, molestation, instrumentation, rape.
- Behavioral coercion, such as forced engagement in practices against the religion of the victim (e.g., forcing Muslims to eat pork).
- Denying necessary medical attention.

The above list is not exhaustive. X-ray methods can be helpful in cases of fractures caused in a torture setting and may also be able to detect foreign objects left in the body. In some cases, whether or not evidence of torture can be identified at physical examination depends on the type, intensity, and duration of the torture; moreover, the point in time of the examination, i.e., the interval between the time of torture and the time of examination, is also of considerable relevance since physical torture-related injuries may heal, even if psychological injuries remain.

Means for detecting evidence of torture were described systematically for the first time in the Istanbul Protocol (see above); specific forms of torture will be discussed here in greater detail:

Blunt Trauma to the Soles of the Feet (Falanga). Graessner and Wenk-Ansohn (2000) state that at least 40–100 blows need to be inflicted in order to be able to reliably diagnose the long-term effects of blunt trauma to the feet. Swelling of the soles of the feet and reactive edema are observed, while in extreme cases reduced fatty tissue, slackening of the plantar aponeurosis, and flattening of the longitudinal arch of the foot are additionally seen. Victims describe a burning sensation in the feet and calves, sometimes many years after the event. At physical examination, the great toes should exhibit at least partial hyperextension, while the skin of the slackened plantar aponeurosis shows washed-out lines. Due to the loss of fatty tissue, the victim may be walking directly on bone. The insides of shoe soles may show distinctive patterns of wear. In differential diagnostic terms, congenital changes, vascular disease in the setting of diabetes mellitus, as well as polyneuropathic symptoms of other etiology should be considered.

Burn Injuries. Also seen outside a torture context, burn injuries are well known in forensic medicine. Burn injuries inflicted in the course of torture are often multiple and vary as a function of the intensity and duration of exposure. Cigarettes and cigars may be used to inflict burns and tend to leave highly characteristic scarring: roundish oval with a central raised area or a sunken, distinctly ring-like margin. In cases of torture, burn scars are found on the back, on the dorsum of the feet, and on the upper legs, often arranged asymmetrically. Heated objects leave patterned scars, while scalding, which is altogether rarer, produces flow marks or splash-like injuries (see Chap. 12). Hot coals cause anthracite-colored particles to become embedded in the skin. In differential diagnostic terms, a distinction needs to be made between accident-related injuries and the effects of torture.

Beatings. The patterns of injury seen in beatings, most notably with a fist to the skull, are well known in forensic medicine (see Chap. 8); in this regard, particular attention needs to be paid to nasal bone fractures, tooth marks and tooth loss, midfacial fractures, fractures of the bony orbit (blow-out fractures), mandibular dislocation, and impression fractures. Beatings can cause chronic post-concussion syndrome, short-term memory impairment, as well as organic personality changes. Headaches and neck pain associated with long-term myelogenesis may be reported many years after the event.

Incision and Stab Wounds. As seen in other forensic medical settings, a preserved ability to defend oneself can result in scarring produced by incision or stab wounds to the flexor side of the lower arms, palms of the hands, as well as the skin between the thumb and index fingers and other fingers (see Chap. 9). From a differential diagnostic point of view, these injuries need to be distinguished from scars produced as part of ritual cutting and self-harm. Sharp force injuries may also be caused when a victim is forced to walk over glass fragments. Subsequently, (hyperpigmented) scarring that is only visible using a magnifying glass is often all that remains.

Inserting Objects into Body Openings. This form of torture includes the insertion of usually sharp objects, e.g., into the urethra. Concomitant injuries can lead to scar-related strictures and may require surgery, while recurrent urethritis is also sometimes seen. The violent insertion of a blunt object or the neck of a bottle into the anus does not necessarily leave visible marks. Uncharacteristic symptoms such as frequent constipation and significant psychological impairment are reported.

Injury to Nails. Both finger- and toenails may be affected, either by extraction or by having sharp objects forced under the nails. Stab tracks may be visible for up to 6 months, after which hyperpigmented striae may be perceptible beneath the nail. The effects of fungal infection need to be considered in the differential diagnosis.

While on the one hand any physical examination of alleged torture victims should include a whole-body examination, careful attention should also be paid on the other hand to injuries known to result from, or which could at

least be explained by, the torture method reported. Numerous torture methods produce little or no physical marks, notable among these being electric shock torture, forced standing on tiptoes, being sprayed with a high-pressure water jet, solitary confinement, forced exposure to—usually acoustic—stimuli, forced immersion of the head in water or other liquids, etc. However, in such cases, psychological abnormalities may point to confirmation of the alleged torture.

26.3 Psychological Torture

Methods of psychological torture, also referred to as white torture, are intended to cause suffering in the victim without leaving visible marks. This is achieved by using measures designed to cause the victim psychological harm. The spectrum of possible torture methods here is broad, ranging from sleep deprivation, insults, interrogation while blindfolded, death threats, mock executions, isolation, threats to torture relatives, sexual humiliation, allowing water on slowly drip on the forehead, forcing the victim to witness torture or atrocities being inflicted on others to threats of attack by animals, such as dogs, cats, rats, or scorpions.

Identifying significant physical and/or severe psychological trauma can be highly challenging. Other information is often required to support the plausibility of alleged torture, such as the location at which the torture took place, the form of abuse or torture, social factors, other torture victims, general political situation, and membership of the torture victim in a persecuted group. In the absence of findings of physical injury, it may be necessary to call upon psychological or psychiatric experts to diagnose or exclude a posttraumatic stress disorder.

Complex posttraumatic stress disorder (C-PTSD) refers to psychological symptoms that can develop as a result of severe persistent trauma, e.g., physical or sexual abuse, experiences of war, torture, natural disasters, psychological and/or emotional neglect in childhood, or life-threatening events. Onset may occur either immediately or in a delayed manner months or years after the traumatic experience.

In contrast to simple posttraumatic stress disorder (PTSD), C-PTSD is characterized by a broad range of cognitive, affective, and psychosocial impairments that persist over a prolonged period of time. The term C-PTSD was introduced in 1992 by the US psychiatrist Judith Herman.

The importance of an expert (forensic) medical and psychiatric appraisal also lies in its ability to identify statements and confessions made under torture, thereby rendering them meaningless in a judicial context and enabling the victim access to appropriate therapeutic support. However, it is not the task of forensic medicine to define the border between hitherto permitted interrogation methods and illegal abuse or torture; this task falls to policy and law makers. Moreover, forensic pathologists are generally unable to draw conclusions about the prognosis of PTSD were it to be treated or left untreated. However, victims of violence may experience some therapeutic benefit in cases where verifiable facts and forensically sound conclusions are (or need to be) upheld by a court and ultimately result in sanctions against the perpetrator.

26.4 Physician Participation in Torture

The participation of physicians in sadistic medical experimentation and/or torture during the Second World War (WWII) led to the Doctors' Trial in Nuremberg. A number of cases involving the participation of physicians in abuse and torture have been proven in the postwar period. Miles et al. (2010) reported on a total of 56 physicians in eight countries who have been punished for complicity in torture or crimes against humanity since the Second World War. The study gathered data from four types of tribunal: international courts, national criminal courts, military tribunals, and medical associations. However, it can be assumed that many of the physicians who participate in torture and crimes against humanity escape detection and that only very few of the physicians who are identified are forced to

answer for their crimes in official proceedings. Regional instances in Rio de Janeiro and São Paulo in Brazil dealt with charges against more than 110 physicians; as of 2010, 17 of these physicians had faced prosecution and been punished. In Chile, more than 80 physicians are believed to have been involved in torture; of these, 10 have been penalized. To date, six of an estimated 200 physicians accused of participation in torture in Argentina have been sentenced.

While on the one hand some countries prevent the issue of physician participation in torture from being addressed altogether, others condemn the involvement of physicians in abuse and torture by official bodies while at the same time failing to apply sanctions where necessary or imposing purely symbolic penalties. However, under pressure from the public as well as non-governmental organizations, a number of South American countries have carried out extensive investigations into and proceedings against physicians, some of which have resulted in penalties. There are also countries where physicians involved in torture, like the actual torturers themselves, are granted amnesty from prosecution or where the argument that a physician was acting on orders is considered acceptable justification.

Regulations contained in the international code of professional conduct for the medical profession address the question of how physicians should deal with the issue of torture. As early as 1975, the World Medical Association (WMA) issued a declaration entitled "Guidelines for physicians concerning torture and other cruel, inhuman or degrading treatment or punishment in relation to detention and imprisonment" (Declaration of Tokyo), which was last amended in 2006. The Declaration states:

Preamble

It is the privilege of the physician to practise medicine in the service of humanity, to preserve and restore bodily and mental health without distinction as to persons, to comfort and to ease the suffering of his or her patients. The utmost respect for human life is to be maintained even under threat, and no use made of any medical knowledge contrary to the laws of humanity. For the purpose of this Declaration, torture is defined as the deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority, to force another person to yield information, to make a confession, or for any other reason.

Declaration

1. The physician shall not countenance, condone or participate in the practice of torture or other forms of cruel, inhuman or degrading procedures, whatever the offense of which the victim of such procedures is suspected, accused or guilty, and whatever the victim´s beliefs or motives, and in all situations, including armed conflict and civil strife.
2. The physician shall not provide any premises, instruments, substances or knowledge to facilitate the practice of torture or other forms of cruel, inhuman or degrading treatment or to diminish the ability of the victim to resist such treatment.
3. When providing medical assistance to detainees or prisoners who are, or who could be later be, under interrogation, physicians should be particularly careful to ensure the confidentiality of all personal medical information. A breach of the Geneva Conventions shall in any case be reported by the physician to relevant authorities. The physician shall not use nor allow to be used, as far as he or she can, medical knowledge or skills, or health information specific to individuals, to facilitate or otherwise aid any interrogation, legal or illegal, of those individuals.
4. The physician shall not be present during any procedure during which torture or any other forms of cruel, inhuman or degrading treatment is used or threatened.
5. A physician must have complete clinical independence in deciding upon the care of a person for whom he or she is medically responsible. The physician´s fundamental role is to alleviate the distress of his or her fellow human beings, and no motive, whether personal, collective or political, shall prevail against this higher purpose.
6. Where a prisoner refuses nourishment and is considered by the physician as capable of forming an unimpaired and rational judgement concerning the consequences of such a voluntary refusal of nourishment, he or she shall not be fed artificially. The decision as to the capacity of the prisoner to form such a judgement should be confirmed by at least one other independent physician. The consequences of the refusal of nourishment shall be explained by the physician to the prisoner.
7. The World Medical Association will support, and should encourage the international community, the National Medical Associations and fellow physicians to support, the physician and his or her family in the face of threats or reprisals

> resulting from a refusal to condone the use of torture or other forms of cruel, inhuman or degrading treatment.

A further declaration was adopted at the 49th General Assembly of the WMA in November 1997 in Hamburg, Germany, entitled "Support for medical doctors refusing to participate in, or to condone, the use of torture or other forms of cruel, inhuman or degrading treatment." The declaration makes reference to additional provisions set out in the international professional code of conduct for physicians and reiterates that "medical doctors throughout the world are prohibited from countenancing, condoning or participating in the practice of torture or other forms of cruel, inhuman or degrading procedures for any reason." The WMA's 1993 statement on "Body searches of Prisoners" (Declaration of Budapest, Hungary) is also worthy of note in this regard. In 2011, the organization Medact—the UK affiliate of the International Physicians for the Prevention of Nuclear War (IPPNW)—published a report on five country case studies. The report concluded with several recommendations to the WMA and other national medical organizations. Among other things, the report called for improvements to and increased promotion of medical training. All national medical associations should ensure that, as part of their training, prospective physicians are made aware of:

- What constitutes torture
- How to implement established ethical codes on nonparticipation in and non-condonement of torture
- Which international regulations are relevant in this regard

The report also calls for national medical associations worldwide to ensure that diagnosing evidence of torture, as well as the professional, ethical, and legal implications thereof, is incorporated in medical curricula.

Furthermore, an amendment to the War Crimes Act that would criminalize all unethical experimentation on humans has been called for. This move is aimed at bringing the Act in line with the Geneva Convention. Finally, greater collaboration between national medical associations and UN Special Rapporteurs on torture and human rights organizations is proposed.

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Forensic Psychopathology

27

Case Study

The heavily inebriated 46-year-old Mr. A became disorderly in front of a pizzeria, throwing a wine bottle that by chance struck one of the guests on the head. At the same time, he was shouting incomprehensible expletives. Initially, he failed to notice the two uniformed police officers called to the scene. As the officers tried to approach, Mr. A started to lash out wildly. On trying to seize him, one of the police officers sustained a violent kick between the legs, while both officers were called names such as "assholes" and "pigs." Mr. A was wrestled to the ground, where he struck his head from a low height against the curb. Once handcuffed, it was possible to take him to the police station, where a blood sample was taken. His blood alcohol concentration was determined to be 3.56‰. Mr. A became somewhat calmer at the police station and, when questioned, reported that a group of guests at the pizzeria had beaten him up in the past and that this was why he had thrown the bottle. In the sobering-up cell, Mr. A suddenly vomited, in addition to complaining of a headache. The guest at the pizzeria and the police officer both brought charges against Mr. A for bodily harm, the police officer additionally for insulting a police officer and resisting arrest. The public prosecutor was able to ascertain from the files that Mr. A had attracted similar attention on 23 occasions in the previous 2 years, always in a state of extreme intoxication. Whereas the public prosecutor applied to the court to place the defendant in a detoxification clinic, the defense alleged that Mr. A could not be held legally responsible for his actions due to his state of intoxication at the time of the incident; that the statement he gave at the police station was not permissible, since it had been made at a time when he was unfit to undergo police questioning; and, moreover, that one must assume that Mr. A was unfit to be held in custody as a result of striking his head on the curb and subsequently vomiting and that he should instead have been taken to the hospital.

A person's ability to gain insight, judge circumstances or situations appropriately, and modify their behavior accordingly, i.e., their accountability and ability to reason, can be impaired as a result of a multitude of factors, including alcohol, drugs and/or medication, neurological and psychiatric disease, as well as possibly also personality disorders. In this context, assessing criminal responsibility is of particular significance and is usually carried out by a forensic psychiatrist in complex cases. The question of criminal responsibility needs to be viewed

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independently of statutory provisions regulating the age of criminal responsibility, which differs from country to country.

In some cases, it is necessary to assess whether the conditions for placement in a psychiatric institution, in a detoxification clinic, or in long-term preventive detention have been fulfilled. Occasionally, prior to the release of offenders, forensic psychiatric risk assessments are carried out to address the question of whether the individual in question still poses a threat. Finally, and particularly in the case of sexual offenses, an assessment of the credibility of witness statements is required.

27.1 Custody

Depending on national laws, a suspect can be admitted to a psychiatric clinic in order to carry out the in-depth psychiatric work-up necessary to assess criminal responsibility. A suspect may also be held temporarily in a psychiatric clinic in the interests of public security. Moreover, statutory regulations make provision for an individual to be held in custody in a reformatory if the perpetrator of the crime is exempt from criminal responsibility or has diminished criminal responsibility. This is also valid when an addiction disorder is the cause of diminished responsibility, and it is assumed that the individual in question will remain in a state of diminished responsibility and is likely to commit crime as a result of their addiction. However, on the other hand, since detention in custody represents a very serious intervention, a judicial review needs to be carried out on a case-by-case basis, and regular reviews need to be made to establish whether prolonging temporary detention is necessary.

27.2 Diminished or Nonexistent Criminal Responsibility

A number of legal conditions need to be fulfilled for diminished or nonexistent criminal responsibility to be established; severe mental illness, profound consciousness disturbances, intellectual impairment, as well as other psychological abnormalities are among these. The disorders or states considered to fulfil these conditions, as stated in German criminal law, are given in Table 27.1.

Table 27.1 Disorders and criteria on which the assessment of diminished or nonexistent criminal responsibility is based (according to German criminal law)

Criterion	Example
Pathological mental illness	Endogenous and exogenous psychoses, psychotic residual syndrome, schizophrenia, organic psychological disorder, (acute) intoxication, disorders following cerebrocranial trauma
Profound consciousness disorders	Shock, impaired consciousness associated with severe emotional agitation (crime of passion)
Intellectual impairment	Congenital intellectual impairment, IQ below ca. 70
Other mental abnormalities	Neuroses, sexual deviation, addiction-related personality changes

Finally, it is necessary to establish whether there is a relevant causal chain between the disorder and the crime committed. For this to be the case, the disorder needs to have caused either a complete absence of—or at least a significant reduction in—an individual's *accountability* and *ability to reason*.

1. *Ability to reason*
   Cognitive knowledge that a crime is as such forbidden. Even perpetrators with psychoses possess this basic knowledge.
2. *Accountability*
   Where a perpetrator understands the wrongness of their deed, their ability to act according to this understanding.

Significantly diminished or *nonexistent criminal responsibility* generally needs to be established retrospectively, partly on the basis of preexisting diseases (e.g., endogenous psychoses, organic brain processes, neuroses, psychopathologies, personality disorders, and drive disorders) and partly on the basis of the symptoms and findings relating to the incident in question. Verifiable facts need to be assessed for their validity and to establish whether they are consistent with the committing of a crime. Indications of intact consciousness include:

- Preparatory actions consistent with the crime.
- Viable chronology of sequence of actions.
- Purposeful behavior.
- No abrupt cessation of action.
- Performing a complex, divisible crime for which obstacles and resistance need to be overcome.
- Perceptible agitation following provocation.
- Orientedness (temporal, spatial, towards oneself, towards others).
- Absence of severe psychomotor abnormalities.
- Appropriate, coordinated behavior following the offense.
- No indication that the act was out of character (motivational association?).
- Failure to provide a patient history (or patient history is of limited validity, since nonverifiable and suppression may play a role).
- Posttraumatic disorders of consciousness can be excluded (verifiable trauma, abnormal behavior, impaired consciousness and disorientation, aggressive behavior?).

Alcohol-related amnesia (possible from around 1.5‰) tends to relate to irrelevancies, while serious incidents are more readily recalled (intact isolated recollections). Medication-induced and craniocerebral trauma-related memory gaps tend to be clearly defined in time, sometimes also as retrograde amnesia, which tends not to be the case in alcohol-related amnesia. In Germany, if nonexistent criminal responsibility is established on the basis of the above grounds, a conviction for intoxication may be considered if there is evidence of alcohol or other intoxicating substances.

Pathological Intoxication. Rare special forms include pathological intoxication and abnormal response to alcohol. The former results in relatively low alcohol levels causing sudden agitation, generalized aggressive behavior, and disturbed orientation. Motor deficits are rarely seen. Pathological intoxication ends in deep sleep followed by a complete lack of recall of the period of intoxication.

Abnormal Alcohol Reaction. An abnormal alcohol reaction (complicated intoxication) is also associated with states of excitement and agitation, which can manifest as aggressive behavior, but may also demonstrate fear-related components and uncharacteristic emotions.

Whereas pathological intoxication fulfils the criteria for criminal responsibility to be nonexistent, an abnormal alcohol reaction is considered grounds only for diminished responsibility and would require further assessment before responsibility was deemed nonexistent.

In addition to criminal responsibility, there are numerous situations in which authorities call upon medical expertise to assist in the decision-making process (see Chap. 17); such situations may require an assessment of the following:

- Fitness to understand a police caution
- Fitness to undergo questioning
- Fitness to be held in custody
- Fitness to be placed in an institution
- Fitness to serve a custodial sentence
- Fitness to travel
- Fitness to testify
- Fitness to stand trial
- Capacity to take legal action
- Fitness to undergo deportation (travel)
- Ability to make a will
- Credibility (also by psychologists)
- Driving ability
- Maturity of children and adolescents (by a psychiatrist)
- Social and criminal prognosis in the case of offenders with mental illness
- Age estimation in asylum seekers
- Age estimation to assess the need for a court-appointed special advocate
- Capacity (or incapacity) to contract (psychiatric assessment)

Expert appraisals, many of which include psychopathological findings, are provided predominantly by psychiatrists or forensic pathologist, including, for example, the assessment of an individual's ability to make a will (Fig. 27.1).

27.3 Ability to Make a Will

Under certain conditions, a person aged 16 and over has the right to make a legally binding will in Germany; full rights to make a will are not granted until the age of 18. The ability to make a will requires that the individual making the will recognizes the full implications of their decisions. That a person is capable of making a will is

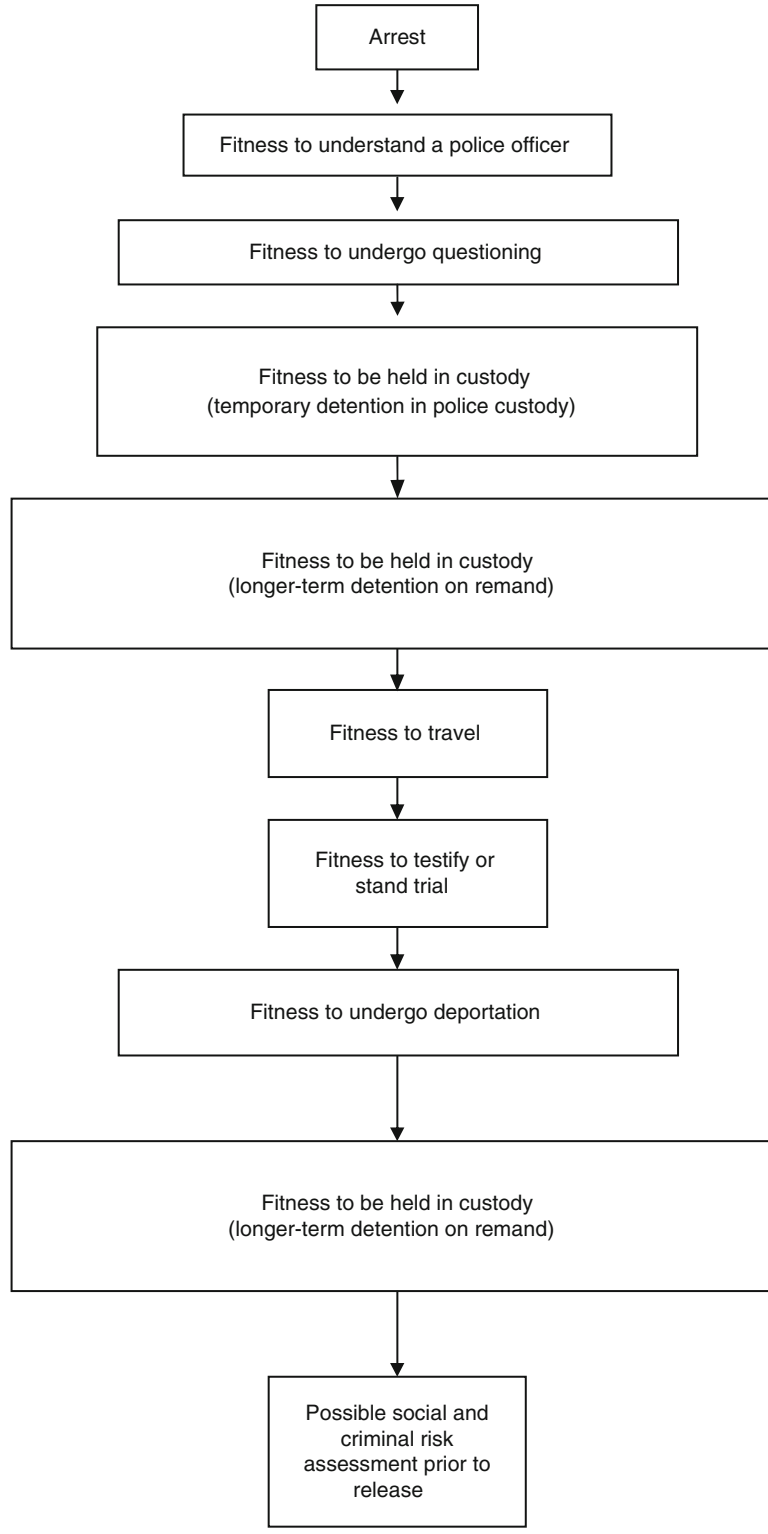

Fig. 27.1 Scenarios requiring expert appraisals during criminal investigation and trial proceedings

considered to be the norm. Not infrequently, however, (potential) benefactors of a will made by an often elderly testator claim that the latter was not in a position to freely state their will or intentions, particularly not without external influence from a third party, at the time the will was written. Any doubt relating to a person's ability to write a will prompts a retrospective assessment, e.g., of alleged states of confusion in the case of cerebral sclerosis (dementia patients) or writing a will under the influence of medication.

27.4 Crimes of Passion

Certain crimes are the result of the perpetrator's state of high-grade excitation or agitation.

Important: A crime of passion is a crime in which the perpetrator is able to wilfully control their actions to only a limited extent due to the influence of emotions, becoming almost a passive victim themselves of functional processes.

Emotional changes occurring in the context of a crime of passion can affect otherwise healthy individuals, making their distinction from "normal" affect-laden acts necessary. Genuine crimes of passion often involve acts that cannot be reconciled with any organized course of action. The usual sequence of an action, i.e., setting a goal, planning the goal, planning the act, and carrying out the act, is often disrupted or incomprehensible; contradictory behavior is also seen. Characteristics of a crime of passion include the following:

- Emotional states such as distress, fear, anger, and rage
- Subsequent and intense emotional convulsions, often associated with concomitant vegetative symptoms
- Affects are of short duration
- Affective changes may dominate to such an extent that an individual's rational persona is completely suppressed

Perpetrators often show a lack of flexibility in the spectrum of their responses and actions, often combined with feelings of inferiority and a low frustration tolerance threshold. Case histories often reveal affect-laden constellations involving slights to and humiliation of the future perpetrator. Finally, a sudden and unexpected explosive unloading of affects takes place; memory lapses or even amnesia are reported after the act, followed by a "sudden awakening in reality." Conversely, a number of characteristics seen in a deed speak against a crime of passion; a number of points are mentioned in the literature, such as the following made by Saß (1983):

1. Aggressive imaginative preoccupation.
2. Announcing the act.
3. Aggressive behavior leading up to the act.
4. Actions designed to prepare for the act.
5. The perpetrator determines the circumstances of the act.
6. Lack of association between provocation, state of agitation, and act.
7. Targeted organization primarily by the perpetrator of how the act will unfold.
8. Precise and detailed description of the act after it has been committed
9. Affirming comments by the perpetrator about the act.
10. Absence of concomitant vegetative, psychomotor, and psychological symptoms.

In the author's opinion, points 4–9 carry the most weight as indicators. One cannot speak of a profound consciousness disorder or of a crime of passion if the characteristics mentioned above are observed.

27.5 Psychopathological Abnormalities: Personality Disorders, Mobbing, Stalking, Narcissism, and Querulousness

The term "psychopathy" was chosen in the past as an umbrella term for a number of personality-related disorders; the term "sociopathy" is sometimes used today. As a rule, when a crime has been committed, a psychiatric expert is called upon. The same applies to the following behavioral abnormalities:

Stalking. A criminal offense in some countries, stalking refers to psychological violence as a means to intimidate a victim in a threatening

manner: sustained sudden personal contact, intimidation via SMS and email, harassing telephone calls, lurking near the victim's home, workplace, or leisure activities. Constant harassment makes victims feel threatened in their private sphere. Various types of stalkers have been identified: the "ex-relationship stalker" (the most common), the "amorous stalker" (female perpetrators also possible), the "obsessed stalker," and the "sadistic stalker." The last two types can be dangerous. The personality of the "sadistic stalker" is often assigned to the same group of psychopathies as the "narcissistic personality disorder."

Narcissistic Personality Disorder. According to the definitions of this personality disorder offered in the literature, the affected individual is not necessarily the person who directly suffers as a result of their disorder, but rather more those around them:

> A narcissist is often characterized by a grandiose sense of self-importance; he craves excessive admiration and believes he can only be understood by special people; he exploits interpersonal relationships, lacks empathy towards others, behaves in an arrogant and haughty manner, and is envious and excessively self-promoting; he consistently believes himself to be in the right and shows disdain for the legal system, believing there to be "another set of rules" for him; he is completely devoid of any sense of remorse or guilt, yet on occasion has a heightened sense of self-pity; after an early period of success, he descends into mediocrity, becoming bored with his work and displaying sudden attacks of vengeful anger in response to supposed insults. [translated from Hochmeister et al. (2007)]

Not all the following criteria of a personality disorder (see also ICD 10 F60.8), or the nine criteria according to the Diagnostic and Statistical Manual of Mental Disorders (DSM IV) of the American Psychiatric Association (APA), necessarily need to be fulfilled:

- An exaggerated sense of importance
- Heavy preoccupation with fantasies about success, power, and beauty
- The belief that he/she is "special" and can only interact with people of equal standing
- A craving for excessive admiration
- A sense of entitlement to preferential treatment
- A tendency to form exploitive and manipulative relationships
- A lack of empathy
- Often experiences envy or believes others to be envious
- Displays arrogant, haughty behavior

It is very rare for all criteria to be met in their fullest degree. The diagnosis is established when at least five characteristics are present. It is important that the clinical picture include a sustained pattern of behavior over time. One specific feature of narcissistic personality disorder is that the affected individual's thoughts, emotions, and behavior are all minutely focused on that individual's sense of their own value and how they compare with others. Narcissists crave recognition at any price; mediocrity is perceived as a threat. At the same time, they break one of the fundamental conditions of interpersonal relationships: "Do unto others as you would have them do unto you." Narcissists, in contrast, treat those around them in precisely the manner they would not wish to be treated themselves. Moreover, the narcissist has learnt to maximize his impact on the world around him: he is often well-read, intelligent, and is quite capable of applying his charm to achieve his goals. Narcissists tend to react to insults in a typical manner: the desire for revenge predominates, accompanied by a need to correct a supposed wrong by whatever means necessary. The subjective perception of a "narcissistic personality disorder" is comparatively mild and remains so as long as the individual's intelligence, charm, talent, and other qualities are able to help him achieve his goals. Thus, it is unsurprising that narcissistic personalities are commonly encountered in top positions in the fields of politics, economics, and science, as well as in the entertainment industry.

Mobbing. Mobbing describes continuous bullying, vexatious, and insulting behavior by colleagues or superiors, for example, with the intention of inducing the "mobbed" individual to resign. Mobbing is often carried out by superiors, assuming their behavior is supported or at least tolerated by others. Behavior of this kind is often caused by, again, a sense of omnipotence, personality deficits, or a personality disorder.

Querulousness. A querulent is defined by Hinsie and Campbell in their 1981 dictionary as follows:

> Querulent. Ever suspicious, always opposing any suggestion, complaining of illtreatment and of being slighted or misunderstood, easily enraged, and dissatisfied with conditions as they exist.

A querulent also tends to be self-opinionated, fanatical, obstinate, stubborn, pigheaded, humorless, and vulnerable. He recognizes general law, respecting it formally insofar as it applies to him. However, he sees himself as a fighter, albeit a nonviolent one, against conditions and society. The querulent appears to work with seemingly legal means, interpretations of the law, written petitions, and notices, etc. In the course of his dealings, he often fails to treat those around him with due consideration. Although the querulent is usually described as being polite, his is a somewhat formal and cool politeness; for the rest, he is distant and avoids close contact. The concept of querulousness, however, is not sufficiently based in science, describing far more a form of abnormal behavior than a definable personality type.

Spree Killing. Spree killers are usually young men displaying parallels in their personal development. They are generally quiet loners who show (relative) performance deficits. They often lack the acceptance of like-minded people. Vilification and humiliation are not dealt with in a confrontational manner, serving instead to silently foster rage, hatred, and the desire for revenge. On occasion, this underlying emotional state is combined with access to firearms, whereby the adults responsible for and in legal possession of firearms contravene the law by making the weapon(s) easily accessible. Careless handling of legal firearms is particularly alarming when obvious behavioral abnormalities in the future spree killer are ignored.

Risk Assessment. Assessing the risk posed by convicted offenders, in particular convicted sex offenders pending release from prison, is among the most challenging tasks in forensic assessment. As such, risk assessment should only ever be assigned to experienced forensic psychiatrists and made subject to the highest possible quality standards.

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Medical Malpractice

28

Case Study

A small surgical towel was retained within the pericardium of a 54-year-old male patient undergoing heart surgery. Wound healing was uncomplicated, and the patient's only symptom was an intermittent sensation of pressure in the retrosternal area. The towel was discovered during routine X-ray follow-up several months after surgery. Removal of the large foreign body was medically indicated, naturally making it necessary to inform the patient about the finding and the need to reoperate. Although the patient withstood surgical removal of the towel well and recovered without complication, he brought charges against the cardiac surgeon responsible for the initial operation.

Depending on the prevailing legal situation, physicians can be made liable for medical malpractice. The majority of cases involve civil claims for damages and compensation. Although rarer, criminal charges are generally brought in the form of claims of negligent bodily harm or negligent homicide. However, claims of breach of a duty of care, illegal bodily harm despite the patient's consent, illegal termination of a pregnancy, issuing an erroneous medical certificate, as well as culpable homicide upon patient request are also seen. In addition, professional sanctions in the case of culpable medical error are also possible.

Expert assessments of medical malpractice claims are dealt with in a variety of ways. While forensic pathologists are active primarily within the context of investigations carried out by the public prosecutor, clinicians specialized in a relevant field may also be called upon by health insurances and other insurance institutions to assess medical malpractice claims. Some professional medical bodies have set up special committees to deal with patient complaints as well as medical malpractice claims brought by surviving relatives.

28.1 The Concept of "Medical Malpractice"

Medical malpractice as a concept is poorly defined; a distinction is sometimes made between "simple malpractice" and "gross malpractice."

Simple Malpractice: An error in treatment is considered to have taken place if a physician, in the performance of his medical duties, either performs those measures objectively deemed necessary by medical opinion improperly, or fails to perform them at all, i.e., when he neglects to use the care and caution normally expected of a competent physician mindful of his duty in a given situation.

Gross Malpractice: Gross malpractice is deemed to have taken place when a physician is in clear breach of the established rules of medical treatment or proven medical knowledge and has committed an error that is incomprehensible from an objective point of view and, moreover, wholly inadmissible in a physician.

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Although it is the task of medical experts in their capacity as assessors appointed by the authorities or relatives to verify whether the rules of medical practice have been breached, a court will ultimately decide whether a physician's actions qualify as simple or gross malpractice.

Various forms of medical malpractice are discussed in the legal literature:

- Organizational culpability, most notably on the part of the clinic/hospital owners, the management, and/or the chief physician
- Contributory negligence liability, particularly in cases where physicians undertake tasks beyond their knowledge and skills
- Collaborative errors, either within one hierarchical level in the case of a horizontal division of tasks (e.g., joint treatment of a patient by several physicians) or by physicians on lower/higher hierarchical levels in the case of a vertical division of tasks
- Incorrect treatment, when medical measures that are indisputably necessary are not taken despite being generally recognized as successful and instead substituted by a patently less successful or incorrect therapy
- Failure to treat, despite a treatment being medically indicated and viable

By way of derogation from this legalistic classification, medical malpractice can also be classified according to a range of medical aspects. Studies in forensic pathology designed to analyze medical malpractice claims involving alleged fatal outcomes have led to a classification of this type of claim (Modified from Dettmeyer (2001)):

- Claims concerning insufficient diagnosis (e.g., failure to perform radiological diagnosis following traumatic brain injury)
- Incorrect medication (e.g., incorrect medication, dose, application method or/and site, as well as failure to observe contraindications)
- Foreign bodies retained in surgical areas (e.g., "forgetting" surgical cloths, clamps, scissors, and needles in the surgical area; Fig. 28.1)
- Patient mix-ups (e.g., due to the same or similar names)
- Incorrect positioning of the patient during surgery
- Refusal to make a home visit despite the clear need to do so
- Failure to insist that a patient presents urgently at the physician's office
- Delayed transfer to hospital
- Incorrect injections, infusions, and transfusions (e.g., AB0 incompatibility)
- Incorrect indication
- Avoidable diagnostic errors (e.g., incorrect interpretation of an otherwise unambiguous X-ray)
- Incorrect treatment information or advice
- Unnecessary medical interventions on the basis of wilful or negligent "manipulation of a medical indication"
- Culpable failure to identify postoperative complications in a timely manner (e.g., postoperative peritonitis)
- Insufficient prophylaxis against decubitus ulcers
- Insufficient venous thromboembolism prophylaxis (e.g., failure to order anti-embolism stockings)
- Claims relating to overlooked myocardial infarction (e.g., failure to perform an ECG despite clear clinical symptoms involving left chest pain radiating to the left arm in a middle-aged heavy smoker)

The first step of any medical malpractice claim involves lodging a complaint or justified suspicion relating to a treatment error. If an error in treatment is proven to have taken place, causality then needs to be established between the error and death, an often challenging if not impossible task. Thus, despite clear evidence that myocardial infarction was overlooked, it is generally not possible to prove with the degree of certainty required by criminal law that a patient would have survived had the heart attack been recognized promptly.

An analysis of forensic appraisals relating to criminal investigations into medical malpractice showed the distribution of malpractice claims across the different medical disciplines, surgery being the discipline most frequently involved. The medicolegal literature provides numerous case studies, alongside large studies showing the different types of medical malpractice claims and

actual instances of medical malpractice. Notable among these are "decubitus ulcer cases," "the overlooked myocardial infarct," and "delayed recognition of postoperative complications," e.g., postoperative peritonitis following abdominal surgery and postoperative hemorrhage following tonsillectomy.

Death on the Operating Table. Iatrogenic error is most readily assumed in cases where a patient dies either during or immediately after medical treatment, most notably if death occurs on the operating table. However, even in such cases, an actual error on the part of the physician can only rarely be proven. Checking the patient's informed consent, during which risks and side effects should have been discussed comprehensively, is particularly important here. This applies, for example, to rare but possibly fatal complications associated with coronary angiography (coronary wall rupture, coronary thrombosis, myocardial infarction, and intraperitoneal hemorrhage originating at the puncture site in the groin region).

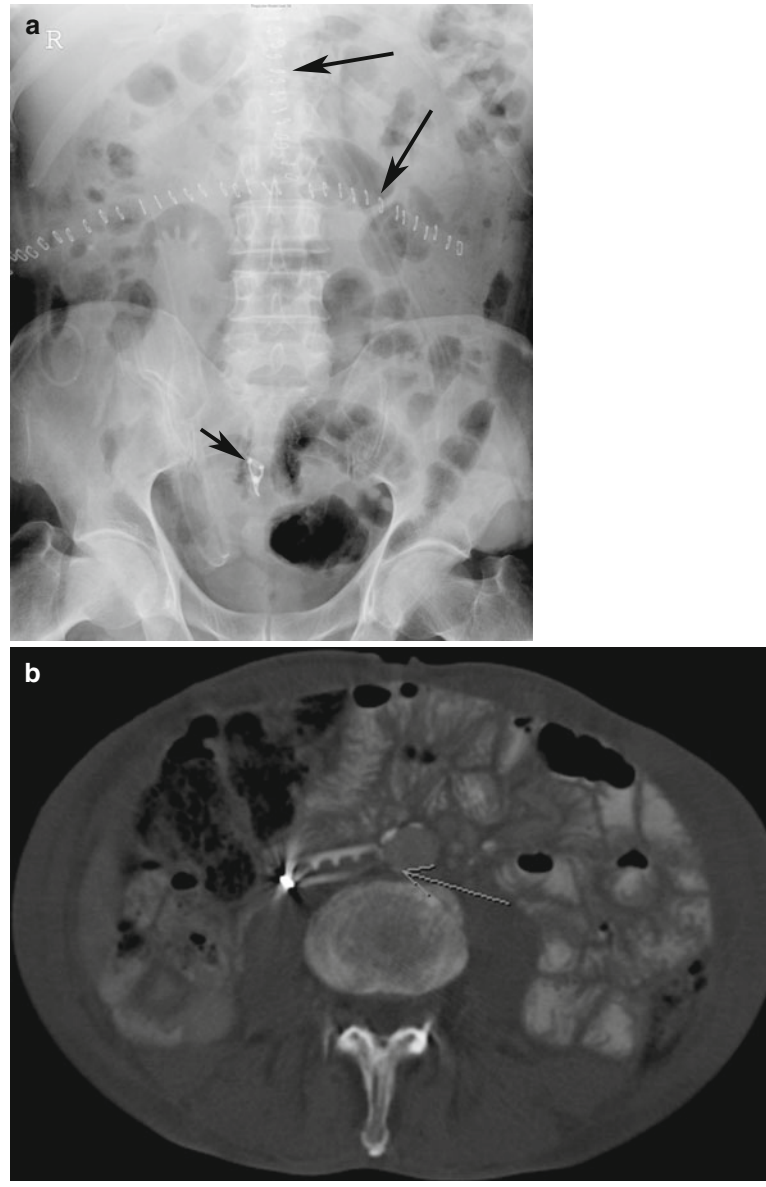

Fig. 28.1 Objects "forgotten" in the surgical area: (**a**) a surgical cloth in the abdomen, (**b**) a plastic clamp, and (**c**) a needle

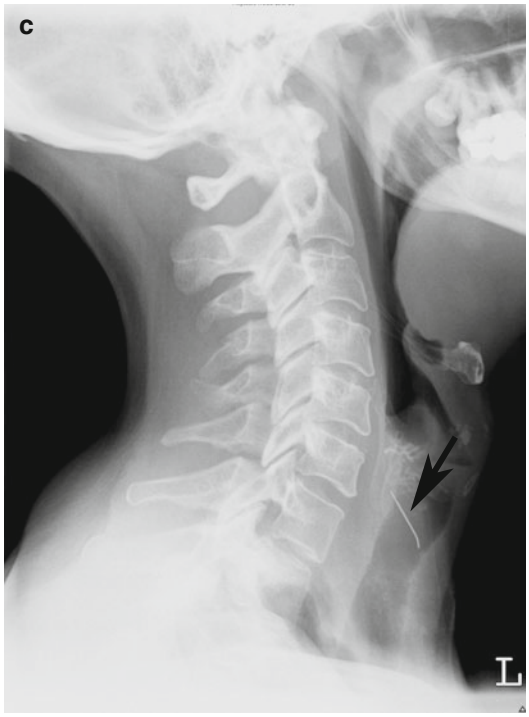

Fig. 28.1 (continued)

Claims Relating to Insufficient Decubitus Ulcer Prophylaxis. All attempts should be made to avoid decubitus ulcers by means of medical/nursing care (Fig. 28.2). Where this fails, the cause of failure needs to be sought. However, it is a recognized fact that some cases of decubitus ulcer cannot be avoided (e.g., obese paraplegic patients, diabetes, fecal and urinary incontinence, and status post-chemotherapy for malignancy). In such cases, all medical and nursing records and documentation need to be checked! Given that even extensive and deep decubitus ulcers can exist for long periods of time, a decubitus ulcer is not necessarily a cause of death in itself. An alternative, unrelated, and plausible cause of death is often found at autopsy. However, in the case of an infected decubitus ulcer accompanied by a febrile or even septic state at the time of death, microbiological comparison of the decubitus ulcer pathogen and blood from the deceased is mandatory. A correlation in the pathogen spectrum may indicate that a decubitus ulcer, otherwise avoidable with appropriate care, was the cause of death.

