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.

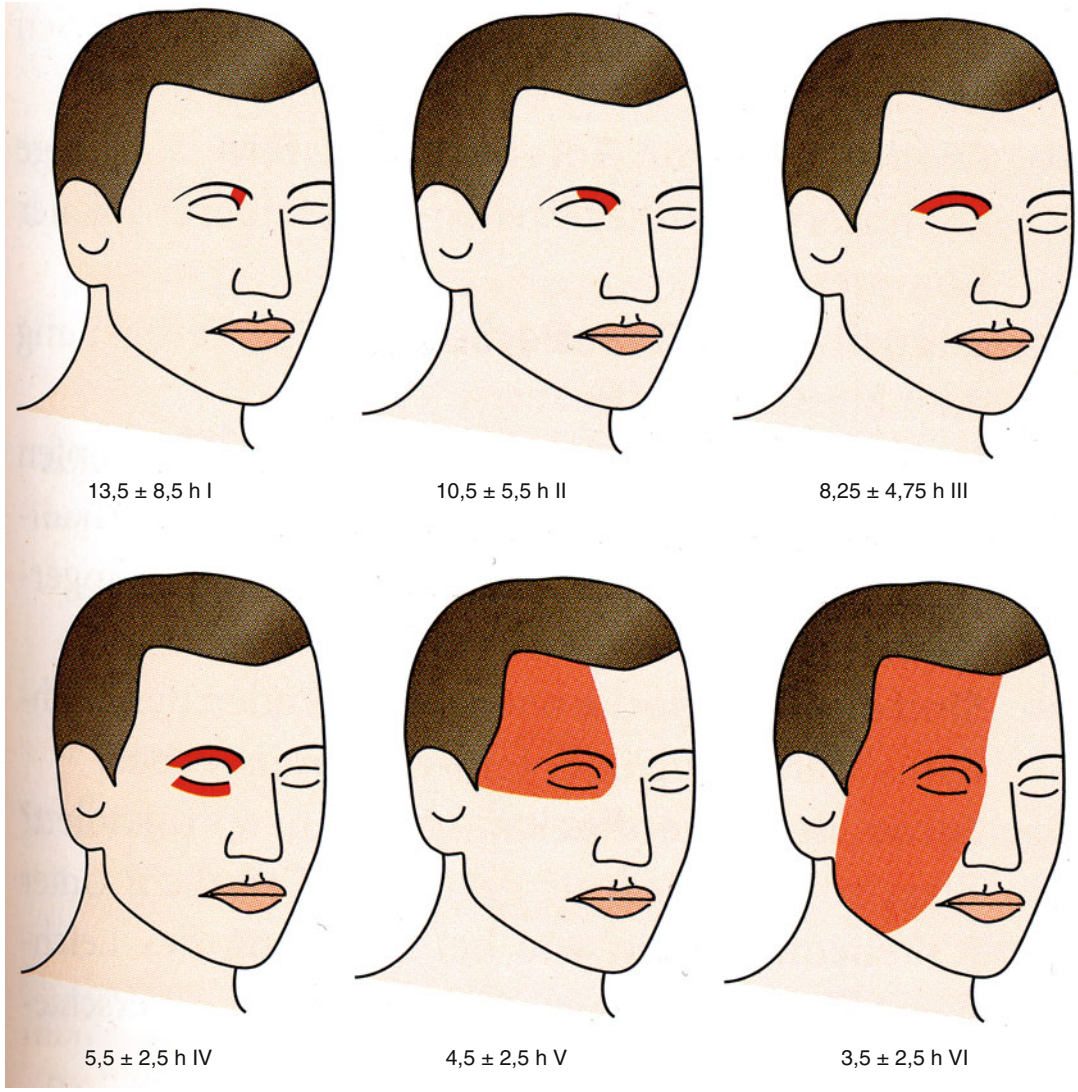

Fig. 3.1 Testing for electrical excitability of the facial mimic muscles. Contraction of the entire ipsilateral facial musculature can be seen in the early postmortem phase. As the postmortem interval increases, muscle excitation is limited to the point of stimulation directly proximal to the electrode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