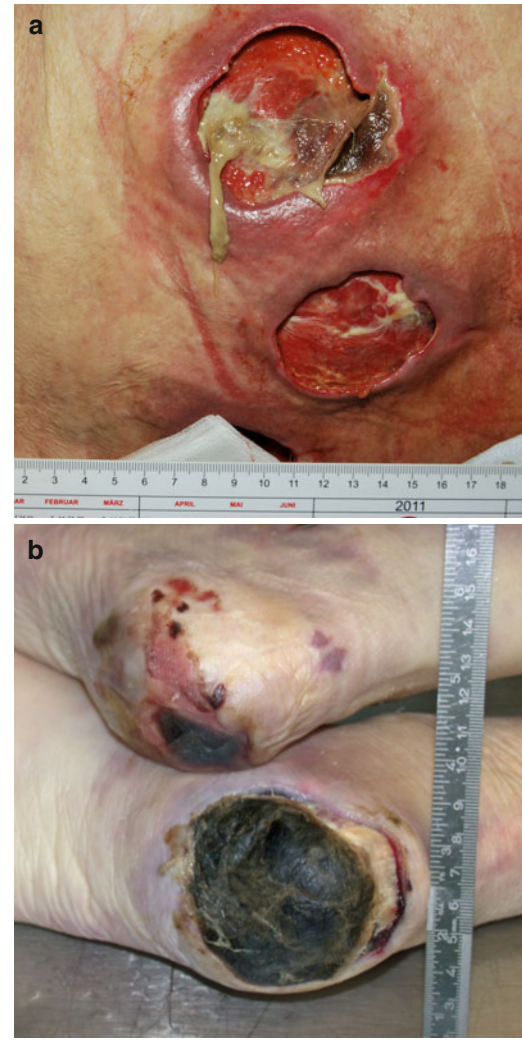

Fig. 28.2 (**a**, **b**) Decubital ulcers

28.2 Handling Medical Malpractice Claims in Forensic Medicine

Forensic pathologists are predominantly concerned with medical malpractice that is suspected or alleged to have caused the death of a patient. Formulating an expert appraisal on medical malpractice claims of this kind is often complex and demands a careful assessment of the facts of the case, including:

(a) Underlying disease in a patient
(b) Medical care that was undertaken or omitted despite medical indication

(c) Checking documentation held in hospital records
(d) A consideration of all examination findings
(e) Evidence of medical malpractice
(f) Establishing the cause of death by means of forensic autopsy
(g) Assessing causality between a proven treatment error and death
(h) The question of culpability for a treatment error (foreseeability and avoidability)

Numerous medical malpractice claims are promptly resolved at autopsy: either there is no evidence to support the alleged or suspected treatment error or a cause of death that is wholly unrelated to the alleged treatment error is found.

Important: In the vast majority of medical malpractice claims, forensic autopsy serves to exonerate the physician in question.

Forensic expert appraisals are able to draw conclusions on a cause of death on the basis of autopsy findings as well as findings from other, subsequent investigations. However, it is frequently necessary to call upon a clinical assessor with expertise in the relevant discipline for the appraisal of alleged medical malpractice. It is at this point that forensic pathologists need to recognize the boundaries of their own competence with a somewhat critical eye.

28.3 Conduct in the Event of a Medical Malpractice Claim

Naturally, the simple fact that a medical treatment fails does not constitute medical malpractice. Rather, evidence showing that a physician was in breach of accepted medical knowledge at the time of treatment is required, whereby the following applies:

Important: In his appraisal of a treatment error, the forensic expert needs to consider the perspective of the accused physician from an ex ante position as well as the standard of medical science at the time of the alleged treatment error.

In the case of a medical malpractice claim, it is essential in the first instance to collect all relevant evidence, i.e., all patient records, including X-rays, and witness statements from co-attending physicians and medical personnel. Making hasty statements to patients, their relatives, superiors, colleagues, staff, and insurance institutions should be avoided at all costs due to the perennial risk the that laypersons will interpret statements incorrectly.

Once hospital records have been thoroughly reviewed, a calm and objective consultation should be held with the patient—or the patient's relatives in the case of a fatality—in the presence of a neutral witness where possible. Making a written record of the consultation is recommended. Under no circumstances should any influence be exerted by either medical or nonmedical personnel. In the case of fatality, the mode of death given in the death certificate should be carefully scrutinized. Ideally, a neutral physician not implicated in the medical malpractice claim should fill out the death certificate. A medicolegal autopsy should be sought if a medical malpractice claim is already known to exist at the time of death. If there is a risk that medical records may be confiscated, copies for own use should be made promptly.

Important: Under no circumstances should medical records be manipulated in any way after the fact!

While on the one hand no physician is obliged to admit to an error and thereby incriminate himself/herself, if there has never been any doubt that a treatment error was made, the physician's liability insurance should be called upon to compensate the damage as soon as possible. No written statement should be submitted to the police until legal counsel has been sought.

However, the following applies in the case of an error that has not (as yet) caused the patient's death: where deemed necessary in the interests of the patient's health, the patient must be informed of a treatment error (e.g., retained surgical cloth in the surgical area; see the "Case Study" above), in order to ensure appropriate decision-making in subsequent treatment.

28.4 Error Prevention Strategies

Recent years have seen a greater willingness to systematically record, analyze, and take precautions against "incidents" and possible medical

errors in the interests of future patients. The registration of "near mistakes" plays an important role to this end. Against the background of the discussion about patient safety, numerous countries have set up error reporting systems, such as the British National Reporting and Learning System (NRLS), its predecessor, the first worldwide Primary Care International Study on Medical Errors (PCISME), or the Swiss Critical Incident Reporting System (CIRS), an online reporting system (www.cirsmedical.ch/kbv/cirs/cirs.php). These reporting systems are able to provide valuable information on the type and incidence or specific treatment errors and how errors could be avoided.

28.5 The Structure of a Forensic Appraisal in Medical Malpractice Claims

In order to satisfy the level of complexity required of an appraisal, forensic appraisals of medical malpractice claims should be structured, possibly beginning with a table of contents. By way of suggestion, the points mentioned in Table 28.1 can be used to structure an appraisal of this kind.

Table 28.1 Proposed structure of a forensic appraisal in medical malpractice claims

1.	Commissioning authority/agent
2.	Commissioned on……. Received on ……
3.	Commissioned appraisal: individual lines of inquiry
4.	Verification of the appraiser's professional and personal suitability to formulate the appraisal (possible conflict of interest?)
5.	Basis of the appraisal
5.1.	Documentation made available by the commissioning agent/authority
5.1.1.	Files (public prosecutor's investigation file, informally communicated case history, other previous appraisals, e.g., technical experts, criminal police records, appraisals from the appraiser's own institute)
5.1.2.	Medical records
5.1.2.1.	Medical records from the family physician
5.1.2.2.	Medical records from the emergency-care hospital (including emergency physician protocols, and findings on admission)
5.1.2.3.	Medical records relating to other previously and/or subsequently treating hospitals or physicians
5.1.2.4.	Medical records from, e.g., a convalescent clinic
5.1.2.5.	Other preliminary medical appraisals, relating most notably to possible long-term effects
6.	A description of the relevant facts relating to the line of inquiry (selective/evaluative samples), including a chronological table in the case of complex facts (general chronology)
7.	Special chronology (e.g., course of the temperature curve, changes in leukocyte numbers or other laboratory values, and sequence of and intervals between radiological findings)
8.	Own investigations
8.1.	Own findings on the patient (medical history, diagnosis)
8.2.	Autopsy findings according to autopsy protocol
8.3.	Histological analysis, in particular of internal organs
8.4.	Chemical-toxicological analysis, in particular of medication levels
8.5.	Postmortem biochemical analysis of, e.g., vitreous humor, cerebrospinal fluid, blood, serum (e.g., in the case of fatalities in diabetics)
9.	Other additional specialist medical investigations/appraisals
10.	Concluding expert opinion
10.1.	Underlying pathology
10.2.	Cause of death
10.3.	Treatment error: breach of medical standards
10.4.	Causality between treatment error and damage (death)
11.	Summary
12.	Appendix: originals of the preliminary and supplementary medical appraisals, bibliography, possibly also image files, etc.

Modified from Dettmeyer and Madea (1999)

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Forensic Alcohology

29

Contrary to popular belief, hashish and heroin are not the most commonly used narcotic drugs but rather ethanol (ethyl alcohol, C_2H_5OH, sometimes also referred to as grain or drinking alcohol). Any use of the term "alcohol" in the following refers explicitly to ethanol. In the case of congener analysis (see Sect. 29.3.4), however, other alcohols can also play a role.

Blood alcohol concentrations (BAC) are given here in per mill (‰), where 1‰ corresponds to 1 g alcohol in 1 kg blood. BACs are often given as a percentage, where 1 % corresponds to 10‰. Many people are affected by the severe health and social problems caused by alcohol, the "everyday drug." Nevertheless, a widespread uncritical and indeed positive attitude towards alcohol prevails in our society. On average, the per capita quantities of pure alcohol given in Table 29.1 are consumed annually.

These numbers are a stark indication that large proportions of the population in many countries have an alcohol problem. Naturally, many road users can be found among these who would be more accurately classified as "driving drinkers" than "drinking drivers."

29.1 Alcohol Detection (Screening)

The following strategies can be used to detect alcohol:

Appearance. Attempts are often made to draw conclusions from a person's outward appearance about their degree of alcohol intoxication or BAC. Two important *cautionary points* should be made here:

- The absence of the *smell of alcohol on a person's breath*, particularly in unconscious individuals, in no way excludes severe alcohol intoxication. Reasons for this absence may include:
  - Low intrinsic odor of certain forms of intoxicating liquor (e.g., vodka)
  - Superficial respiration in the patient
  - Impaired sense of smell in the attending person due to:
    Cold or flu
    Other impairments

  In contrast, the consumption of small quantities of intoxicating liquor (e.g., a sip of beer) can, in certain cases, produce a strong odor of alcohol.

Table 29.1 Annual alcohol consumption (liters) in over 15-year-olds The Organisation for Economic Co-operation and Development-OECD (2009, actual data published regularly)

Ireland	13.4	OECD	9.7
Hungary	13.2	The Netherlands	9.6
France	13.0	New Zealand	9.6
Austria	12.9	Greece	9.0
Denmark	12.1	Slovak Republic	8.9
Czech Republic	12.1	United States	8.6
Spain	11.7	Italy	8.1
Portugal	11.4	Canada	8.1
United Kingdom	11.2	Korea (Rep.)	8.0
Belgium	10.7	Japan	7.7
Finland	10.5	Iceland	7.5
Switzerland	10.4	Sweden	6.9
Poland	10.3	Norway	6.5
Germany	9.9	Mexico	4.6
Australia	9.9	Turkey	1.2

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Table 29.2 Frequently observed stages of alcohol intoxication

BAC (‰)	Stage of alcohol intoxication	Symptoms
0–0.3		Generally no clinically apparent changes (except in cases of intolerance)
0.3–0.5		Performance deficits can already be observed
0.5–1.5	Mild intoxication	Euphoria, lack of judgement, reduced attention and concentration, increased impulsivity, logorrhea, mildly impaired equilibrium, slow pupil reaction, nystagmus, weakened spinal reflexes
1.5–2.5	Medium intoxication	Symptoms of the previous stage are aggravated and accompanied by impaired vision, impaired gait, behavioral disinhibition, lack of insight
2.5–3.5	Serious intoxication	Marked difficulties walking and talking (staggering, slurring), increased mental confusion, impaired orientation, loss of memory
Over 3.5	Severe intoxication	Imminent risk of death, generally strongly impaired or absent consciousness, "alcoholic narcosis," absence of reflexes, risk of vomitus aspiration and suffocation in the absence of assistance, death frequently occurs as a result of hypothermia or respiratory paralysis

Modified from Schwerd (1992)
BAC blood alcohol concentration; 1‰ corresponds to 0.1 %

- It can be highly challenging and problematic to attribute a certain constellation of symptoms to a certain level of alcohol intoxication. Classification tables (see Table 29.2) can be helpful in establishing an association between a particular BAC and the alcohol-related performance deficits *frequently* seen. However, exceptions to these comparatively approximate classification rules are common.

The extent of clinical symptoms is affected by numerous psychological and physical factors, such as age, sex, physical constitution, fatigue, alcohol tolerance (in particular, genetic alcohol hypersensitivity), as well as the phase of alcohol intoxication (accumulation or elimination). Similar symptoms may be triggered by other non-alcohol-related factors, such as the effects of medications or drugs, metabolic imbalance, or craniocerebral trauma (CCT). From a clinical chemical perspective, metabolic acidosis, hyperlactatemia, elevated serum osmolality, increased serum enzymatic activity of GGT and CK, as well as hypoglycemia can all represent chemically detectable effects of severe alcohol intoxication.

Alcohol in Breath, Urine, and Saliva. The validity of samples often used to estimate BAC is classified as follows:

The Diagnostic Value of Breath Alcohol Concentrations (BrAC). This method is based on the principle that a transfer of alcohol from arterial blood to aspirated fresh air takes places in the alveoli. Alcohol is thus released on expiration, detectable from the smell of alcohol on a person's breath. Due to the invasive nature of taking a blood sample and the significant costs of measuring BAC, it is sometimes expedient to measure the alcohol content of breath, which can to a certain degree enable inferences about BAC to be made. A number of electronic devices are suitable for alcohol *screening* (e.g., Alcomat®, Alcometer®, and Alcotest® 7010/7310/7410); the Alcotest® 7110 Evidential MK III, which also enables relatively accurate BrAC measurements, has been particularly widely used in recent years. However, converting BrAC into BAC remains problematic, since there is no constant factor. Due to the multitude of influencing factors, each BrAC value can correspond to a certain range of BAC values. BrAC is often higher than venous BAC in the absorption phase, while on the other hand lower in the later elimination phase.

The Diagnostic Value of Urine Alcohol Concentrations (UAC). Urine can also be used as a sample for alcohol screening purposes. Statistically, there is a direct correlation between urine alcohol and blood alcohol; however, in isolated cases this can vary to such an extent that a reliable

conversion of UAC to BAC is not possible. The principle derived from experience is that the UAC "lags behind" the BAC following absorption. This means that alcohol can still be detected in urine even after absorption in the blood is complete. Determining alcohol levels in morning urine samples from patients during detoxification treatment enabled this discrete observation relating to alcohol consumption of the previous evening (which patients had possibly spent outside of the institution).

The Diagnostic Value of Saliva Alcohol Concentrations (SAC). Saliva can also be used as a sample for alcohol testing. There is a significantly high correlation between BAC and SAC, irrespective of the manner of alcohol consumption (ingestion, infusion), even shortly after consumption has stopped and the mouth has been thoroughly rinsed.

29.2 Toxicokinetics of Alcohol

The toxicokinetics of alcohol can be divided into the following stages:

Absorption phase
Distribution phase
Elimination phase and biotransformation (metabolism)

29.2.1 The Absorption Phase

Even in the absence of an external supply, tiny amounts of alcohol from the intermediary metabolism of the organism can be found. This *endogenous* (i.e., the body's own) alcohol, however, produces a blood alcohol level of a mere 0.015‰.

At most, *inhalation* accounts for maximum BAC values of 0.2‰. Even absorption via intact skin (transdermal absorption) produces no forensically relevant concentrations. However, fatal poisoning in children following extensive application of poultices soaked in alcohol has been described.

Alcohol is introduced into the body almost exclusively by oral ingestion. Following *oral* ingestion, however, only small amounts of alcohol are absorbed from the mouth and a maximum of 15 % from the stomach. The principle sites of alcohol absorption include the duodenum and, in particular, the small intestine, whereby the speed of absorption depends on the concentration gradient between the stomach and the bloodstream. Absorption depends on a multitude of factors, key among which are general physical condition and constitution, the type of intoxicating liquor (concentration, congeners), food components in the gastrointestinal tract (fat content, consistency, spices, pH value), the degree of stomach filling, the temperature of intoxicating liquor, ingestion of certain medications, possibly also previous stomach surgery and diseases, simultaneous nicotine uptake, changes in peristalsis, general motility and perfusion, as well as psychological factors.

Taken together, all these factors make a mathematically exact determination of absorption impossible.

Absorption is usually completed within 60–90 min, and frequently less when high-proof forms of alcohol are ingested "on an empty stomach." Significant stomach filling (e.g., following a large meal) can result in longer absorption times, which is also true if the pylorus closes and the stomach acts as a long-working storage organ. This kind of pyloric spasm can be triggered by, e.g., high-proof spirits. Conversely, following gastric resection, the stomach's storage function can be lost and rapid absorption therefore takes place. In the case of moderate consumption of small amounts of alcohol over a prolonged period of time, it is possible that absorption and consumption conclude simultaneously.

Thus, the speed and duration of absorption are subject to the wide fluctuation margins typical for a living organism and can only be approximated.

Particularly if food is consumed before or during alcohol consumption, a portion of the alcohol ingested does not appear in the blood, i.e., the absorption quota is below 100 %. The difference, the so-called absorption or alcohol deficit, in which immediate absorption due to the first-pass effect as well as gastric alcohol dehydrogenase (ADH) clearly also play a role, can be as high as

10–20 %, under certain circumstances possibly even 30 % or more. The reason for this, however, remains unclear.

The average *absorption deficit* following ingestion of "normal" quantities of alcohol:

Approximately 10 %: spirits in a concentration range of ca. 40 vol.-%

Approximately 20 %: wine and champagne in a concentration range of ca. 10 vol.-%

Approximately 30 %: beer in a concentration range of 5 vol.-%

29.2.2 Distribution Phase

Alcohol is by no means distributed only in blood but rather in total body water, which accounts for between 60 and 70 % of body weight (body mass) in a male of normal build. Variations are seen in individuals with pyknic body types (50–60 %) and of slim build (70–80 %). Water content is generally around 10 % lower in women due to the higher fat content related to their physical constitution.

The Widmark factor "*r*" (also referred to as a reduction or constitution factor) derived from various water contents is used to calculate volumes of alcohol and BAC using the Widmark formula (see Sect. 29.2.6). Average values in men and women are $r = 0.7$ and $r = 0.6$, respectively.

Body weight multiplied by the Widmark factor *r* is referred to as "reduced body weight" and corresponds approximately to the volume of alcohol distribution (body water).

29.2.3 The Elimination Phase and Biotransformation (Metabolism)

Elimination begins immediately after commencement of drinking. Animal studies have shown that radioactive $^{14}CO_2$ (carbon dioxide) was exhaled immediately after administration of ^{14}C-labeled alcohol. At most, around 10 % of ethanol present in blood leaves the organism without having undergone intensive biotransformation (metabolism). This process takes place primarily in the liver, where ethanol is oxidized to acetaldehyde by the enzyme alcohol dehydrogenase (ADH). This main biotransformation pathway accounts for around 90–95 % of biotransformation in humans. Subsequent metabolism via the aldehyde dehydrogenase enzyme (ALDH) produces acetic acid, which is metabolized in the citric acid cycle to carbon dioxide and water (Fig. 29.1).

If ALDH activity is restricted, acetaldehyde may accumulate in the organism, leading to *flush syndrome*, which is characterized by redness of the skin, nausea, and low blood pressure, among other symptoms, all of which fall under the term *alcohol intolerance reaction*. Causes may be genetic (as observed in many Asians) or related to foreign substances (administration of antabus in the treatment of alcoholism or ingestion of the fertilizer calcium cyanamide).

The microsomal ethanol-oxidizing system (*MEOS*) represents an alternative biotransformational pathway. The system does not depend on the quantity of nicotinamide dinucleotide (NAD^+) required for further oxidation to acetic acid and can be induced by chronic alcohol consumption. Increased hourly rates of absorption (up to 0.20‰) are seen in habitual drinkers, going up to 0.29‰ (average) or even 0.35‰ (although exceptional) in severe alcoholics.

Ethyl glucuronide (EtG), another alcohol metabolism product, has recently been playing an important role in *abstinence control* (see Sect. 29.2.10).

Ethanol *kinetics* has a distinctive feature which is extremely relevant in forensic practice and which lies in the fact that, largely independent of the BAC per time unit, almost always the same amount is eliminated, the BAC going down on average by 0.15‰/h to the point where alcohol has almost completely disappeared. This is also referred to as a linear absorption characteristic, since the graphic representation of BAC against time produces a straight line. This is very unusual for a biotransformational process, since in the case of many other foreign substances (e.g., medications) the volume

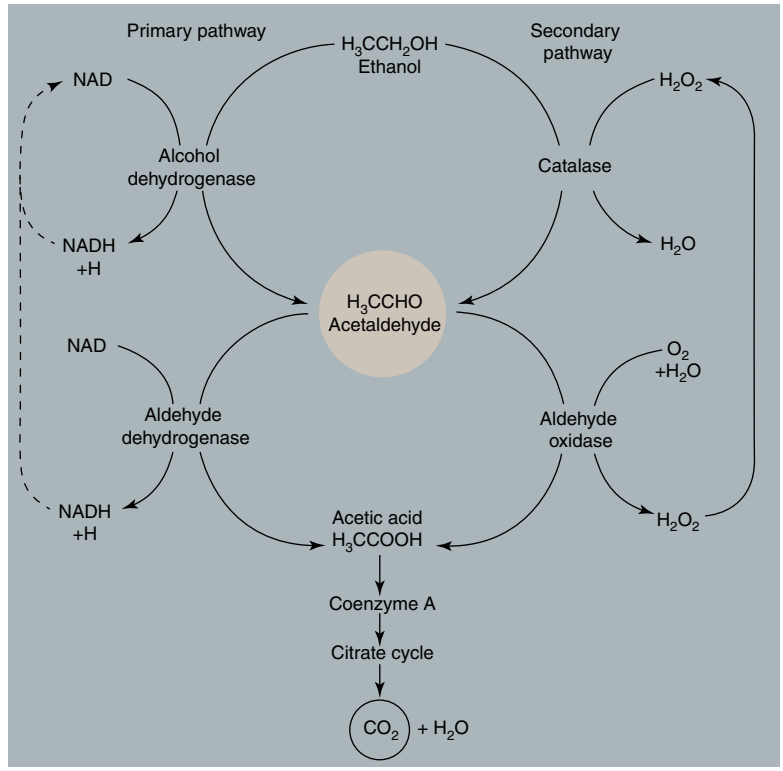

Fig. 29.1 The biotransformation of ethanol

eliminated within a time unit conforms to the concentration of the substance. The reason for ethanol's linear elimination characteristic lies in the limited quantity of NAD^+. However, there are indications that a mildly exponential course appears in relatively high BACs, which has been explained by the concentration dependence of oxidases also available for ethanol elimination. However, the extent of this deviation from the linearity of alcohol elimination is in practice negligible. Nevertheless, a divergence in the blood alcohol curve in the presence of high concentrations can also be cause by increased excretion (e.g., via the kidneys), which is concentration dependent.

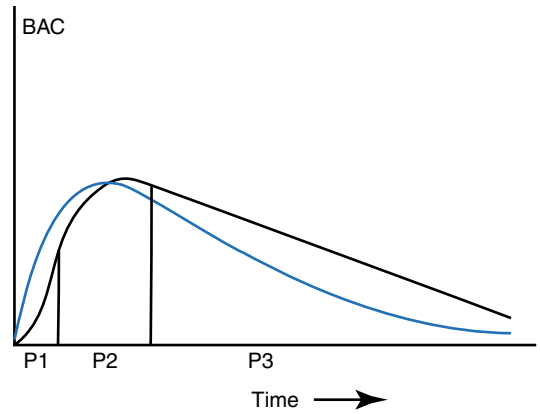

Fig. 29.2 The blood alcohol curve (*black curve*)

Note: Below a BAC of 0.15‰, elimination no longer follows a uniformly linear but instead an asymptotic curve to a greater or lesser extent. Thus, values under 0.15‰ can no longer be used as a starting point for retrograde calculations.

29.2.4 The "Blood Alcohol Curve"

The curve shown in *blue* in Fig. 29.2 (Bateman function) clearly illustrates the antagonistic nature of invasion (absorption) and elimination. It applies to most medications with nonlinear elimination; it does not apply, however, to the almost linear elimination of alcohol.

The *blood alcohol curve* is a representation of the temporal course of blood alcohol concentrations. The speed of ethanol absorption is concentration and therefore also volume dependent, while elimination follows a linear course to a great extent. By transposing these two phenomena, the blood alcohol curve shown in *black* in Fig. 29.2 is produced. Following an *absorption phase* (*P1*), during which elimination already takes place, a *distribution phase* (*P2*) begins, which is completed only after the peak of the curve. In this latter phase, ingested alcohol is equally distributed to the blood from the main absorption site, the small intestine. However, *late absorption* (e.g., of alcohol contained in food) may occur even after the peak of the curve has been reached.

The next phase is characterized mainly by *elimination*. A linear fall in the blood alcohol concentration is typical (*P3*). However, elimination has already long started (effectively on arrival of the first ethanol molecule in the liver, immediately after ingestion of the intoxicating liquor). Thus, it would not be entirely correct to characterize the ascending slope of the blood alcohol curve as purely the absorption phase and the descending slope as purely the elimination phase. One can only say that absorption dominates the ascending and elimination the descending slopes of the curve. The segment of the curve which descends almost in a straight line could be seen as a pure elimination phase, since absorption is clearly complete at this point (postabsorption phase). Around the peak, the balance between the increase and the drop in BAC may be level, causing the blood alcohol curve to follow a comparatively horizontal course [Gréhantsches plateau (GP); Fig. 29.3].

A further effect commonly seen is the "diffusion drop," caused by the following mechanism: in the case of extremely rapid absorption following ingestion of concentrated alcohol, ethanol accumulates largely in blood; diffusion to tissues does not occur at the same speed. After reaching the peak of the blood alcohol curve, the alcohol diffuses out of the blood into tissue, initially causing a marked drop in the blood alcohol curve ["diffusion drop" (DS); Fig. 29.4]. Uniform elimination can begin only after this phase. In the case of slow ingestion (e.g., several units of spirits/liqueurs during the course of a prolonged meal), the blood alcohol curve, without reaching a clear peak, can cross over into the descending "postabsorption" segment.

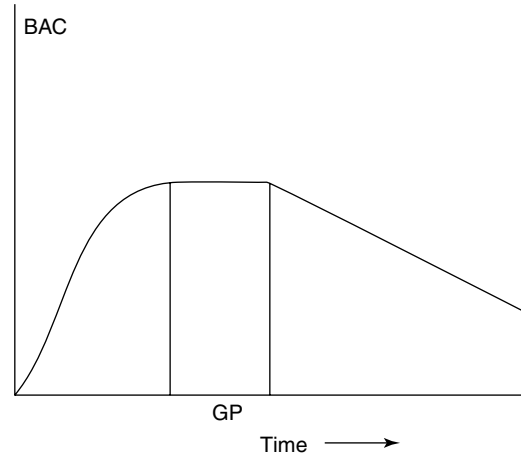

Fig. 29.3 Gréhantsche's plateau (GP)

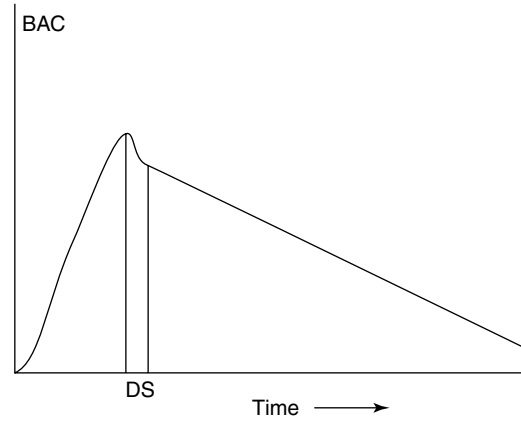

Fig. 29.4 Diffusion drop (DS)

29.2.5 Controversial and Actual Variables Influencing the BAC

"Sobering-Up" Agents. A distinction should be made here between those compounds/agents which cause a drop in BAC and those which reduce the affects of alcohol while leaving the BAC unchanged. These agents are usually capsules, powders, or soft drinks that have been authorized by the food and drug authorities as so-called food supplements and are for sale, e.g., on the Internet. No forensically relevant effect could be scientifically proven in any of these products to date.

Alcopops. It is also a widely held misconception that "Alcopops" contain no or only small quantities of alcohol, since there is often no taste of alcohol. Indeed, the alcohol content in these beverages is usually 5–10 vol.-% (occasionally more). The intrinsic odor and flavor of the alcohol is simply masked by adding fruit aromas (soft drinks).

The Formation of Alcohol by Means of Fermentation in the Body. The formation of significant volumes of alcohol via fermentation (e.g., of strawberries in the stomach or intestines) that could lead to detectable BACs is not possible.

Alcohol-Free and Diet Beers. Classifying beverages with an alcohol content of up to 0.5 vol.-% as "alcohol-free" is permitted in many countries. Such low alcohol levels are barely relevant even from a traffic medicine perspective. Thus, an adult male weighing 75 kg and of normal constitution ($r = 0.7$) would have to ingest at least 26 g of ethanol in order to theoretically (!) reach 0.5‰, and in order to ingest this quantity of ethanol, approximately 6.5 l of the beverage in question would need to be ingested. However, one should beware of the widespread belief that "diet beers" contain little or no alcohol. Indeed, only their carbohydrate content is lower, while their ethanol content can sometimes be higher than that of normal beers.

Sleep and Residual Alcohol. There is no significant difference in the speed of alcohol elimination when asleep or awake.

A phenomenon frequently requiring expert testimony in court is that of *residual alcohol*. In the case of a person going to bed at 1 a.m. after significant alcohol consumption (producing a BAC of 2.5‰), one can expect that at 7 a.m. (e.g., when driving to work) the person is *likely* to still have a BAC of 1.6‰ (2.5‰–6 h × 0.15‰). Even in the case of fast alcohol elimination (0.2‰/h), a *minimum* BAC of 1.3‰ can be expected (2.5‰–6 h × 0.2‰).

Renewed alcohol consumption (e.g., a "pre-lunch drink") on top of residual alcohol can prove particularly ill-advised, since high BACs can result without these being subjectively perceivable.

Work, Sport, Sauna, and Showering. Two earlier assumptions required clarification here: On the one hand, it needed to be established whether perspiration or other loss of fluids produced changes in body water (and thus in *r*). The alcohol distribution factor *r* plays, for example, a significant role in BAC calculation using Widmark's formula (see Sect. 29.2.6). On the other hand, it was believed that increased metabolic activity would also lead to faster alcohol elimination. However, numerous studies failed to produce any evidence that different retrograde extrapolation values needed to be applied in the case of work, sport, rest or sleep, perspiration, or exposure to cold.

Coffee, Tea, and Caffeine. Coffee and the caffeine contained therein have none of the "sobering" properties frequently ascribed to them by popular opinion. Although it is often possible to shorten the longer reactions times caused by alcohol, this is usually at the price of "reaction quality," i.e., experimental subjects react faster following coffee consumption than subjects having consumed alcohol only; however, they make more errors.

Particular Aspects in the Setting of Diabetes. Although high blood acetone concentrations can be seen in diabetics with severe disease in a precomatose or comatose state, the specific methods available for measuring BAC (ADH) and, in particular, gas chromatography give true BAC values. In severe cases of disease, consideration should be given to whether the disease itself causes performance deficits prohibiting participation in road traffic.

Particular Aspects in the Setting of Liver Disease. A relatively common problem is seen when a person, due to severe liver disease (e.g., hepatitis, fibrosis, or liver cirrhosis), is unable to eliminate alcohol, leading to accumulation and the subsequent development of high BACs. However, even in the case of severe liver disease (e.g., decompensated liver cirrhosis), a minimal rate of alcohol elimination of 0.1‰ is still possible. If even this low elimination rate is no longer possible, the affected person faces imminent hepatic coma, in which case one can assume that the affected individual is unfit to drive.

The Effects of Blood Loss, Volume Replacement, Anesthesia, and Blood Sampling Techniques. Significant blood loss and subsequent volume replacement can lead to relevant

shifts in body fluids. There are two principal factors that can affect a preexisting BAC:

- On the one hand, hemorrhage causes an absolute loss of alcohol, i.e., precisely the amount present in the blood lost. However, a 1-l loss of blood and alcohol intoxication of 2‰ corresponds to a loss in alcohol of only 2 g, the quantity found in 50 ml of beer.
- On the other hand, volume replacement agents cause a replenishing of the total body water. In certain cases, volume replacement can lead to a replenishing which exceeds the volume prior to hemorrhage; thus, volume replacement generally always results in a reduction in BAC.

Moreover, circulatory conditions in shock, with circulatory centralization in the early shock phase, play a role.

In animal experiments, decreased blood alcohol levels were observed following death due to hemorrhage. Investigations in humans with BACs of up to 2.6‰ and who had given temporary blood samples of up to 750 ml produced no abnormal results.

In 20 surgical patients in intensive care (volume replacement), increases in blood water content were greater than in serum water content. Thus, a normal conversion would have proved favorable for these patients, since the divisor is often below 1:1.

Furthermore, a slowdown in alcohol metabolism due to impaired liver perfusion, as well as increased metabolism via elimination of body fluid spaces, has been discussed.

No affect on BAC is expected from anesthetic agents frequently administered in a trauma setting; similarly, commonly administered volume replacement agents have no alcohol content and thus do not contribute to increased BACs. In addition, blood collection, when performed correctly, does not produce distorted results.

Thus, a BAC detected in a blood sample is as a rule attributable to the ingestion of alcohol.

Forensically Relevant Factors Influencing the Course of the Blood Alcohol Curve. As made clear by numerous examples, there is no patented recipe or special agent to lower BAC. The course of the blood alcohol curve is determined almost exclusively by the quantity of alcohol ingested, a person's body weight, the distribution factor r,

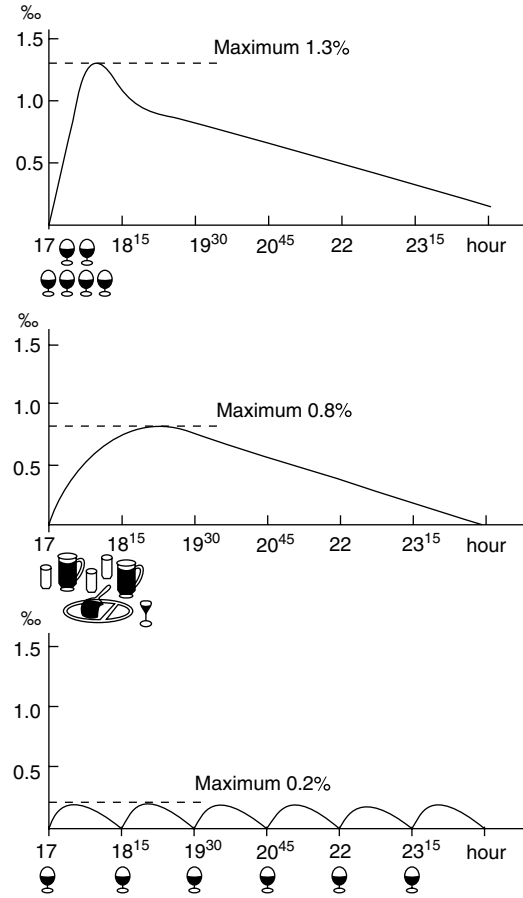

Fig. 29.5 The affect of drinking patterns on the course of the blood alcohol curve (From Schwerd (1992))

the ingestion period, and absorption conditions. The latter can be influenced to a limited extent, whereby under certain circumstances forensically relevant BACs are not achieved. Figure 29.5 shows the course of the blood alcohol curve in the same person following ingestion of the same volumes of intoxicating liquor depending on the ingestion period, concentration, and additional food intake. The short-term ingestion of the total alcohol volume (upper diagram) on an empty stomach produces—following a rapid rise in BAC—a peak that is far higher than that seen on ingestion of the same volume of alcohol in the context of a large meal (middle diagram). Thus, the tin of sardines widely rumored to be an old home remedy could indeed affect the course of the BAC in individual cases; having said that, other foodstuffs can also produce the same effect.

If the alcohol volume is divided into individual units over several hours (lower diagram), the highest BAC achieved is certainly significantly lower. Consumed over a long enough period of time, the peak value corresponds to that of an individual unit.

29.2.6 Calculating Blood Alcohol Concentrations from Data on Alcohol Consumption (the Widmark Formula)

In principle, the option to estimate a BAC on a purely mathematical basis is available. This is necessary when, e.g., no blood sample could be taken or when the time interval between an offense and blood testing is so long that values would be invalid. However, the course of alcohol ingestion needs to be accurately reconstructed after the fact for this kind of calculation, which frequently proves impossible since statements (e.g., from relatives) are often incomplete and divergent.

The basis for this calculation is the *Widmark formula*:

$$A = c \times p \times r$$

where A is the volume of alcohol in the organism in grams (with the exception of possibly unabsorbed amounts), c the BAC [in grams of ethanol per kilogram body weight = per mill (‰)], while p refers to body weight in kilograms, and r the reduction factor or distribution factor. r depends mainly on the individual's physical constitution. People with a *relatively high level of fatty tissue* (pyknic body types and most women due to their particular physical makeup) have a relatively low r value (0.55–0.60) and thus a higher BAC despite otherwise identical parameters in the Widmark formula, while *leaner* (*leptosome*) types can have r values of 0.80 or more. Experimentally, a calculation where $r = 0.70$ for a male of *normal build* (and $r = 0.60$ for a female of normal build) usually provides confirmatory values (see also Sect. 29.2.2).

If c, p, and r are known, the Widmark formula in the form given above can be used (approximately) to calculate the volume of alcohol absorbed.

Table 29.3 shows the alcohol content of a selection of popular intoxicating liquors.

Conversely, by transforming the formula thus:

$$c = \frac{A}{p \times r}$$

it is possible to calculate the BAC if A, p, and r are known.

Example

A sample calculation can be used to clarify how the Widmark formula is used:

If a child (10-kg body weight) ingests two large tablespoons (30 ml) of an ethanol-containing tincture (62 vol.-%, corresponding to 500 g ethanol per liter), the following maximum BAC can be calculated according to the Widmark formula:

$$\frac{15 \text{ g Ethanol}}{10 \text{ kg Body weight} \times 0.7} = 2.1 \text{ ‰ (gram ethanol per kilogram blood)}$$

In contrast, an adult male with a body weight of 70 kg would only reach approximately 0.3 ‰. This example clearly demonstrates that even small volumes of ethanol can be extremely hazardous for children.

Table 29.3 The alcohol content of a selection of intoxicating liquors

Type of beverage	Vol.-%	g/l
"Alcohol-free" beer	0,5	4
Export or pilsner beer	5	40
Doppelbock beer	8	64
White wine	9–13	72–104
Red wine	10–14	80–112
Champagne	8–12	64–96
Liqueur wine	14–20	112–160
Corn schnapps	32	256
Herbal liqueurs	35	280
Double-distilled corn schnapps	38	304
Brandy	36	288
Vodka/whisky	40	320
Fruit liqueurs	40–60	320–480
Stroh rum	40–80	320–640

Note: Values given are averages, significant variation is possible

In order to achieve more realistic values, other important parameters (various absorption deficits and elimination values) need to be taken into consideration. These correlations can be clearly shown using practical sample calculations (see Sect. 29.2.7).

In this context, however, it should be expressly pointed out that retrograde calculations on the basis of a BAC ascertained using analytically precise methods generally yield more reliable values than calculations based on information on the course of alcohol ingestion.

Alcohol content is given in vol.-% on bottle labels!
Conversion: vol.-% × 8 = grams of alcohol per liter
Example: 40 vol.-% × 8 = 320 g alcohol/l

29.2.7 Retrograde Extrapolation of BAC to the Time of the Offense Using Blood Samples

Alcohol concentrations measured in blood samples relate to the point in time at which the blood sample was taken. In cases where there is a time interval between the time at which blood samples were taken and the incident in question (e.g., an accident), a certain value needs to be added to the ethanol concentration determined in the blood sample, reflecting the elimination of ethanol up to that point. One prerequisite of this, however, is that the point in time to which the retrograde extrapolation relates was *no longer* in the absorption phase.

A reliable minimal retrograde extrapolation value is 0.1‰, the maximum being 0.2–0.3‰. The probable value is approximately 0.15‰; however, in the case of alcoholics without advanced liver disease, this value is somewhat higher.

As shown in Fig. 29.6, there is a particular problem associated with retrograde extrapolations: Although a retrograde extrapolation from the blood sample (BS) value to the time of the incident TI2 is relatively straightforward and permissible, since this time point lies in the linear segment of the blood alcohol curve (where absorption is to a large extent complete), a linear retrograde extrapolation to an incident time in the absorption phase TI1 would yield an excessively high value and be disadvantageous to a person suspected of a traffic offense. The prerequisite of correct retrograde extrapolation, therefore, is that the absorption phase is complete at the time of the incident. Thus, establishing the time at which alcohol consumption ended (e.g., for the purposes of judicial proceedings) is more relevant than determining the actual time of the incident.

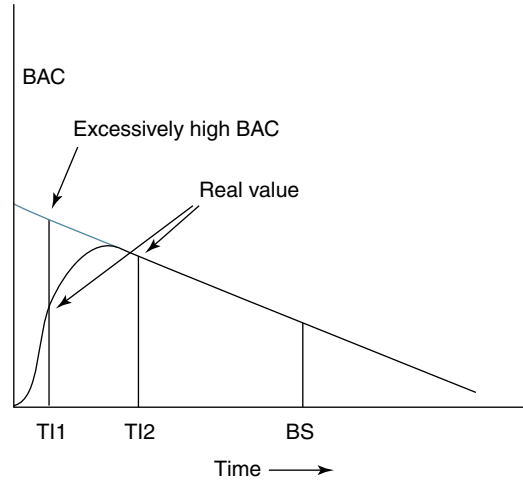

Fig. 29.6 Varying absorption rates

While there is largely consensus on the minimum value of 0.1‰/h, the maximum retrograde extrapolation value on which to base an assessment of criminal liability in Germany is controversial. A value of 0.2‰/h is often used, but higher extrapolation values are occasionally also assumed (e.g., 0.22‰/h or even 0.29‰/h). Schematic retrograde extrapolation using 0.29‰/h is rejected by most blood alcohol experts as excessively high, since it leads to unrealistic values in the case of long retrograde extrapolation times it (much like retrograde extrapolation with 0.1‰). On the basis of statistical considerations in numerous drinking investigations for which 1,486 blood alcohol curves

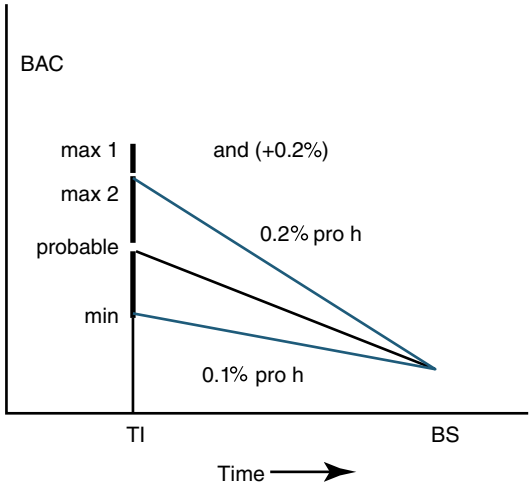

Fig. 29.7 The various options for retrograde extrapolation

were evaluated, Zink and Reinhardt found that the maximum BAC, calculated according to the formula (where t is time in hours between the incident and blood sampling),

$$\mathrm{BAC}_{\max} = \mathrm{BAC}_{(\text{blood sample})} + t \times 0.20‰ + 0.20‰$$

eliminates the possibility of a disadvantage (i.e., assuming excessively low maximum concentrations) in over 99 % of cases.

Figure 29.7 shows a graphic representation of the individual options available for retrograde extrapolation: Taking the blood sample (*BS*) value as a starting point, retrograde extrapolation to the incident time point (*TI*) using the extremely low value of 0.1‰/h leads to the most effective minimal value (*min*) for establishing unfitness to drive. Retrograde extrapolation using 0.2‰/h yields a maximum value (*max 2*) which, according to Zink and Reinhardt's method, needs to be increased by 0.2‰ (*max 1*) and which can be used as a basis to assess criminal liability at the time of an incident (*TI*). The most probable value (probable), however, lies somewhere between the two. The difference between minimum and maximum values increases as the time interval between blood sampling and the time of the incident widens, often leading to unrealistic values.

Finally, the question of the minimum BAC from which retrograde extrapolation is even feasible arises. In general, retrograde extrapolation from a blood test value of 0.15‰ using a retrograde value of 0.1‰ is considered unproblematic, assuming the starting value was obtained using an alcohol-specific procedure (see "Test Specificity" below).

29.2.8 Sample Calculations

The following sample case demonstrates the varying effects of influencing variables on BAC.

A defendant (body weight 65 kg, $r = 0.7$) claims to have consumed six 0.4-l beers (5 vol-% × 8 = 40 g/l) and four 2-cl shots of spirits (32 vol.-% × 8 = 256 g/l) between 4 and 8 p.m. What could the *maximum*, *minimum*, and *probable* BAC at the time of the accident at 11.30 p.m. have been?

Calculation. Firstly, the *theoretical maximum* BAC can be calculated from the alcohol contained in the intoxicating liquor stated. Six 0.4-l beers contain approximately 96 g alcohol (2.4 l × 40 g/l) and four 2-cl shots of spirits contain approximately 20 g alcohol (4 × 0.02 l = 0.08 l × 256 g/l = approx. 20 g). This gives a total alcohol volume of 116 g. In a person with the reduction factor $r = 0.7$ and a body weight of 65 kg, this volume can produce a *theoretical maximum* BAC of

$$c = \frac{A}{p \times r} = \frac{116\,\mathrm{g}}{65\,\mathrm{kg} \times 0.7} = 2.55‰$$

Calculating the *Maximum* BAC: Firstly, working on the assumption that the highest possible value is beneficial (e.g., when establishing criminal liability), it can be expected that almost all alcohol was absorbed, leaving a minimum absorption deficit of 10 %. This corresponds to an alcohol volume of 116 g × 0.9 = approx. 104 g. Moreover, it is beneficial to use as low an hourly retrograde extrapolation value as possible (0.1‰) from the time alcohol consumption began. The latter time point was allegedly at 4 p.m., the incident at 11.30 p.m.; during the intervening time, at least 7.5 × 0.1‰ = 0.75‰ was eliminated, which should be deducted from the maximum value. This gives

a *maximum* BAC at the time of the incident (11.30 p.m.) of 2.29‰–0.75‰=approx. 1.54‰.

$$c = \frac{A \times 0.9}{p \times r} = \frac{104\ \text{g}}{65\ \text{kg} \times 0.7} = 2.29‰ - 0.75‰ = 1.54‰$$

Calculating the *Minimum* BAC: It is in the defendant's interests here if as little alcohol as possible has been absorbed. Assuming a high absorption deficit of 30 % (i.e., only 70 % of the whole alcohol volume of 116 g, i.e., 81 g in actual terms, has been absorbed), an initial BAC of 1.78‰ is calculated.

Furthermore, it is beneficial when calculating a minimum value, and therefore generally also when establishing fitness to drive, to assume that the maximum possible volume of alcohol has been eliminated since the time point at which alcohol consumption began. Assuming in this particular case that, for the entire time between the commencement of drinking (4 p.m.) and the incident (11.30 p.m.), 0.2‰ was eliminated hourly, a concentration of 7.5×0.2‰=1.50‰ for 7.5 h of elimination would need to be deducted from 1.85‰, yielding a (relatively unrealistic) *minimum* value of 1.78‰−1.50‰=0.28‰ at the time of the incident.

$$c = \frac{A \times 0.7}{p \times r} = \frac{81\ \text{g}}{65\ \text{kg} \times 0.7} = 1.78‰ - 1.50‰ = 0.28‰$$

Calculating the *Probable* BAC: This calculation should be based as far as possible on actual physiological conditions, such as an absorption deficit of 20 % and an hourly retrograde extrapolation value of 0.15‰. The calculation then looks as follows: 80 % of the entire alcohol volume is equivalent to 0.8×116 g=an actual 93 g. This volume can produce a BAC of approximately 2.04‰. The probable volume of eliminated alcohol (7.5×0.15‰=1.13‰) needs to be deducted from this, yielding a *probable* BAC of 2.04‰−1.13‰=0.91‰.

$$c = \frac{A \times 0.8}{p \times r} = \frac{93\text{g}}{65\text{kg} \times 0.7} = 2.04‰ - 1.13‰ = 0.91‰$$

Thus, the following BAC values for the time of the incident can be calculated for this sample case:

Maximum 1.54‰
Minimum 0.28‰
Probable 0.91‰

This example shows how to calculate a BAC when working on the principle "in dubio pro reo" ("when in doubt, for the accused"), whereby the most beneficial scenario for the defendant is alleged (minimum values when establishing fitness to drive, maximum values when establishing criminal liability) and variables for the calculation are chosen such that the extreme values desired are achieved. In actual fact, however, in an experimental investigation attempting to reconstruct this particular case with a larger number of test subjects, only few if any would achieve the extreme values calculated here. By far the largest proportion of test subjects would have a BAC in the region of the probable value (approx. 0.9‰), whereby the distribution would show significant similarities to a Gaussian curve: the further one moves away from the probable value, the more unlikely this BAC becomes. Thus, although extreme values are more hypothetical in nature, they cannot be fully excluded in court.

29.2.9 Post-Offense Alcohol Consumption and Double Blood Sampling

Post-offense alcohol consumption, i.e., ingesting intoxicating liquor *following* a legally relevant incident (e.g., a traffic accident with subsequent abscondence) plays a significant role in forensic expert appraisal. A typical case is illustrated here by means of a caricature (Fig. 29.8). A driver is involved in a traffic accident following an evening of heavy drinking (pre-offense alcohol

Fig. 29.8 Alleged post-offense alcohol consumption (From Bonte (1987) with permission; pencil sketches by H. Kluger, Kiel, Germany)

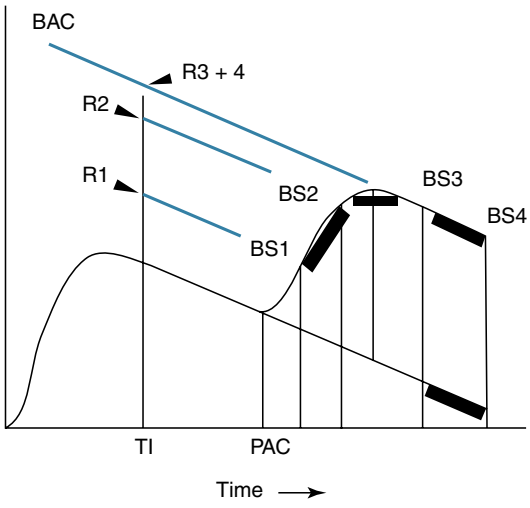

Fig. 29.9 The course of a BAC curve in the case of post-offense alcohol consumption

consumption). The driver absconds from the scene of the accident and later presents the police with a bottle emptied only a short time previously, claiming that he consumed the contents of the bottle after returning to his home (post-offense alcohol consumption).

Figure 29.9 illustrates the most important correlations of post-offense alcohol consumption: alcohol consumption took place prior to an incident (TI) and absorption was effectively complete at the time of the incident, as can be seen from the largely linear course of the BAC (postabsorption phase). At some point after the incident (TI), post-offense alcohol consumption (PAC) took place, resulting in a rise in BAC. Thus, a new blood alcohol curve was effectively "grafted" onto the elimination branch of the first blood alcohol curve.

Blood was sampled at varying time points after post-offense alcohol consumption (between one and four blood samples taken); the empirical significance of these samples is discussed below:

Firstly, it can be seen that retrograde extrapolation to the time point of the incident TI (for more details on retrograde extrapolation and, in particular, retrograde extrapolation values, see Sect. 29.2.7), without taking post-offense alcohol consumption into consideration, necessarily yields a higher BAC (for the time point of the incident) than the actual BAC at that time. Linear retrograde calculation would give the retrograde extrapolation concentrations R1, R2, and R3+4.

Thus, a proportion of these excessively high values, which are not conducive to an assessment of, e.g., fitness to drive, need to be subtracted in consideration of post-offense alcohol consumption. To determine a *minimum* value suitable for the assessment of a defendant's fitness to drive, the *maximum* value of post-offense alcohol consumption calculated using the Widmark formula is subtracted from the *minimum* value without post-offense alcohol consumption.

Double blood sampling should be briefly mentioned at this point. In the present case (Fig. 29.9), if two blood samples (BS1 and BS2) were to be taken at an interval of 20 min and analyzed shortly following post-offense alcohol consumption, it is to be expected that the BAC of the first sample (BS1) would be lower than that of the second (BS2). This could be interpreted as clear evidence of

post-offense alcohol consumption; this is also true of approximately equivalent values in both samples. The latter would indicate that double blood sampling was performed during the peak of the (post-offense) blood alcohol curve. When both samples are taken a significant time after post-offense alcohol consumption, i.e., in the postabsorption phase, a largely identical course is seen in the blood alcohol curve—with or without post-offense alcohol consumption—characterized by the fact that the blood sample taken earlier (BS3) shows a higher BAC than the sample taken later (BS4). This finding, therefore, neither excludes nor confirms post-offense alcohol consumption. Even in the peak area of the blood alcohol curve, a drop measured in the curve in no way excludes post-offense alcohol consumption: a diffusion drop (see Sect. 29.2.4) could be responsible for this fall in the curve. Double sampling is only reliable when the time interval between the consumption of post-offense alcohol and the first blood sample is less than 1 h and should be used as flanking evidence in an overall evaluation of a case, taking other criteria (e.g., performance deficits observed during detainment and blood sampling or changes in condition over time) into consideration.

Due to the limited significance and reliability of double blood sampling, *congener analysis* (see Sect. 29.3.4) has meanwhile come to represent a more valid basis on which to formulate an expert appraisal on data relating to post-offense alcohol consumption.

29.2.10 Markers of Alcohol Consumption

Alcohol is eliminated relatively rapidly from the organism, and even people with an alcohol problem are often able to attend an examination, for example, in a nonintoxicated state. Thus, indicators which are able to tell us something about alcohol habituation and other possibly correlated problems are of particular interest. These issues are associated with, for example, therapy monitoring or the assessment of a person's fitness to drive.

An overview published by Iffland and Grassnack (modified according to Thierauf et al.) can be used as a guide to the significance and reliability of individual indicators (Table 29.4):

Particular Features of Carbohydrate-Deficient Transferrin. Carbohydrate-deficient transferrin (CDT) is a transferrin variant. In the case of long-term alcohol abuse (generally daily volumes of over 60 g ethanol over several weeks), structural changes in transferrin molecules can be seen and measured as serum CDT. The diagnostic specificity and sensitivity of CDT are relatively high.

Particular Features of Methanol. Elevated methanol concentrations in blood are indicative of chronic alcohol abuse. Methanol is found in almost all intoxicating liquors. The toxicokinetic basis of methanol as a marker for alcoholism lies in the fact that *methanol* concentrations in blood only really start to drop at blood *ethanol* concentrations below approximately 0.4‰. This means that an *accumulation of methanol* can be expected in people with BACs above this value over long periods of time. Meanwhile, a decision limit of 10 mg methanol per liter of blood has been suggested. Assuming no methanol-rich beverages (e.g., some brandies) have been ingested, values above this level indicate chronic alcohol abuse. Conclusions of this kind, however, should not be drawn in isolation; indeed, a number of other assessment criteria should be considered, e.g., the medical report made at the time of blood sampling (normal behavior despite high BAC).

Particular Features of Ethyl Glucuronide *(EtG).* Ethyl glucuronide is a highly stable metabolic product made up *entirely* of ethanol; as such, it is also stored in *hair* and can be used to monitor long-term alcohol abstinence. Detecting EtG in *blood* and *urine* indicates alcohol ingestion even when alcohol is no longer detectable in blood.

Table 29.4 Important markers for alcohol consumption

Indicator (marker)	Indication
Blood alcohol concentration (BAC)	Acute alcohol consumption Snapshot of alcoholization
CDT	Massive alcohol consumption of several weeks' standing (Approximately 60–80 g alcohol daily)
γ-GT	Long-term (chronic) alcohol consumption (Approximately 80–200 g alcohol daily)
Mean corpuscular erythrocyte volume (MCV)	Long-term consumption of moderate volumes of alcohol Alcohol volumes (<40 g alcohol/day)
Methanol	Alcohol consumption with no significant phases of sobriety
Acetone and isopropanol	Alcohol-induced metabolic disorders
Fatty acid ethyl esters (FAEE)	Short-term markers in blood
Ethyl glucuronide (EtG)	Reliable marker for abstinence (specifically for alcohol)
Ethyl sulfate (EtS)	Also a reliable marker for abstinence

In addition to elevated γ-glutamyl-transferase (*γ-GT*) and *MCV*, an additional alcohol consumption marker (see Table 29.4) is the presence of *acetones* and *isopropanol*, which is considered an indicator for alcohol-induced metabolic disorders (although these can also have *other causes*). Therefore, elevated liver values seen in isolation are under no circumstances conclusive for alcoholism or alcohol abuse.

29.3 Analysis

This section discusses the procedures necessary for the precise determination of a BAC.

29.3.1 Blood Sampling

Blood samples should be taken by a physician according to accepted medical practice. Blood should be taken from the cubital vein (elbow) in living persons and from the femoral vein (upper thigh) in cadavers. Taking samples from a site remote to the stomach ensures that, in the case of long postmortem intervals, no alcohol has diffused through the stomach to the sampling site where it could affect the BAC, as seen with blood taken from the heart.

29.3.2 Analysis Methods

First of all, a distinction must be made between alcohol detection for *clinical* purposes and alcohol detection for *forensic* purposes, since there are widely varying requirements in terms of the accuracy of measurement results depending on the objectives of the investigation. A clinical evaluation of the degree of alcohol intoxication does not require blood alcohol determination to a precision of two positions after the decimal point, as required for forensic purposes.

However, it can be assumed in principle that measurement results could become *forensically relevant*, i.e., used in legal proceedings, such as in accidents in the workplace. In this context, questions arise regarding the validity of determining BACs on the basis of only a *single* measurement taken using an enzymatic procedure. Since many clinical chemical laboratories do not have the facilities to determine blood alcohol levels for forensic purposes, i.e., according to the strict guidelines of the respective authorities, a special blood sample should be taken using a suitable venule, closed securely, and stored in a fridge (under no circumstances frozen) at approximately +4 °C.

The following methods are commonly used:

The Widmark Method. This method uses ethanol's ability to reduce yellow potassium

dichromate (Cr^{6+}) in sulfuric acid, which reacts to green Cr^{3+}. Measurements are then made on the basis of isothermal microdistillation followed by photometric or titrimetric determination. First developed in 1922, Widmark's method is still occasionally used today, although its significance is waning.

The ADH Method. This technique of enzymatic ethanol determination is similar to the biotransformation of alcohol in the liver. Alcohol dehydrogenase catalyzes the oxidation of ethanol to acetaldehyde while reducing nicotinamide adenine dinucleotide (NAD^+) to NADH, which is measured photometrically at its maximum wavelength of 340 nm.

$$\text{Ethanol} + \text{NAD}^+ \xrightarrow{\text{ADH}} \text{Acetaldehyde} + \text{H}^+ + \text{NADH}$$

Headspace Gas Chromatography. This method is based on headspace sampling rather than blood sampling, whereby sample gases are broken down into their various components (alcohol and other volatile substances) on gas chromatographs. The temporal sequence of signals on the gas chromatograph indicates the type of substance, while signal height relates to its volume or concentration.

Test Specificity. Blood alcohol analysis using gas chromatography is the most specific of all methods mentioned here. While the Widmark method responds to all volatile and easily oxidized substances, and even the ADH method can be affected by other alcohols (e.g., isopropanol found in cosmetics), correctly performed gas chromatographic blood alcohol analysis is guaranteed to measure only ethanol, even when blood samples have undergone autolysis and putrefaction.

29.3.3 Determining Alcohol Concentrations in Other Samples

The Relevance of Alcohol Concentrations in Vitreous Humor. The advantage of investigating vitreous humor lies mainly in its distance from the stomach (thus avoiding diffusion), as well as it prolonged resistance to putrefaction. Experimental studies on ethanol neogenesis showed the following maximum (up to 1.5‰): Weinig and Lautenbach, 1‰; Mebs and May, 1‰; Gilliland and Bost, 1,5‰; Canfield et al., 1.5‰; Bonte et al., 1.5‰.

The Relevance of Alcohol Concentrations in Hematomas. In principle, it is possible to measure a BAC using hematoma blood, which would give a good indication of the actual level of alcohol intoxication at the time of injury.

The Relevance of Alcohol Concentrations in Other Samples. The possibilities and advantages/disadvantages of determining alcohol in stomach contents, synovial fluid, bone marrow, gallbladder fluid, cerebrospinal fluid, as well as testicular, brain, liver, and kidney tissues are also reported. Muscle tissue is particularly suited to alcohol analysis. The BAC to tissue ratio is given as 1:30 ($n = 51$) for cardiac muscle tissue (standard deviation ±0.25).

29.3.4 Congener Analysis

Measuring ethanol concentrations in blood only allows inferences to be made about the *volume* of intoxicating liquor consumed. Numerous intoxicating liquors contain, in addition to ethanol, other volatile substances (e.g., methanol, 1-propanol, isobutanol, 1-butanol, and 2-butanol), all of which can also be detected and quantified using gas chromatography for congener analysis (Fig. 29.10). Since the congener profiles for most types of intoxicating liquors in terms of their fluctuation margins are known (Table 29.5), or can be found using comparison samples, it is possible to make assertions about the *type* of intoxicating liquor consumed. Due to the considerable variability of concentrations sometimes seen, the "original beverage" consumed post-incident should be investigated where possible. Even in the absence of the latter, congener analysis still enables concrete assertions, for example, post-offense consumption

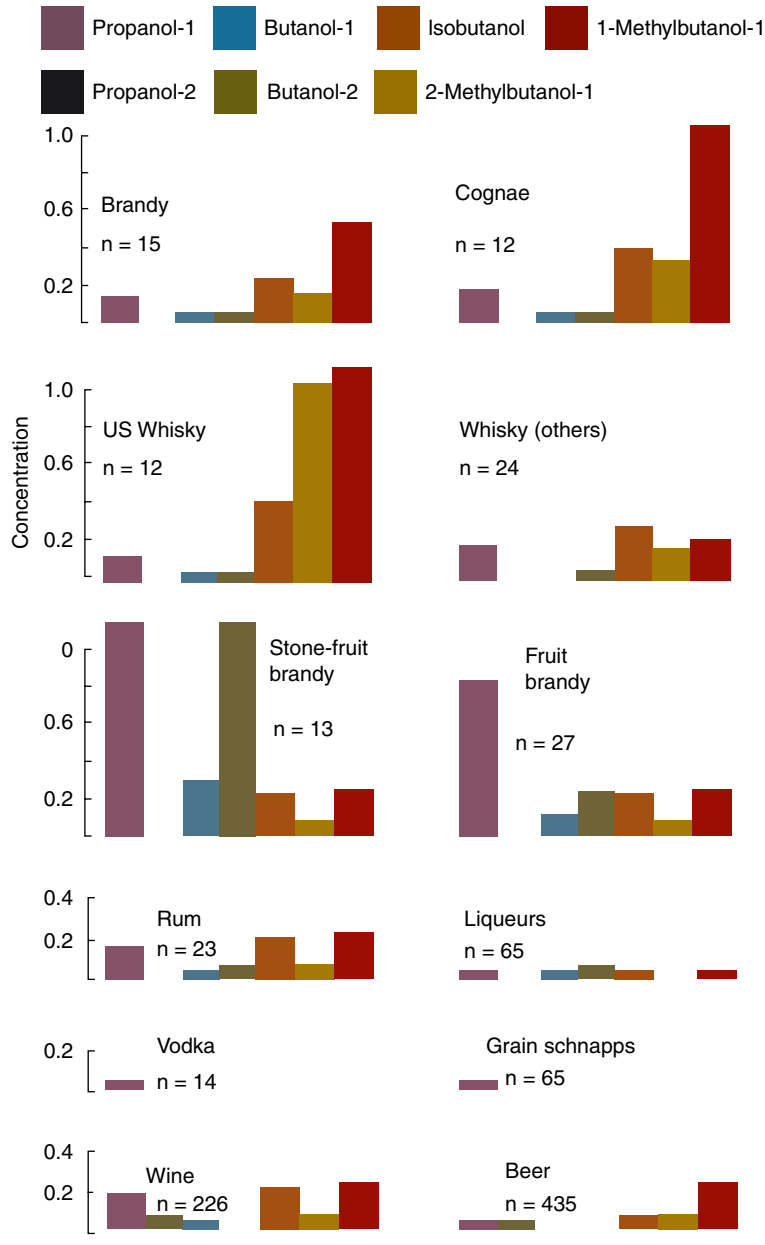

Fig. 29.10 The congener profiles of common intoxicating liquors

of vodka is alleged; however, the finding of high concentrations of congener alcohols argues against the defense plea, since vodka is almost free of congener alcohols. Conversely, high levels of numerous congener alcohols are expected following the ingestion of brandy, the absence of which argues against the alleged post-offense alcohol consumption.

Congener analysis has meanwhile become an established method for the verification of alleged post-offense alcohol consumption, while double blood sampling has been relegated to the sidelines.

Table 29.5 The congener content of a selection of intoxicating liquor

Beverage	EtOH	MeOH	1-PropOH	2-ButOH	IsobutOH	1-ButOH	2/3-MetbutOH-1
Beer[a]	3–8	ND–2	9–27	ND–ND	8–54	ND–2	43–110
White wine[b]	NM	29 ± 16	31 ± 8	ND	56 ± 18	ND–8	139 ± 31
Red wine[b]	NM	104 ± 30	30 ± 7	ND–1	47 ± 15	MD–7	170 ± 32
Sparkling wine[b]	NM	16 ± 8	31 ± 5	ND	53 ± 15	ND–9	156 ± 35
Brandy (D)[a]	35–38	128–338	44–177	ND–12	49–410	ND–10	247–1,160
Williams pear brandy[a]	31–43	724–5,556	77–2,776	ND–504	127–3,253	17–102	320–1,613
Schnapps/fruit schnapps[a]	37–46	101–3,533	104–2,490	ND–357	162–468	23–93	841–1,822
Kirschwasser[a]	39–45	1,127–2,552	223–4,424	3–474	112–411	5–14	392–1,734
Topinambur[a]	38–45	1,313–2,418	650–1,836	13–126	227–609	6–18	522–990
Grappa[a]	37–43	958–5,034	111–456	3–135	97–410	7–222	252–1,359
Rum[a]	10–54	ND–73	ND–1,933	ND–317	ND–206	ND–7	ND–768
Vodka[a]	35–40	ND–64	ND–ND	ND–ND	ND–6	ND–3	ND–7
Tequila[a]	38–40	269–725	97–271	ND–6	60–314	2–23	187–776
Gin[a]	37–47	ND–7	ND–6	ND–ND	ND–ND	ND–ND	ND–7
Ouzo[a]	37–40	ND–15	ND–3	ND–ND	ND–ND	ND–ND	4–6
Raki[a]	44–45	63–459	ND–135	ND–4	ND–246	ND–11	6–437
Aquavit[a]	37–40	6–40	ND–ND	ND–ND	ND–ND	ND–ND	3–5
Liqueurs[a]	13–40	ND–140	ND–135	ND–5	ND–118	ND–4	ND–414
Sherry[b]	NM	11–149	14–59	ND–2	8–74	1–4	5–213
Rice wine[b]	NM	7–48	23–43	ND–4	8–41	ND–1	8–117
Vermouth[b]	NM	9–98	9–58	ND–1	16–71	ND–9	13–186
Scotch whisky (blended)[c] $n = 22$	40–43	17–51	100–369	2–4	187–386	2–6	164–797
Scotch whisky (pure malt)[c] $n = 2$	40–43	23	197–222	0.5–0.6	324–370	7–9	951–1,106
Scotch whisky (single malt)[c] $n = 185$	40–63	19–68	147–407	ND–14	204–117	ND–1,117	644–2,949
Irish whiskey[c] $n = 28$	40–54	23–39	140–390	ND–4	57–712	1–36	225–2,127
US whiskey (Kentucky straight bourbon)[c] $n = 31$	40–57	33–107	58–298	ND–2.4	216–1,322	3–9	1,519–5,088
US whiskey (Tennessee)[c] $n = 5$	40–45	46–74	114–127	ND–0.4	333–923	3–5	1,737–2,949
Canadian whisky[c] $n = 5$	40	19–29	26–165	ND–0.6	60–336	1–3	195–448
Whisky liqueur[c] $n = 2$	17–40	15–22	6–7	3–20	ND	ND	6–43

[a]Lachenmeier and Musshoff (2004)
[b]Bonte (1987)
[c]Zinka et al. (2011)
ND not detectable, *NM* not measured, *EtOH* ethanol (vol.-%), *MeOH* methanol (mg/l), *1-PropOH* 1-propanol (mg/l), *2-ButOH* 2-butanol (mg/l), *IsobutOH* isobutanol (mg/l), *1-ButOH* 1-butanol (mg/l), *2/3-MethylbutOH-1* 2/3-methylbutanol-1 (mg/l)

29.4 Establishing Suspicion and Evidence Recovery

A more detailed discussion of *substance-specific* tests can be found in Sect. 29.1 (alcohol) and Sect. 30.29 (preliminary drug tests).

These are usually performed on the basis of concrete *suspicion* and for the purposes of *recovering evidence*. Special *strategies* for recognizing the effects of alcohol or drug abuse, based on the following situations or characteristics, have proven to be particularly effective:

Case Study

The female driver of an all-terrain vehicle caused an accident in which her car came into contact with and significantly damaged numerous parked cars. She was able to abscond. Approximately 2 h later she was found at her home by the police. When questioned about alcohol consumption, she claimed she had consumed no alcohol prior to the accident but that she had consumed fruit schnapps after the incident. Congener analysis results supported her allegation of consumption of this particular congener-rich beverage shortly prior to blood sampling. However, an analysis of the residual blood sample subsequently ordered by the court yielded evidence of a high concentration of the psychotropic drug lorazepam, which had been taken prior to the accident and which, according to the court, was the cause of the accident.

Phase 1: A vehicle in moving traffic

Criteria

Driving style: e.g., snaking, orientation towards the center of the carriageway, driving on the central line, inappropriate speed, disregarding the right of way of other traffic users, blatant use of detours or blocked roads, driving too close to the vehicle in front, stopping for no reason, abrupt breaking and acceleration, and, in particular, *accidents* caused due to the abovementioned driving styles or nonobservance of traffic regulations.

Vehicle operation: Driving without lights, absent or incorrect use of indicators, and unnecessary use of high (full) beam and horn

Vehicle defects and condition: e.g., conspicuous design

Passenger behavior: e.g., throwing drugs or drug utensils out of the vehicle

Observational ability while stationary: Reacting to optical and acoustic signs

Phase 2: Contact with the driver

Criteria

In particular, the driver's reaction to being asked to stop, unusual characteristics in the driver and/or passengers or their behavior, getting out of the vehicle, gait, or suspicious utensils

Phase 3: Detainment, tests, medical examination, and sample taking

Criteria: The *medical report* often drawn up at the same time as blood sampling generally records important observations and findings; however, these relate to the time of blood sampling and are not necessarily representative of the person's condition at the time of the incident (Warning: post-offense alcohol consumption). Tests are often involved here, such as writing specimens, gait tests, pupil size and reaction, rotary nystagmus, as well as finger-to-finger and finger-to-nose tests. Speech, awareness, thought processes, behavior, mood, whether or not symptoms of inebriation are feigned, as well as overall impression, are often also assessed.

Abnormalities observed during this phase may provide the grounds on which to base a suspicion of drug and/or medication abuse. However, numerous impairments can be mistaken for the effects of alcohol and/or drugs, although they are in actual fact disease-related or due to other impairments *unrelated* to foreign substances; examples of impaired performance include gait (physical disability) and speech (speech impediment, stammer). Should these impairments be present while the person in question is nonintoxicated (e.g., during subsequent questioning by the police or at a judicial hearing), they can no longer be used as evidence of foreign substance abuse.

29.5 Toxicodynamics of Alcohol

An important distinction needs to be made here between traffic medicine aspects and clinical-toxicological aspects.

Fig. 29.11 Alcohol-related disinhibition and increased risk-taking. *Left*, a nonintoxicated driver; *right*, a disinhibited driver

29.5.1 Traffic Medicine Aspects

A number of deficits and impairments relevant to traffic medicine are given in Table 29.2 and discussed in Sect. 29.1. Investigations have shown that the traffic accident risk doubles at 0.6‰ and is as much as 25-fold higher at 1.5‰ compared with a nonintoxicated state.

In particular, the following impairments occur after alcohol and drug consumption and often represent the cause of driving errors:

- *Alcohol-related disinhibition* and *increased risk-taking*, often leading to excessive speed, "showing off," and inconsiderate behavior (Fig. 29.11)
- *Impaired distributive attention*: Inability to perform more than one task at a time, e.g., operating the radio or picking a cigarette up off the ground while driving
- *Prolonged reaction time*: e.g., rear-end collisions or identifying dangerous situations late (Fig. 29.12)

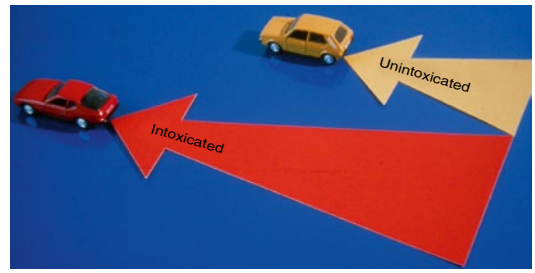

Fig. 29.12 Delayed reaction time under the influence of alcohol

- *Reduced field of vision*: "Tunnel vision" (Figs. 29.13 and 29.14)
- *Impaired light–dark adaption*: Optical blackouts
- *Increased sensitivity to high (full) beams*: Particularly in wet road conditions (Fig. 29.15)
- *Reduced visual acuity*: e.g., distant visual acuity reduced by approximately 15 % at

Fig. 29.13 Wide perception range in nonintoxicated drivers

Fig. 29.14 "Tunnel vision" under the influence of alcohol and drugs

0.4–0.7‰ and by 25–30 % at between 0.8 and 1.1‰

- *Reduced depth perception*: e.g., correct final adjustment at between 0.6 and 1.2‰, but in twice the time, reduced to around half the ability of an nonintoxicated driver at between 0.8 and 1.1‰
- *Reduced perception of red lights*: e.g., traffic lights, brake lights
- *Loss of orientation*: e.g., when negotiating corners

Important: Alcohol-related driving errors occur more frequently during the absorption phase **than at comparable values during the** postabsorption **phase (elimination phase) due to the accumulation effect**

The *effects of alcohol* (on psychophysical performance) are determined not only *interindividually*, i.e., acquired alcohol tolerance, but can also be affected to a greater or lesser extent intraindividually as a result of, e.g., tiredness, stress, illness, medication, or a "hangover."

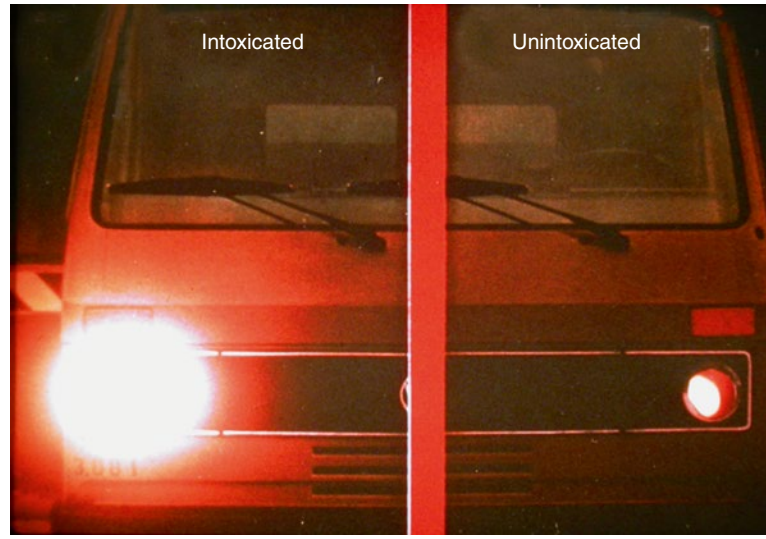

Fig. 29.15 Increased sensitivity to high (full) beams under the influence of alcohol and numerous drugs

29.6 Clinical-Toxicological Aspects

Due to its widespread use and easy accessibility, alcohol also plays an important role in clinical-toxicological practice.

Autopsies carried out in the course of forensic practice at the Free University Berlin produced a diagnosis of fatal alcohol intoxication in 155 cases (15 % of all intoxication cases). BACs were below 3.0‰ in 41 % of these cases, between 3.0 and 4.0‰ in 37 %, between 4.0 and 5.0‰ in 18 %, and over 5.0 in 4 %. A BAC of as high as 7‰ is reported to have been measured in one case.

General physical condition and (existing) disease(s) play a significant role in the course of intoxication, as well as external factors such as ambient temperature. Death usually occurs after a number of hours as a result of respiratory paralysis or progressive cerebral edema.

For a more detailed discussion of interactions between alcohol and other foreign substances, see Sect. 29.7.3. and Chap 30, Sect 30.2.13, 14 and 15

29.7 Forensic Aspects

Forensic aspects relate mainly to issues of fitness to drive and criminal liability.

29.7.1 Assessing Fitness to Drive (Driving Safety)

General *fitness to drive* (suitability to drive) is required of all vehicle drivers; this fitness may be absent in the presence of certain physical, mental, or character-related deficits. The performance deficits leading to a person's unfitness to drive generally extend over a prolonged period of time.

In terms of concrete traffic situations, a distinction is generally made between *driving ability* (driving skills) and *fitness to drive* (driving safety). In principle, even a considerably intoxicated driver is "capable of driving," assuming he is able to move his vehicle; however, he is only considered "fit to drive" if he is capable of psychophysically meeting the considerable requirements of driving in modern road traffic in a timely manner appropriate to the situation.

As a general rule, the higher the driving performance required at the time of an incident, the greater the likelihood that a driver performs poorly under the influence of alcohol. In addition, the lower the BAC measured, the greater the need for high-quality evidence becomes.

The following limit values are in force for vehicle drivers in Germany (compiled from the German Association against Alcohol and Drugs in Road Traffic, B.A.D.S.) (Table 29.6).

In Germany and numerous other countries, there is a basic 0‰ limit for the following:

- Pilots, copilots, and flight engineers
- Drivers transporting hazardous materials
- Taxi, bus/coach, and rail vehicle drivers
- Masters/coxswains are considered absolutely unfit to be in charge of a vessel at a BAC of 0.5‰ or higher
- Wheelchair users?

Furthermore, with a BAC of 1.6‰ or higher, or a BrAC of 0.8 mg/l or higher, a positive *medical/psychological examination* is generally required in Germany before a driving license can be recovered (§ 13 Nr. 3c of the German driving license regulations; FeV).

29.7.2 Assessing Criminal Liability

While BAC limit values can generally be defined for traffic offenses, this level of clarity is not possible when assessing *criminal liability*. The main reason for this is the variations in alcohol tolerance. Thus, a person unaccustomed to intoxicating liquor may show significant performance deficits at a BAC well below 2‰, while in the case of chronic abuse of large volumes of alcohol, even a BAC of over 3‰ may not produce any obvious deficits. Therefore, the individual psychophysical appearance of a person before, during, and after an incident should form the main basis of any expert appraisal.

Table 29.6 Limit values for blood alcohol concentrations in Germany

BAC (‰)	Explanatory notes
0.0	Drivers still in their provisional driving period according to § 2a para 1 of the German Road Traffic Act (StVG), or who are under the age of 21, are prohibited from consuming intoxicating liquor while in charge of a vehicle in road traffic or attempting to drive a vehicle while under the influence of intoxicating liquor. A level of up to 0.2‰ will be tolerated (see note on § 24c StVG)
0.3	If any driver demonstrating *poor driving performance*, causing a hazardous situation in traffic, or causing an actual accident is found to have a BAC of 0.3‰ or more at the time of the contravention, the possibility that intoxicating liquor was the cause of the *relative unfitness to drive* must be taken into consideration according to consistent case law. In the event of positive findings, a conviction for drunk driving (§316 of the German Criminal Code) or dangerous driving (§ 315c para 1 No. 1a of the German Criminal Code), depending on whether the contravention was without consequence, caused an actual hazard, or indeed caused an accident, may be imposed
0.5	Any driver found to have a BAC of 0.5‰ or more, or a BrAC of 0.25 mg/l or more, will be prosecuted for a misdemeanor according to § 24a para. 1 StVG, even if there was no alcohol-related impairment to their driving performance. The "hazard limit values" of 0.5‰ BAC and 0.25 mg/l BrAC replaced the former "0.8‰ limit" on 01.04.2001
1.1	According to consistent case law, a BAC of 1.1‰ or higher denotes *absolute unfitness to drive*. On the basis of this alone and without recourse to prove the opposite, any driver with this level of alcohol (or higher) in their blood or breath is considered unfit to participate in motor traffic and is liable to prosecution for intoxication while in charge of a motor vehicle (§ 316 StGB) or, in the case of an accident occurring or almost occurring, dangerous driving (§ 315c para 1 Nr. 1a StGB)
1.6	A person is considered as absolutely unfit to ride a bicycle with a BAC of 1.6‰ or higher

29.7.3 Alcohol and Medication

A distinction needs to be made here between toxicokinetic and toxicodynamic interaction properties.

Toxicokinetic Interaction. This denotes the effect a foreign substance (e.g., medication) has on the kinetics of alcohol, i.e., the course of the blood alcohol curve. It has been rumored on the Internet, for example, that the H_2-receptor blockers cimetidine and ranitidine can cause a sharp rise in BAC. However, investigations were unable to find any forensically relevant effect of these drugs. A maximum increase of 0.02‰ can occur only in BAC values below 0.5‰.

With the exception of preparations containing alcohol (e.g., plant extracts), there is no known medication which, when dosed therapeutically, produces, simulates, or masks alcohol or which impairs the validity, precision, or reliability of analysis methods used to determine BAC.

Toxicodynamic Interaction. Alcohol is, however, a frequent *interaction partner* in mixed intoxications, generally when other foreign substances with a central effect are involved, such as medications and drugs. In this context, the interaction may be *additive* (the overall effect corresponds to the sum of the individual effects) or *overadditive* or *potentiated* (the overall effect is greater than the sum of the individual effects). Moreover, an interaction may not only increase a substance's effect, it may also change its active profile, sometimes even causing paradoxical reactions.

29.8 Alcohol-Related Histopathology

Pathological changes are routinely observed where collagen connective tissue is replaced by toxic, damaged liver tissue; this process begins with truncation and fibrosis of the portal fields, leading to liver fibrosis and the development of pseudolobules demarcated by connective tissue in the case of fine to medium-coarse nodular portal liver cirrhosis. In addition, alcohol-induced pathological changes of the pancreas can occur. When portal congestion is present, splenomegaly will also develop, and varices of the esophagus may be seen.

Alcoholic cardiomyopathy should be mentioned separately; with regard to differential diagnoses, inflammatory cardiomyopathy must be considered in connection with dilative cardiomyopathy. The intracerebral reduction in Purkinje cells which may be detected morphometrically leads to the diagnosis of chronic alcohol abuse. In cases of death with a relatively low BAC, cardiomyocytic microvesicular steatosis and lipid droplet discharge in the adrenal cortex at the time of death have been reported surprisingly often.

29.8.1 Alcoholic Liver Pathology

With additional alcohol use, the degree of fibrosis may increase and the liver may already show fatty liver macroscopically if >50 % of the hepatocytes are fatty. Due to steatosis, the liver can weigh up to 4,000 g compared to its normal weight of approximately 1,500 g. Unlike fibrosis and cirrhosis, steatosis is always reversible and may quickly regress within 3–4 weeks once the person stops alcohol consumption. In advanced stages, portal liver cirrhosis with pseudolobules occurs (Table 29.7).