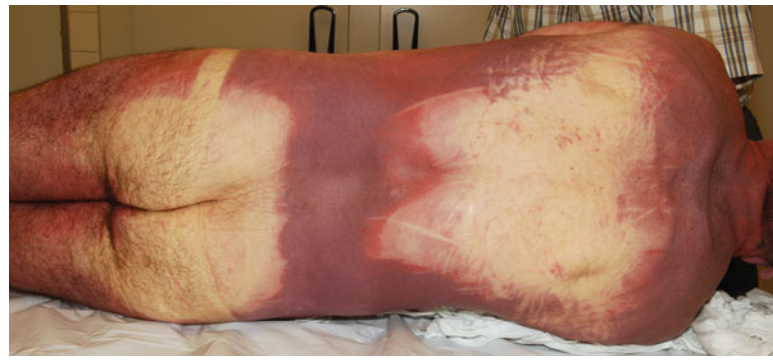

Fig. 3.2 Livor mortis. Butterfly-shaped area of sparing from *bluish-violet* livor mortis due to the supine position of the body. An additional area of sparing is seen in the belt area. Areas of lighter livor mortis are a sign of early postmortem reoxygenation of hemoglobin due to cold storage

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

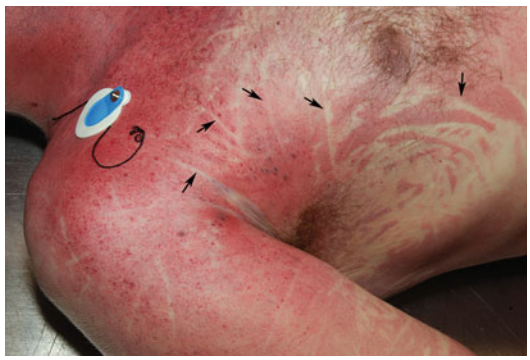

Fig. 3.3 Livor mortis. Linear sparing (*arrows*) within livor mortis due to folds in clothes

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

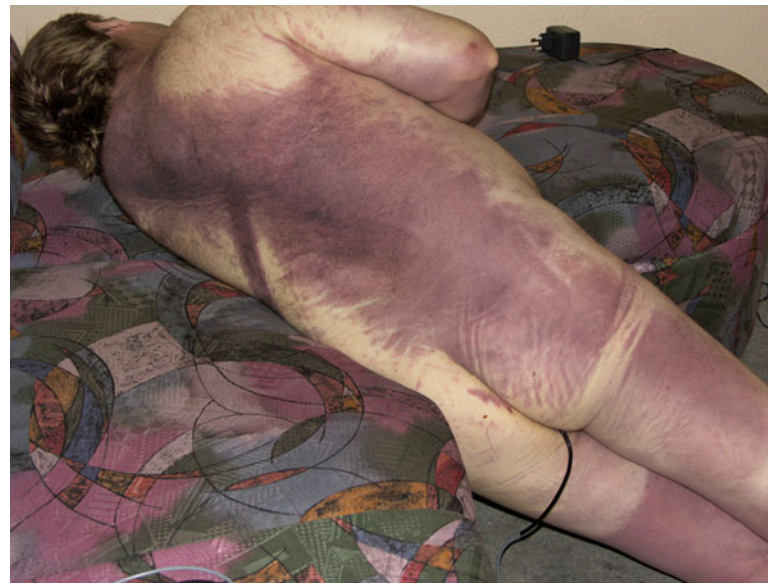

Fig. 3.4 The distribution of livor mortis is consistent with the (former) position of the body on the sofa with linear sparing matching the folds in the sofa fabric

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).