As a result of alcohol consumption, enlarged and multiplied Kupffer stellate cells may also appear, often containing iron pigments and termed "alcoholic's iron."

In the advanced stage of liver cirrhosis with signs of decompensation, intrahepatic cholestasis is seen along with bile pigment deposits in the cytoplasm of the remaining hepatocytes, as well as retained bile thrombi in smaller bile ducts in some cases. As a rule, autopsy reveals icterus of the skin, eyelid connective tissue, mucous membranes, and vessel intima in all body regions. Epithelial cells of the renal tubules may reabsorb bile pigment, leading to the histopathological picture of so-called cholemic nephrosis.

29.8.2 The Pancreas

Alcohol-related damage to pancreas tissue is usually chronic damage. Fibrosis and lipomatosis

Table 29.7 Classification of alcoholic histopathological findings in liver tissue

Degree	Fibrosis/cirrhosis	Degree of steatosis (%)	Inflammatory activity
0	No pathological findings	0–5	No increase in infiltrates
1	Mild fibrosis of the portal fields; from the differential diagnostic perspective, a condition following hepatitis or ascending cholangitis should be considered	5–25	Mild increase in lymphocytes and monocytes at portal field margins
2	Portal field fibrosis with connective tissue-like branching into the hepatic sinus; however, no communication between branches (pre-cirrhotic restructuring of liver parenchyma)	25–50	Lymphocytic and monocytic infiltrates in the portal fields and around neighboring liver parenchyma
3	Thin fibrotic strands, which arise from the portal fields and continue along the hepatic lobules, coalesce while developing pseudolobules which surround intact liver tissue	50–75	Dense infiltrates with lymphocytes and monocytes in the portal fields and in the liver parenchyma
4	Wide fibrotic strands surround small pseudolobules with sparsely remaining intact hepatocytes, intense bile duct proliferation, inflammatory infiltrates with hepatocellular necrosis	75–100	"Fatty liver hepatitis" with hepatocellular necrosis and hyaline (Mallory bodies)

Dettmeyer (2011), According to Ferries and Thompson (1981)
Steatosis = % of hepatocytes with intracytoplasmic fatty vacuoles with fine and coarse droplets

of the pancreas with perilobular and intralobular fibrosis may develop, along with atrophy of the parenchyma and moderate inflammatory infiltration. Advanced stages show thickened fibrous walls of the gland excretory ducts with flattened epithelial cells and an often loose lymphocytic inflammatory infiltrate in the context of unspecific chronic fibrous pancreatitis. In addition, duct ectasia is present in which retained secretion may accumulate, as well as microscopically small concrements. In the case of severe damage, there may be predigestion of the surrounding tissue. In this case, one may observe tryptic fatty tissue necrosis which resembles candle-grease spots and which may go on to calcify.

29.8.3 Alcoholic Cardiomyopathy

Toxic myocardial damage caused by prolonged alcohol abuse producing electron microscopic findings, results in alcoholic cardiomyopathy falling into the category of secondary cardiomyopathies. Histologically, cardiomyocytes are partially hypertrophic, partially degenerated. Interstitial fibrosis zones may appear, along with patchy endocardial fibrosis. The clinical and morphological presentation corresponds to dilative cardiomyopathy, which may lead to sudden death in the case of chronic alcohol abuse. However, in the case of sudden death of an alcoholic, other causes should also be discussed, e.g., alcoholic ketoacidosis. Alcohol-related effects on skeletal muscle have also been examined in regard to an association with alcoholic cardiomyopathy.

There are often other histological findings which correspond to alcohol consumption, particularly in the liver. Excessive alcohol consumption can apparently lead to impairment of cardiac pump function even in people who otherwise abstain from alcohol consumption, potentially resulting in acute lethal cardiac arrhythmia.

Thus, alcoholic cardiomyopathy is a dilative-type cardiomyopathy caused by chronic alcohol abuse. In this context, a differentiation has been made between alcoholic and idiopathic cardiomyopathy (Bulloch et al. 1972). The toxic effects of alcohol were the first to be considered as a cause of dilative cardiomyopathy (Richardson et al. 1986; Regan et al. 1969), but the volume effects linked to beer consumption on the cardiovascular system have also been discussed as a cause (Morin and Daniel 1967).

29.8.4 Other Alcohol-Related Histopathological Findings

Nervous System. Although alcohol-related damage to the central nervous system shows no specific histomorphological findings, it produces (occasionally severe) deviations from the norm compared with control brains. Notable among these is Wernicke encephalopathy, which is clinically associated with Korsakov's psychosis, eye muscle palsy, and ataxia. There are morphological changes within the margins of the third and fourth ventricle and the aqueduct involving glial cell proliferation accompanied by branched capillary blood vessels presenting with thickened vascular walls. The clinical picture resembles inflammation but affects less than 1 % of all alcoholics. Red wine consumption is more likely to result in central pontine myelinolysis (Marchiafava syndrome). With the consumption of fusel alcohol, particularly methanol, neurological symptoms may appear, including cerebral hemorrhage and necrosis. Other histomorphological changes are described in the neuropathology literature.

Bone Marrow. With chronic alcohol abuse, alcohol-induced impairment of hemopoietic bone marrow is seen in the form of vacuolized bone marrow cells at an early stage of erythropoiesis and leukopoiesis. However, the entire hemopoietic system may be affected, also as a result of a lack of folic acid and vitamin B12. The result is megaloblastic anemia, genetically determined to sideroblastic anemia. Macrocytosis may disclose concealed alcohol abuse.

Oral Cavity, Esophagus, and Gastrointestinal Tract. With chronic alcohol consumption, mucous membrane inflammation of the oral cavity often appears, including caries and parodontosis, but also cancer of the tongue, hypopharynx, and larynx. Chronic esophagitis leads to an increased incidence of esophageal cancer. Gastritis of varying severity is also found more frequently with chronic alcohol consumption. With repeated vomiting, there may be longitudinal tears in the cardia region of the stomach with associated bleeding (Mallory–Weiss syndrome). This syndrome involves upper gastrointestinal hemorrhage due to longitudinal mucosal lacerations in the esophagogastric junction.

Ethylene glycol intoxication leads to neurological deficits and microscopically visible deposits of oxalate crystals. Although alcohol undoubtedly causes functional deficits, e.g., in the small intestine and other organs such as the kidney, there are as a rule no characteristic conventional histological findings. Severe alcohol-induced changes appear with heavy alcohol abuse that may lead to testicular atrophy. In forensic practice, the effects of alcohol on the liver, pancreas, and heart are highly significant in terms of determining cause of death.

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Forensic Toxicology

30

General toxicology (from the ancient Greek τοξικολογία, *toxikologia*: toxicology) deals in the broadest sense with toxic substances (toxins) and poisoning (intoxication). It is an interdisciplinary field often associated with pharmacology. *Clinical toxicology*, on the other hand, focuses on clinical aspects. The national poison information centers established in numerous countries play a significant role in this field.

Forensic toxicology is the study of toxins (drugs, medications, and other foreign substances) and their detection in and effect on the human organism in a legal context, thus making it a field of forensic medicine. Its areas of application mainly include the investigation of poisoning in living subjects and fatalities (postmortem toxicology), as well as drug and medication abuse. Other subfields of toxicology include food toxicology, industrial toxicology, environmental toxicology, and radiation toxicology.

International standards for the analysis of alcohol and drug consumption are lacking, with the result that practices differ significantly. An EU guideline deals with the analysis of residues in animal products (Commission Decision of 12 August 2002 implementing Council Directive 96/23/EC concerning the performance of analytical methods and the interpretation of results), much like a Food and Drug Administration (FDA) regulation in the USA (mass spectrometry for confirmation of the identity of animal drug residues). WADA guidelines address the analysis of doping in sport (WADA Technical Document—TD2003IDCR—Identification criteria for qualitative assays. Incorporating chromatography and mass spectrometry). Finally, mention should be made of the Forensic Laboratory Guidelines of the SOFT AAFS (USA). Since there are no internationally recognized regulations, legally binding or otherwise, to ensure the quality of analysis, reference can only be made to national regulations.

30.1 Information and Basic Principles

The volume and concentration of a substance is usually relevant when considering its toxicity. Some substances have a beneficial effect on the organism at therapeutic doses, while higher concentrations are toxic. In general, the principle set down by Paracelsus (1493–1541) applies:

> All things are poison, and nothing is without poison; only the dose permits something not to be poisonous.

Poisoning is a pathophysiological condition resulting from the ingestion of toxic substances (poisons) and can be classified as either "chronic" or "acute." The clinical picture is referred to as toxicosis (Greek τοξίκωση). Poisoning with more than one substance is referred to as mixed or polyintoxication. Estimates put the annual number of poisonings (generally emergencies) seen in hospitals in Germany, for example, at between 150,000 and 200,000. Patients often need to be treated symptomatically with invasive methods on the basis of mere suspicion, since methods of

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Table 30.1 Distribution of the most common forms of poisoning in children and adults

Poisonings in children	Poisonings in adults
27.1 % human pharmaceuticals	59.8 % human pharmaceuticals
17.7 % plants	8.5 % cleaning and care products
13.4 % cleaning and care products	3.1 % plants
8.2 % cosmetics	1.5 % cosmetics

rapid toxicological analysis are generally lacking. However, invasive methods are mandatory, since in approximately 25 % of all cases of poisoning, not the substance initially suspected but rather a completely different or even additional substance has been ingested. A recent report from a national poison information center indicates that the rate of lethal poisonings is equally distributed among adults and children, with a mortality rate of approximately 0.2 % (1.5 % according to other statistics). The report cites the circumstances of poisoning as accidental (65 %), suicidal (22 %), addiction-related (5.2 %), industrial (2.5 %), and due to adverse drug (side) effects (1.8 %). The varying distribution of poisonings among children and adults according to these statistics is interesting (Table 30.1).

In many countries, the number of poisonings is as high as the number of heart attacks, even if the statistical information available here is controversial. Toxicological analysis is often not performed in the case of poisoning, symptomatic treatment being preferred instead. This approach is comparable to treating a heart attack without clinical–chemical diagnostic methods or a bone fracture without an X-ray.

30.2 Important Pharmaco- and Toxicokinetic Parameters

The course and severity of intoxication are determined mainly by pharmaco- and toxicokinetic parameters. In addition to the *administered dose* (volume of poison), these primarily include *bioavailability* (depending on the galenic formulation and absorbability, among others), *distribution* (liver, kidneys, and brain, for example, are better perfused than fatty tissue), *distribution volume*, *clearance*, and *elimination*. The *elimination half-life* is of particular relevance here, referring to the time interval in which a concentration in blood or plasma sinks to half its initial concentration. This interval can vary significantly, measuring only a few minutes in the case of GHB (liquid ecstasy), for example, or almost 300 h in the case of metabolic products such as diazepam and flurazepam. With an elimination half-life of 30–90 min, nicotine is also eliminated relatively rapidly, otherwise chain-smoking would be fatal. Moreover, elimination half-lives are highly dependent on other parameters, such as age or preexisting damage to the elimination organs (liver and kidneys) and may exceed the values mentioned significantly. Even in the case of use according to the recommended dose, the danger of *cumulation* remains.

Note: The most relevant symptoms of poisoning have generally faded after between two and three elimination half-lives. Foreign substances with irreversible mechanisms of action are an exception.

Biotransformation (*metabolization*) also influences the toxicity of a substance significantly, the goal being to convert the toxic substance into metabolites that can be better eliminated from the body. A distinction is made between phase I reactions (functionalization) and phase II reactions (hydrophilization). The reaction products from these reactions are referred to as *metabolites*. Thus, detoxification or elimination is not always necessary, since metabolites can also be pharmacologically active (Table 30.2).

When blood concentrations drop only slowly, an *enterohepatic circulation* should always be considered, whereby foreign substances in blood (e.g., morphine) accumulate in the gallbladder when passing through the liver and are then excreted in the intestine. From here, substances can be reabsorbed into the bloodstream and thus possibly circulate repeatedly and for a prolonged period of time. In the case of poisoning, it is

helpful to be familiar with the toxicokinetics of a toxin in order to predict the effects of intoxication as well as the need for and usefulness of therapy.

30.2.1 Classifying Poisonings

Classifying the severity of poisonings is carried out according to the Poisoning Severity Score (PSS) (Table 30.3).

The *Glasgow Coma Scale* (GCS) offers a simple scale for the assessment of impaired consciousness. It is often used in intensive care medicine to assess craniocerebral trauma; however, it is also helpful in the classification of general impairments of consciousness. This widely accepted classification method to describe the conscious state uses three categories, in each of which points are awarded:

- Eyes
- Verbal
- Motor

Points are awarded for each category individually and then added up. The maximum score is 15 (fully conscious), while the minimum score is 3 (death or deep coma). A score of 8 points or less indicates severely impaired cerebral function and the risk of life-threatening respiratory failure; thus in the case of a GCS of 8 or less, securing the respiratory pathway by means of endotracheal intubation should be considered. The widely accepted GCS (Table 30.4) also forms part of other scoring systems, such as the Mainz Emergency Evaluation Score or the APACHE II Score.

Table 30.2 Examples of how biotransformation influences effects

Mother substance	Metabolic product (metabolite)
Diazepam (effective)	Oxazepam (also effective)
Procaine (effective)	p-Aminobenzoic acid (ineffective)
Parathion (barely effective)	Paraoxon (effective)

Table 30.3 Classifying the severity of poisonings according to the Poisoning Severity Score (PSS)

Score	Classification	Symptoms
0	None	Asymptomatic course, no symptoms
1	Mild	Mild, transient symptoms that spontaneously resolve
2	Moderate	Clear or protracted symptoms
3	Severe	Severe or life-threatening symptoms
4	Fatal	Poisoning results in death

30.2.2 Symptoms and Syndromes

Poisonings are generally characterized by typical symptoms. However, some toxins produce no *initial symptoms* (e.g., paracetamol) in the early stages, while others appear immediately following ingestion (e.g., carbon monoxide). Examples of symptoms of poisoning are given in Table 30.5.

Poisoning is often characterized by the onset of more than one simultaneous symptom, i.e., a complex of symptoms, also referred to as a *toxidrome*. If several symptoms can be classified as belonging to a single toxidrome, the reliability of a suspected diagnosis is increased considerably. Examples of symptoms of poisoning are given in Table 30.6.

Furthermore, information can be gained from a number of clinical chemical parameters in terms of particular poisons or groups of active substances. For example, acidosis is often seen

Table 30.4 The Glasgow Coma Scale (GCS) for adults

Points	Eyes open	Verbal communication	Motor reaction
6	–	–	Obeys commands
5	–	Converses normally, oriented	Localizes pain
4	Spontaneously	Confused, disoriented	Withdraws from pain
3	On command	Inappropriate words	Abnormal flexion to pain
2	In response to pain	Incomprehensible sounds	Extension to pain
1	No reaction	No verbal reaction	No reaction to pain

Impairment severity: 14–15 points, mild; 9–13 points, moderate; 3–8 points, severe

Table 30.5 Major symptoms associated with poisonings

Central nervous system (CNS)	
Symptoms	*Possible causes* (*selected*)
Somnolence to coma	Ethanol, barbiturates, benzodiazepines, ß-receptor blockers, butyrophenones, calcium channel blockers, chlorohydrate, carbon monoxide, GHB, H_1-antihistamines, opiates, opioids, organophosphates, phenothiazines, tricyclic antidepressants, Z drugs (e.g., zaleplon, zolpidem, zopiclone)
Agitation and disorientation	Amphetamines, atropine, scopolamine, cocaine, H_1-antihistamines, lithium, phenothiazines, salicylates, selective serotonin reuptake inhibitors, tricyclic antidepressants, theophylline
Euphoria	Ethanol, amphetamines, cannabinoids, cocaine, opiates, opioids
Hallucinations	Amphetamines, atropine, scopolamine, H_1-antihistamines, LSD, psilocin, tricyclic antidepressants
Motor system	
Symptoms	*Possible causes* (*selected*)
Seizures	Amphetamines, ß-receptor blockers, butyrophenones, calcium channel blockers (verapamil-like), carbon monoxide, cocaine, cyanide, ethylene glycol, H_1-antihistamines, nonsteroidal anti-inflammatory drugs, organophosphates, phenothiazines, selective serotonin reuptake inhibitors, tricyclic antidepressants, theophylline
Ataxia, dysarthria	Ethanol, barbiturates, benzodiazepines, H_1-antihistamines, phenothiazines, Z drugs (e.g., zaleplon, zolpidem, zopiclone)
Extrapyramidal motor disturbances	Butyrophenones, lithium, phenothiazines, selective serotonin reuptake inhibitors, tricyclic antidepressants
Malign neuroleptic syndrome	Butyrophenones, phenothiazines
Muscle twitching/tremors	Lithium, organophosphates, selective serotonin reuptake inhibitors, tricyclic antidepressants
Rhabdomyolysis	Amphetamines, barbiturates, carbon monoxide, H_1-antihistamines
Tremors	Amphetamines, butyrophenones, cocaine, insulin/glinides/sulfonylurea, lithium, organophosphates, phenothiazines, selective serotonin reuptake inhibitors
Respiratory system	
Symptoms	*Possible causes* (*selected*)
Central respiratory depression	Ethanol, barbiturates, opiates, opioids, phenothiazines, tricyclic antidepressants
Increased bronchial secretion	Organophosphates
Dyspnea, respiratory distress	Carbon monoxide, cyanide
Hyperventilation, hyperpnea, tachypnea	Cocaine, ethylene glycol, methanol, nonsteroidal anti-inflammatory drugs, salicylates
Pulmonary edema	Barbiturates, carbon monoxide, paraquat, salicylates, irritant gases
Cardiovascular system	
Symptoms	*Possible causes* (*selected*)
Sinus bradycardia	Amiodarone, ß-receptor blockers, clonidine, calcium channel blockers, digitalis, opiates, opioids, organophosphates
Sinus tachycardia	Tricyclic antidepressants, amphetamines, atropine, scopolamine, butyrophenone, calcium channel blocker, cocaine, H_1-antihistamines, L-thyroxine, nonsteroidal anti-inflammatory drugs, organophosphates, phenothiazines, selective serotonin reuptake inhibitors, theophylline
Atrial tachycardia Flutter and "flickering"	Digitalis, class-1 antiarrhythmics, L-thyroxine, tricyclic antidepressants
Ventricular tachycardia Flutter and "flickering"	Cocaine, cyanide, digitalis, H_1-antihistamines, class-1 antiarrhythmics, phenothiazines, tricyclic antidepressants

Table 30.5 (continued)

Extrasystole	
Torsade de pointes	
QT lengthening	Amiodarone, ß-receptor blockers, butyrophenones, H_1-antihistamines, class-1 antiarrhythmics, phenothiazines, tricyclic antidepressants
QRS widening	ß-Receptor blockers, H_1-antihistamines, selective serotonin reuptake inhibitors, tricyclic antidepressants
Delayed SA, AV, and ventricular nervous conduction block	ß-Receptor blockers, calcium channel blockers, digitalis, H_1-antihistamines, class I antiarrhythmics, and ventricular tricyclic antidepressants
Angina pectoris symptoms	Carbon monoxide, cocaine, cyanide
Hypertension	Amphetamines, cocaine, organophosphates
Hypotension, circulatory shock	Ethanol, amiodarone, amphetamines, ACE inhibitors, barbiturates, benzodiazepines, ß-receptor blockers, butyrophenones, calcium channel blockers, H_1-antihistamines, class-1 antiarrhythmics, nonsteroidal anti-inflammatory drugs, opiates, opioids, phenothiazines, tricyclic antidepressants, theophylline
Gastrointestinal tract	
Symptoms	*Possible causes* (*selected*)
Abdominal pain/cramps/diarrhea	Amanitin, chloral hydrate, colchicine, digitalis, nonsteroidal anti-inflammatory drugs, organophosphates, salicylates
Nausea/vomiting	Amanitin, amphetamines, ACE inhibitors, benzodiazepines, carbon monoxide, cocaine, colchicine, cyanide, digitalis, H_1-antihistamines, nonsteroidal anti-inflammatory drugs, opiates, opioids, organophosphates, paracetamol, salicylates, selective serotonin reuptake inhibitors
Gastrointestinal atony, obstipation	Atropine, scopolamine, barbiturates, opiates, opioids, phenothiazines, tricyclic antidepressants
Ulceration, gastrointestinal hemorrhage	Amanitin, nonsteroidal anti-inflammatory drugs
Impaired liver function, icterus, hepatic encephalopathy	Colchicine, amanitin, paracetamol
Increased salivation, salivary gland secretion	Organophosphates
Kidneys and urinary tract	
Symptom	*Possible causes* (*selected*)
Acute kidney failure, anuria	Amanitin, calcium channel blockers, colchicine, ethylene glycol, nonsteroidal anti-inflammatory drugs, paracetamol, salicylates, valproic acid
Impaired miction, urinary retention	Atropine, scopolamine, H_1-antihistamines, phenothiazines, tricyclic antidepressants
Oliguria	Amanitin, barbiturates, ethylene glycol, nonsteroidal anti-inflammatory drugs, opiates, opioids, paracetamol, valproic acid
Skin and mucous membranes	
Symptom	*Possible causes* (*selected*)
Cyanosis, pallor	Chloral hydrate, clozapine, carbon monoxide, cyanide, ergotamine, opiates, opioids, paraquat
Hypersalivation	Clozapine, organophosphates
Dry mouth	Atropine, scopolamine, carbamazepine, H_1-antihistamines, phenothiazines, selective serotonin reuptake inhibitors, tricyclic antidepressants
Perspiration	Amphetamines, cocaine, insulin, glinides, sulfonylurea, salicylates, selective serotonin reuptake inhibitors
Dry mucous membranes	Atropine, scopolamine, antihistamines, phenothiazines, tricyclic antidepressants
Pinkish skin color	Cyanide (early), carbon monoxide
Hot, red skin	Atropine, scopolamine, antihistamines, phenothiazines, tricyclic antidepressants

(continued)

Table 30.5 (continued)

Metabolic, electrolyte, and temperature levels	
Symptom	*Possible causes* (*selected*)
Hyperthermia	Amphetamines, butyrophenones, clozapine, colchicine, cocaine, phenothiazine, selective serotonin reuptake inhibitors, nonsteroidal anti-inflammatory drugs
Hypothermia	Alcohol, barbiturates, clonidine, nonsteroidal anti-inflammatory drugs
Hypoglycemia	Alcohol, amanitin, insulin, glinides, sulfonylurea, paracetamol
Hypokalemia	Clozapine, colchicine, nonsteroidal anti-inflammatory drugs, salicylates
Metabolic acidosis	Alcohol, salicylates, colchicine, ethylene glycol, nonsteroidal anti-inflammatory drugs, methanol, barbiturates
Increased transaminases	Amanitin, paracetamol, paraquat, valproic acid
Lactic acidosis	Caffeine, cyanide, carbon monoxide, metformin
Impaired coagulation	Colchicine, paracetamol, barbiturates, anticoagulants

Selected from Peters and Mall (2009)

Table 30.6 The major toxidromes associated with poisonings

Toxidrome	Symptoms	Selected causative agents
Opiate syndrome	Somnolence to coma, miosis, cyanosis, respiratory depression	Opiates and opioids (e.g., morphine, heroin, oxycodone, methadone, fentanyl)
Anticholinergic syndrome	Somnolence to coma, hallucinations, mydriasis, impaired vision, tachycardia, dry skin and mucous membranes, hyperthermia and urinary retention, reduced gastrointestinal motility, thirst, swallowing and respiratory difficulties	Anticholinergic substances (e.g., atropine, scopolamine, diphenhydramine, doxylamine, tricyclic antidepressants, plants containing atropine/scopolamine (e.g., angel's trumpet (*Datura*), belladonna, datura, henbane))
M-Cholinergic syndrome	Miosis, bradycardia, defecation and urination vomiting, increased lacrimation and salivation	Acetylcholinesterase inhibitor (e.g., organophosphates, E 605, muscarine, e.g., from crack mushrooms (*Inocybe*))
N-Cholinergic syndrome	Tachycardia, hypertension, fibrillary contractions, paralysis	Acetylcholinesterase inhibitors (e.g., organophosphates, E 605)
(Nor)adrenergic or sympatho-mimetic syndrome	Agitation, euphoria, fear, confusion, tremor, seizures, hypertension, tachycardia, hyperthermia, perspiration	Sympathomimetics (e.g., amphetamines, cocaine)
Serotonin syndrome	Agitation, confusion, hyperthermia, myoclonias, tremor	Serotonergic substances (particularly in combination) (e.g., selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, MDMA (ecstasy))

Selected from Peters and Mall (2009)

following ethyl glycol, methanol, salicylate, and cyanide poisoning, whereas hypoglycemia should arouse suspicion of insulin, oral antidiabetic agent, and salicylate poisoning. Hemoglobin changes are evident in carbon monoxide, cyanide, and agents causing methemoglobinemia (e.g., nitrate, nitrite, aniline derivatives, and phenacetin metabolites in particular). In addition to these primarily clinical symptoms and syndromes, there are a number of conspicuous states or warning signals that are recognizable even to the layperson and may provide relevant information. The possibility of poisoning should always be considered in the following cases:

- Unexpected death of a young, otherwise healthy person
- Any unconscious patient
- Sudden illness in a child with no known previous disease
- Simultaneous illness in or death of multiple persons (e.g., following carbon monoxide emissions)
- Drug addicts

- Persons with easy access to toxins (chemicals, pesticides, pharmaceuticals)
- Particular constellations, such as inheritance disputes, threats, and enmities
- Following exposure to smoke gas or fires
- All disease types with symptoms similar to those of a particular form of poisoning (e.g., the clinical picture of arsenic poisoning is similar to that of cholera)

The circumstances under which a person is found can often arouse concrete suspicion of foreign substance involvement. For example, medication packaging and drug paraphernalia, injection sites, or even needle tracks (i.e., a line of needle-entry wounds produced by repeated injection along the course of a vein, mainly scarred and of differing age), as well as psychophysical deficits, are a direct indication of foreign substance abuse. In the absence of indications of this kind, it may prove challenging to detect medication and drug abuse, since a practical "marker," like the smell of alcohol on breath in alcohol abuse, is lacking. This situation is particularly relevant in the field of traffic medicine when, for example, a police official wishes to prohibit a vehicle driver from continued participation in traffic after suspecting drug abuse during a traffic check. Against the background of this problem, strategies to recognize the effects of drugs have been developed based mainly on changes in *pupil size* and *pupil response*. However, it should be borne in mind that these types of test are not specific for drugs and can also be affected by the correct and indicated use of medications (e.g., dilated pupils due to atropine applied in an ophthalmological setting) or other effects (e.g., contracted pupils due to certain disease patterns). In principle, it should be noted that drug consumers are not always conspicuous and do not always correspond to the widespread "junkie" image. The effects of a number of foreign substances on pupil size and response are given in Table 30.7.

Contracted pupils (*miosis*) are observed following exposure to parathion (E 605) and other pesticides. In the case of mixed intoxications (e.g., heroin and cocaine), pupils often appear normal, since their respective effects are largely able to offset one another. Other signs which may indicate foreign substance abuse include *rotary nystagmus*: rotating the body on its axis five times in 10 s with the eyes open, then focusing on the index finger, which should be held up at a distance of 30 cm; eye movement lasting in excess of 6 s is considered abnormal. This test is used mainly as a field sobriety test; however, results may be affected by disease and fatigue, among other effects.

Table 30.7 Effects of drugs and other foreign substances on pupil size and pupil response

Drug type	Pupil size	Pupil response
Amphetamines	Dilated	Delayed
Sedatives	Normal	Delayed
Cannabis	Normal to dilated	Normal to delayed
Designer drugs	Dilated	Delayed
Hallucinogenics	Dilated	Almost normal
Cocaine	Dilated	Delayed
Opiates/opioids	Contracted	Barely detectable response
Sleeping aids	Normal	Delayed
Inhalants	Normal to dilated	Almost normal

Observations on "divided attention," which can be significantly impaired in the case of drugs and medication in particular, and thus represent a relatively reliable identifying sign, are of particular importance in traffic medicine. This relates to the interplay of several individual actions that can be performed effortlessly by a nonintoxicated, healthy, and relaxed driver (e.g., coordinated acceleration, braking, steering, clutch engagement, and gear shifting). A driver under the influence of drugs often drives like a beginner. Numerous attention evaluation tests involving, e.g., asking for two things at once or asking simple but unusual, distracting, interrupting questions, all belong to drug and other psychotropic substance detection programs. Furthermore, the following warning signals may be an indication of drug abuse and, as such, are also important for parents and educators:

- Sudden drop in school performance in all subjects (not only in one subject!)
- Giving up or constantly changing friends
- Withdrawal or isolation

- Giving up previous interests, apathy
- Unexplained lack of money

However, the distinctive features described above (e.g., pupil size, unsure speech and gait, needle tracks, drug paraphernalia) would be insufficient evidence to prove drug abuse in the context of legal proceedings for instance. Thus, reliable drug detection and identification using modern methods of analysis is mandatory.

30.2.3 Classifying Poisons

Numerous poisons can be classified according to their effect. Examples of fast-acting poisons include:

- *Cyanide*: Sudden death only in the case of acidic conditions in the stomach, causing rapid release of hydrocyanic acid. If the stomach is full, potassium cyanide is converted to potassium hydroxide by hydrolysis, followed by an agonizing death from chemical burns.
- *Carbon monoxide*: Exhaust fumes, as well as any form of incomplete combustion of organic material. Therapy: hyperbaric oxygen treatment.
- *Carbon dioxide*: For example, in mines, fermentation cellars, and food silos.
- *Chloroform* and *ether*: Used as an anesthetic in victims of crime.
- *Parathion* (E 605): A pesticide that can also prove fatal after strong cramps of long standing. Antidote: atropine.
- *Alcohol*: In the context of irresponsible drinking bets, for example.

Examples of poisons with no early symptoms, their effect beginning only after hours or days, include:

- *Paracetamol*: An analgesic available without prescription in many countries. Lethal dose ca. 8 g in adults, while as little as 2 g can be lethal in children. Death as a result of fulminant liver failure. Antidote: *N*-acetylcysteine.
- *Paraquat* (herbicide): Lethal damage to the lung parenchyma (lung fibrosis) occurs following a latency of several days. Now rare in many countries.
- *Ethylene glycol* (antifreeze agents): Risk of severe kidney damage following a period of latency. Therapy/antidote: controlled administration of approximately 1‰ ethanol, causing substrate competition and blocking metabolization of ethyl glycol to highly toxic glyoxylic acid and oxalic acid; surviving ethylene glycol ingestion often means severe kidney damage in the form of oxalosis or anuria and the need for dialysis (see Fig. 30.1).
- *Methanol*: Methylated alcohol, poorly distilled liquor, and cleaning agents. Risk of blindness at 10 ml. Therapy/antidote: controlled administration of approximately 1‰

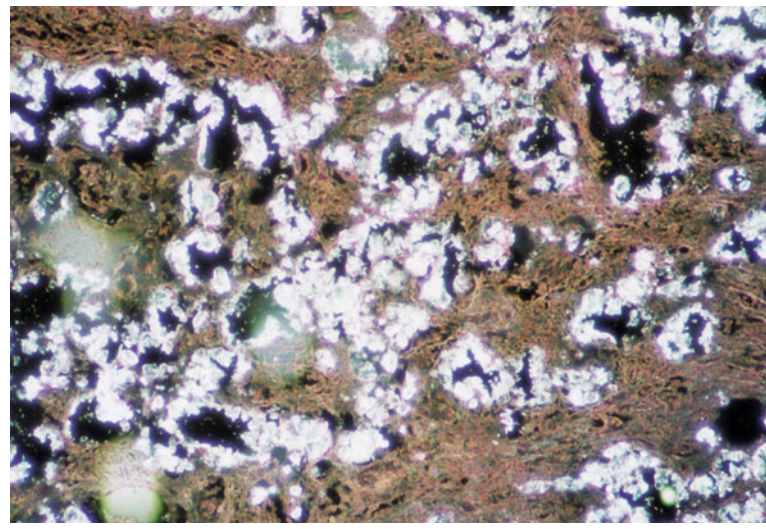

Fig. 30.1 Multiple polarization optical, birefringent oxalate crystals in chronic oxalosis due to ethylene glycol intoxication (From Dettmeyer (2011))

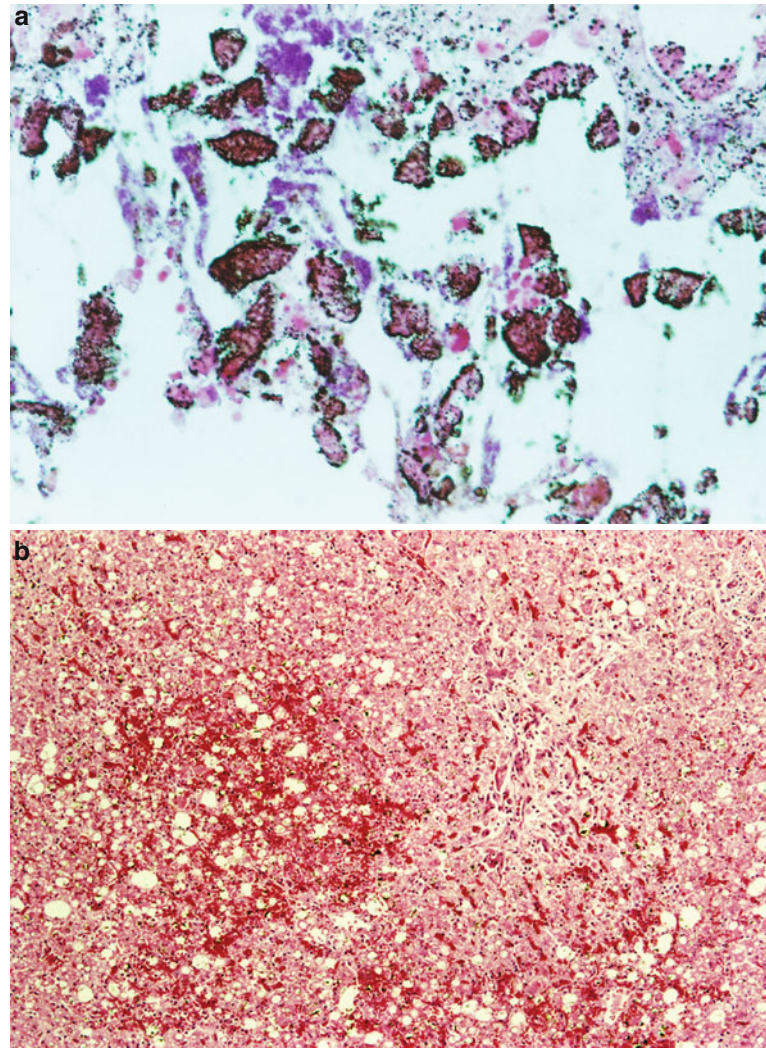

Fig. 30.2 (**a**) Intestinal mucosa with retained fungal components (**b**) and extensive destruction and hemorrhage in hepatic tissue in acute death cap intoxication (From Dettmeyer (2011))

ethanol, causing substrate competition and blocking metabolization to highly toxic formaldehyde and formic acid (damage to the optic nerve).

- *Thallium* (pesticides/rodenticides): Neurotoxic symptoms, often full-blown after 2–3 days, hair loss generally after 2–3 weeks, and Mees' lines after 4–8 weeks. Antidote: potassium iron(III) hexacyanoferrate(II).
- *Fungal poisons* (e.g., death cap): Following a latency period of approximately 12 h, stomach cramps and sometimes bloody diarrhea and vomiting ensue; acute liver dystrophy after 4–7 days, which usually follows a lethal course. Fungal components can often be detected microscopically in the gastrointestinal contents as well as on gastrointestinal mucosa (Fig. 30.2a); the liver usually shows extensive necrosis at the time of death (Fig. 30.2b).

30.2.4 Therapeutic Margin

It is important to know the therapeutic margins of foreign substances (e.g., medications). The smaller the therapeutic margin, the more dangerous a drug is. When using drugs with a narrow therapeutic margin, it is essential to ensure that the therapeutic range has not been exceeded; this

is done by regularly checking blood levels in the context of *therapeutic drug monitoring* (TDM). However, treatment should also not fall below the therapeutic margin.

Examples of toxins with *narrow therapeutic margins* include:

- *Alcohol* (ethanol): A blood alcohol concentration (BAC) of 2‰ usually carries no complications (with the exception of a possible "hangover" and/or police investigation) and causes no late effects; a BAC of 4‰, in contrast, can already be in the lethal range.
- *Coumarin-like anticoagulants*: Risk of (fatal) hemorrhage in the case of overdose. Antidote: vitamin K.
- *Cardiac glycoside*: Cardiotoxic effects in the case of overdose.
- *Lithium* (psychopharmaceutical): Agitation, confusion, as well as coarse tremor, dizziness, and cardiac arrhythmias in the case of overdose.
- *Paracetamol* (see Sect. 30.2.3, "poisons with no early symptoms").
- *Theophylline* (bronchospasmolytic agent): Tremor, tachycardia, arrhythmias, and cramps in the case of overdose.

Examples of toxins with *wide therapeutic margins* include:

- *Benzodiazepines* (e.g., diazepam): But only as mono-intoxication with no involvement of other centrally active agents
- *Glucocorticoids* (cortisol)
- *Penicillin* (antibiotic)

Finally, toxins can also be classified on the basis of their late effects in the case of survival. Benzodiazepine intoxication generally follows a largely uncomplicated course if suitable therapy is administered, while surviving even hydrocyanic acid or carbon monoxide poisoning is not necessarily associated with severe late effects. On the other hand, paracetamol or paraquat, for example, is greatly feared due to its high incidence of long-term late effects. Particularly challenging issues are faced when lifesaving medication (e.g., cardiac glycosides or insulin) are discontinued in "helpless" patients, either by the patient themselves or by relatives.

30.2.5 Therapeutic Strategies

As a basic principle, all cases of poisoning require medical treatment. Emergency poison hotlines provide rapid round-the-clock information on estimated risk and therapy options for affected individuals as well as medical professionals. The primary goal of first aid is to maintain vital functions. Unspecific removal of the poison can be performed by rinsing with water (eyes, skin) or by diluting orally ingested substances and vomiting with the aid of emetics. However, vomiting is contraindicated in the case of unconsciousness and certain intoxications, e.g., acids, alkalis, or solvents. Specific antidotes are available for use in some intoxications (Table 30.8).

Note: In emergencies, it should always be borne in mind that a detected foreign substance (e.g., atropine, biperiden, ethanol, or propranolol) was possibly not ingested by the patient themselves, but is instead attributable to antidote administration.

In principle, even following the absorption of toxins, *secondary poison elimination* is still possible by means of hemodialysis, hemoperfusion, plasmapheresis, peritoneal dialysis, forced diuresis, interrupting enterohepatic circulation, or exchange transfusion. In order for these largely invasive methods to be effective, information as accurate as possible about the toxicokinetic characteristics of the substance to be eliminated is essential. For some of the more commonly used toxic foreign substances, there are simple detection and identification methods, which can be applied rapidly even in small clinical–chemical laboratories in cases where emergency analyses are required. These, however, are not able to substitute (subsequent) analyses using valid analysis methods (Table 30.9).

Reliable diagnosis and effective therapy of suspected poisoning is not possible in the absence of clinical-toxicological or forensic toxicological analysis. However, excluding the possibility of poisoning may also be important, for example, in cases where this would lift incriminating suspicion in a forensic context or where this precludes the use of high-risk or invasive methods in the course of clinical treatment. Excluding poisoning

Table 30.8 Important antidotes, commercial products, and effectiveness in poisonings

Antidote (INN)	Commercial products	Effective against
Acetylcysteine	Fluimucil antidote	Paracetamol
Atropine sulfate	Atropine sulfate	Organophosphates
Biperiden	Akineton	Neuroleptic agents
Calcium-trinatrium-pentetate	Ditripentat-Heyl	Au, Cr, Fe, Mn, Pb, Pu, Zn
Deferoxamine	Desferal	Fe
Digitalis antitoxin	Digitalis antidote	Cardiac glycosides
Dimercaptopropanol sodium sulfonate (DMPS)	Dimaval, Mercuval	Hg, Pb
Dimethylaminophenol	4-DMAP	Cyanide, hydrogen sulfide
Ethanol		Ethylene glycol, methanol
Ferric hexacyanoferrate (II)	Antidotum Thallii-Heyl	Tl, Cs
Flumazenil	Anexate	Benzodiazepines
Fomepizole	Antizol	Ethylene glycol, methanol
Glucagon	GlucaGen	Antidiabetic agent, insulin
Hydroxocobalamin	Cyanokit	Cyanide
Potassium iodide		Iodine (radioactive)
Naloxone	Narcan	Opiate (excl. buprenorphine)
Sodium thiosulfate		Cyanide
Neostigmine bromide	Prostigmin	Curare alkaloids, muscle relaxants
Obidoxime chloride	Toxogonin	Organophosphates
Penicillamine	Metalcaptase, Trisorcin 300	Au, Co, Cu, Hg, Pb, Zn
Physostigmine salicylate	Anticholium	Parasympatholytic agents, phenothiazines, tricyclic antidepressants
Phytomenadione	Konakion	Vitamin K antagonists
Propranolol	Dociton	ß-Sympathomimetic agents
Pyridoxine	B_6-Vicotrat, Hexobione	Isoniazid
Oxygen		CO, CO_2
Snake poison serum polyvalent		Snake toxins
Silibinin	Legalon SIL	Death cap
Tolonium chloride	Toluidine blue	Methemoglobinemia-causing agents

From Mutschler et al. (2008)

can often even be lifesaving in cases of other severe and hitherto undetected diseases (e.g., brain hemorrhage).

30.2.6 Postmortem Toxicology

Poisoning can usually be recognized in living persons by identifying the symptoms and syndromes described in Sect. 30.2.2. Naturally, postmortem, information of this kind is lacking or has been obscured by antemortem therapeutic measures (e.g., antidotes). What is important here is that the possibility of poisoning is nevertheless taken into consideration. As a fundamental principle, poisoning should be considered in all cases of unexplained death!

Case Study

Serial poisoning with strychnine

Two young (consecutive) husbands of the same woman both died shortly after marriage. The woman's third husband was then taken to the hospital as an emergency. The preliminary diagnosis was classic extensor seizures with heart failure. Forensic toxicological investigations were able to demonstrate strychnine poisoning, resulting in the exhumation of the woman's two late husbands: both had been poisoned with strychnine.

In some cases of poisoning, external signs alone are able to provide information about a

Table 30.9 Simple and rapid analysis methods for cases of suspected poisoning in an emergency setting

Qualitative detection	
Chlorinated hydrocarbons	Fujiwara reaction
Cyanide	Test tubes (e.g., Dräger)
Ketones (acetone bodies)	Test strips (e.g., Ketur test)
Nitrite	Diazonium stain (Griess reaction)
Paracetamol	Indophenol reaction
Paraquat	Blue radical formation with sodium dithionite
Phenothiazines	Heavy-metal staining (Forrest)
Salicylates	Iron-III chloride staining (Trinder)
Acids and alkalis	pH value test strips
Quantitative detection	
Bromide	Phenol red test (Kisser)
Carboxyhemoglobin (COHb)	Spectrophotometric determination (Vierodt, Hüfner)
Cyanide	Spectrophotometric determination (isothermal distillation according to Conway or Widmark)
Iron	Spectrophotometric determination (Kleesiek, Solem et al.)
Ethanol	Enzymatic methods (ADH) (Bücher, Redetzki)
Paracetamol	Spectrophotometric determination using indophenol stain (Price)
Paraquat	Spectrophotometric determination of blue radical stain (Daldrup and Fowinkel)
Salicylates	Spectrophotometric determination with iron-III chloride staining
Thallium	Spectrophotometric determination through ion pair

certain poison; for example, commercially available preparations of the pesticide parathion (E 605) are intense blue in color, intended to produce a warning stain around the victim's mouth. Signs of chemical burns (e.g., from acids or alkalis) and abnormal odors (e.g., bitter almonds) are also a direct indication of poisoning. Empty medication packages, syringes, or other utensils found near the deceased may provide valuable and

Table 30.10 Examples of abnormal findings at autopsy

Abnormal findings	Possible causes (examples)
Dark line along gums	Lead or mercury (chronic)
Hair loss	Thallium
Cerebral edema	Nonspecific indication of intracranial pressure in the case of delayed onset of death
Icterus	Hepatotoxic agents
Injection site	Narcotics (i.v.), insulin (s.c.)
Pulmonary edema	Hemorrhagic, e.g., in the case of opiates
Miosis	Barbiturates, morphine, nicotine, opioids, organophosphates
Mydriasis	Alcohol, amatoxin, atropine, cannabinoids, colchicine, cocaine, methanol, scopolamine, cyanide
White or hemorrhagic edema fluid	Parathion (E 605), opiates
Livor mortis	Carbon monoxide (light red), methanol (gray), methemoglobinemia-causing agents, e.g., nitrite, phenacetin (brownish)
Mees' lines	Arsenic or thallium (chronic!)

time-saving information on the type of poison and be sufficient to support a primary suspicion, which, however, may prove unfounded. It is often the case that third parties (as in assisted suicide) or even the victim disposes of material of this kind themselves (common motives: fear of life insurance complications, questions of "family honor," desire to avoid an autopsy). A suicide note may be able to provide a clear indication of poisoning and its motive. While typical signs of lethal poisoning are often absent, an autopsy investigation is able to reveal findings to support the suspicion of certain poisons or poison groups (Table 30.10).

When evaluating quantitative toxicological findings (in particular blood values), it should be borne in mind that significant changes in foreign body concentrations can occur during and after the agonal period. This phenomenon, also known as postmortem release, is caused mainly by the interruption of steady states that can only be maintained in a living organism. This process of postmortem redistribution can make the interpretation of toxicological findings particularly challenging. Significant differences in femoral

and heart blood concentrations are found, for example, in digoxin, propoxyphene, and tricyclic antidepressant poisoning, while poisoning with cocaine, paracetamol, and salicylates produces milder differences.

The suspicion of poisoning is often aroused after death. In the case of burial, forensic toxicological investigations can be carried out following exhumation. In order to establish whether—and to what extent—foreign substances have been absorbed (e.g., heavy metals from neighboring earth) or released (e.g., via postmortem excretions), samples of earth from several areas, objects placed in the coffin, pillows, wood shavings, saw dust, as well as several coffin components should be obtained in addition to samples from the cadaver. Following cremation, during which all organic material (including most toxins) is destroyed, an investigation to detect anorganic toxins (e.g., thallium and other metals) can be carried if necessary.

30.2.7 The Diagnostic Value of Individual Sample Types

Stomach Contents and Gastric Lavage Fluids. This specimen material accumulates in acute poisonings. Shortly after ingestion, traces of tablets are often found; these can be classified as a particular product according to external characteristics (e.g., shape, color, inscriptions, or markings), thereby supporting a concrete suspicion. Quantitative determination of the as yet unabsorbed volume of a toxin can also influence the nature and extent of further detoxification measures.

Urine. Urine is usually required for screening analyses with no concrete suspicion of a foreign substance. The advantages of this specimen material include (among others):

- Foreign substances are usually present in higher concentrations in urine than in blood.
- Foreign substances are usually detectable in urine over longer time periods than in blood.
- The detection of particularly high levels of metabolites in urine may provide valuable additional information.
- Urine can be obtained from conscious individuals without the need for sampling techniques.

Using urine as a sample material for screening analyses has few disadvantages, other than that detection in urine may be impossible shortly after foreign substance ingestion (lag times) despite detectable concentrations in blood. Indeed, quite the reverse is more often the case, i.e., while drugs (amphetamines, cocaine, or opiates) or other foreign substances can no longer be detected in blood after only a few hours, they remain detectable in urine for several days or even weeks (e.g., cannabinoids). In many cases, although the originally ingested substance can no longer be identified, its characteristic metabolites can be.

Blood. The advantages of blood as a specimen material lie in the timeliness of the information it provides, i.e., when blood has been obtained shortly after an incident (e.g., an offense or poisoning). The blood concentration of a foreign substance, either directly detected or retrogradely calculated, reflects the action or toxic status at around the time of an incident far better than urine concentrations.

In the case of cadaver investigations, it should be borne in mind that femoral blood generally yields more valid information about concentrations at the time of death than for heart blood, for instance, where there is a greater risk of postmortem release and the migration of foreign substances, such as alcohol, from the stomach.

Head Hair. A wide variety of foreign substances (medications, drugs, metal toxins, and even alcohol in the form of its metabolite ethyl glucuronide) are stored in the follicles and roots of hair, which grows at a rate of approximately 1 cm/month. Thus, hair analysis permits a long-term reconstruction of foreign substance ingestion (drug career), while segmental analysis yields insights into particular periods of time, as in chronic poisoning. However, hair analysis meets its limitations in the case of one-off ingestion prior to an incident (e.g., as in "knockout" drugs). Several individual hairs are by no means sufficient for analysis; moreover, special sampling techniques need to be applied

Case Study

An investigator undercover as a heroin consumer

An undercover police investigator was found unconscious in bed in a hotel. He was resuscitated, and heroin metabolites were detected in the blood and urine samples taken in the course of emergency measures. The investigator made a statement to the effect that he had never consumed heroin before in his life and that the heroin had instead been injected with intent to kill by heroin dealers after they discovered him to be an undercover investigator and wished to eliminate him. The dealer, who had been arrested in the meantime, alleged in contrast that the undercover investigator had been addicted to heroin for several months and was one of the dealer's regular customers. Hair analysis results were able to reconstruct a period of long-term heroin abuse after opiate derivatives were detected in a long section of hair.

Case Study

Clozapine administration to an infant

The infant in question had been admitted to the hospital on an inpatient basis on four separate occasions at short intervals apart due to hyperthermia and blood count changes. The suspected medical diagnosis was a rare metabolic defect. After the child's death at the age of 2 years, the cause of death was given as "natural" in the death certificate. A rare metabolic disease was also suspected on clinical postmortem. Interment subsequently took place. Initial suspicions were aroused more than 1 year later when siblings started to show similar symptoms of poisoning. As a result, the body was exhumed. The highly toxic psychiatric drug (neuroleptic) clozapine could be detected by means of hair analysis. A charge of homicide was brought and a conviction for grievous bodily harm and manslaughter achieved.

Positive hair analysis was made possible since the offender (the mother) had administered the substance clozapine on several occasions prior to the child's death in order to test the effects of gradually increasing the dose (hence the four hospital stays). In the case of one-off administration, positive detection on hair analysis could not have been expected.

and possibly requested at a forensic toxicological laboratory.

Other specimen materials that can be considered include:

- *Materials and objects found around the victim*: For instance, tablets, fluids, empty packaging; garbage cans and medicine chests should be searched! A thorough inspection of the place at which the body is found should be carried out. Medications belonging to relatives, as well as the victim's professional field or opportunities to obtain substances, should be investigated.
- *Saliva and perspiration* can be obtained non-invasively in the case of suspected drug abuse and used for screening purposes.
- *Liver and kidneys*: To determine the overall distribution and estimate toxin volumes.
- *Muscle fiber*: An important sample in cases of putrefaction and burns.
- *Gallbladder*: Offers information on enterohepatic circulation and late detection of opiates.
- *Brain*: Detection of lipophilic, volatile gases, estimation of survival time.
- *Lungs*: Detection of volatile foreign substances, such as combustive agents.
- *Gastrointestinal contents*: Detection of plant toxins (fungi), as well as rectal introduction.
- *Fingernails*: Detection of metal toxins.
- *Bones*: Chronic metal poisoning.
- *Injection site*: Detection of insulin and other injected substances.
- "Exotic" exhibits: Such as tampons (see Case Study below).

Case Study

Cyanide in a tampon

A young woman was found dead by her husband on his return from a long business trip (thus with an airtight alibi). Results obtained at autopsy and from chemical–toxicological blood analyses showed cyanide poisoning. Initially, the case remained unsolved, since no cyanide could be detected in the stomach; only in blood could lethal concentrations be found. Finally, it could be shown that the cyanide had been applied to a tampon by the victim's husband prior to his business trip and that the cyanide had been absorbed through the vaginal mucosa.

Note: In the case of nonspecific suspected poisoning, sample material should be collected particularly carefully and extensively.

Storing and Transporting Sample Material. Transport containers should neither add to (note: empty medication bottles) nor detract from (note: infiltration of lipophilic substances in plastic containers) a sample. Keeping a sample cool is usually sufficient until a laboratory is reached. The former recommendation to add acids or preserving agents is no longer valid and may even be harmful. Special procedures, such as phase separation or deep freezing, are carried out at the laboratory depending on the type of foreign substance and specimen.

30.2.8 Sampling Strategies and Quantities

Due to the generally serious consequences of drug detection for the individual involved, attempts are occasionally made to manipulate results.

Manipulating and Falsifying Samples. This usually involves attempts to mimic drug abstinence. However, there have been cases where, for instance, tablets dissolved in urine have been used to simulate massive drug effects. Manipulation is mainly a problem when an individual has time to prepare for a particular sampling appointment. For this reason, many investigating centers give appointments on a random basis. The risk of manipulation is far smaller in the narrower forensic field, where sampling is unforeseeable, e.g., following a traffic accident.

In principle, a distinction is made between three different manipulation possibilities:

- An individual dilutes his/her own urine sample with water, tea, drug-free urine, or other suitable fluids in order to fall below the detection limit.
- An individual attempts to modify his/her own urine sample by *adding* or *ingesting particular substances or agents* in order to produce a false-negative screening result.
- An individual attempts to provide a fraudulent, drug-free urine sample by using products available on the Internet (e.g., the "Whizzinator," an artificial penis).

Important: **Urine samples should be obtained under supervision**. **In addition**, **no water**, **toilet cleaner**, **liquid soap**, **disinfectant**, **or similar agents should be located in the sampling room**, **since items of this kind may be used to falsify results**.

As a basic rule, samples of the following should always be obtained in living persons:

- *Urine* (where possible, 50–100 ml). Question: *What* has been ingested?
- *Blood* (where possible, 5–10 ml). Question: *How much* has been ingested?
- *Gastrointestinal contents or vomit* (in poisonings). Question: How long ago was the poison ingested, and how much of it has not yet been absorbed?
- *Head hair*, possibly to clarify cases of suspected long-term administration, e.g., by estimating tolerance and compliance or monitoring abstinence in cases of driving license recovery. Sampling regulations should be strictly observed here: a clump of hair approximately pencil-size in diameter should be fixed using adhesive tape and cut from the crown of the head; a shift lengthways could impair the temporal classification of foreign substance intake.

Important autopsy *samples* include:

- *Heart blood* (ca. 100 ml)
- *Femoral vein blood* (ca. 10 ml, e.g., to determine BAC)
- *Urine* (ca. 100 ml; for equilibration, determine total volume beforehand)
- *Gastric contents* (ca. 100 ml; again, total volume should be measured)
- *Liver*, *kidney*, and *lung tissues* (ca. 100 g each)
- *Bile fluid* (to detect substances in the enterohepatic circulation)
- *Brain* and *fatty tissue* (to detect lipophilic and volatile foreign substances)
- *Skin*, *nails*, and *bones* (in suspected metal poisoning)
- *Feces* (in suspected plant poisoning, such as fungi)
- *Vitreous humor*
- *Hair*
- *Liquor*

The question of detection periods plays an important role in the selection of samples and the interpretation of results. This parameter, also referred to as the diagnostic window, depends on the type of foreign substance, the quantity (dose), and frequency of administration, alongside a multitude of other pharmaco- or toxicokinetic parameters. Table 30.11 provides a general, sample-specific overview, while details on substance-specific lag times can be found below with the relevant descriptions of important individual substances.

Table 30.11 Detection periods (diagnostic windows) for a number of foreign substances found in important sample materials in living individuals

Sample	Detection period (depending strongly on method sensitivity!)
Stomach contents	*Begins* immediately after oral ingestion *Ends* once absorption is complete *Note*: residual absorption of basic substances from blood (i.v. injection) in acidic stomach contents is possible, producing risk of false interpretation (incorrect assumption of oral administration)
Blood	*Begins* immediately after i.v. injection and inhalation; after oral ingestion, following several minutes up to 1 h or even more, depending on the absorption parameters also known for alcohol *Ends* depending on pharmaco- and toxicokinetic parameters (in particular, the elimination half-life)
Urine	*Begins* after several minutes up to 1 h or even more, depending on the absorption parameters also known for alcohol *Note*: even after ingestion of high doses, lag times of up to 1–2 h or even more may be seen For this reason, both urine *and* blood should be analyzed in cases of poisoning *Ends* depending on pharmaco- and toxicokinetic parameters (in particular, the elimination half-life). Urine pH value is an important factor: base substances are eliminated rapidly at acidic pH values, slowly at alkaline); the converse is true for acidic foreign substances (e.g., barbiturates). Also, a foreign substance's lipophilicity plays an important role (following absorption in fatty compartments, THC is released after a certain delay; hence THC-COOH can be detected for up to several weeks in the case of long-term use, occasionally even up to 3 months)
Saliva	Similar to blood (values often correlate well with blood values)
Perspiration	Similar to blood *Note*: lag times are possible due to transfer to the skin surface, in some instances with considerable time delays. In addition, poor hygiene can result in traces of dried drugs producing positive results after significant periods of time, although drugs are no longer present in blood and urine
Hair	*Begins* within days following what is usually repeated ingestion, isolated instances are difficult to detect; exceptions have been reported, particularly following poisoning *Ends* after months, occasionally years, depending greatly on hair length and hair treatment

Maximum Detection Period. The question of how long ingestion of a substance lies in the past is often posed and difficult to answer, since elimination (and hence also the detection period)

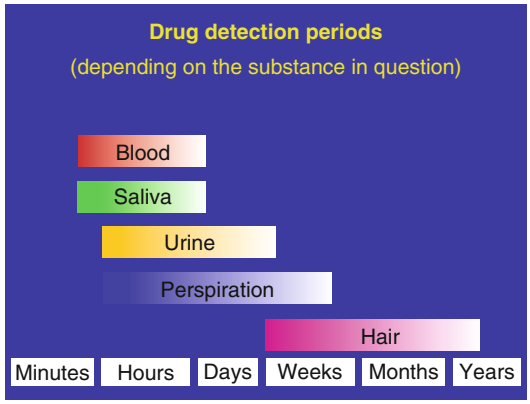

Fig. 30.3 Diagnostic window for various sample materials

depends on numerous intra- and interindividual factors. Dose plays a particularly important role here, as well as damage to the organs of elimination, the liver and kidneys. The latter results in slower and thus also longer elimination, making detection periods longer compared to healthy individuals.

The overview shown in Fig. 30.3 has proved helpful as a *practical guide* to detection periods. However, variations upwards or downwards to a greater or lesser extent should always be expected! For example, the THC metabolite THC carboxylic acid can still be detected more than 100 days (!) following drug cessation.

Vital elimination of foreign substances ceases on death. Detection periods are determined to a great extent by cadaveric conditions, such as postmortem interval, temperature, moisture, and pH value. The following are guideline values for the maximal detection time of toxins in *cadavers*:

- *Anorganic* toxins, such as arsenic, lead, and thallium: centuries.
- *Organic* toxins, such as morphine and strychnine: decades.
- Even carbon monoxide can often be detected as a COHb complex after weeks or months.

Note: There is always a danger of toxins in the surrounding area migrating to a cadaver.

In certain autopsy cases, prior consultation with a forensic toxicologist is advisable, since some cases require special samples, such as vitreous humor or liquor, or sampling strategies, as in the case of some moderately volatile or unusual toxins.

30.2.9 Analytical Detection and Determination Methods

Targeted and Nontargeted Analysis. In cases where there is concrete evidence—due to certain symptoms or substances found—of a toxin type, any analysis strategy will generally focus on this primary suspicion. This is known as targeted analysis. If a drug addict is discovered with typical utensils (heroin paraphernalia) and miosis is present, opiate intoxication should be the considered first. However, there are often no signs of foreign substances whatsoever. Moreover, it may not be clear whether mono-intoxication is being dealt with or whether other substances are involved. In such cases, a wide range of foreign substances need to be tested for as part of an untargeted analysis strategy (*general unknown analysis*) using special chromatography and spectroscopy search software. This type of investigation, which can often cover several thousand substances, is also known as systematic toxicological analysis (STA). Analyses of this kind, which can be very time-consuming and highly dependent on equipment, are also referred to as *screening tests*. Even in the case of negative results, intoxication with an extremely rare or even a new substance cannot be fully ruled out.

Immunochemical Screening Methods (*Immunoassays*). Particularly in the field of drug analysis, immunoassays are used in the first instance as a screening method, enabling high numbers of samples to be classified as "negative" or "positive" rapidly and with minimal preparation. As a basic principle, the following applies: screening methods should yield specific and sensitive qualitative or semi-qualitative results in a straightforward manner, i.e., results should provide orientation.

In general, no further investigations are carried out once a *negative* screening result has been obtained, since modern screening methods rarely yield false-negative results. A false-negative result would mean that a test gave a negative result despite the presence of a drug. This strategy involves the often correct assumption that a false-negative test can have no prejudicial consequences for the individual involved in terms

of their being wrongly suspected or incriminated. However, a false-negative test bears the inherent risk that a public transport bus driver, for instance, is not promptly barred from driving and may undergo drug therapy only after further, possibly fatal, incidents.

In the case of a *positive* screening result, in contrast, investigations to confirm and differentiate this initial result are required and carried out using analytical methods based on a different physicochemical principle. Such tests usually involve chromatography, e.g., gas chromatography (GC), high-pressure liquid chromatography (LC, HPLC), or spectroscopy methods (generally mass spectroscopy, MS).

The need to use *confirming methods* is established mainly on the basis of the following:

- Although it is rare with modern immunoassays for positive immunochemical screening results to not be confirmed by more valid methods, e.g., MS, and thus false-positive, the disclosure of an unconfirmed immunohistochemical finding of this kind can have far-reaching consequences for the individual in question, such as therapy discontinuation or long-term license revocation, representing a threat to the livelihoods of certain professional groups (e.g., long-haul truck drivers, pilots).
- Most immunochemical screening methods used for toxicological analysis detect only groups of active substances (e.g., opiates), whereas they are unable to differentiate between individual substances within a group, such as the legal opiate codeine, which is contained in numerous prescription cough syrups, and the non-tradable and hence illegal opiate heroin. A reliable distinction between the two substances, which is of the utmost importance in the context of criminal liability, is possible and straightforward using mass spectroscopy for instance. It should be pointed out here that immunoassays for opiates are unable to detect the opiate-like opioids dextropropoxyphene, levomethadone, nefopam, pentazocine, pethidine, tilidine, and tramadol, since these have insufficient cross-reactivity due to their considerable structural and chemical differences.

Basic Principles, Performance, and Evaluation of Common Immunoassays. Immunochemical methods are based on antigen–antibody reactions. Antibodies, also referred to as immunoglobulins or gamma globulins, are the immune system's response to foreign substances invading the organism. They are formed to bind to the latter and render them harmless. Antibody formation, however, is only triggered when the invading molecule measures a minimum of approximately 5,000 Da. Daltons (Da) are used to describe unified atomic mass, whereby 1 Da is 1/12 of the mass of the carbon isotope ^{12}C. Therefore, only high-molecular substances cause the organism to react by forming antibodies. Hence these substances are referred to as antigens (antibody-generating). Even outside the organism, antibodies are capable of antigen-specific binding, a phenomenon capitalized upon for immunochemical testing. To generate antibodies against low-molecular substances, such as drugs and medications measuring far below 5,000 Da, large carrier molecules are used to achieve the size of reactive molecules (5,000 Da), thus enabling measurement. The technique used for immunochemical detection can vary widely. In general, so-called competitive immunoassays are used, whereby antigens in the specimen to be tested compete with labeled reagent antigens for a limited number of antibodies. The following tests are often applied in practice:

- Competitive radioimmunoassay (RIA)
- Enzyme-multiplied immunoassay technique (EMIT)
- Cloned-enzyme donor immunoassay (CEDIA)
- Fluorescence polarization immunoassay (FPIA)
- Enzyme-linked immunoassay (ELISA)
- Microparticle enzyme immunoassay (MEIA)
- Luminescence immunoassay (LIA)
- Ascent multi-immunoassay (AMIA)
- Gold-labeled optically read rapid immunoassay (GLORIA)

The *radioimmunoassay* initially used has been almost completely replaced—due to its numerous disadvantages, including the handling and disposal of radioactive material—by nonradioactive immunoassays used in routine drug analysis. The ease with which modern, rapid drug tests are

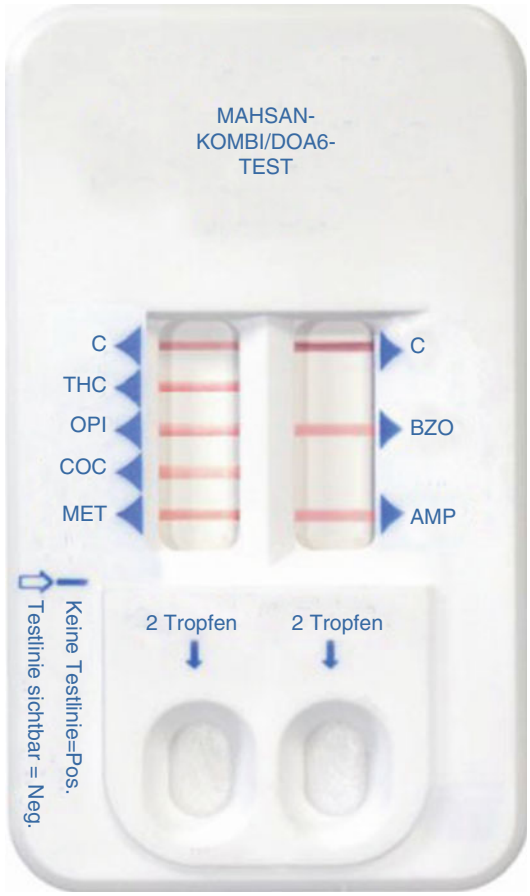

Fig. 30.4 The MAHSAN® test (source: MAHSAN®-Diagnostika, Reinbek, Germany)

performed, primarily by police for pretesting purposes, is best illustrated with the widely used and trusted MAHSAN® test (Fig. 30.4).

The MAHSAN device utilizes the principle of immunochromatography; the test is based on a physicochemical competition between the membrane-bound drug and the drug in urine for a limited number of gold-antibody conjugates.

Test Procedure: Two drops of urine are placed in each sample well; results appear within 3–10 min.

Test evaluation:

Negative: When a clearly identifiable red line appears level with the respective abbreviation: THC, cannabinoids; OPI, opiates; COC, cocaine; MET, methadone; BZO, benzodiazepine; AMP, amphetamine.

Positive: When *no* (!) detectable red line appears level with the respective abbreviation (see above).

In the case study illustrated in Fig. 30.4, all tests proved negative. Moreover, the test includes a control function whereby it cannot be evaluated if the control line "C" is undetectable; if this is the case, either insufficient urine was used or the test was performed incorrectly.

In the event of a positive result on police pretesting, such as the MAHSAN® test, the nal von minden Drug-Screen®, the saliva Drug Test®, or DrugWipe®, the investigating authorities will usually request an investigation to confirm and differentiate findings, e.g., at a forensic medicine institute. Here, the forensic toxicological department will check the external screening result, generally using immunochemical methods (immunoassay), in order to better develop a strategy for the confirming analysis. Figure 30.5 shows two examples of typical results from immunochemical control screening.

Cutoff Value. A cutoff value can be defined as a threshold value, such as a unit of concentration or a dimension, beneath or above which analysis results are considered "negative" or "positive," respectively. Values immediately below the cutoff value are sometimes referred to as "borderline negative," while values immediately above are referred to as "borderline positive." The cutoff value is determined according to the questions posed by the commissioning authorities.

Case Study

Toxicological analysis in a case of polytoxicomania

A young woman was arrested while dealing cocaine (COC). During questioning she admitted to having also sniffed the opiate (OPI) heroin to relieve strong nasal pain. (The practice of sniffing heroin over a long period of time causes acids formed by hydrolysis of the salt form to attack the nose, in particular the nasal septum, causing chronic pain.) In addition, it emerged that the young woman was also on a methadone

program (MET) as an approach to ending to her drug addiction. Meanwhile, she was obtaining the benzodiazepines (BENZ) diazepam (Valium®) and flunitrazepam (Rohypnol®), as well as the tricyclic antidepressant (TCA) doxepin (Aponal®) in high doses (hence the strongly positive test result marked with "AMAX") as a bridgeover against withdrawal symptoms between individual administrations of cocaine and heroin. Finally, cannabis derivatives (THC) were also detected. All immunochemical screening results could be reliably confirmed using gas chromatography/mass spectroscopy (GC-MS) and/or high-pressure liquid chromatography/mass spectroscopy (LC-MS). However, it was not possible to establish with an immunoassay alone whether the detected opiates were illegal heroin or legal codeine. The same applied to the differentiation of benzodiazepines and tricyclic antidepressants.

Confirming Methods. A substance can be conclusively identified with the aid of mass spectrometry (Fig. 30.6). There is complete concurrence between the signals in the actual case and the reference substance (here MDMA (ecstasy) from the spectrum library).

The diagnostic value of toxicological analysis can be effectively explained using intoxication with the analgesic paracetamol as an example—a particularly dangerous example due to its absence of initial symptoms, putting it at the top of poison information center statistics for both accidental and intentional intoxications. The nomogram shown in Fig. 30.7 is based on extensive data from cases of poisoning. Hepatotoxicity is classified according to the blood paracetamol concentration and the interval since ingestion, both of which also directly influence the application of therapeutic measures and antidote administration (*N*-acetylcysteine).

The serious consequences of a non-confirmed false-positive immunoassay are illustrated in the following case study.

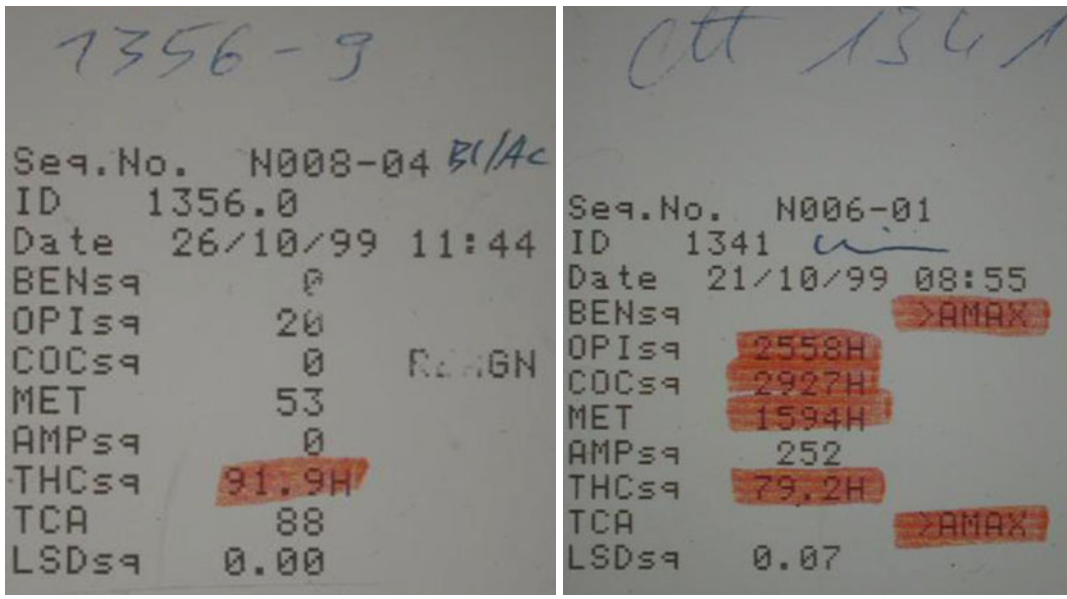

Fig. 30.5 Results of an immunochemical screening test using fluorescence polarization immunoassay (FPIA). The values marked in *red* indicate a positive screening result since they are above the cutoff value, while the low values are irrelevant from a forensic toxicological perspective, being attributable to either minute concentrations or artifacts. The printout on the *left* shows a test that was positive only for cannabis (*THC*), while the *right-hand* results are those of a polytoxicomanic drug abuser

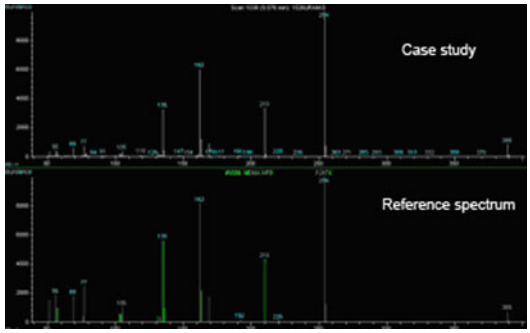

Fig. 30.6 Conclusive identification of MDMA using mass spectroscopy

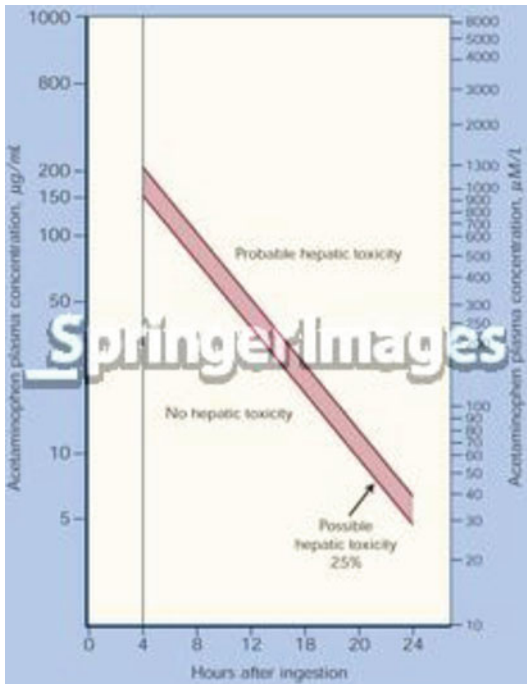

Fig. 30.7 A nomogram to estimate paracetamol hepatotoxicity (From Ito et al. (2006))

Case Study

Misdiagnosis "opiates in urine" in a case of false-positive immunoassay

The public prosecution department commissioned an expert appraisal after an already extremely time-consuming and expensive investigation had been undertaken. A 35-year-old man complained of upper abdominal discomfort, upon which his primary care physician suggested analyzing a urine sample for toxins. The uncommented findings report from the laboratory physician read: Opiates in urine "20 ng/ml." The public prosecutor subsequently opened an investigation into the man's sister-in-law on the grounds of suspected administration of poison and contravention of narcotic regulations, since, in his opinion, a concrete value (i.e., 20 ng/ml) had been detected. Only after months of extensive and costly investigation was a valid forensic toxicological review of the case undertaken, since residues of the sample material were available. Confirming analysis using gas chromatography/mass spectroscopy (GC-MS) yielded no evidence of opiates or other toxins.

30.2.10 Quality Control and Plausibility

The often far-reaching consequences of detecting drugs necessitate the best possible safeguards in the form of quality control to ensure the reliability of analysis results. This applies to positive results in particular. On the other hand, false-negative results carry their own risks, for example, when a case of poisoning is overlooked and lifesaving therapy (e.g., an antidote) is not administered.

External quality controls (*interlaboratory tests*) for blood alcohol and numerous drugs are offered in Germany by the Society for Toxicological and Forensic Chemistry (*GTFCh*), as well as many other organizations, under qualitative and quantitative aspects. In addition, *internal quality control* is an integral part of any modern quality management system; it requires positive and negative samples in all analysis series and should be organized according to good laboratory practice (GLP) guidelines. As clearly illustrated above, this latter aspect relates not only to actual measuring techniques but more to the somewhat peripheral issues, such as writing detailed descriptions of analysis procedures (whereby any deviation from standard methods should be recorded), archiving measurements,

as well as carefully documenting the course a sample takes to and within a laboratory (the so-called chain of custody). Above all, however, all analysis results should be checked for plausibility. The following questions should be asked as part of this procedure:

Is the result compatible with the individual's condition?

This would not be the case if, for example, a patient showed no performance deficits of any kind despite an allegedly measured BAC of 4‰. In this case, a sample mix-up or an analysis error should be considered.

Can a trend be logically explained?

In the course of trend monitoring, the question arises as to how much a parameter value can change within a certain period of time and remain reasonably explainable. A BAC of 3‰ which has failed to drop to physiological levels after 30 h, at the latest, is implausible given that the human organism metabolizes and eliminates at least 0.1‰/h (see Chap. 29). In order for this to be the case, repeat alcohol consumption must have taken place.

Is a result analytically plausible?

Example: Large quantities of cocaine were detected in a urine sample, but not the main metabolite benzoylecgonine. The patient, however, showed no obvious signs of intoxication. This toxicologically implausible finding could be explained by the subsequent addition of cocaine to the urine sample "in vitro."

Case Study

Red cabbage rather than parathion

The image shown in Fig. 30.8 was taken during autopsy. Due to the conspicuous blue color seen on a section of the small intestine, poisoning with parathion (E 605), which usually contains benzidine blue as a warning dye, was initially suspected. However, this suspected diagnosis was implausible since the advanced stage of passage through the

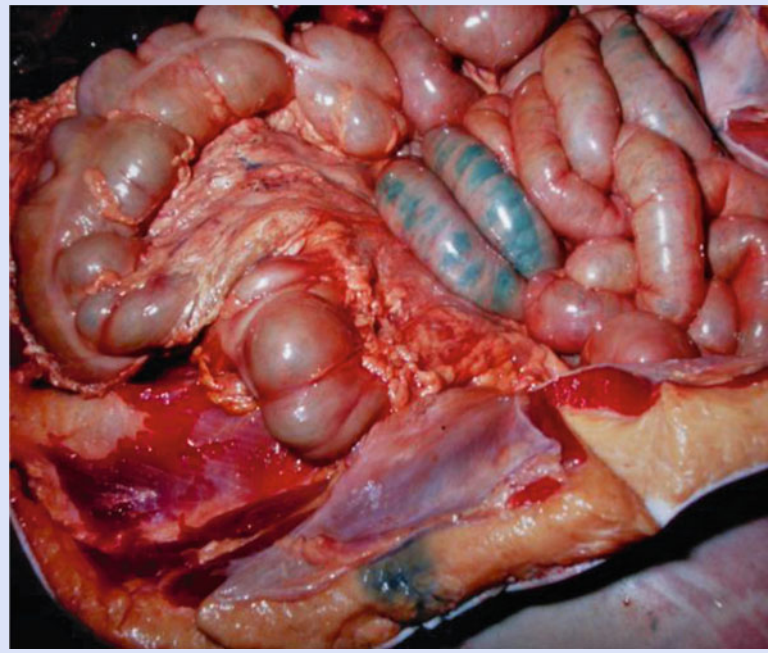

Fig. 30.8 Suspected diagnosis of parathion poisoning due to blue staining of the stomach contents

small intestines was incompatible with this rapid-working toxin. In reality, this was not a case of poisoning; the intestinal contents proved instead to be red cabbage, which had turned blue in the alkaline environment due to its indicator characteristics.

30.2.11 Toxicology of Special Substances and Groups of Substances

The toxicokinetics and toxicodynamics of alcohol are discussed in detail in Chap. 29, as are the clinical-toxicological aspects of alcohol intoxication.

Special Forensically Relevant Foreign Substances. The distribution range of toxins is subject to constant change. When a hitherto easily available substance becomes a prescription drug, it will often be replaced by a nonprescription substance. On the other hand, media coverage of celebrity intoxications or discussions in Internet forums often influences an individual's choice of toxin or suicide strategy. Medications still represent the substances most frequently detected in association with intoxications, followed by household products, recreational substances, and poisonous plants. A varying distribution between adults and children is seen here (see Table 30.1).

"*Exotic*" *Substances*. Depending on their availability for the purposes of suicide, in the work environment (e.g., an electroplater with access to numerous metal toxins), or in other special circumstances, highly unusual substances are occasionally used, making their identification challenging. In addition to rare medications, these may include numerous plant and animal toxins, as well as more "exotic" substances, for example, polonium (^{210}Po), as in the spectacular poisoning of the ex-Russian agent Alexander Litvinenko in November 2006, or ricin, which was administered via an umbrella tip in the notorious 1978 "umbrella assassination" of Georgi Markov in London. In addition, primarily illegal substances relevant in traffic medicine are described, although these are often also associated with intoxications. In another section, medications and other substances primarily of forensic toxicological interest are discussed, although these are also relevant in traffic medicine.

30.2.12 Dependence and Abuse

The World Health Organization (WHO) defines "dependence" as a psychological, possibly also physical, condition characterized by a strong desire or sense of compulsion to continually and periodically take a substance. Dependence is recognized today as a disease and is usually treated with therapeutically supported inpatient withdrawal.

Definitions

Abuse: The administration of medications, drugs, or other foreign substances that leads to physical and psychosocial damage. A dependence, however, is generally not (yet) present.
Psychological Dependence: The individual experiences an intense desire for an addictive substance and only feels content following administration of the relevant substance. Once self-control has been eroded, all thoughts and actions are focused on procuring medications or drugs.
Physical Dependence: The body reacts to the risk of intoxication with counter-regulatory mechanisms (e.g., faster elimination of foreign substances by enzymes). This is associated with an increase in both tolerance and dose. Intoxication increasingly becomes normality. Cessation of the addictive substances leads to withdrawal symptoms, such as cramps, agitation, sleeping disorders, headaches, anxiety attacks, and outbreaks of sweating.
Multiple Dependencies: Dependence on several addictive substances is referred to as polytoxicomania.

The WHO classifies forms of dependence into the types shown in Table 30.12.

Table 30.12 Forms of dependence with various classes of substance (WHO classification)

Type	Tolerance	Physical	Psychological
Amphetamines	++	(+)	++
Alcohol/barbiturates/sedatives	+++	++	++
Cannabis/marihuana	(+)	(+)	++
Cocaine	(+)	(+)	+++
Morphine/opiates	+++	+++	+++
Hallucinogens (LSD)	++	(+)	++

30.2.13 Threshold Values in Drug Consumption

In contrast to alcohol, most countries have no legally established threshold values for illegal drugs and medications above which a *relative or absolute unfitness* to drive is assumed. The principal reasons for this are the following:

- The physical and psychological effects of drugs and medications depend more greatly on intra- and interindividual parameters than with alcohol. These parameters include primarily tolerance, as well as varying toxicokinetic values, such as the elimination half-life's dependence on age and preexisting disease.
- Although much insight into the effects and kinetics of alcohol has been gained in extensive studies using voluntary participants, the same is hardly possible for illegal drugs due to the unwillingness of ethics committees in many countries to authorize such research. This unwillingness is based on the significant risk of dependencies being formed with many drugs, as well as their unknown kinetics.

Liability Under the Influence of Drugs. The type of offense plays a significant role in the assessment of liability in individuals under the influence of drugs. In the case of criminality directly for the purposes of obtaining drugs, there is usually a direct link between the offense and an addiction. In the course of what can be violent offenses, generally only the individual's needs are covered, and drug consumption usually takes place immediately thereafter. Depending on the case, the individual's ability to gain insight into or control of a situation may be reduced, if not completely absent. In the case of crime indirectly for the purposes of obtaining drugs, on the other hand, it is assumed that a certain level of self-control is required to carry out the offense, e.g., drug dealing, in order to ensure the successful completion of often complex processes. The dealer is often not a drug abuser himself/herself; however, where this is the case, reduced ability to act responsibly, i.e., reduced liability, may be considered. Any assessment of criminal liability in common criminal offenses should be oriented by the psychophysical deficits seen in close temporal association with the offense.

30.2.14 Major Drugs and Substances

Important: **Due to the wide variance in pharmacokinetic parameters and a strong dose relationship**, **the values given in Tables** 30.13, 30.14, 30.15, 30.16, 30.17, 30.18, 30.19, 30.20, 30.21, 30.22, 30.23, 30.24, **and** 30.25 **are intended for orientation only**.

Amphetamine Derivates and Designer Drugs. Derived from catecholamines and ephedrine, amphetamines are the oldest synthetically produced drug with a stimulatory effect. Unlike with most other substance groups, this particular substance has both positive and negative effects, depending on the method of administration and/or indication. In the case of attention deficit hyperactivity syndrome (ADHS) in children and adolescents, as well as in narcolepsy, these substances provide a valuable therapeutic effect and generally do not lead to dependence. In the absence of an appropriate indication, however, they do carry a significant risk of dependence when taken, e.g., in the context of the drug scene. Important data on amphetamines, methamphetamines, and the designer drugs MDMA, MDA, MDEA, and MDE are given in Tables 30.13, 30.14, and 30.15.