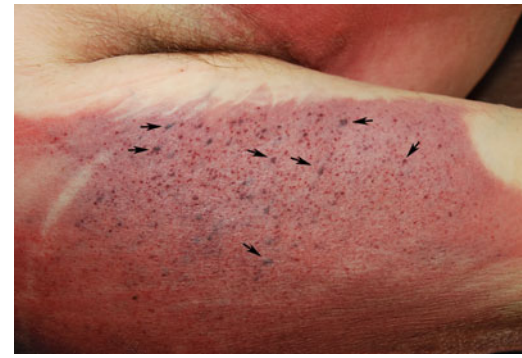

Fig. 3.5 Vibices. Abundant small purple vibices (*arrows*) within an area of livor mortis

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

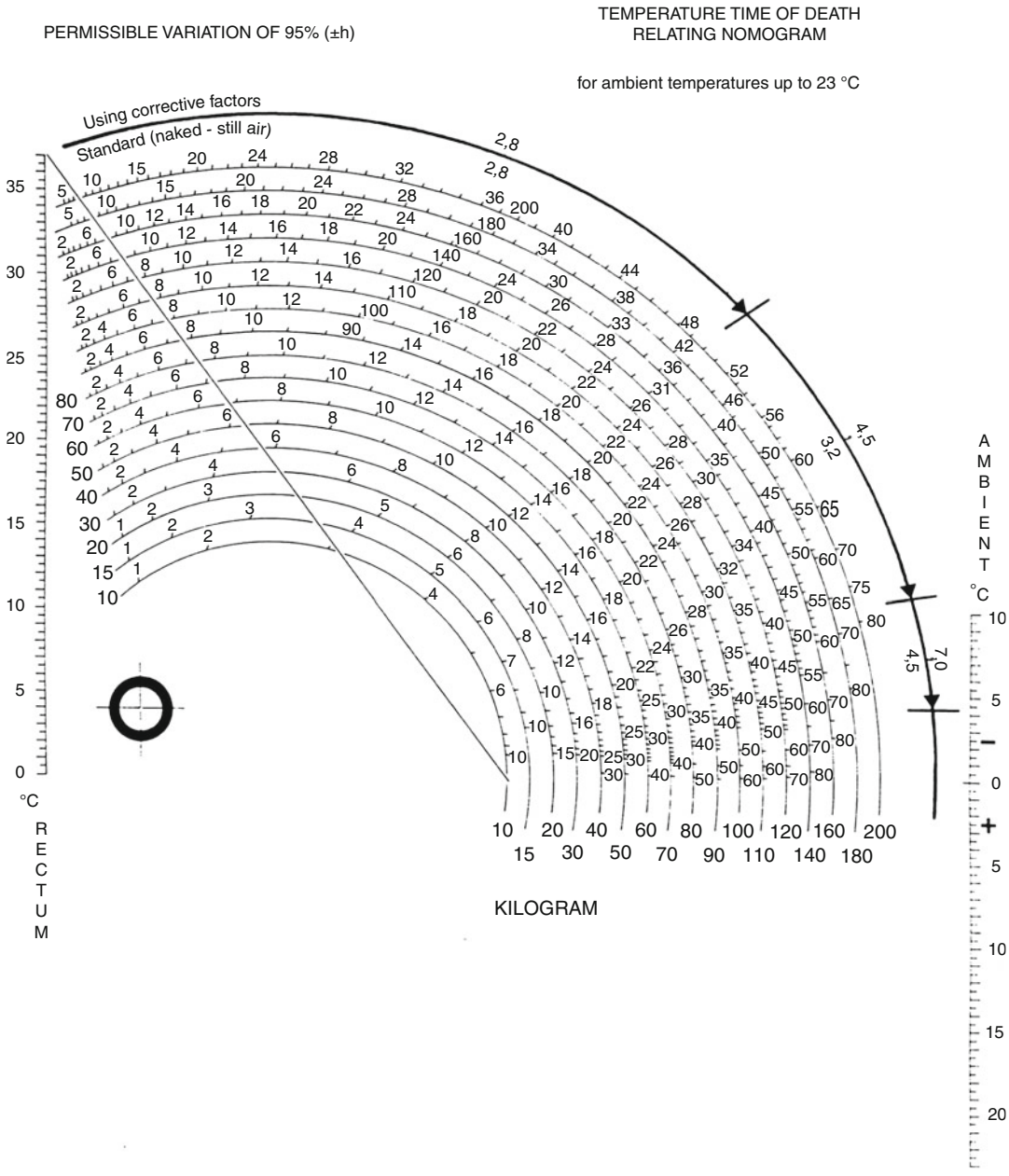

Fig. 3.6 (**a**) Henssge's nomogram method for estimating time since death from a single rectal temperature where the environmental temperature is below 23 °C (Henssge et al. 2002). (**b**) Henssge's nomogram method for estimating the time since death from a single rectal temperature where the environmental 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)