By means of generally illegal synthesis, derivates regularly appear with new substitution patterns; these are considered to be narcotic agents only once they have been adopted in the relevant

Table 30.13 Amphetamine data

Street names (examples)	Speed, pep, among others
Forms of administration	Primarily oral and nasal
Range of effects	Central stimulant effect (hence used for doping), also produces euphoria and suppresses the need for sleep, followed by marked exhaustion
Side effects	Depression, psychoses, hallucinations
Usual dose	10–50 mg (extreme dose, 2–5 g/day)
Effective period	1–3 h
Biotransformation	May be eliminated unchanged Major metabolite: phenyl acetone
Kinetic data	Half-life: 4–12 h
Detection period in blood	ca. 6–10 h
Detection period in urine	ca. 1–3 days (strongly pH-dependent)

Note: So-called prodrugs, such as amphetaminil (psychoanaleptic), benzphetamine, clobenzorex, dimethylamphetamine, ethylamphetamine, famprofazone, fencamine, fenethylline, fenproporex, furfenorex, mefenorex, mesocarb, prenylamine, and selegiline (antiparkinson agent), can result in the production of methamphetamine or amphetamine in the organism

Caution: False-positive immunoassays may be seen following ingestion of cyclamate (sweetening agent), as well as the formation of phenylethylamine due to autolytic and putrefactive changes to the specimen

Table 30.14 Methamphetamine data

Street names (examples)	Crystal ice, crystal speed, meth, Yaba, among others
Forms of administration	Primarily oral and nasal
Range of effects	Effect approximately double that of amphetamines (see Table 30.13), followed by marked exhaustion
Side effects	Depression, psychoses, hallucinations
Usual dose	10–50 mg (extreme 2–5 g/day)
Effective period	1–3 h
Biotransformation	May be eliminated unchanged Major metabolites: amphetamine, 4-hydroxymethyl-amphetamine
Kinetic data	Half-life: 4–12 h
Detectability in blood	ca. 6–10 h
Detectability in urine	ca. 1–3 days (strongly pH-dependent)

Note: See Table 30.13

Table 30.15 Data for 3,4-methylenedioxymethamphetamine (MDMA), 3,4-methylenedioxyamphetamine (MDA), and 3,4-methylenedioxyethylamphetamine (MDEA, MDE)

Street names (examples)	MDMA: ecstasy, Adam, XTC MDA: love pills, speed for lovers MDEA/MDE: Eve, Eva
Forms of administration	Primarily oral and nasal
Range of effects	Central stimulant effect, produces euphoria and suppression of the need for sleep, heightened sensory perception, reduced communication barriers, increased self-confidence, followed by extreme exhaustion
Side effects	Depression, psychoses, and hallucinations
Usual dose	Up to approximately100 mg
Effective period	1–3 h
Biotransformation	May be eliminated unchanged
Kinetic data	Half-life: ca. 7–25 h
Detectability in blood	6–10 h
Detectability in urine	24–48 (maximum, 72) h

Note: Ecstasy is a commonly used collective term for methylenedioxyamphetamines: MDMA, MDA, and MDEA/MDE. Liquid ecstasy, on the other hand, is a completely different structure, i.e., gamma-hydroxybutyric acid (GHB), the so-called knockout drug

Table 30.16 Data on tetrahydrocannabinol [THC/THC (Δ^9-tetrahydrocannabinol)]

Street names (examples)	For hashish: dope,[a] hash, hemp, kif, pot, shit For marihuana: grass
Forms of administration	Usually smoked in a joint, bong, or pipe (chillum); more rarely, ingested orally (biscuits or tea); effect easier to "control" with smoking than with oral ingestion
Range of effects	*Acute phase*: ca. 1–2 h following consumption Central depressant effect, impaired motor function and perceptivity, often wide and fixed pupils, impaired gait and speech *Subacute phase*: ca. 4–6 h following consumption

(continued)

Table 30.16 (continued)

	Euphoric mood, negative aspects suppressed, increased well-being, reduced ability to be critical, overestimation of self *Particular relevance in traffic medicine*: Hazardous driving style at excessive speeds, easily distracted and with low concentration, inadequate reaction to unexpected events, disregarding right of way and traffic signals Postacute phase: ca. 14–24 h following consumption Largely passive state with numerous performance deficits relevant in traffic medicine
Side effects	See "Range of effects" Personality disorders, particularly in the case of chronic consumption
Usual dose	Smoking: 5–40 mg THC Oral: up to 20 mg THC
Onset of effects	Smoking: ca. 5–30 min Oral: ca. 2–3 h
Duration of effects	Smoking: ca. 3–4 h (diminishing thereafter) Oral: several hours (an approximately threefold weaker effect than with smoking)
Biotransformation	Rapid metabolization of THC via 11-hydroxy-THC to THC-COOH (free and as glucuronide)
Kinetic data	Half-life in absorption phase ca. 45 min Half-life in distribution phase ca. 3–4 h Half-life in terminal elimination phase: up to 24 h Distribution and elimination: Rapid uptake in blood following inhalation, followed by marked loss of concentration due to distribution in blood-rich compartments. Lipophilic cannabinoids then accumulate in fatty tissue; from that point onwards, blood levels begin to drop slowly. In a final phase, THC is once again released from depots Overall, elimination takes a polyphasic course
Detectability in blood	In occasional users, ca. 10–24 h; in heavy users, over 24 h and occasionally up to 48 h, and in individual cases with a high body mass index (BMI), even longer than 48 h. THC-COOH concentrations can provide information on the frequency of consumption
Detectability in urine	Dependent on quantity and frequency of consumption

Note: Under realistic conditions, passive smoking does not lead to the intake of forensically relevant quantities of cannabinoids. A level of 50 ng/ml THC carboxylic acid in urine has been proposed as the threshold between "active" and "passive" smoking. Flashbacks, i.e., renewed states of intoxication following a drug-free interval, have not been conclusively proven for cannabinoids. However, indications of this phenomenon can be seen in association with LSD consumption

[a]The term "dope" is also used to refer to illegal drugs in general

Table 30.17 Data on 11-OH-THC (11-hydroxy-THC)/(11-hydroxy-Δ^9-tetrahydrocannabinol)

Range of effects	Also has a psychotropic effect
Biotransformation	Effective metabolite of THC
Detectability in blood	4–20 h following one-off use, longer in the case of habituation
Detectability in urine	Several hours
Kinetic data	Half-life in terminal elimination phase: 12–18 h

Note: In addition to THC und THC-carboxylic acid, 11-hydroxy-THC is used to calculate the cannabis influence factor (CIF) which, under certain conditions, correlates to performance deficits. 11-hydroxy-THC is an important indicator for the identification of recent cannabis intake

Table 30.18 Data on THC-carboxylic acid (11-nor-9-carboxy-Δ^9-tetrahydrocannabinol)

Range of effects	No psychotropic effects
Kinetic data	Half-life: 25–40 h Half-life in terminal elimination phase: up to 6 days
Biotransformation	Inactive metabolite of THC

Table 30.18 (continued)

Detectability in blood	2–3 days following one-off intake, longer in the case of habituation ca. 3 weeks in the case of regular use
Detectability in urine	2–3 days following one-off use Up to 3 months in the case of heavy chronic use

Note: For the purposes of expert appraisals, a serum concentration of 150 ng THC-carboxylic acid/ml is set as the threshold between occasional and regular use of cannabis products. However, according to recent data, this threshold could also be lowered

Caution: Even once consumption has ended, strong variations including renewed increases in the THC-carboxylic acid concentration in urine may mimic a relapse. Remedy: divide the THC-carboxylic acid concentration by the creatinine value

Table 30.19 Data on cocaine

Street names (examples)	Coke, powder, snow, white stuff, among others Speedball (heroin)
Forms of administration	Primarily nasal or intravenous
Range of effects	Central stimulant effect Three phases of intoxication are often seen: *Euphoric stage*: strongly positive mood ("high phase" lasting minutes to hours) *Stage of decreasing euphoria*: anxiety, impaired coordination, tremors, mania, paranoia, and hallucinations *Depressive stage*: feelings of depression, including hopelessness, anxiety, marked exhaustion, extreme fatigue, as well as a strong compulsion to use cocaine again *Important*: intoxication depends strongly on the individual's personality and psychophysical status prior to intoxication
Side effects	see "Range of effects"
Usual dose	Nasal: between 20 and 100 mg and several grams per day (one line/h) Intravenous: 30–150 mg (2–5 g in severe addiction) Smoking: 50–200 mg per single dose (ca. 2/3 loss) Oral (rare): 100–300 mg per single dose
Lethal single dose	From about 1 g (in the case of tolerance, up to 5 g/day has been survived), while a dose in excess of 30 mg can be lethal in the case of idiosyncrasy
Duration of effects	Varies for the individual phases
Biotransformation	Only small amounts are directly eliminated Major metabolites include benzoylecgonine and methylecgonine (ecgonine methyl ester), as well as ecgonine and norcocaine Simultaneous alcohol consumption produces ethyl cocaine (cocaethylene)
Kinetic data	Half-life: 40–90 min
Detectability in blood	ca. 4–6 h
Detectability in urine	ca. 6–8 h

Note: Under usual storage conditions, cocaine is unstable (remedied by adding sodium fluoride)

A value determined in an unstabilized or uncooled blood sample does not correspond to the concentration at the time the blood sample was taken

Caution: Heating cocaine base produces methylecgonidine, which can then be used as a marker for smoking. It may also be produced in the heated components of the gas chromatograph (the injector)

Table 30.20 Data on benzoylecgonine

Kinetic data	Half-life: 4–7 h
Detectability in blood	A few days depending on dose
Detectability in urine	ca. 3–6 days

Table 30.21 Data on ecgonine methylester (methylecgonine)

Kinetic data	Half-life: 3–5 h
Detectability in blood	A few days depending on dose
Detectability in urine	ca. 3–6 days

Table 30.22 Data on heroin

Street names (examples)	Brown sugar, hero, H, hit, junk, powder, among others With cocaine: cocktail, happy pills, speedball With cocaine and LSD: frisco speedballs
Forms of administration	Mostly intravenous, also smoking (thus avoiding risk of infection via dirty needles and utensils!), as well as nasal ("sniffing")
Range of effects	Initially only analgesic. Repeated consumption brings euphoriant component to the fore, including balanced mood and an intensive feeling of happiness
Side effects	Impaired cognitive ability and dulled awareness or apathy
Usual dose	Intravenous: 50–250 mg (several doses in the case of dependence) Smoking: significantly higher dose due to loss
Lethal single dose	Minimum 200 mg (higher doses in the case of tolerance)
Duration of effects	Heroin is more lipophilic than morphine and breaches the blood–brain barrier more rapidly (hence "flush" or "kick")
Biotransformation	Small quantities (0.1 %) are eliminated directly, major metabolites are 6-monoacetylmorphin (6-MAM) and morphine, which is metabolized to morphine-3-, morphine-6-, and morphine-3,6-glucuronide
Kinetic data	Half-life: 2–10 min (from heroin to 6-MAM) Half-life: ca. 40 min (from 6-MAM to free morphine) Half-life: 1–4 h (morphine)
Detectability in blood	Several hours
Detectability in urine	2–3 days

Note: Important marker function—6-MAM is evidence of heroin use. Repeated use of heroin and other opiates leads to psychological and physical dependence with severe withdrawal symptoms (e.g., diffuse pain, hypertension, hyperthermia, hyperglycemia, tachycardia, tachypnea, cramps, and shock) which, if left untreated, can lead to death as a result of circulatory failure. Miosis, coma, and respiratory depression are typical of opiate intoxication

Table 30.23 Data on morphine

Street names (examples)	"Base," among others
Forms of application	Primarily intramuscular, subcutaneous, intravenous, oral
Range of effects	Analgesic, central depressant, sedative, hypnotic, hypoventilatory, and antitussive
Side effects	Risk of habituation (tolerance development); unfitness to drive during withdrawal phase
Usual dose	5–20 mg (several daily doses in the case of dependence)
Lethal single dose	Minimum 120 mg (higher doses may be survived in the case of tolerance)
Duration of effects	4–5 h following therapeutic doses
Biotransformation	ca. 10 % as free morphine ca. 75 % as inactive morphine-3-glucuronide ca. 5 % as low-action normorphine Less as active morphine-6-glucuronide Renal elimination
Kinetic data	Half-life: 1–4 h (free morphine)
Detectability in blood	Several hours to a few days (strongly dose-dependent)
Detectability in urine	2–3 days

Note: Morphine can enter the organism by means of poppy seed consumption (e.g., in pastries). Detection of 6-MAM, however, is evidence of heroin use

Table 30.24 Data on codeine

Street names (examples)	Codies, among others
Forms of administration	Primarily oral, also intramuscular and rectal
Range of effects	Antitussive effect. Analgesic effect weaker than with morphine
Side effects	Risk of habituation (tolerance development); unfitness to drive during withdrawal phase
Usual dose	Oral 30–60 mg (several daily doses in the case of dependence)
Lethal single dose	Minimum 800 mg (higher doses may be survived in the case of tolerance)
Duration of effects	Several hours

Table 30.24 (continued)

Biotransformation	Over 95 % of a single dose is eliminated via the kidneys within 48 h, of which: ca. 50 % as codeine, ca. 15 % as norcodeine ca. 10 % as morphine Is eliminated in the first 24 h, all free or mainly conjugated
Kinetic data	Half-life: 2–4 h
Detectability in blood	Several hours to a few days (strongly dose-dependent)
Detectability in urine	ca. 2–4 days (also strongly dose-dependent)

Note: Codeine is not a metabolite of morphine or heroin. If no codeine product has been ingested, the detection of codeine in urine can be attributed to codeine already present in drugs as an "impurity." At the end of the elimination phase, more morphine than codeine may be detected in urine; hence the risk of incorrectly concluding that heroin or morphine has been used. Codeine can enter the organism by means of poppy seed consumption (e.g., in pastries). Detection of 6-MAM, however, is evidence of heroin use

Table 30.25 Data on dihydrocodeine

Street name (examples)	"Remmis," among others
Forms of administration	Primarily oral, also intravascular, intramuscular, and rectal
Range of effects	Antitussive effect, analgesic effect weaker than with morphine
Side effects	Risk of habituation (tolerance development), unfitness to drive during withdrawal phase
Usual dose	Oral 60–120 mg (several daily doses in the case of dependence)
Lethal single dose	Comparable with codeine
Duration of effects	Several hours
Biotransformation	Comparable with codeine
Kinetic data	Half-life: 3–5 h
Detectability in blood	Several hours to a few days (strongly dose-dependent)
Detectability in urine	ca. 2–3 days (also strongly dose-dependent)

riders of the respective narcotic regulations. Until such time, in Germany at least, these substances are subject to German drug law.

Cannabis. This term is used to describe all psychoactive components of or products from the plant *Cannabis sativa*. Hashish is composed mainly of compressed stalked resin glands, as well as parts of the leaf and flower. It is often compressed to sheets, which contain approximately 2–10 % of the main active substance Δ^9-tetrahydrocannabinol (THC). A THC content of up to around 20 % can be achieved by cultivating so-called turbo hashish. Marihuana refers to air-dried leaves, flowers, and stalks with an average THC content of 4 %. Hashish oil is a concentrate obtained by solvent extraction with a THC content of up to 30 % (rarely, as high as 60 %). Other psychoactive contents include cannabidiol (CBD) and cannabinol (CBN). The collective term "cannabinoids" describes the structurally similar compounds found in cannabis, over 60 of which are now known. The substances most relevant in terms of effect and analysis [THC; 11-hydroxy-THC or 11-hydroxy-Δ^9-tetrahydrocannabinol (11-OH-THC); and 11-nor-9-carboxy-Δ^9-tetrahydrocannabinol (THC-carboxylic acid)] are shown in Tables 30.16, 30.17, and 30.18 (see also Fig. 30.9).

Cocaine. Cocaine is obtained via extraction from the leaves of the coca plant, *Erythroxylum coca*. Commercial forms and their usage include:

- *Cocaine hydrochloride* (cocaine HCl): While freebase cocaine (cocaine base) is pharmacodynamically the most effective form, it is unstable. Therefore, the significantly more stable salt form (e.g., cocaine hydrochloride) is used for the purposes of transportation and storage; the freebase is then "released" by the end user by the process of "freebasing."
- *Freebase*: "Freebasing" involves dissolving the cocaine salt in an alkaline solution and extracting the freebase using a suitable solvent that, when vaporized, leaves only freebase material, which is usually consumed immediately.
- *Crack*: Freebase cocaine is also the active substance in "crack," which is produced by heating the salt with an alkaline material (e.g., baking soda) and small amounts of water. This form gets its name from the "cracking" sound often heard during this process.

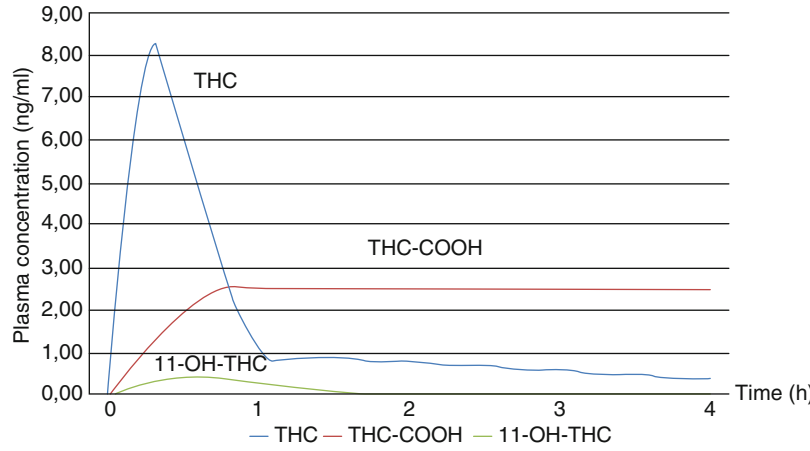

Fig. 30.9 Temporal course of the blood concentration–time curve (blood) for THC, 11-OH-THC, and THC-carboxylic acid after smoking a one-off joint

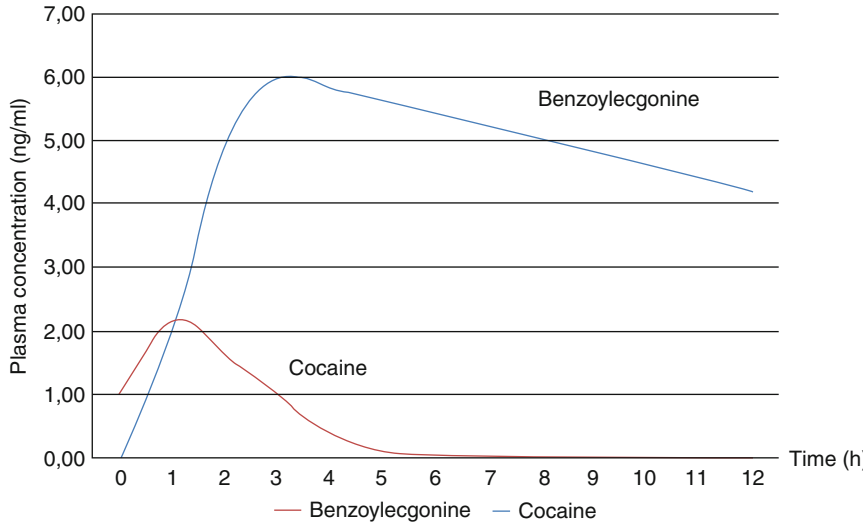

Fig. 30.10 Temporal course of the blood concentration–time curve for cocaine and benzoylecgonine following intranasal administration of cocaine

The most important data in terms of effect and analysis are given in Tables 30.19, 30.20, and 30.21 (see also Fig. 30.10).

Opiates. Opiates are found in opium, which is obtained from the opium poppy (*Papaver somniferum*). The major active substances here include morphine, codeine, thebaine, noscapine, papaverine, and narceine. The term "opioid" refers to all substances with morphine-like properties that attack opioid receptors. In addition to opiates, these include the body's own opioids, such as endorphins and enkephalins, as well as numerous semisynthetic and fully synthetic substances.

Caution: Due to their opiate-like action, substances which are completely different in structure from a pharmacological perspective are included in the opiates group (e.g., dextropropoxyphene, levomethadone, nefopam, pentazocine, pethidine, tilidine, and tramadol, among others) and classified as opiates. These substances and their metabolites are *not* detected in opiate immunoassays.

Opiates are used in a therapeutic setting as strong analgesic agents, while in the case of abuse, they are used primarily for their euphoriant effect. Caution is often exercised when prescribing opiates for pain patients due to the fear of addiction development. Experience with the medical use of morphine has shown, however, that discontinuing its use following legitimate pain therapy is far easier than withdrawal following purely hedonistic use. Heroin (diacetylmorphine) obtained from morphine via acetylation is particularly addictive.

The most important data in terms of effect and analysis are given in Tables 30.22, 30.23, 30.24 and 30.25 (see also Fig. 30.11).

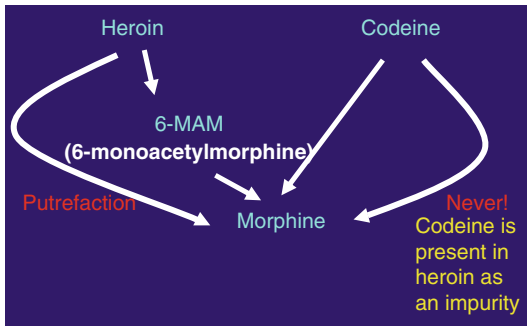

Fig. 30.11 Overview of morphine formation

6-MAM is evidence of heroin use. Morphine, on the other hand, can be formed by codeine, heroin, and other opiates, such as pholcodine. However, non-detection of 6-MAM does not exclude heroin use, since 6-MAM can be rapidly eliminated or destroyed by microbial factors, such as autolysis. Codeine, on the other hand, is not a metabolite of morphine, but is often present in street heroin as a by-product or impurity (Fig. 30.11). To these should be added a number of other substances that are used as extenders in heroin and injected intravenously. Frequently used additives/diluents to adulterate street drugs include quinine, mannitol, lactose, glucose, procaine, caffeine, inositol, lidocaine, starches, methapyrilene, sucrose, acetyl procaine, dextrose, scopolamine, paracetamol, phenobarbital, and methaqualone. Following intravenous drug use of many years' standing and embolic spread to the terminal vessels of the lungs, nondegradable crystalline particles lead to abundant foreign body granulomas (pulmonary granulomatosis) with increasing hypertension in the pulmonary circulation, so-called junkie pneumopathy (Fig. 30.12; see also Fig. 7.5). Severe toxic pulmonary edema, with lungs weighing >1,000 g/lung and hemorrhagic foam in the airways, is

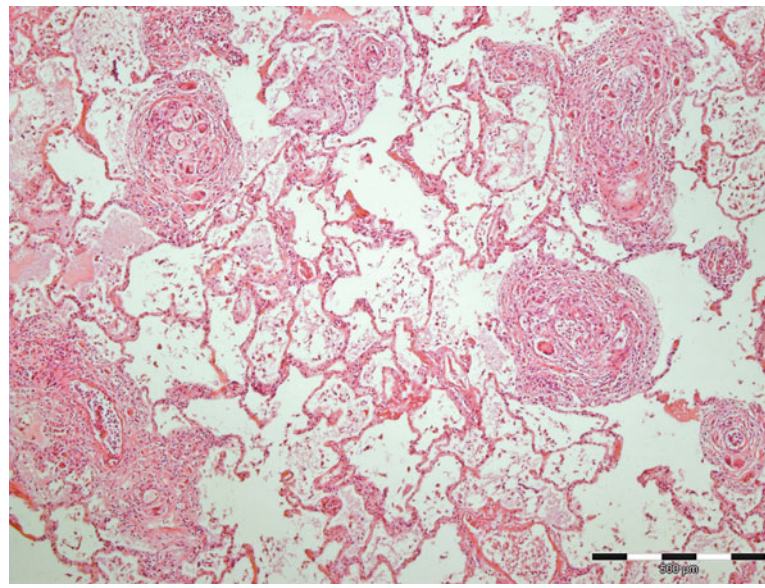

Fig. 30.12 Loosely spread pulmonary granulomas in the case of junkie pneumopathy in a 36-year-old male (From Dettmeyer (2011))

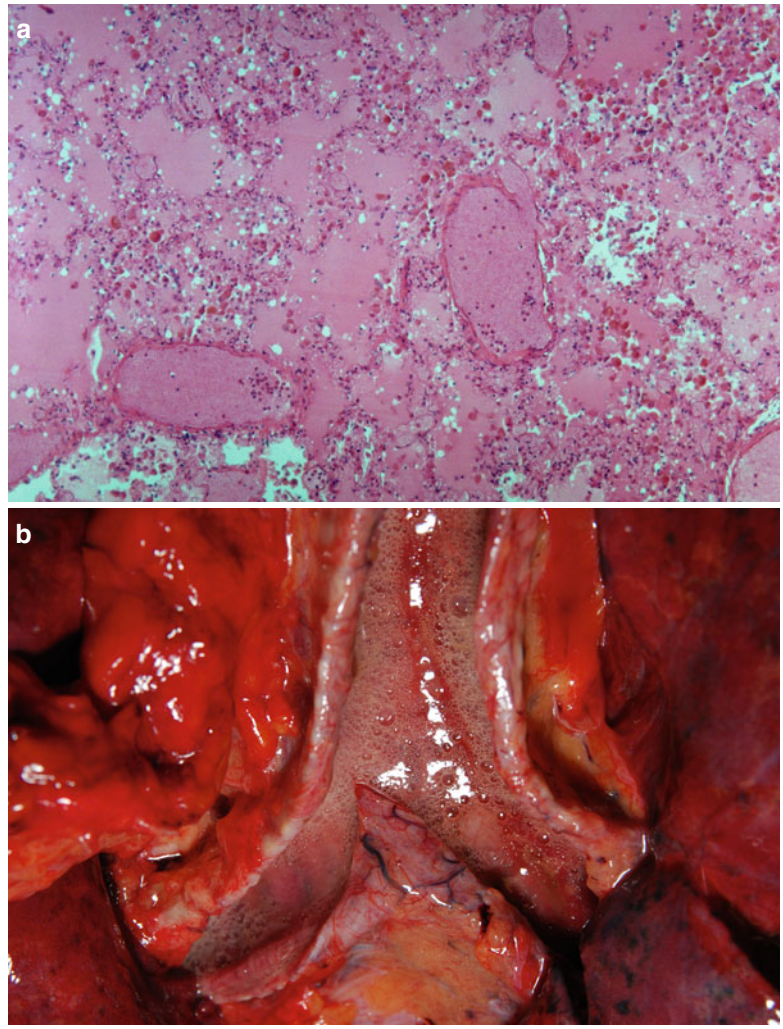

Fig. 30.13 (**a**) Lethal heroin/morphine intoxication with toxic pulmonary edema seen microscopically and (**b**) macroscopic hemorrhagic foam in the airways

often seen during the agonal phase in the case of heroin/morphine intoxication (Fig. 30.13).

30.2.15 Other Narcotics

LSD. Lysergide (LSD; (6a*R*,9*R*)-*N*,*N*-*diethyl*-7-methyl-4,6,6a,7,8,9-hexahydroindolo-[4,3-*fg*] quinoline- 9-carboxamide) is obtained from indole alkaloids of ergot (*Claviceps purpurea*).

Forms: The substance is a white or beige crystalline powder. In contrast to most other drugs, even very small quantities (less than 0.3 mg) of LSD can be effective. Since the substance can only be precisely dosed using special scales when in powder form, it is common practice to dissolve a weighable amount in a volume of liquid, which can then be dosed relatively accurately by counting drops (e.g., on a sugar cube) or by applying the liquid to plotting paper from which sections corresponding to an individual dose are then cut. The most important data in terms of effect and analysis are given in Table 30.26.

Spice. Although initially marketed legally as incense, spice has been increasingly abused in Germany as a cannabis-like intoxicant since late 2008. The main active substance responsible for intoxication is a modification of the synthetic cannabinoid CP-47,497. Spice also contains the synthetic cannabinoid mimetic JWH-018 (1-pentyl-3-naphthoylindol). These substances bind to the cannabinoid receptors in the brain,

Table 30.26 Important data on lysergide (LSD)

Street names (examples)	Ace, acid, crackers, D, dots, frisco speedballs With cocaine and heroin: ghost, hawk, L, morning glory, pink dots, pink Jesus, purple haze, purple wedges, sunshine acid, the bast, the chief, trips, 25+, yellow submarine
Forms of administration	Primarily oral
Range of effects	Hallucinogenic
Side effects	Hyperthermia, sweating, vegetative symptoms such as tachycardia and bradycardia, depression, fatigue, exhaustion, irritability, followed by lethargy
Usual dose	0.02–0.3 mg
Duration of effects	Onset of effects after 15–45 min Effects can last hours to days (flashbacks)
Biotransformation	Extensive metabolization by *N*-demethylation, *N*-deethylation, and hydroxylation
Kinetic data	Elimination half-life: ca. 3–4 h
Detectability in blood	Strongly dose-dependent (maximum 12 h)
Detectability in urine	Strongly dose-dependent (ca. 1–2 days)

Note: Particular relevance in traffic medicine: In view of its extreme hallucinogenic effects (shifts in perception and sense of time, hallucinations, and tendencies towards depersonalization) and the risk of flashbacks, an individual under the influence of LSD is considered categorically unfit to drive. Significant performance deficits may also be seen during the withdrawal phase
Caution: False-positive LSD immunoassays following ingestion of ambroxol and sertraline are possible

triggering a state of intoxication. Their pharmacodynamic effects are significantly greater than those of THC. Since they bear no resemblance to normal cannabinoids, current immunochemical drug tests are usually negative.

Desomorphine ("*Crocodile*"). Desomorphine, also known as "crocodile" or "croc" for short on the drug scene, is a highly potent (and also highly addictive) opioid that was first made in the USA in 1932. Due to its illegal synthesis from codeine, iodine, and red phosphor in a process similar to the production of methamphetamine using a pseudoephedrine base, the end product is impure and contains many highly toxic by-products. On injection, these by-products can result in severe tissue damage, vein inflammation, necrosis, gangrene, or even organ failure. Irreversible damage, such as neurological, renal, or vascular lesions, may develop immediately on first use. According to statements made by the Yekaterinburg anti-drug initiative, "Town without Drugs," desomorphine is so aggressively toxic that the average life expectancy among users once they begin regular intravenous use is not more than 1 year. The drug is referred to as "crocodile" in Russia, since it damages the body from within and often leaves green skin discoloration at the injection site. Known as "the poor man's drug," crocodile is widely used in Russia, although cases in other countries have been reported.

"*Bath Salts*" *Drugs*. The collective term "bath salts drugs" refers to a variety of substances that are often also illegally marketed as mixtures. The major component is usually mephedrone (4-methylmethcathinone (4-MMC)), in addition to other active substances, such as ethylcathinone, flephedrone, methylone, methylenedioxypyrovalerone (MDPV), or also lidocaine. Illegal trading is carried out mainly via the Internet, although in the USA bath salts drugs are said to be available in supermarkets and gas stations. The substance is consumed either nasally or orally, producing a range of effects including euphoria, alertness, mobilization of energy reserves, and appetite suppression. Patterns of strong entactogenic effects are primarily seen. The following side effects have been described: skin irritation and burning, in particular damage to (including perforation of) the nasal septum, severe perspiration, tachycardia, strongly impaired cognitive function including amnesia, and cravings.

Khat. A "natural amphetamine," khat is a herbal drug with cathinone and cathine as its main active agents. The khat plant is grown in the south of the Arabian Peninsula and in East and South Africa. The main active agents cause a mild psychological dependence. Psychotic symptoms have been described following extensive long-term use. Khat is a socially accepted

drug in its traditional countries of origin. Although primarily the leaves and young branches are chewed, khat may also be mixed with tobacco and smoked or made into a tea. Khat is best consumed fresh, since it loses its hallucinogenic effect during long storage. Given the general narcotic drug situation and its limited stability, khat plays only a minor role in many countries.

Psilocybin (*Magic*) *Mushrooms*. Psilocybin mushrooms contain the hallucinogenic compound psilocybin, as well as psilocin in smaller quantities. Both substances are tryptamine derivates related to the likewise hallucinogenic LSD. Psilocybin-/psilocin-containing types of the *Psilocybe* species are by far the most frequently used hallucinogenic fungi. A dose of as little as 3–6 mg can produce early changes in perception; pronounced euphoric states, as well as visual and auditory hallucinations, are also described. Experiments on the drug scene involving poisonous mushrooms (active substance: ibotenic acid) are reported. Like khat, psilocybin mushrooms also play a minor role in many countries as a narcotic drug.

30.2.16 Medicinal Drugs and Other Substances of Particular Relevance in Forensic Toxicology and Traffic Medicine

The present section deals with substances that are also, but not only, of particular relevance in traffic medicine. Basically, any substance can be of forensic toxicological relevance, whereby dose and mechanism of action play a predominant role. The following substances or groups of substances are those most frequently seen at the top of statistics on medications in terms of intoxication incidence:

- Psychotropic drugs
- Analgesics, antirheumatic agents
- Hypnotic agents, sedatives
- Antiepileptic agents
- Beta-receptor blockers, calcium antagonists, ACE inhibitors

However, there are numerous substances which, at first glance, would not be considered relevant in terms of traffic medicine, not least because special warnings relating to impaired ability to react are often not given on the patient information leaflet.

Case Study

Laxatives as the cause of a traffic accident

Following a traffic accident, the driver reported how he had suffered dizziness immediately before departing from his lane. No alcohol, drugs, or other substances of particular relevance in traffic medicine (e.g., psychotropic drugs) could be detected. However, the driver admitted chronic laxative use (bisacodyl, among others), which had obviously led to hypokalemia, significantly impaired circulation, and possibly also a psychosyndrome.

In the case of overdose, individual side effects can often come more sharply into focus than the main intended therapeutic effect and, when interactions with other medications are also present, may take a critical course. Protein binding of medications is an important factor. Competition for the same binding site can lead to suppression of a substance from protein binding and thus to increased blood concentrations of the suppressed drug. Antidiabetic agents, which are highly protein-bound, are an important example of this: simultaneous administration of sulfonamide or salicylates causes the antidiabetic drug to be released and, as a result of the increased blood concentration, leads to increased insulin secretion. This results in hypoglycemia, which can significantly reduce an individual's fitness to drive. The biotransformation of a medication can also be affected by interactions with other drugs, causing inhibition in particular. In addition, chronobiological effects or circadian rhythms may impair the effectiveness of medications and other substances, with similar effects on fitness to

drive. In this context, shift work or travelling across several time zones (i.e., east to west or west to east with the resultant jet lag) can be relevant factors.

Vehicle drivers and individuals operating machinery should always read the *warnings* given about medications in the patient information leaflet and on packaging. Moreover, the prescribing physician is obliged to advise and inform patients about the effects of a medication, how it should be taken and for how long, frequent side effects, as well as measures to take in the event of complications. Consultations should be documented by the consulting physician and, where necessary, supplemented with written information.

The following medications and substances are of particular relevance in forensic toxicology and traffic medicine:

Analgesics/Antirheumatic Agents. A distinction is made primarily between narco-analgesics (morphine, morphine derivates, and other narco-analgesics), other centrally acting analgesics, and analgesics/antirheumatic agents. According to the WHO classification, narco-analgesics should only be used in cases of severe pain. These are followed by extremely potent analgesic agents, such as dextropropoxyphene, dihydrocodeine, tilidine, and tramadol, which in turn are followed by the substances often referred to as "small analgesics," such as paracetamol, metamizole, and salicylates (but which are nonetheless of no little relevance in forensic toxicology and traffic medicine, since they are frequently found in combination products, particularly with caffeine and codeine). Discontinuing this kind of product can lead to withdrawal symptoms, such as headaches, which are then self-treated using increased doses, thus creating a vicious circle. All substances cause significant performance deficits, particularly at the start of therapy; thus detailed instructions should be given by the treating physician (or pharmacist in the case of nonprescription drugs).

Antidiabetic Agents. Relevant substances here include insulin, glibenclamide, glimepiride, metformin, and acarbose. The risk of overdosage-related hypoglycemia and underdosage-related hyperglycemia is of particular relevance in traffic medicine. Insulin, particularly among insulin-dependent diabetics, is relatively frequently used in suicide attempts, while cases of its use in homicide have also been reported. Determining glucose levels, however, is inconclusive due to rapid postmortem elimination. Where possible, detection at the injection site or for C-peptide should be carried out. The risk of occasionally life-threatening lactic acidosis following metformin administration is also of toxicological and traffic medicine relevance.

Caution: Blood glucose concentrations measured in postmortem or stored blood samples should be interpreted with great caution due to autolytic or enzymatic factors.

Antiepileptic Agents. Even after seizures have ceased, this disorder may still cause impaired performance. Particularly careful dosing of medication is required in epileptics participating in road traffic. Both overdosage and underdosage represent risk factors in this context, hence the particularly important role played by patient compliance.

Beta-Receptor and Calcium Channel Blockers. In accordance with numerous guidelines, these agents are used either as a monotherapy (ß-blockers, diuretics, calcium antagonists, ACE inhibitors, or α_1-blockers) or in a dual or triple combination of the substances mentioned here. These agents can lower blood pressure and cause sedation as well as possible orthostatic side effects. Detailed patient information about risks, particularly at the start of therapy, is essential.

Hypnotic Agents/Sedatives. Widely used in the past, barbiturates and bromcarbamides have lost their significance in Europe as sleeping aids and sedatives. Phenobarbital, on the other hand, is an important antiepileptic agent.

The following sections discuss the benzodiazepine class of medications in more detail, since

they play a particularly important role in forensic toxicology and traffic medicine.

Benzodiazepines. Benzodiazepines remain among the most widely used medications, and their detection is a routine procedure in any chemical toxicology laboratory. Although their therapeutic range is broad, i.e., lethal mono-intoxications with classic benzodiazepines are rarely seen, benzodiazepines as an interaction partner in mixed intoxications (e.g., with alcohol or other centrally acting foreign substances) are feared. With the exception of cases of acute poisoning, detection plays a significant role in the monitoring of addicts as well as in the field of traffic medicine. Some benzodiazepines are used on the drug scene as replacement or supplementary drugs, i.e., "downers"; the principal drug to be mentioned in this context would be flunitrazepam (e.g., Rohypnol®).

The range of effects seen with benzodiazepines include anxiolytic (antianxiety), sedative (calming), hypnotic (sleep inducing and sleep sustaining), anticonvulsive (antispasmodic), and central muscle relaxant components, among others. These effects can produce performance deficits relevant in traffic medicine; for example, anxiolysis is associated with disinhibition and increased risk-taking behavior, while the sedative and hypnotic effects lead to reduced concentration and impaired ability to react. Finally, muscle relaxant effects may lead to motor function impairment. Personality changes have been reported following long-term abuse.

Equivalent Doses. The effects of benzodiazepines vary in degree; the *equianalgesic potency conversion chart* given in Table 30.27 can be used for the purposes of comparison.

Dosages: Individual therapeutic doses depend on receptor affinity.

Examples: 0.25 mg, alprazolam, triazolam; 0.5–2 mg, lorazepam, lormetazepam; 2 mg, flunitrazepam; 2–10 mg, diazepam; 5 mg, nitrazepam; 6 mg, bromazepam; 7.5 mg, midazolam; 10 mg, chlordiazepoxide; 10 mg, medazepam; 10–20 mg, clobazam, prazepam; 10–50 mg, oxazepam; 30 mg, flurazepam; 50 mg, clorazepate.

Note: Extreme overdoses may be tolerated once tolerance has developed!

Table 30.27 Data on 1.4- and 1.5-benzodiazepines

Benzodiazepine (e.g., found in:)	Equivalent dose to 10 mg diazepam (mg)	Elimination half-life $t_{1/2el}$ (h)
Alprazolam (e.g., Tafil®)	1	10–25
Bromazepam (e.g., Lexotanil®)	3	8–20
Chlordiazepoxide (e.g., Librium®)	20	5–30
Clobazam (e.g., Frisium®)	20	10–30
Clonazepam (e.g., Rivotril®)	2	12–60
Clorazepate (e.g., Tranxilium®)	20	2–5[a]
Diazepam (e.g., Valium®)	10	21–48[a]
Flunitrazepam (e.g., Rohypnol®)	2	9–25
Flurazepam (e.g., Dalmadorm®)	30	1–3[a]
Lorazepam (e.g., Tavor®)	2	10–20
Lormetazepam (e.g., Noctamid®)	1	8–17
Medazepam (e.g., Rudotel®)	20	1–2[a]
Midazolam (e.g., Dormicum®)	5	1–4
Nitrazepam (e.g., Mogadon®)	5	15–40
Nordazepam (e.g., Tranxilium N®)	20	50–90
Oxazepam (e.g., Adumbran®)	40	4–15
Prazepam (e.g., Demetrin®)	20	1–3[a]
Triazolam (e.g., Halcion®)	1	2–5

[a]The half-life of the metabolite primarily responsible for effects can be significantly higher (40–90 h or more in the case of cumulation)

Biotransformation and Kinetics: During phase I, benzodiazepines may be either mildly (e.g., oxazepam) or intensively (e.g., flurazepam) metabolized, although the unchanged proportion excreted in urine can vary significantly. The fact that identical metabolites can be formed from different substances is relevant to the interpretation of findings, e.g., nordazepam from chlordiazepoxide, clorazepate, diazepam, ketazolam, medazepam,

and prazepam. Elimination half-lives also depend heavily on chemical structure (see Table 30.27).

The following effects, which are closely linked to elimination half-life, are of particular forensic relevance:

Benzodiazepines with metabolites that have long elimination half-lives (e.g., clorazepate, diazepam, flurazepam, medazepam, and prazepam) tend to accumulate. This means an unequal balance between quantities ingested and quantities eliminated, leading to significantly increased levels of the substances when elimination—as determined by long half-lives—is slow. Drug concentrations of this magnitude can lead to severe impairments. On the other hand, benzodiazepines with extremely short half-lives (e.g., triazolam) surge away from the receptor site particularly rapidly, often causing so-called rebound phenomena, which may manifest as, e.g., agitation, anxiety, increased irritability, and paranoid states and frequently lead to renewed use of the drug.

Detectability in Urine:

- Classic benzodiazepines (e.g., diazepam and oxazepam): up to 3 days following therapeutic dosage; up to 1 week using sensitive detection methods
- Benzodiazepines with metabolites that have a long half-life (e.g., flurazepam): often several days to weeks
- Benzodiazepines with a short half-life (e.g., triazolam): only a few hours following ingestion

Detectability in Blood: Between several hours and a few days (depending strongly on dose and method).

Zaleplon, *Zolpidem*, *and Zopiclone* (*the Z Drugs*). Zaleplon (Sonata®), a pyrazolopyrimidine; zolpidem (Bikalm®, Stilnox®), an imidazopyrimidine; and zopiclone (Optidorm®, Somnosan®, Ximovan®), a cyclopyrrolone derivative, have the same mechanism of action as benzodiazepines in that they all act as agonists at the same subunit of the $GABA_A$ receptor–chloride channel complex. The difference in their action profile compared to benzodiazepines is negligible. Although they are believed to be less addictive than benzodiazepines, this has been called into question by numerous case studies.

Local and General Anesthetics. These drugs can be divided into three main groups:

- Local anesthetics
- Inhalation anesthetics
- Intravenous anesthetics

All these substances are of fundamental interest in forensic toxicology, e.g., when providing an expert appraisal on an incident involving anesthesia. Primarily anesthesia used in the setting of outpatient treatment, following which a patient may drive home, for example, is of particular relevance in traffic medicine. Marked impairments are seen in the first 2 h and for significantly longer in the case of concomitant administration of centrally acting drugs, e.g., benzodiazepines. Patients need to be fully informed about the hazards not only of driving a vehicle, but also of moving around on foot. Ideally, patients should be advised prior to anesthesia to have an accompanying person with them.

Psychotropic Drugs. Noteworthy among this group of drugs are antidepressants, neuroleptics, and tranquilizers.

Antidepressants. The involvement of antidepressants in poisonings continues to increase. This could have to do with the now widely acknowledged fact that benzodiazepines do not possess antidepressant properties and that the use of antidepressants in some diseases is more beneficial, as reflected in the frequency with which antidepressants are prescribed. Although administration is usually oral, intramuscular or intravenous administration is also possible; dose depends strongly on the type of substance and the severity of disease. Major substances and products include amitriptyline (Amineurin®, Laroxyl®, Novoprotect®, Saroten®, Syneudon®), amitriptylinoxide (Equilibrin®), clomipramine (Anafranil®), desipramine (Pertofran®), dibenzepin (Noveril®), doxepin (Aponal®, Sinquan®), imipramine (Tofranil®), lofepramine (Gamonil®), nortriptyline (Nortrilen®), and trimipramine (Stangyl®).

Poisoning with tricyclic and structurally related antidepressants produces serious cardiovascular symptoms (marked drop in blood pressure, tachycardia, and cardiac arrhythmia), as well as hyperthermia and delirium. Cardiac

and respiratory arrest may be seen in severe cases. Impaired ability to react and disorientation are of particular relevance in traffic medicine.

Neuroleptic Agents. Poisoning with neuroleptics primarily causes somnolence, agitation, hypo- and hyperthermia, hypertension, tachycardia, and bradycardia. In addition, generalized seizures and agranulocytosis may be seen. Due to reduced attention and visual perception, impaired fitness to drive should be expected particularly at the start of therapy. However, these undesired side effects may diminish after the first or second week of therapy. Detailed patient information is of the utmost importance, whereby the underlying disease should not be lost out of focus.

Tranquilizers. See "Hypnotic Agents/Sedatives" and "Benzodiazepines" above.

In addition to the substances hitherto described, numerous substances in other fields of application can be relevant in poisonings or affect fitness to drive, including, for example:

- Antiallergic agents
- Antiarrhythmic agents
- Antihypertensive drugs
- Antihypotensive agents
- Diuretics
- Anti-addiction drugs
- Cold and flu remedies
- Cardiac drugs
- Laxatives
- Muscle relaxants
- Ophthalmic agents
- Antiparkinson agents
- Spasmolytic/anticholinergic agents
- Cytostatic agents

Major and minor side effects as well as interactions are generally listed in the patient information leaflet, which also provides information about restrictions on use, contraindications, and the risk of poisoning. There are usually also instructions and recommendations on drugs that impair fitness to drive or to operate machinery, as well as special information on other drug groups. These warnings are based on a drug's -pharmacodynamic profile, its known side effects, and the impairments observed in fitness to drive and/or operate machinery.

30.2.17 Anorganic and Organic Substances

Metal Toxins. Although anorganic substances, e.g., metal toxins or medications containing heavy metals, used to be in wide circulation, they are less relevant today in terms of acute toxicity, becoming more relevant in terms of chronic exposure. However, metal toxins should be considered in any "general unknown" search program, otherwise there is a danger that a toxin such as arsenic (*poudre de succession*, or "inheritance powder"), which has been widespread for centuries, could be overlooked. Relatives belonging to particular professional groups, such as laboratory personnel or electroplaters, have access to occasionally rare metal toxins; thus, an offender's or a victim's environment should always be taken into consideration when planning analysis concepts. Heavy metals are usually capillary and enzyme poisons, with often varying toxic reaction mechanisms.

Case Study

Attempted suicide by means of thallium ingestion

Increasing hair loss was observed in an elderly man who had already been an inpatient for 14 days. The police were informed due to the suspicion that relatives were bringing poisoned cakes into the hospital. Toxicological analysis of the foodstuff, however, was negative. On the advice of the laboratory, a search of the man's home was undertaken, where a drinking vessel was found with remnants of the thallium-containing rodenticide (rat poison) Zelio paste, which had been deliberately ingested with suicidal intent. The patient had been admitted to the hospital for acute abdomen and paresthesia, which had initially been attributed to food poisoning.

Table 30.28 Data on metal toxins

Substance	Lethal dose	Major symptoms and observations
Arsenic	0.1–0.3 g As_2O_3 (arsenite)	More toxic is the trivalent arsenic formed in the organism by pentavalent arsenic. Headaches, abdominal pain, cramps, vomiting, and diarrhea begin shortly after ingestion; death usually occurs within 24 h Chronic intoxication: polyneuropathy with muscle atrophy, hair loss, Mees' lines (grayish-white lines on fingernails and toenails) within ca. 6–8 weeks, hyperkeratosis on hands and feet
Lead	ca. 1 g	Intestinal colic with nausea and vomiting; hypothermia with decreased blood pressure; severe liver, kidney, and CNS damage; hemolytic anemia and hemoglobinuria Chronic intoxication: blue line on gums; damage to the bone marrow, CNS, kidneys, and muscles (in particular, smooth muscles of the GI tract); skin and mucosal changes
Mercury	0.2–1 g	*Important*: metallic mercury, when ingested orally, is less toxic than highly toxic mercury vapor. However, mercury(II) compounds ingested orally are more toxic than mercury(I) compounds *$HgCl_2$ (sublimate)*: strongly corrosive, causing gastroenteritis with violent vomiting and bloody diarrhea, short-term polyuria followed by anuria due to severe renal tubule damage *Chronic intoxication*: nephrotic syndrome, stomatitis with dental loosening, mercurial line on gums, mercurial tremor, psychological disorders, cachexia
Thallium	ca. 1 g	Monovalent thallium formed in the organism by trivalent thallium is toxic. Short symptom-free interval of 2–3 days with a short phase of obstipation, severe gastrointestinal disorders (gastroenteritis with vomiting, cramps, and diarrhea) Chronic intoxication: polyneuropathy, hyperesthesia, thallium encephalopathy, impaired vision, hair loss after 2–3 weeks, Mees' lines

The heavy metals listed in Table 30.28 are (or once were) of particular relevance.

Acids and Alkalis. Alkali poisoning is considerably more dangerous than acid poisoning, since tissue liquefaction prevents the development of corrosion scabs (formation of colliquative necrosis). Due to necrotized tissue, bases can penetrate to deep tissue layers. Should alkali come in contact with the eyes, complete loss of eyesight may result.

- Lethal doses of alkali: 10–15 ml 15 % solutions
- Lethal doses of acids: ca. 5 ml (concentrated H_2SO_4), ca. 15–20 ml (concentrated HCl)

Pesticides. In the narrower sense, pesticides are agents to combat pests; however, pesticides have been equated to insecticides to a certain extent. Pesticides in the broader sense include pest control agents as well as all plant protection products. This broader term has meanwhile been adopted by the US Environmental Protection Agency. Thanks to better information, more restrictions on commercial practices, as well as greater environmental awareness, pesticides now play a much smaller role than formerly. Nevertheless, they include the following major substance groups:

Organophosphates (*e.g.*, *Parathion*, *E 605*)

- Mechanism of action: Irreversible acetylcholinesterase blockade, leading to an accumulation of acetylcholine and continuous stimulation of the vegetative nervous system.
- Clinical symptoms: Salivation, miosis, cramps, bradycardia.
- Death is usually caused by apnea. High doses cause rapid onset of action, while borderline lethal doses (300–500 mg) often cause an agonal phase lasting several hours during which the individual retains consciousness.
- Antidote: High-dose atropine.

Carbamates

- Mechanism of action: Reversible acetylcholinesterase blockade (see above, "Organophosphates")

Organochlorine Insecticides and Pyrethroids

- Mechanism of action: Hyperexcitability of the CNS due to sodium channels being kept open.

- Clinical symptoms: Agitation, mydriasis, paresthesia, tremors, disorientation, coma, cerebral seizures, apnea.
- Special "intelligent" substances inhibit chitin biosynthesis or delay pupation or adult emergence (juvenile hormone analogs) of insects.

Household Chemicals, Alcohol, and Tobacco. Annual reports from poison emergency centers indicate that easily accessible cleaning and care products, alcohol and tobacco, and plant toxins are frequently involved in pediatric cases of poisoning at home.

- Eating as little as half a cigarette can be lethal for an infant.
- Two tablespoons of spirit of Melissa (79 % alcohol by volume), mixed, for example, with juice, can produce a BAC of almost 2‰ in a child weighing 10 kg.
- Lamp oils are particularly attractive to children due to their color and often pleasant-smelling aroma. (Caution: thinned lamp oil may enter the lungs and cause severe infections.)
- "Attractive" household chemicals, such as washing-up liquid with a lemon on the label or caustic cleaning agents in duck-shaped bottles.
- The household medicine cabinet often contains medications that are particularly toxic for children or "attractive alternatives" for addicts, e.g., codeine-containing cough syrup and psychotropic drugs.

As a basic universal rule, children are curious about anything they find in the home, the garden, or in their wider surroundings, e.g., syringes on the playground.

Plant and Animal Toxins. Many ornamental plants contain toxic substances that can cause poisoning, particularly in infants. Moreover, different parts of plants can have different degrees of toxicity; examples are given in Table 30.29.

Table 30.29 Plant toxins

Plants	Particularly toxic parts	Toxin(s)
Yew (*Taxus baccata*)	Needles	Taxine
Aconitum	Leaves and seeds	Aconitine
Angel's trumpet (*Brugmansia*)	All parts	Belladonna alkaloids
Green beans (*Phaseolus vulgaris*)	Raw beans	Phasin
Laburnum	Seeds	Cytisine
Autumn crocus (*Colchicum autumnale*)	Seeds	Colchicine
Lily of the valley[a] (*Convallaria majalis*)	All parts	Cardiac glycosides
Oleander (*Nerium oleander*)	All parts	Cardiac glycosides
Spindle tree (*Euonymus europeaus*)	Fruits	Cardiac glycosides
Winter daphne (*Daphne odora*)	Flower and berries	Diterpenoids
Thorn apple (*Datura*)	Seeds	Belladonna alkaloids
Belladonna (*Atropa belladonna*)	Berries	Belladonna alkaloids

[a]Often confused with bear's garlic (*Allium ursinum*)

Case Study

Severe intoxication with angel's trumpet seeds

A young man recounted to his friends how he had experienced a pleasant "high" after ingesting a number of angel's trumpet seeds. However, he forgot to mention that he had taken the seeds from a plant on the north side of the house; his friends then collected seeds from a potted plant on the south side, which, due to its optimal position in the sun, demonstrated significantly increased levels of the substances atropine, hyoscyamine, and scopolamine. The young man's friends were subsequently taken to the hospital with acute poisoning.

Snake poisons play only a secondary role in Europe compared to tropical countries. European venomous snakes, including the adder, belong to the *Viperidae* family. Against this background, the risk of anaphylactic reactions to snake venom serum is greater than the risk of actual poisoning. The risk of poisoning by exotic snakes and other animals kept as pets—and which occasionally escape—is significantly greater.

Table 30.30 Stages of carbon monoxide poisoning

COHb concentration	Symptoms
Up to 10 %	No significant symptoms (smoker)
10–15 %	Possible shortness of breath on physical exertion (heavy smoker)
15–25 %	Usually no effects when resting, but shortness of breath on physical exertion
25–35 %	Headache, dizziness, vomiting, impaired judgement, impaired vision
35–45 %	Confusion, signs of paralysis, syncope on mild exertion
45–55 %	Severely impaired consciousness or loss of consciousness, collapse, life-threatening situation after prolonged exposure
55–65 %	Cramps, apnea
From ca. 65 %	Imminent threat of death

30.2.18 Gases, Solvents, and Industrial Chemicals

Carbon Monoxide (*CO*). CO poisoning is one of the most common causes of lethal poisoning. Since CO has a between 200- and 300-fold greater affinity for hemoglobin than oxygen, it accumulates in blood even at low ambient air concentrations, leading to anoxic asphyxiation (Table 30.30). Sources of poisoning include inhalation of exhaust fumes and incomplete combustion of gases, such as natural gas, which does not primarily contain CO. The fact that CO is a colorless and odorless gas that can permeate through ceilings and walls makes it particularly dangerous. Even exhaust fumes from vehicles fitted with catalytic converters contain CO.

Autopsy Findings Following CO Intoxication: Light red livor mortis (which, however, can also be attributed to cold), liquid cherry red blood, salmon red coloration of the musculature, light red conjunctivae and fingernail beds, and cerebral edema. Increased COHb and possibly also cyanide concentrations, as well as inhalation or swallowing of soot, generally indicate that an individual was *alive* at the time of the fire. The absence of these signs supports the suspicion of death prior to the outbreak of fire and thus also of criminal activity (removal of a dead body). However, anoxia may also be the cause of death, for example, in cases where all the oxygen in a car is used up by a fire.

Caution: CO poisoning needs to be excluded in all cases of unexplained death, particularly in closed rooms (e.g., bathroom, kitchen, garage, car, lorry, tent) heated by an open flame, such as a fireplace or continuous-flow water heater, not least for the purposes of self-protection and the protection of third parties.

Carbon Dioxide (CO_2). Since carbon dioxide is heavier than air, it can collect in fermentation cellars, wells, and silos to an extent where it poses an asphyxiation hazard.

Symptoms: Initially hyperventilation. At a concentration of 8–10 %: headache, dizziness, respiratory acidosis, cramps, and coma; concentrations over 20 % can be lethal.

When recovering a cadaver, always consider the safety of helpers!

Hydrogen Sulfide (H_2S). H_2S is often found in sewers and cesspools. At low concentrations, H_2S is easy to identify by its odor of bad eggs; higher concentrations, however, cause olfactory paralysis.

Hydrogen sulfide is more toxic than hydrogen cyanide!

Hydrogen Cyanide (*Prussic Acid*). Hydrogen cyanide (HCN) is one of the most potent poisons known to man. It is often used for suicides (as recommended by the German Society for Humane Death, *DGHS*), as well as for homicides. Additionally, given that as few as 10 bitter almond kernels can be lethal, accidental poisoning in children is observed. Cyanide salts (e.g., potassium cyanide, KCN) react with stomach acids to form HCN, which rapidly blocks the cell respiratory enzyme cytochrome peroxidase (internal asphyxiation, possible sudden death). Anacidity of the stomach contents often leads to a protracted course as well as chemical burns via hydrolysis to potassium hydroxide solution. Cyanide is also produced by the burning of plastics, wool, feathers, as well as many other nitrogenous compounds. Thus cyanide is often additionally detected in CO poisoning. Approximately half the population of Germany, for example, is unable to smell cyanide due to a genetic predisposition.

30.2.19 Organic Solvents

Aliphatic Compounds. These compounds, which are found in petrol, petroleum, and heating oils, are relatively nontoxic. However, due to their lipophilic properties, they are able to breach the blood–brain barrier. Acute poisoning causes agitation, drowsiness, and coma. Aspiration can additionally lead to hemorrhagic pneumonia and pleurisy. Chronic poisoning is frequently seen in "sniffers," as a result of which there is a danger of peripheral neuropathy and damage to the kidneys in particular (glomerulopathies).

Aromatic Compounds. Foremost among these is benzene, which has now largely been replaced as a solvent by the less toxic toluol. However, it is still found in fuels due to its properties as an antiknock agent. A lethal dose is around 25 ml. Acute poisoning is characterized by agitation, tremors, cramps, and cardiac arrhythmias, followed by respiratory paralysis and circulatory failure. Chronic benzene poisoning causes bone marrow damage resulting in a reduction in erythrocytes as well as aplastic anemia and leukemia. *Toluol* and other alkyl homologues of benzene can be metabolized by side-chain oxidation and eliminated as conjugates, making them less toxic.

Halogenated Compounds. The effect of these compounds is greater than that of comparable nonhalogenated compounds. There are two principal phases of acute poisoning: the first resembles the narcotic picture seen with nonhalogenated compounds. This is followed by a second phase which can result in toxic hepatitis with kidney damage, yellow liver atrophy, as well as uremic coma. All halogenated aliphatic compounds sensitize the myocardium to catecholamines, thereby triggering life-threatening cardiac arrhythmias. In the case of chronic poisoning, kidney, liver, and bone marrow lesions of varying degree, as well as central nervous and carcinogenic effects, are primarily seen.

Alcohol (*Ethanol*). For a detailed discussion of the effects of alcohol, see Chap. 29.

Methanol. Methanol, which is found in numerous *cleaning products* (including household cleaning products), is sometimes confused with ethanol. A lethal dose can be anything between 30 and 100 ml. Like ethanol, methanol is distributed to total body water, its particular toxicity lying in its biotransformation to formaldehyde and formic acid, which in turn cause marked acidosis. Symptoms of poisoning are characterized by gastrointestinal disorders, which appear following a latency of several hours. The impaired vision typical of methanol poisoning generally begins after 3 days with initially reversible retinal edema, followed by damage to the optic nerve (optic atrophy) which, if left untreated, is irreversible and can lead to complete loss of vision. One of the main therapeutic strategies here consists in inhibiting methanol oxidation via simultaneous administration of ethanol as a natural substrate for alcohol dehydrogenase, whereby a BAC of around 1‰ (by means of infusion if necessary) should be maintained for approximately 5 days. Alternatively, the alcohol dehydrogenase inhibitor 4-methylpyrazol (Antizol®) can be administered.

Ethylene Glycol. Ethylene glycol, while also found in cosmetics, is used primarily as an antifreeze agent. However, its sweet taste makes it attractive to children. With a lethal dose of approximately 100–200 ml, both accidental and suicidal poisonings are seen. Although ethylene glycol is itself largely nontoxic, it is converted during biotransformation to the toxic metabolites glycolaldehyde and glycolic acid, as well as glyoxylic acid and oxalic acid in particular. Together with calcium ions, the latter forms hardless soluble calcium oxalate that can block the kidneys (calcium oxalate kidney stones) and cause urinary blockage. In turn, glyoxylic acid can cause uremic coma with lethal outcome. Ethylene glycol poisoning also follows a polyphasic course, sometimes with no initial symptoms. The first phase begins approximately 12 h following ingestion and is characterized by neurological symptoms and metabolic acidosis. Primarily cardiopulmonary disorders, such as tachypnea and cyanosis, are seen in the second phase. Between 24 and 72 h after ingestion, onset of the nephrotoxic symptoms described above is observed, at which point oxalate crystals, in addition to protein and blood, can be found in urine. Therapy is the same as with methanol poisoning.

30.2.20 Food and the Environment

Foodstuffs stored at insufficiently low temperatures or without conservatives, particularly ready-prepared foods, present a food poisoning hazard. Two main clinical pictures are observed:

Enterotoxin Poisoning. Enterotoxins are formed by some strains of *Staphylococci*, *Enterococci*, Listeria, *Salmonellae*, as well as *Escherichia coli* and *Proteus* bacteria. The clinical picture is characterized by vomiting, diarrhea, low blood pressure, headache, and often also a raised temperature. In mild cases, symptoms resolve spontaneously within several days. In the absence of treatment, however, severe cases may develop hypokalemia, hyponatremia, and hypochloremia, often associated with exsiccosis. Therapeutic measures in the case of recent ingestion consist in primary toxin elimination using activated carbon, followed by adequate fluid and electrolyte intake, parenterally if necessary.

Botulinum Toxin Poisoning. Poisoning with the botulinum toxin produced by the bacterium *Clostridium botulinum* is a far more dangerous entity than enterotoxin poisoning and always represents a life-threatening situation. These toxins form in incorrectly conserved meat, fish, and vegetable products, preferring an alkaline environment. Botulinum toxins are among the most potent toxins known to man (as little as 10 µg ingested orally can be lethal). In contrast to thermostable enterotoxins, botulinum toxins are thermolabile and can be destroyed by boiling for 5–10 min. The clinical picture comprises symptoms of acetylcholine deficiency, such as accommodation paralysis, double vision, mydriasis, respiratory distress, and cramps. The latency period to symptom onset is 12–24 h or more, while full recovery may take many weeks. Artificial respiration is required in many cases. In severe cases, death occurs between the second and the tenth day as a result of respiratory and cardiac paralysis as well as bronchopneumonia. Therapeutic measures consist primarily in toxin elimination using activated carbon, rapid gastric emptying, and administration of polyvalent botulinum antitoxins.

30.2.21 Doping Agents

The most common doping agents include:

- *Stimulants*, such as amphetamines, ephedrine, and caffeine, which have a central nervous effect and increase motor activity. Often, the body's own warning system no longer functions, with the effect that all other body reserves are used up without the athlete noticing. This can lead to marked exhaustion, syncope, and even death in extreme cases.
- *Narcotic drugs* and other sedative substances are used primarily for their calming effect, particularly in golf and shooting sports.
- *Anabolic Agents*. The term anabolic agent usually refers to anabolic steroids, which are usually derivates of the male sexual hormone testosterone. Their main effect is to increase muscle mass. Anabolic steroids are used in running sports, broad/long jump, weightlifting, and bodybuilding, given that these disciplines require large muscle mass. They are also used in endurance sports. Common side effects in men include gynecomastia (abnormal mammary gland growth and breast development), impaired semen production, and testicular atrophy. Side effects in women include virilization (facial hair growth, balding, breast reduction), laryngeal development, deepening of the voice, as well as irreversible development of the clitoris. Adolescents may experience inhibited growth. Other side effects include hypertension, cardiac hypertrophy, altered coagulation, myocardial damage, acne, hair loss, liver damage, depression, and hallucinations.
- *Diuretic Agents*. Examples of diuretics include acetazolamide, furosemide, and mersalyl. These agents are used primarily in combat sports, such as judo, boxing, and wrestling, where weight classes are mandatory and failure to observe limitations results in exclusion from participation.
- *Erythropoietin* (*Epo*). Using erythropoietin significantly increases the number of red blood cells, thus promoting oxygen transport, which in turn improves an athlete's endurance. Erythropoietin is used mainly in cycling, skiing, and long-distance running, such as marathons.

- *Increasing Oxygen Transport Capacity* (*Blood Doping*). During altitude training, when there are a greater number of red blood cells in blood than usual, a large volume of blood is taken, stored, and subsequently transfused back into the athlete shortly before the next competition. The increased number of red blood cells results in improved athletic performance. This method is beneficial when blood is taken sufficiently in advance of an important competition. Other methods aimed at increasing oxygen transport capacity are also prohibited, e.g., hyperbaric chambers.
- *Gene Doping*. Any use of cells, genes, or their components is prohibited insofar as they are capable of increasing sporting performance.

Alcohol, cannabis products, local anesthetics containing cocaine, corticosteroids as anti-inflammatory agents, as well as beta blockers are all subject to certain restrictions. The latter are frequently abused in sporting disciplines requiring composure and concentration.

30.2.22 Knockout Drugs

Although benzodiazepines (generally flunitrazepam) were frequently used illegally in the past to induce unconsciousness, they have now been replaced by liquid ecstasy. Due to its special properties, liquid ecstasy represents the "ideal" knockout drug. Compared with most other drugs, it has a relatively simple chemical structure: gamma-hydroxybutyric acid (GHB; see Fig. 30.14), making it comparatively straightforward to produce illegally.

From a chemical structure perspective, liquid ecstasy is not related to "normal" ecstasy (MDMA) or the similar-acting drugs MDA, MDEA/MDE. A colorless, odorless liquid, GHB is available in small plastic ampoules or bottles. In contrast to other knockout drugs such as benzodiazepines, which often have a bitter taste, GHB has a mildly salty taste at most, whereby this is usually masked by the taste of accompanying drinks. Finally, since GHB does not need to be injected, an offender is able to introduce it to a victim's drink unnoticed. GHB's effects range from euphorizing, energy boosting, and sexually arousing to relaxing, soporific, and disorienting. Onset of action can be expected after as little as 15 min. GHB is chemically related to the neurotransmitter responsible for the nervous conduction of stimuli, dopamine, the so-called feel-good hormone.

Important: **GHB has a narrow therapeutic range**, **i.e.**, **the margin between its effects perceived as pleasant and its highly toxic**, **if not lethal**, **effects is extremely small**.

Simple and cheap immunochemical screening tests, some of which are already used by police in the field, are available for most illegal drugs, with the exception of GHB. The screening and detection of GHB, in contrast, is complex and requires analysis methods only available in specialized laboratories. However, even using these methods, GHB can only be detected for a few hours after an incident.

Detectability of Liquid Ecstasy (*GHB*) *in Blood and Urine*: Analytical detection of GHB is only possible within a small diagnostic window due to its short elimination half-life. This period is defined as maximally 8 h for blood and 20 h for urine following administration.

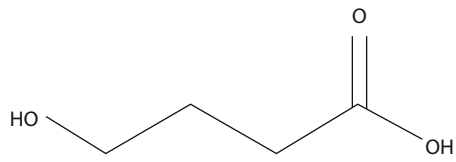

Fig. 30.14 Gamma-hydroxybutyric acid (GHB, 4-hydroxybutanoic acid, liquid ecstasy; CAS No.: 591-81-1)

The main reasons for this are the following:

- GHB is eliminated rapidly from the body (the elimination half-life is only 20 min–1 h).
- GHB is metabolized in the organism to water and carbon dioxide, thus producing no specific metabolites as with other drugs.

- *GHB's particularly sinister nature* lies in the fact that it is present in the body even when no GHB has been ingested, since it is produced by the gamma-aminobutyric acid (GABA) neurotransmitter that occurs naturally in the body (endogenous production).

Victims of knockout drugs often wake up only hours after the incident. Their recollection, if any, is generally slow and fragmented. They are often tortured for days by what they do remember before confiding in parents or friends, on whose advice they finally seek help from a victim support organization, the police, a hospital (gynecology), or a forensic institute. As a result, it is often no longer possible to gather important evidence, such as blood samples and swabs, or to document injuries. Not infrequently, this leads to victims being accused of fabricating an incident in order to account to their parents for coming home late or staying away from home for a long period.

"Advantages" of GHB from the offender's perspective

GHB can be administered surreptitiously. It acts fast, initially producing a state of sexual arousal and euphoria, followed by the soporific effect desired by the offender for the purposes of criminal activity (theft, rape, etc.). Loss of memory (amnesia)—which can be beneficial to the offender—often occurs.

Moreover, GHB is difficult, if not impossible, to detect or is simply overlooked. Even when detection is successful, exogenous administration of GHB can only be reliably diagnosed from a minimum blood concentration.

Finally, the offender benefits from the victim's sense of shame or fear, insofar as this prevent early charges being brought and prompt GHB detection being carried out. Thus time is on the offender's side!

Case Study

Knockout drug used for the purposes of committing theft

A birthday celebration among "fellow countrymen" was simulated in a service station. A man (the owner of a luxury car) from the same ethnic group happened to pass the group; not wanting to be a "spoil sport," he was persuaded to drink to the health of the person celebrating a birthday. The knockout drug had been added to the drink he was offered. The man's car was subsequently stolen and the man "discarded" at the side of a carpark where, due to the cold weather conditions, he froze to death during the night.

Case Study

Using a knockout drug as a "date rape drug"

Given the "private" nature of these offenses, they rarely take place in clubs and discos; indeed, carrying a victim out would attract unwanted attention. Where possible, victims are lured to a home with an invitation to have "another drink"; the knockout drug is then added to the victim's drink, for example, while they visit the restroom. In one particularly macabre case, a girl sedated with GHB was subjected to heinous abuse; a film of the abuse was then put on the Internet, and fellow students were encouraged to watch the scenes for "entertainment."

One problem with the detection of knockout drugs in drink remnants consists in the fact that GHB is found naturally in, for example, wine; thus traces may be detected even in cases where GHB has not been used as a knockout drug.

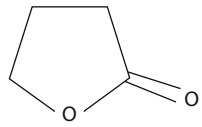

Fig. 30.15 γ-Butyrolactone (GBL, butyro-1,4-lactone; CAS No.: 96-48-0)

The following are the GHB concentrations found in:

Red wine: 4.1–21.4 mg GHB/l

White wine: <3–9.6 mg GHB/l

These values, however, are way below the values detected in drinks when GHB is used illegally and which lie in the gram/liter concentration range. In principle, the possibility of detecting GHB in hair could be considered, given that hair analysis of numerous medications and drugs has become a tried and trusted method. However, medications and drugs are generally taken on a repeated basis, while the intake of a knockout drug usually only happens once. Nevertheless, there are new approaches for GHB detection in hair, albeit in their infancy. Meanwhile, γ-butyrolactone (GBL; Fig. 30.15) is gaining in importance, since it is converted to GHB both in vivo and in vitro.

The background to GBL's particular forensic relevance is described as follows: in contrast to GBL, GHB has been classified as a narcotic agent in many countries. In the case of GBL, however, distribution is monitored only by manufacturers and distributors. A clear liquid with little odor and taste, GBL can also be found at low concentrations in wine. Estimates put the annual quantity used worldwide at 50,000 t. Thus, on the basis of the quantities produced alone, GBL is all but impossible to control. It is used mainly as a solvent, e.g., for the removal of graffiti (at a concentration of 5–10 %) or as nail polish remover, as well as a raw material in numerous industrial chemicals, including medications. By giving these intended uses, GBL can be easily procured over the Internet once guarantees have been given in an electronic form that the substance is not intended for use as a drug, but rather as a cleaning product for the removal of graffiti, for instance. Quantities of 250, 500, and 1,000 ml are generally available at an average price of 50€/500 ml; this makes 100 5-ml portions, which can then be resold on the drug scene at a significant profit.

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Appendixes

Preliminary Remarks

The present Appendixes provide methods, conversion tables, reference values, and charts that may be useful in routine practical work. Due to the volume of data, it was necessary for the authors to be selective, choosing only those data most relevant to forensic practice wherever these have not been included in the individual chapters. The reader is referred to the relevant literature for details on staining methods for tissue and cell samples or diatom detection in suspected death by drowning. It should be mentioned that a portion of the data quoted in tables is based on measurements made in individuals of Central European origin and therefore cannot be directly extrapolated to individuals of other ethnic origins in other parts of the world. From a modern perspective, some of the data is based on relatively scant case numbers. More detailed information, for instance, relating to the appearance of skeletal ossification centers and epiphyseal plate ossification, should be sought in the relevant literature.

Graphic representations in the form of body charts can be copied and used as templates for documenting, e.g., scars, injuries, the localization of tattoos, and congenital lesions. Dimensions are given in units of measurement commonly used in Central and Continental Europe; however, conversion tables for units of measurement generally used in other countries are provided here in the Appendixes.

While the information provided here on the collection of evidence guarantees the highest possible standards of evidence recovery, it presupposes appropriate on-site capacity to perform further investigations, a prerequisite by no means fulfilled in all countries.

Appendix A: General Data

Table A.1 Converting degrees Fahrenheit to degrees Celsius and vice versa

Degrees Fahrenheit (°F)	Degrees Celsius (°C)
23	−5
32	0
41	5
50	10
59	15
68	20
77	25
86	30
95	35
104	40

Conversion:

Degrees Fahrenheit minus 32 divided by 1.8 = degrees Celsius

Degrees Celsius multiplied by 1.8 plus 32 = degrees Fahrenheit

Table A.2 Lengths

Length in cm/m/km	Alternative units of length
2.54 cm	1 inch (in)
30.48 cm	1 foot (ft)
0.91 m	1 yard (yd) = 3 ft
1.61 km	1 mile = 1,760 yd

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Table A.3 Measures of capacity

Capacity in ml/l	Alternative capacities
29.57 ml	1 fluid ounce
0.47 l	1 pint = 16 fl. Oz.
0.95 l	1 quart = 2 pints = 32 fl. Oz
3.79 l	1 gallon = 4 quarts = 8 pints
158.97 l	1 barrel = 42 gallons = 168 quarts

Table A.4 Surface area measurements

Surface area in cm^2/ha/km^2	Alternative surface area measurements
6.45 cm^2	1 square inch (in^2)
929 cm^2	1 square feet (ft^2)
0.84 m^2	1 square yard (yd^2)
0.405 ha = 4,046.8 m^2	1 acre = 4,840 square yards (yd^2)
2.59 km^2	1 square mile = 640 acres

Table A.5 Weights

Weight in grams and kilograms	Alternative units of weight
28.35 g	1 ounce
453.59 g	1 pound (lb)
907 kg	1 ton = 2,000 lb

Table A.6 A comparison of lengths in inches, centimeters, and millimeters

Inches	Centimeters	Millimeters
0.25	0.63	63
0.5	1.27	127
1.0	2.54	254
1.5	3.81	381
2.0	5.08	508
2.5	6.35	635
3.0	7.62	762
4.0	10.16	1,016
5.0	12.70	1,270

Table A.7 Clothing and shoe sizes (these need to be recorded in the case of unidentified deceased individuals!). A comparison of German and US sizes

Men's shirts							
German	36	37	38	39	40/41	42	43
US	14	14.5	15	15.5	16	16.5	17
Men's shoes							
German	39	40	41	42	43	44	45
US	6.5	7.5	8.5	9	10	10.5	11
Women's clothing							
German	36	38	40	42	44	46	
US	6	8	10	12	14	16	
Women's shoes							
German	36	37	38	39	40	41	42
US	5.5	6	7	7.5	8.5	9	9.5
Children's clothing							
German	98	104	110	116	122		
US	3	4	5	6	6x		

Appendix B: Data on Embryos, Fetuses, and Live/Deceased Neonates

Body length is measured (crown–heel length, CHL) when examining stillborn, liveborn, and abandoned infants to estimate gestational age. Data collected in Central Europe for this purpose show significant variations, as is likely to also be the case for embryos (up to and including the third gestational month) and fetuses (from the fourth gestational month) in pregnancies among other ethnicities. The values shown in Table B.1 are based on data collected at the beginning of the last century. Since then, improved nutrition and medical care in Europe have prompted a tendency towards somewhat greater body length values in embryos and fetuses (with exceptions, e.g., smokers); however, the considerable variations in body length in embryos and fetuses, particularly in the first and last trimesters, still need to be considered when estimating pregnancy duration. Some authors suggest also measuring the crown–rump length (CRL), since this offers somewhat greater reliability in the first 4 gestational months due to the early embryonic curvature of the body axis. In general, measuring both the CHL and the CRL is recommended.

Table B.1 Average length of an embryo or fetus (crown–rump length) according to pregnancy duration in days or weeks

Average length of an embryo or fetus	Probable age in days	Probable age in weeks	Probable deviation	Probable deviation
			Maximum	Minimum
	7	**1**		
	14	**2**		
0.5	21	**3**	4.5	0
2.5	28	**4**	10.0	0
5.5	35	**5**	15.0	0
11	42	**6**	25	4
19	49	**7**	37	9
30	56	**8**	50	15
41	63	**9**	66	22
57	70	**10**	86	33
76	77	**11**	105	45
98	84	**12**	135	68
117	91	**13**	155	80
145	98	**14**	178	113
161	105	**15**	190	130
180	112	**16**	210	155
198	119	**17**	230	172
215	126	**18**	243	187
233	133	**19**	265	207
250	140	**20**	283	222
268	147	**21**	305	245
286	154	**22**	320	256
302	161	**23**	323	270
315	168	**24**	345	282
331	175	**25**	362	300
345	182	**26**	380	315
358	189	**27**	400	327
371	196	**28**	413	340
384	203	**29**	430	355
400	210	**30**	447	370
415	217	**31**	473	385
425	224	**32**	485	393
436	231	**33**	500	403
448	238	**34**		413
460	245	**35**		421
470	252	**36**		430
484	259	**37**		440
494	266	**38**		445
500	270	**39**		450

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 12. See also Potter EL (1961) Pathology of the fetus and infant, 2nd ed. Year Book Medical Publishers, Chicago

Another source provides data on the length of an embryo or fetus in centimeters according to pregnancy duration (Table B.2).

As a rule, estimates of pregnancy duration made on the basis of body length are considered to be somewhat more reliable than those based on embryo/fetus weight.

Table B.2 Embryo/fetus length according to pregnancy duration

Pregnancy duration	Body length (cm)
End of month 1	1
End of month 2	4
End of month 3	9
End of month 4	16
End of month 5	25
End of month 6	30
End of month 7	35
End of month 8	40
End of month 9	45
End of month 10	50

Fügel B, Greil H, Sommer K (1986) Anthropologischer Atlas. Verlag Tribüne Berlin, Leipzig, p 27

Table B.3 Range of variation in body weight in relation to body length in neonates

Body length (cm)	Lower limit of normal body weight (g)	Upper limit of normal body weight (g)
48	2,682.8	3,072.8
49	2,765.3	3,180.8
50	2,972.3	3,437.5
51	3,112.4	3,572.4
52	3,298.3	3,775.9
53	3,453.7	3,921.2
54	3,635.7	4,160.4
55	3,848.2	4,425.5
56–59	4,180.9	4,665.4

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 110

Table B.4 Average diameter of the anterior fontanel from birth to closure in boys and girls

Age	Average diameter (cm)	Percentage of fontanel closure
0–3 months	2.6	0.0
3–6 months	2.4	0.3
6–9 months	2.1	1.1
9–12 months	1.8	4.5
12–15 months	1.2	18.6
15–18 months	0.55	46.2
18–21 months	0.31	53.5
21–24 months	0.08	80.0

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 115

Table B.5 Placental weight in relation to neonate weight

Neonate weight (g)	Placental weight (g)
2,500–3,000	483.3
3,000–3,500	578.9
3,500–4,000	653.3
4,000–4,100	698.0
4,100–4,300	730.0
4,300–4,500	761.5
4,500–5,000 and above	880.9

Knaus HH (1962) Das Gewicht der Plazenta und seine forensische Bedeutung. Archiv für Gynäkologie 198:73

Appendix C: Data on Children, Adolescents, and Adults

The data shown in Table C.2 were collected in a separate survey on body length and weight in neonates, children, and adolescents up to the age of 18. As a general rule of thumb, an infant doubles its birth weight by the age of 5 months and triples it by the age of 1 year. Body growth occurs in bursts, with growth spurts taking place at 1 year, 5–7 years, and 12–15 years (earlier in girls than in boys).