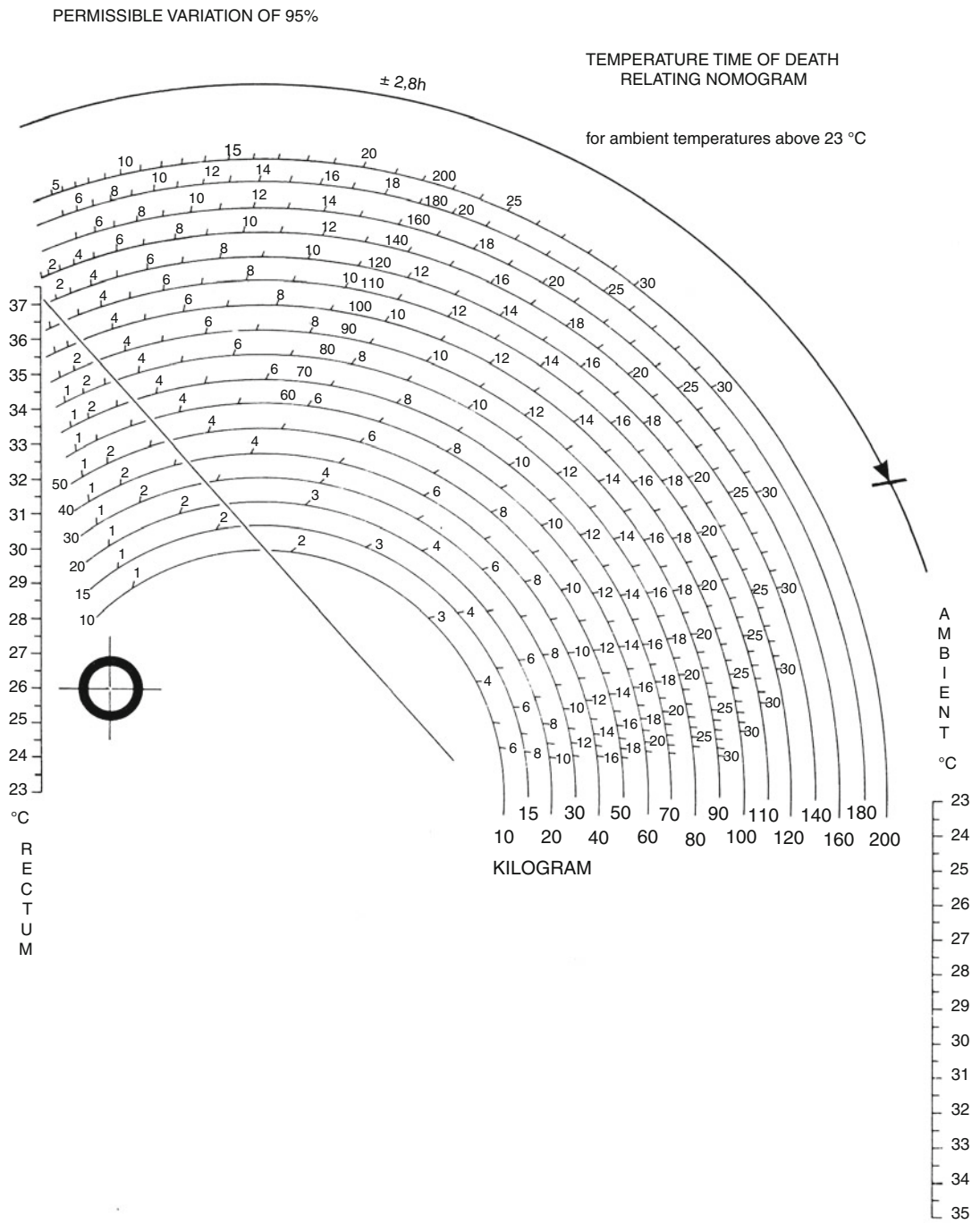

Fig. 3.6 (continued)