Table C.1 Average values for weight, age, and height in boys and girls up to the age 16 (in Central Europe)

Boys		Girls		
Weight (kg)	Age (months, years)	Height (cm)	Age (months, years)	Weight (kg)
3.48	Birth	50	Birth	3.5
3.7		51		3.7
3.9		52		3.9
4.1		53	1 month	4.1
4.4	1 month	54		4.3
4.7		55		4.5
5.0		56	2 months	4.8
5.3	2 months	57		5.1
5.6		58		5.4
5.9		59	3 months	5.7
6.2	3 months	60		6.0
6.5		61	4 months	6.3
6.8	4 months	62		6.6
7.0		63	5 months	6.9
7.3	5 months	64		7.1
7.6		65	6 months	7.4
7.9	6 months	66		7.6
8.2		67	7 months	7.8
8.5	7 months	68		8.0
8.7		69	8 months	8.2
8.9	8 months	70	9 months	8.5
9.2	9 months	71	10 months	8.8
9.5	10 months	72		9.1
9.7		73	11 months	9.4
9.9	11 months	74	1 year	9.7
10.20	1 year	75	1 year, 1 month	9.95
10.45	1 year, 1 month	76	1 year, 2 months	10.20
10.70	1 year, 2 months	77	1 year, 4 months	10.45
10.95	1 year, 4 months	78	1 year, 5 months	10.70
11.20	1 year, 5 months	79	1 year, 6 months	10.95
11.45	1 year, 6 months	80	1 year, 7 months	11.20
11.70	1 year, 7 months	81	1 year, 8 months	11.45
11.95	1 year, 8 months	82	1 year, 10 months	11.70
12.20	1 year, 10 months	83	1 year, 11 months	11.95
12.45	1 year, 11 months	84	2 years	12.20
12.70	2 years	85	2 years, 2 months	12.45
12.95	2 years, 2 months	86	2 years, 3 months	12.70
13.20	2 years, 3 months	87	2 years, 5 months	12.95
13.45	2 years, 5 months	88	2 years, 6 months	13.20
13.70	2 years, 6 months	89	2 years, 8 months	13.45
13.95	2 years, 8 months	90	2 years, 9 months	13.70
14.20	2 years, 9 months	91	2 years, 11 months	13.95
14.45	2 years, 11 months	92	3 years	14.20
14.70	3 years	93	3 years, 2 months	14.45
15.00	3 years, 2 months	94	3 years, 4 months	14.70
15.3	3 years, 3 months	95	3 years, 6 months	14.95
15.6	3 years, 6 months	96	3 years, 8 months	15.30
15.9	3 years, 8 months	97	3 years, 10 months	15.45
16.2	3 years, 10 months	98	4 years	15.70
16.5	4 years	99	4 years, 2 months	15.95
16.8	4 years, 2 months	100	4 years, 5 months	16.20
17.1	4 years, 5 months	101	4 years, 7 months	16.45
17.4	4 years, 7 months	102	4 years, 10 months	16.70
17.7	4 years, 10 months	103	5 years	17.0
18.0	5 years	104	5 years, 3 months	17.5
18.5	5 years, 2 months	105	5 years, 6 months	18.0
19.0	5 years, 5 months	106	5 years, 9 months	18.5
19.5	5 years, 7 months	107	6 years	19.0

Boys		Girls		
Weight (kg)	Age (months, years)	Height (cm)	Age (months, years)	Weight (kg)
20.0	5 years, 10 months	108	6 years, 2 months	19.3
20.5	6 years	109	6 years, 4 months	19.7
21.0	6 years, 2 months	110	6 years, 6 months	20.0
21.4	6 years, 4 months	111	6 years, 8 months	20.3
21.8	6 years, 6 months	112	6 years, 10 months	20.7
22.2	6 years, 8 months	113	7 years	21.0
22.6	6 years, 10 months	114	7 years, 2 months	21.4
23.0	7 years	115	7 years, 5 months	21.8
23.4	7 years, 2 months	116	7 years, 7 months	22.2
23.8	7 years, 5 months	117	7 years, 10 months	22.6
24.2	7 years, 7 months	118	8 years	23.0
24.6	7 years, 10 months	119	8 years, 2 months	23.4
25.0	8 years	120	8 years, 5 months	23.8
25.5	8 years, 2 months	121	8 years, 7 months	24.2
26.0	8 years, 5 months	122	8 years, 10 months	24.6
26.5	8 years, 7 months	123	9 years	25.0
27.0	8 years, 10 months	124	9 years, 2 months	25.4
27.5	9 years	125	9 years, 5 months	25.8
28.0	9 years, 2 months	126	9 years, 7 months	26.2
28.5	9 years, 5 months	127	9 years, 10 months	26.6
29.0	9 years, 7 months	128	10 years	27.0
29.5	9 years, 10 months	129	10 years, 2 months	27.4
30.0	10 years	130	10 years, 5 months	27.8
30.5	10 years, 2 months	131	10 years, 7 months	28.2
31.0	10 years, 5 months	132	10 years, 10 months	28.6
31.5	10 years, 7 months	133	11 years	29.0
32.0	10 years, 10 months	134	11 years, 2 months	29.5
32.5	11 years	135	11 years, 4 months	30.0
33.0	11 years, 2 months	136	11 years, 6 months	30.5
33.5	11 years, 5 months	137	11 years, 8 months	31.0
34.0	11 years, 7 months	138	11 years, 10 months	31.5
34.5	11 years, 10 months	139	12 years	32.0
35.0	12 years	140	12 years, 2 months	32.7
35.5	12 years, 2 months	141	12 years, 3 months	33.4
36.0	12 years, 5 months	142	12 years, 5 months	34.1
36.5	12 years, 7 months	143	12 years, 7 months	34.8
37.0	12 years, 10 months	144	12 years, 9 months	35.5
37.5	13 years	145	12 years, 10 months	36.2
38.0	13 years, 2 months	146	13 years	37.0
38.6	13 years, 4 months	147	13 years, 2 months	37.8
39.2	13 years, 6 months	148	13 years, 3 months	38.6
39.8	13 years, 8 months	149	13 years, 5 months	39.4
40.4	13 years, 10 months	150	13 years, 7 months	40.3
41.9	14 years	151	13 years, 9 months	41.2
41.6	14 years, 2 months	152	13 years, 10 months	42.1
42.3	14 years, 4 months	153	14 years	43.0
43.0	14 years, 6 months	154	14 years, 2 months	44.0
43.6	14 years, 8 months	155	14 years, 5 months	45.0
44.3	14 years, 10 months	156	14 years, 7 months	46.0
45.0	15 years	157	14 years, 10 months	47.0
45.7	15 years, 2 months	158	15 years	48.0
46.4	15 years, 3 months	159	15 years, 6 months	50.0
47.1	15 years, 5 months	160	16 years	52.0

Table C.1 (continued)

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, S. 18

Table C.2 Height and weight (average values) according to Stuart and Stevenson

Boys			Girls		
Age (months, years)	Weight (kg)	Height (cm)	Age (months, years)	Weight (kg)	Height (cm)
Birth	3.4	50.6	Birth	3.36	50.2
1 month	4.25	54.5	1 month	4.15	53.8
2 months	5.01	57.6	2 months	4.91	56.8
3 months	5.72	60.4	3 months	5.62	59.5
4 months	6.40	62.7	4 months	6.21	61.6
5 months	7.02	64.6	5 months	6.75	63.5
6 months	7.58	66.4	6 months	7.26	65.2
7 months	8.10	68.1	7 months	7.78	67.0
8 months	8.61	69.7	8 months	8.27	68.6
9 months	9.07	71.2	9 months	8.71	70.1
10 months	9.49	72.6	10 months	9.10	71.6
11 months	9.82	74.0	11 months	9.45	73.0
12 months	10.07	75.2	12 months	9.75	74.2
15 months	10.75	78.5	15 months	10.43	77.6
18 months	11.43	81.8	18 months	11.11	80.9
2 years	12.56	87.5	2 years	12.29	86.6
2.5 years	13.61	99.1	2.5 years	13.43	91.4
3 years	14.61	96.2	3 years	14.42	95.7
3.5 years	15.56	99.8	3.5 years	15.38	99.5
4 years	16.51	103.4	4 years	16.42	103.2
4.5 years	17.42	106.7	4.5 years	17.46	106.8
5 years	18.37	108.7	5 years	18.37	109.1
5.5 years	20.68	114.4	5.5 years	19.96	112.8
6 years	21.91	117.5	6 years	21.09	115.9
6.5 years	23.22	120.8	6.5 years	22.41	119.1
7 years	24.54	124.1	7 years	23.68	122.3
7.5 years	25.9	127.1	7.5 years	25.04	125.2
8 years	27.26	130.0	8 years	26.35	128.0
8.5 years	28.62	132.8	8.5 years	27.67	130.5
9 years	29.94	135.5	9 years	28.94	132.9
9.5 years	31.3	137.9	9.5 years	30.44	135.8
10 years	32.61	140.3	10 years	31.89	138.6
10.5 years	33.93	142.3	10.5 years	33.79	141.7
11 years	35.2	144.2	11 years	35.74	144.7
11.5 years	36.74	146.9	11.5 years	37.74	148.1
12 years	38.28	149.6	12 years	39.74	151.9
12.5 years	40.23	152.3	12.5 years	42.37	154.3
13 years	42.18	155.0	13 years	44.95	157.1
13.5 years	45.5	158.9	13.5 years	47.04	158.4
14 years	48.81	162.7	14 years	49.17	159.6
14.5 years	51.66	165.3	14.5 years	50.35	160.4
15 years	54.48	167.8	15 years	51.48	161.1
15.5 years	56.65	169.7	15.5 years	52.30	161.7
16 years	58.83	171.6	16 years	53.07	162.2
16.5 years	60.33	172.7	16.5 years	53.57	162.4
17 years	61.78	173.1	17 years	54.02	162.5
17.5 years	62.41	174.1	17.5 years	54.20	162.50
18 years	63.05	174.5	18 years	54.39	162.5

Feer E (1966) Lehrbuch der Kinderheilkunde. VEB G. Fischer, Jena, p 2

Table C.3 Normal heart weight in children up to the age of 12 years; data on boys (slight downward variations in girls)

Age	Body length (cm)	Heart weight (g)
0–3 days	49	17
3 weeks	52	19
3 months	57	30
6 months	62	40
12 months	74	50
24 months	84	60
6 years	114	105
12 years	139	124

Schulz DM, Giordano DA (1962) Hearts of infants and children: weights and measurements. Arch Pathol 74:464–471. From: Caesar R (1984) Herz. In: Remmele W (ed) Pathologie, Band 1. Springer, Vienna Berlin Heidelberg, p 43

Table C.4 Average data on height and weight, as well as heart, liver, and kidney weights in male children and adults according to age. The data, some of which were gathered from only small collectives, are intended merely as a guide

Age	Height (cm)	Body weight (kg)	Heart (g)	Liver (g)	Kidneys (g)
2	83	12.98	63.9	456	118
3	94	13.88	68.5	468	98
4	97	15.43	83	516	105
5	105	16.01	91.3	556	117
6	109	18.86	101.9	628	118
7	122	23.6	127.6	824	146
8	124	21.4	128.8	720	150
9	125	24.5	141.4	795	148
10	136	25.78	144.2	782	162
11	137	28.4	164.8	1,021	199
12	143	31.1	172.5	848	133
13	143	30.7	169.8	1,120	182
14	146	32.8	169	945	186
15	153	48.0	246.6	1,384	257
16	163	48.2	245.7	1,380	245
17	137	52.9	293	1,597	278
18	169	53.3	283.2	1,580	275
19	169	57.3	283.3	1,621	286

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 59

Table C.5 Average data on height and weight, as well as heart, liver, and kidney weights in female children and adolescents according to age. The data, some of which were gathered from only small collectives, are intended merely as a guide

Age	Height (cm)	Weight (kg)	Heart (g)	Liver (g)	Kidneys (g)
2	84	11.57	57.4	410	85
3	89	14.47	69.9	434	92
4	98	15.8	71.9	504	102
5	110	16.01	97.5	594	122
6	109	17.24	90.8	725	157
7	117	20.73	106.5	611	126
8	121	26.5	126.3	743	150
9	125	22.6	119.4	620	131
10	125	22.75	115	701	128
11	136	26.7	138.7	765	167
12	138	31.18	166.4	1,070	180
13	141	34.3	191.3	953	177
14	150	38.75	197	1,020	202
15	153	49.3	227.9	1,378	243
16	155	44.7	217.8	1,341	241
17	158	49.35	247.5	1,385	281
18	157	54.3	271.7	1,479	265
19	157	51.7	261.7	1,493	279
20	155	52.8	251.4	1,599	292

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, pp 59–60

Appendix D: Data on Organs

Other authors provide slightly different measurements, as illustrated in Table D.4.

Table D.1 The average weights of vital organs

Organ	Full-term neonates (g)	40-year-olds		80-year-olds	
		Men (g)	Women (g)	Men (g)	Women (g)
Heart	24	320	270	370	320
Right lung	27	430	370	380	320
Left lung	22	370	310	340	290
Both kidneys	24	280	240	250	220
Spleen	11	150	135	120	100
Liver	150	1,600	1,450	1,400	1,300
Brain	380	1,380	1,250	1,200	1,100
Both adrenal glands	6	14	13	13	12
Thymus	13 (10–25)				

Kettler LH (1965) Lehrbuch der speziellen Pathologie. VEB G. Fischer Verlag, Jena, p 794

Table D.2 Normal heart weight in adult men and women from a height of 150 cm

Height (cm)	Heart weight in men (g)	Heart weight in women (g)
150	243–323	215–275
160	262–342	233–293
170	281–361	251–311
180	300–380	269–329
185	309–389	277–337

Zeek PM (1942) Heart weight. I. The weight of the normal human heart. Arch Pathol 34:820–832. From: Caesar R (1984) Herz. In: Remmele W (ed) Pathologie, Band 1. Springer, Vienna Berlin Heidelberg, p 43

Table D.3 Cardiac wall thicknesses and valvular sizes

Wall thicknesses (mm)	Valvular sizes (mm)
Atrium, 2–2.5	Tricuspid valve, 110–130
Right ventricle (conus), 3	Pulmonary valve, 75–85
Left ventricle (beneath the posterior mitral cusp), 12–15	Mitral valve, 90–110
	Aortic valve, 70–80[a]

From Caesar R (1984) Herz. In: Remmele W (ed) Pathologie, Band 1. Springer, Vienna Berlin Heidelberg, p 43

[a]In 30- to 40-year-old men, 5.8–7.5 cm; in 80-year-old men, 7.9–9 cm; in women, 2 mm less in both ranges

Table D.4 Normal measurements of the heart and vessels in adults

Heart valves in blood flow direction	Dimensions (cm)
Tricuspid valve	12
Pulmonary valve	8.5
Pulmonary conus	8
Mitral valve	10
Aortic valve	7.5
Segments of the aorta	
Ascending aorta ascendens	8.5
Descending aorta	4.5–7
Abdominal aorta	3.5–4.5
Ventricular wall thickness	
Right ventricular wall	0.5
Left ventricular wall	1.5

Sunderman FW, Boerner F (1949) Normal values in clinical medicine. W.B. Saunders Company, Philadelphia

Table D.5 Weight of the spleen according to age (adapted from Lubarsch)

Age	Weight (male/female) (g)
Full-term stillborn	9.5/9.6
Infant	15.7/15.3
1–2 years	29.9/29.0
2–3 years	33/30
3–5 years	42/40
5–7 years	60/58
7–10 years	59/61
10–15 years	82/73
15–20 years	120/122
20–30 years	142/145
30–40 years	142/146

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 49

Table D.6 Average weight of the lungs in men and women according to age; these values, however, are subject to significant upward and downward variations

	Men		Women	
Age	Right lung (g)	Left lung (g)	Right lung (g)	Left lung (g)
At birth	27.23	22.74	25.73	21.45
Up to 6 months	31.75	31.80	34.62	30.50
7–12 months	61.66	51.66	–	53.00
1 year	96.11	75.77	96.00	82.00
2 years	106.83	84.70	95.92	85.08
3 years	115.25	97.75	106.00	98.00
4 years	131.9	113.50	142.80	110.33
5 years	–	–	139.66	127.00
6 years	–	–	131.33	113.66
7 years	156.0	136.75	–	–
8 years	–	–	198	151
9–10 years	189.5	164.00	145	127.5
12 years	201	203.33	173.5	160
13 years	322	–	243.33	248
15–20 years	379	291.00	322.75	297.66
21–30 years	421.75	354.28	346.20	287.09
31–40 years	435.07	377.71	336.40	286.40
41–50 years	429.23	363.20	387.15	319.30
51–60 years	445.90	374.80	340.60	291.60
61–70 years	410.92	378.53	361.60	265.90
71–90 years	377.25	375–320	350–356.85	270.33–277.42

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, pp 53–54

Table D.7 Average combined lung weights in adults (15–70 years), broken down according to lung and total lung weight

Men			Women		
Left lung (g)	Right lung (g)	Total (g)	Right lung (g)	Left lung (g)	Total (g)
420.31	356.58	776.89	349.11	291.32	640.43

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 55

Table D.8 Average liver weight values and body weight according to age and sex. The data, some of which were gathered from only small collectives, are intended merely as a guide

	Men		Women	
Age	Liver weight (g)	Body weight (kg)	Liver weight (g)	Body weight (kg)
At birth	134.65	3.373	133.73	3.059
Up to 6 months	164.46	3.789	139.87	3.910
7–12 months	258.75	–	267	–
1 year	379.75	12.366	332	8.226
2 years	420.33	13.422	346.75	11.725
3 years	417.16	15.300	421	12.450
4 years	490.57	17.766	425.75	12.800
5 years	605.33	17.325	457.60	–
6 years	–	–	528.50	16.650
7 years	611.50	20.200	602.50	17.000
8 years	560	–	694	19.200
9–10 years	745	23.400	831	22.700
11 years	–	–	1,010	32.350
12 years	820.75	37.600	1,051	–
13 years	1,030	37.350	895	36.300
14 years	–	–	1,170	40.65
15 years	1,270	44.228	926	35.5
16 years	1,287.87	43.562	1,121	48.38
17 years	1,525	49.175	1,203.33	53.63
18 years	1,431.25	44.780	1,126.25	44.05
19 years	1,512.10	56.787	1,376.66	47.83
20 years	1,607.75	53.076	1,616.25	56.1
20–25 years	1,563.50	55.09	1,323.12	50.08
26–30 years	1,610.68	53.517	1,418.10	52.31
31–35 years	1,511.14	57.68	1,355.9	51.65
36–40 years	1,606.51	58.939		
41–45 years	1,557.35	57.567	1,359.09	55.300
46–50 years	1,650.16	62.558	1,530.34	61.44
51–55 years	1,546.73	59.278	1,407.69	60.54
56–60 years	1,490.40	56.652	1,459.16	56.916
61–65 years	1,495.20	54.772	1,215.00	56.42
66–70 years	1,513.07	61.900	1,440	56.85
71–75 years	1,367.81	57.909	1,058.62	47
76–80 years	1,321.11	54.588	1,289.00	48.34

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, pp 58–59

Table D.9 Cerebrum and cerebellum weights in relation to height without taking sex into consideration (according to Rieger)

Height (cm)	Cerebrum weight (g)	Cerebellum weight (g)
130	820	110
140	955	125
150	1,080	135
155	1,130	140
160	1,210	150
165	1,270	150
170	1,310	160
175	1,360	170
180	1,440	180
185	1,570	200

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 91

Table D.10 Heart weight correlated with body length/height, liver weight, and kidney weight in men

Age	Heart weight (g)	Body weight (kg)	Body length/height (cm)	Quotient heart/liver weight	Quotient heart/kidney weight
Neonate	23.37	3.37	51.57	0.173	0.973
1–12 months	33.5	–	60.88	0.158	0.826
1–5 years	75.87	15.123	92.34	0.177	0.935
6–10 years	127.5	24.06	125.93	0.201	1.093
11–15 years	184.96	36.837	145.93	0.177	0.945
16–20 years	263.2	48.708	165.07	0.179	1.012
21–30 years	308.75	53.371	168.46	0.194	1.090
31–40 years	313.24	55.174	166.97	0.200	1.090
41–50 years	326.43	56.827	166.72	0.203	1.229
51–60 years	340.19	56.433	165.80	0.223	1.235
61–70 years	341.87	56.39	164.2	0.227	1.296
Over 70 years	315.77	45.42	162.53	0.234	1.359

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 121

Table D.11 Heart weight correlated with body length/height, liver weight, and kidney weight in women

Age	Heart weight (g)	Body weight (kg)	Body length/height (cm)	Quotient heart/liver weight	Quotient heart/kidney weight
Neonate	21.4	3.198	48.96	0.160	0.913
1–12 months	28.85	4.288	60.09	0.141	0.707
1–5 years	67.24	12.719	96.70	0.164	0.829
6–10 years	103.73	21.500	122.16	0.156	0.861
11–15 years	174.66	39.400	141.39	0.172	0.892
16–20 years	233.37	49.273	155.54	0.181	1.059
21–30 years	259.20	51.073	157.05	0.189	1.000
31–40 years	251.32	52.14	156.06	0.185	0.874
41–50 years	293.62	55.822	153.83	0.203	1.105
51–60 years	304.36	58.74	154.12	0.212	1.302
61–70 years	334.84	51.932	153.82	0.252	1.416
Over 70 years	292.04	39.358	154.27	0.248	1.446

Roessle R, Roulet F (1932) Mass und Zahl in der Pathologie. Julius Springer, Berlin Vienna, p 121

Table D.12 Kidney weights in neonates and children

Age	Right kidney (g)	Left kidney (g)
1 month	16	16
2 months	19	18
4 months	22	21
6 months	26	25
8 months	31	30
10 months	32	31
1 year	36	35
2 years	47	46
3 years	48	49
4 years	58	56
5 years	65	64
6 years	68	67
7 years	69	70
8 years	74	75
9 years	82	83
10 years	92	95
11 years	94	95
12 years	95	96

Sunderman FW, Boerman F (1949) Normal values in clinical medicine. W.B. Saunders Company, Philadelphia

Appendix E: Data on Forensic Alcohology and Toxicology

Table E.1 Common sizes (filling volumes) of glasses, bottles, and cans

Liter	ml	cl
0.02	20	2
0.05	50	5
0.1	100	10
0.2	200	20
0.25	250	25
0.35	350	35
0.5	500	50
0.7	700	70
1.0	1,000	100
1.5	1,500	150
2.0	2,000	200
3.0	3,000	300
5.0	5,000	500

Table E.2 Special champagne bottle sizes

Bottle size	Filling volume (liters)
Quarter bottle	0.187
Half bottle	0.375
Normal bottle	0.750
Magnum	1.5
Double magnum or Jeroboam	3
Rehoboam	4.5
Methuselah	6
Salmanazar	9
Balthazar	12
Nebuchadnezzar	15

Table E.3 Common dosages

Dosage	Filling volume (ml)
1 teaspoon	ca. 5
1 tablespoon	ca. 15
1 cup	100–150
1 tumbler	100–150
1 sip	10–50
1 gram tincture	ca. 60 drops

Schütz H (1983) Alkohol im Blut. Nachweis und Bestimmung, Umwandlung und Berechnung. Verlag Chemie, Weinheim

Appendix F: Informed Consent Form for Victims of Violence

Information for Victims of Violence Prior to Medical Gynecological Examination

Dear patient,

You have come to us because you have been the victim of violent crime.

We will make every effort to relieve any pain or discomfort you may be in. We are also here to answer your questions and assist with any problems you may have.

The planned examination and documentation of findings is intended to collect evidence that may be crucial to further investigations or subsequent criminal proceedings, assuming you wish to pursue this avenue.

You may have already made a full statement regarding the offense to the police. Although we do not wish to burden you unnecessarily by asking you to recount the incident again, we do require some details for the purposes of a thorough examination and documentation of findings. This often reduces the time needed for the examination and makes it easier to establish a relationship between the offense and injury findings.

If recounting the incident is too stressful for you, we will naturally respect this; the examination will then be performed on the basis of our experience in past cases.

We assume that today's examination, including the collection of potentially relevant trace evidence, will take place with your consent. None of the findings will be passed on to other persons or institutions, including the investigating authorities, without your explicit consent. You will be given the relevant consent form, as well as a form for your consent to disclose medical information.

It is likely that, in addition to this preliminary medical examination, you will require further assistance to deal with recent events. We will be happy to answer any questions you may have

and/or refer you to other bodies: …… If you require any further information, please do not hesitate to ask us!

Your physician

Informed Consent

I hereby give my informed consent for today's examination. Where relevant to the reported incident, I additionally give my consent for photographic documentation to be compiled, blood samples taken, and a pregnancy test performed, as well as tests for HIV, sexually transmitted diseases, and hepatitis to be carried out. I consent to the collection of trace evidence, the analysis of which may be relevant as evidence at some later stage.

Patient signature..... Date.....

Consent to Disclose Medical Information

The examination in question, including the collection of trace evidence, has been carried out in the context of medical treatment and is thereby subject to medical confidentiality. A disclosure of information about the examination or examination results to third parties, in particular the investigating authorities, can therefore only take place with your explicit consent to disclose medical information or by order of a court.

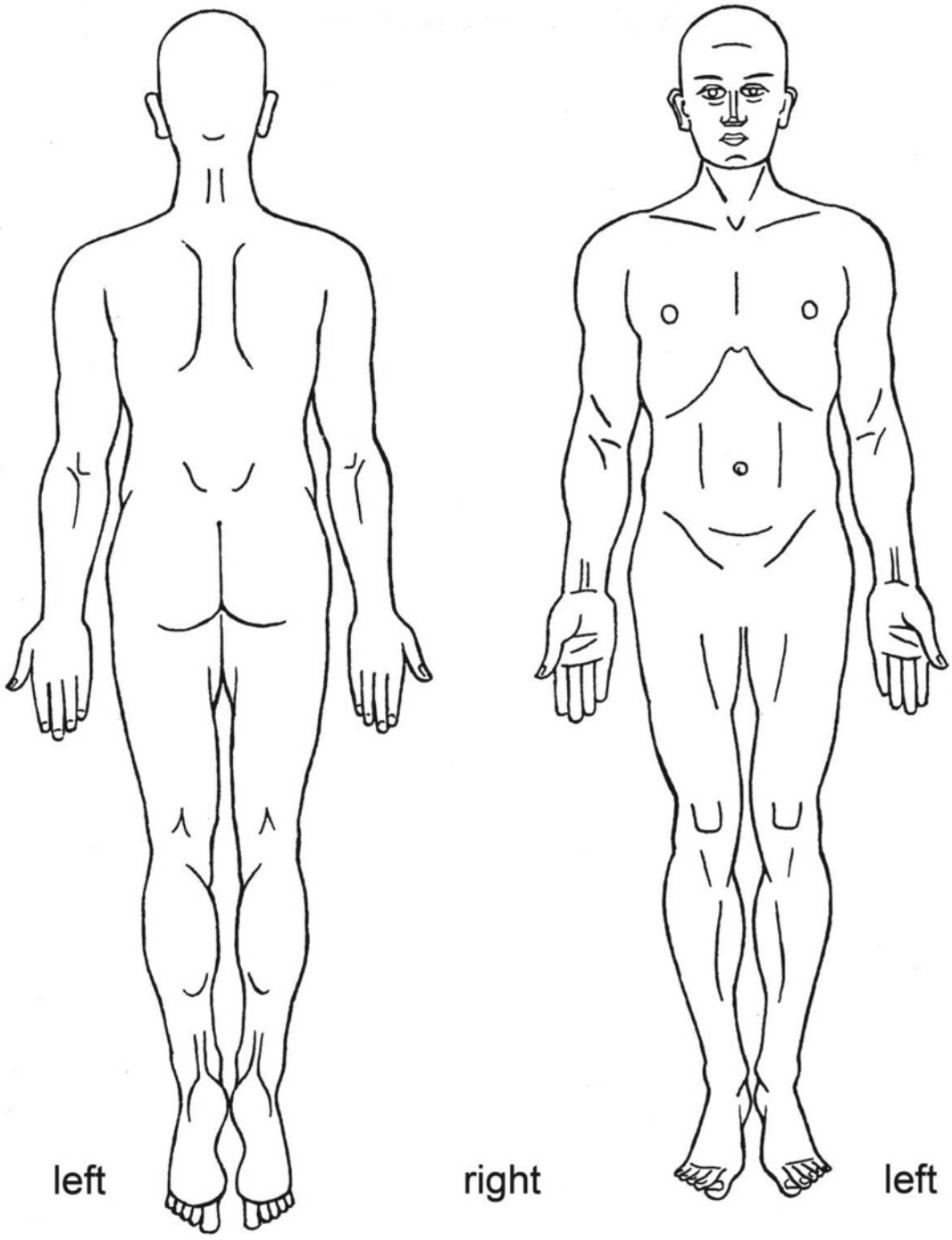
I hereby give/decline (please cross out as appropriate) my consent for the treating physicians and assisting personnel to disclose my medical information to the investigating authorities (e.g., the police) and the court.

I am aware that I have the right to give or withdraw consent at any stage (with only limited effectiveness for further steps).

Patient signature..... Date.....

Appendix G: Body Charts for the Graphic Documentation of Injuries

In addition to body charts designed to document injuries graphically, special forms for documenting domestic and sexual violence are recommended. These should include details on general patient history, gynecological history, the reported incident, psychological and physical findings, the manner of evidence collection, and the results of serological tests. In special cases, a physician with forensic experience should be consulted. Where possible, patients can be provided with information on local assistance organizations.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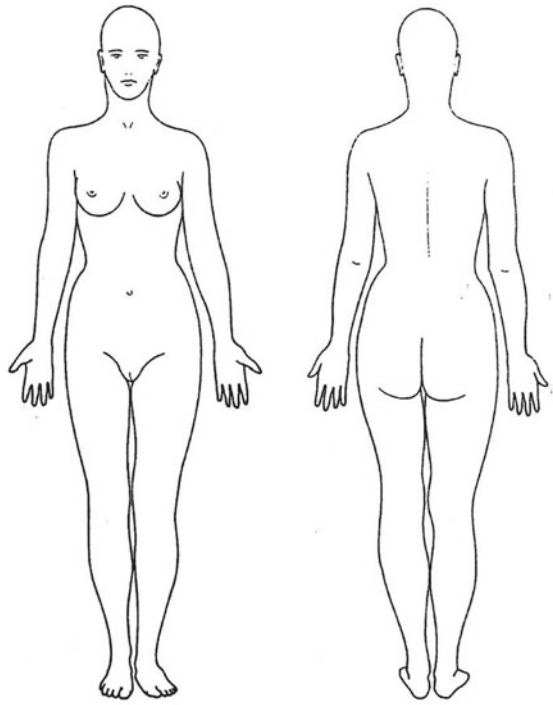
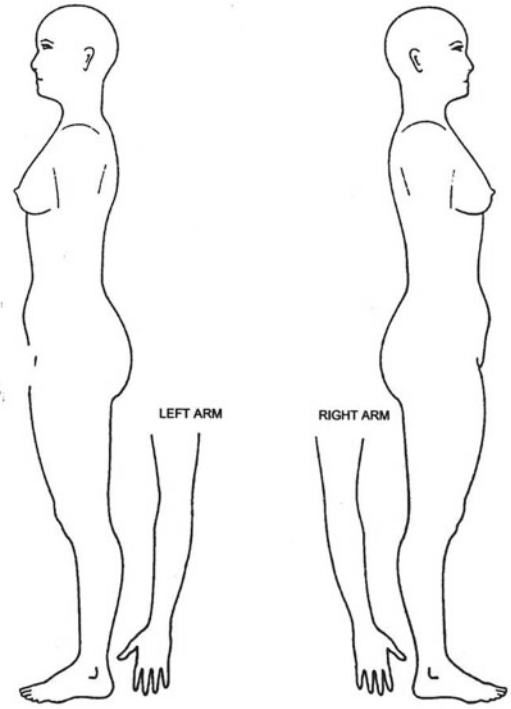
FULL BODY, FEMALE—ANTERIOR AND POSTERIOR VIEWS



Name _____ Case No. _____
Date _____

FULL BODY, FEMALE—LATERAL VIEW



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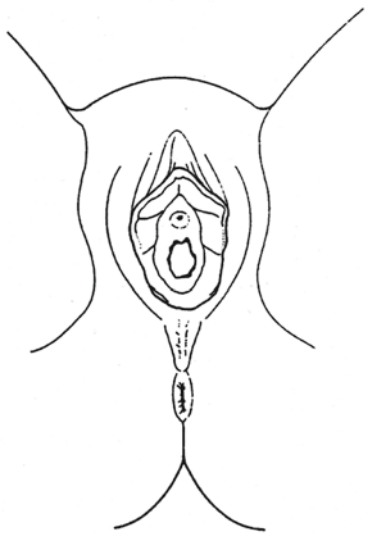
PERINEUM—FEMALE



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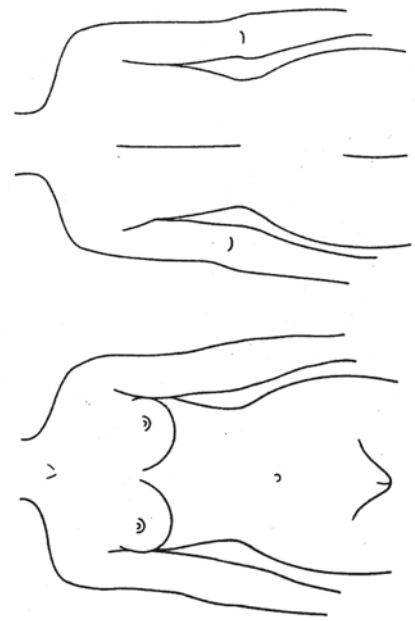
THORACIC ABDOMINAL, FEMALE—ANTERIOR AND POSTERIOR VIEWS



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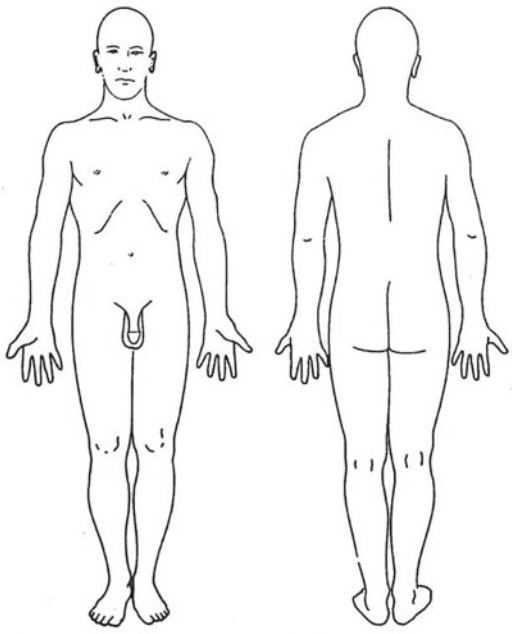
FULL BODY, MALE—ANTERIOR AND POSTERIOR VIEWS (VENTRAL AND DORSAL)



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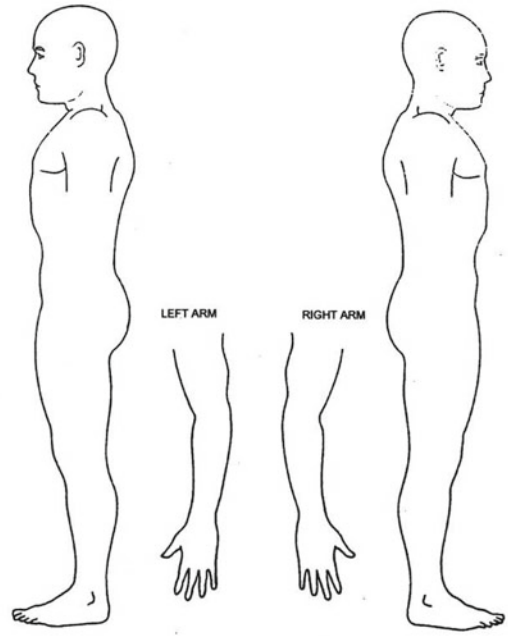
FULL BODY, MALE—LATERAL VIEW



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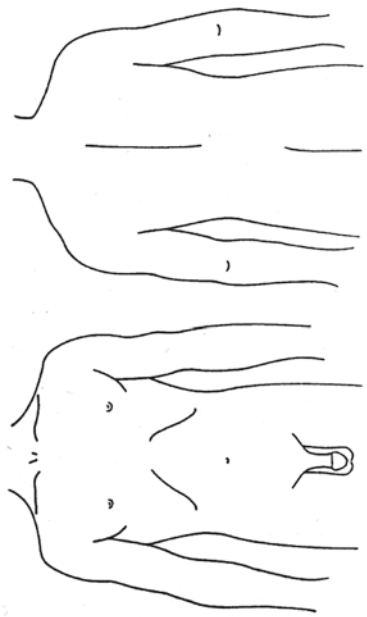
THORACIC ABDOMINAL, MALE—ANTERIOR AND POSTERIOR VIEWS



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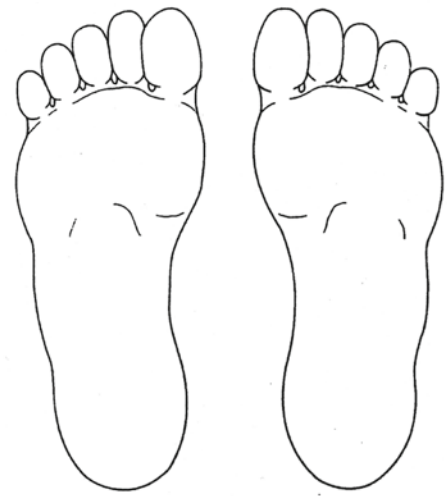
FEET—LEFT AND RIGHT PLANTAR SURFACES



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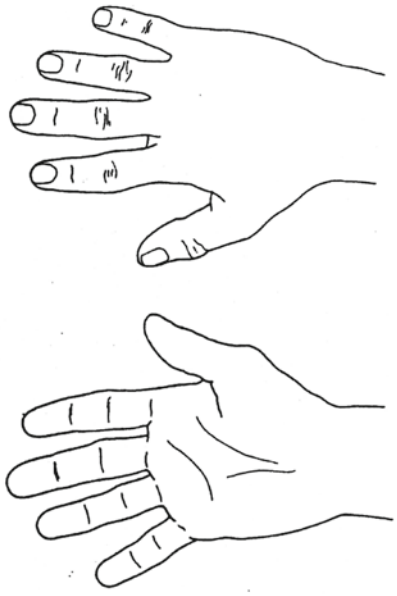
RIGHT HAND—PALMAR AND DORSAL



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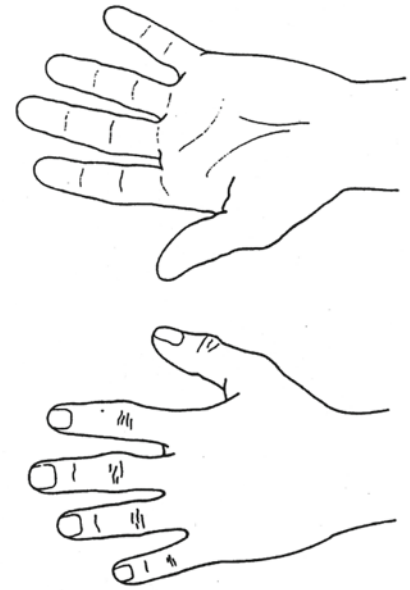
LEFT HAND—PALMAR AND DORSAL



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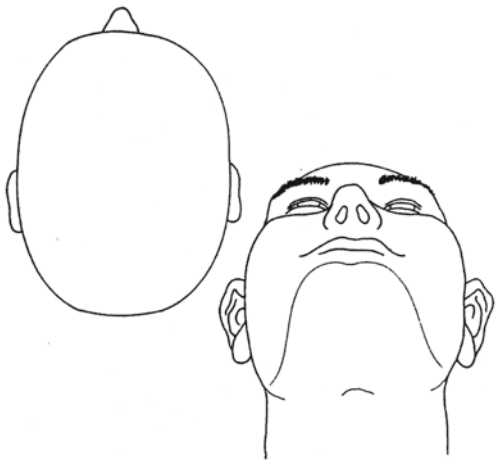
HEAD—SURFACE AND SKELETAL ANATOMY, SUPERIOR VIEW—INFERIOR VIEW OF NECK



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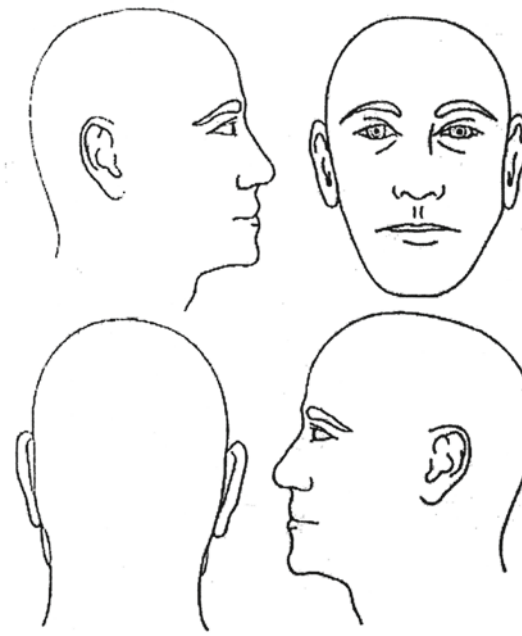
HEAD—SURFACE AND SKELETAL ANATOMY, LATERAL VIEW



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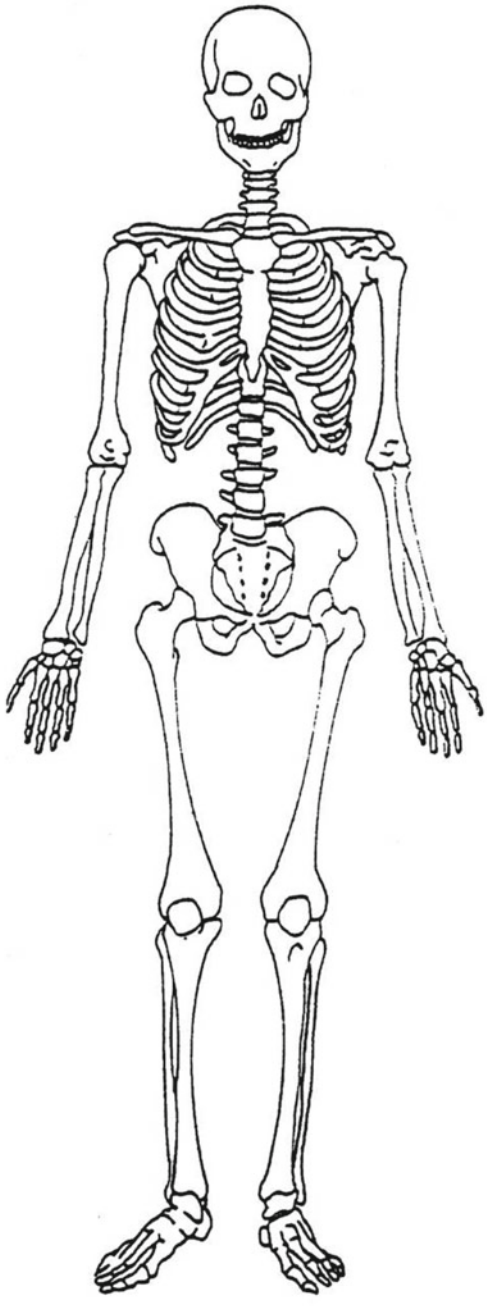
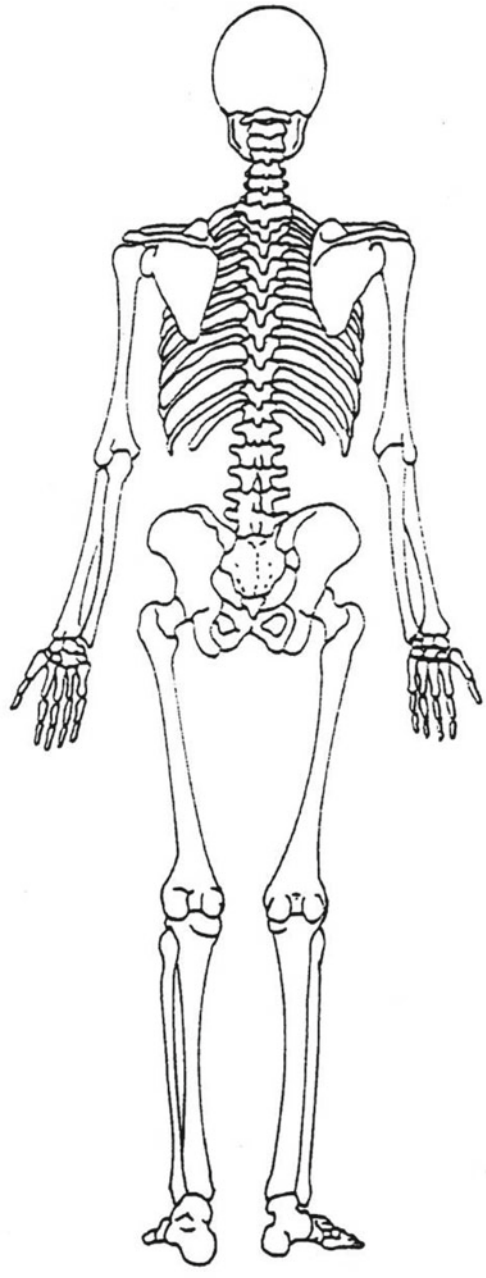
SKELETON—ANTERIOR AND POSTERIOR VIEWS





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