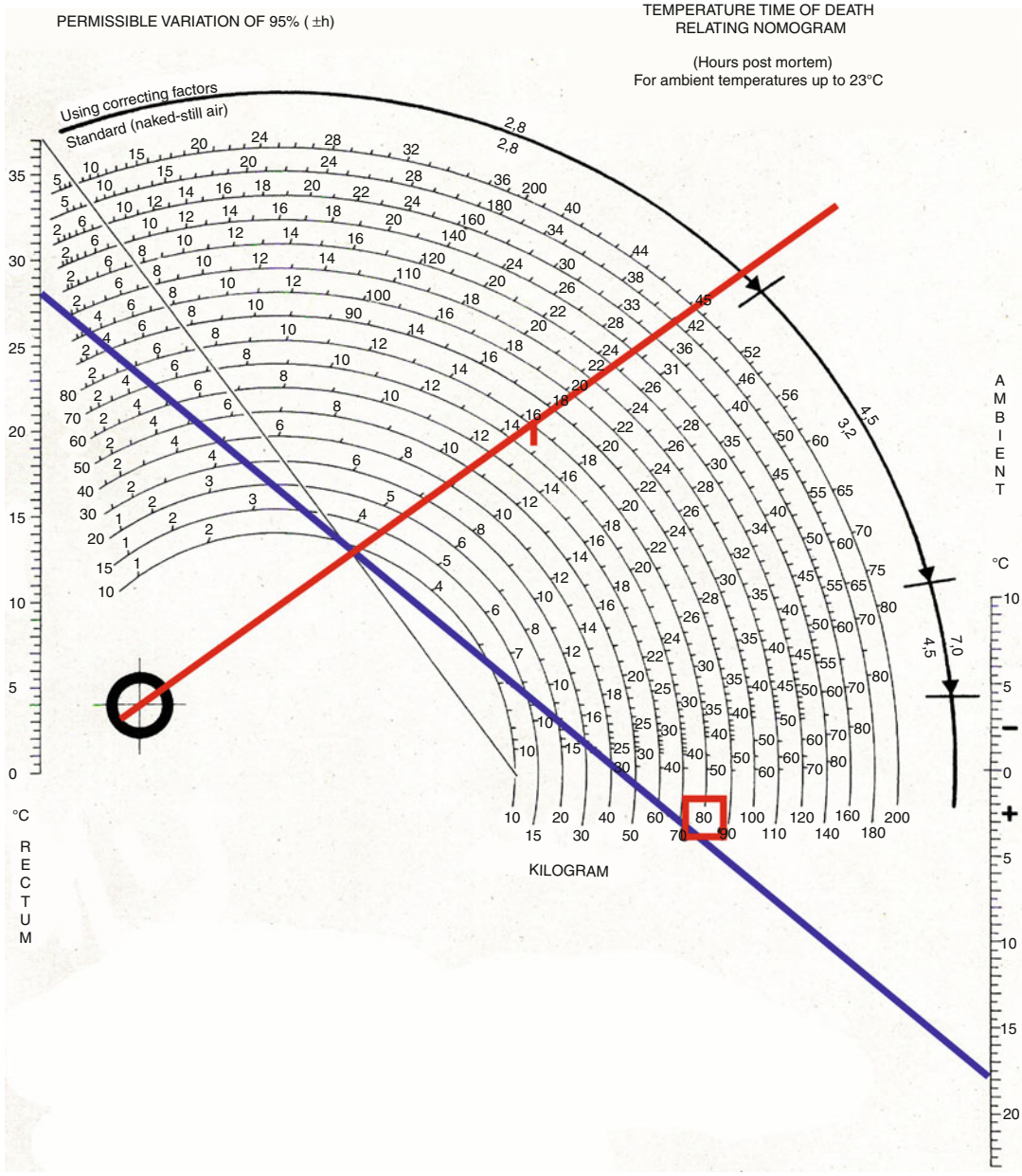

Fig. 3.7 Henssge's nomogram method for estimating time since death. A rectal temperature of 28 °C and an ambient temperature of 18 °C at a bodyweight of 80 kg yield an average time since death of approximately 16 h with 95 % tolerance levels of ±2.8 h (Modified from Reimann et al. (1990))

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,

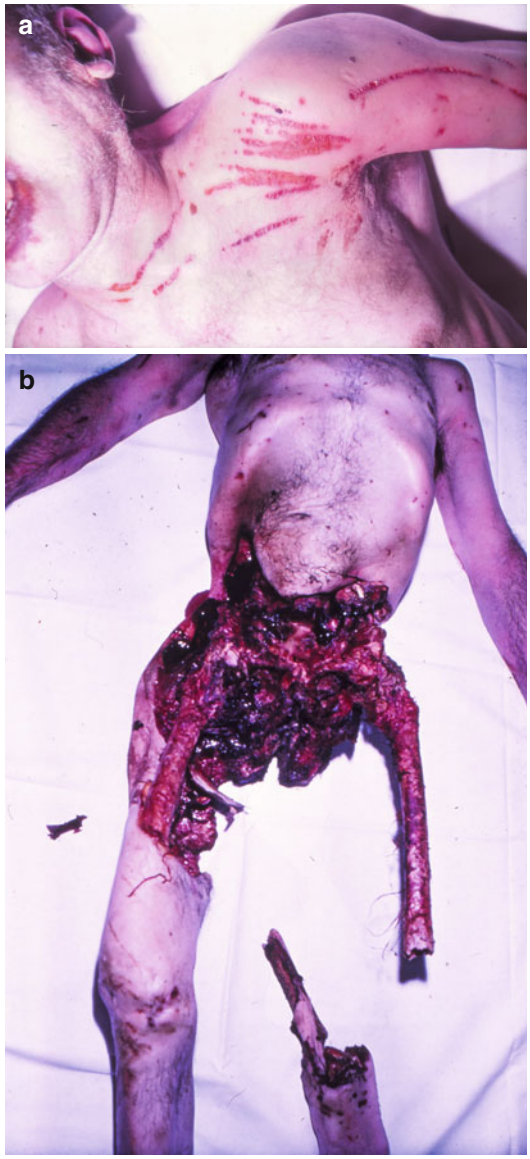

Fig. 3.8 (**a**) Striated and dried-out skin abrasions following dog predation (Alsatian). (**b**) Extensive traces of scavenging next to the body after it had been enclosed at home for an extended period of time

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

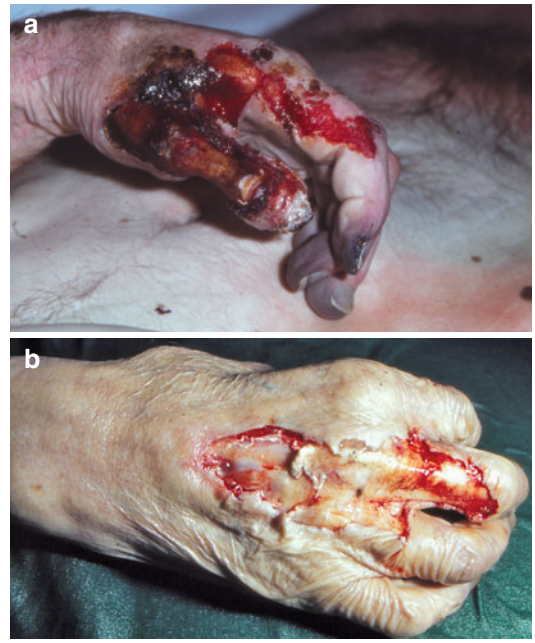

Fig. 3.9 (**a**, **b**) Rat scavenging defects on the hand with gently curved wound edges

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.

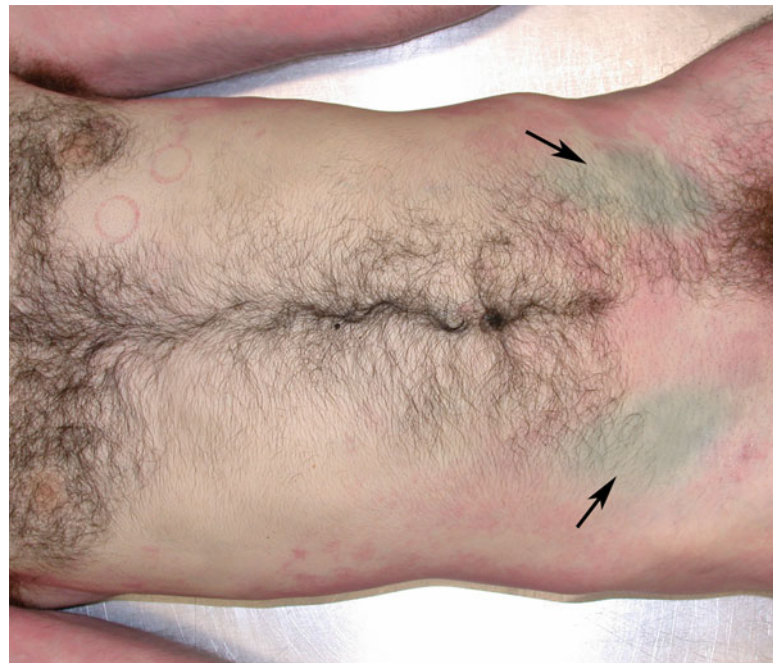

Fig. 3.10 Putrefaction. Early greenish putrefaction of the skin in the right and left lower abdomen (*arrows*)

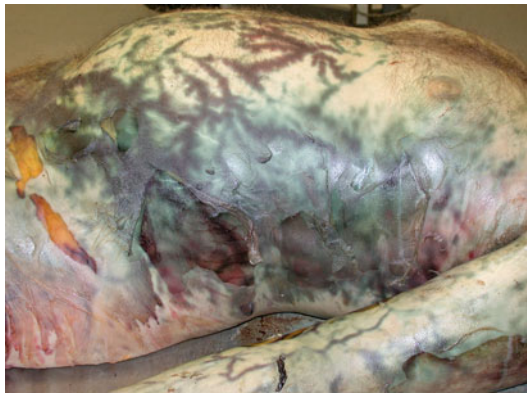

Fig. 3.11 "Marbling," whereby the venous network becomes visible through the skin. Postmortem putrefaction with abdominal distension due to gas formation, marbling, and the rupture of previously fluid-filled putrid blisters

- 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.

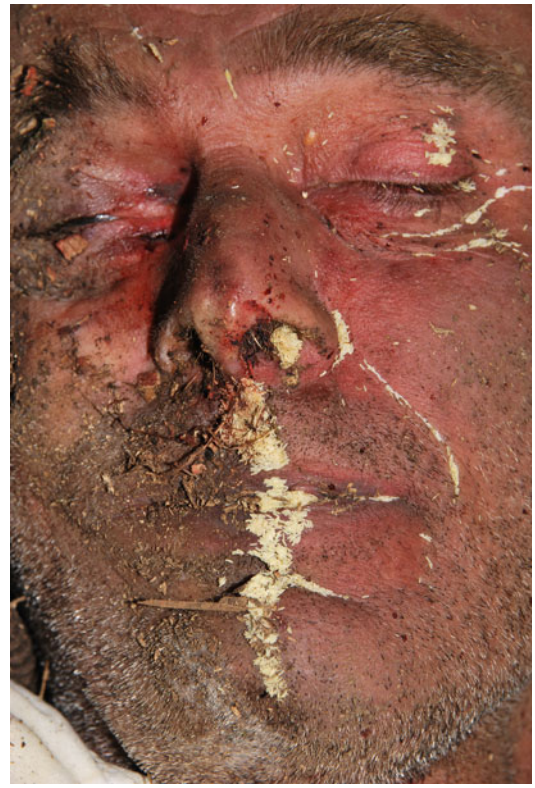

Fig. 3.12 Fly egg deposition. Abundant whitish-gray fly eggs deposited on the eyelids, nostrils, and mouth region post-mortem

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.

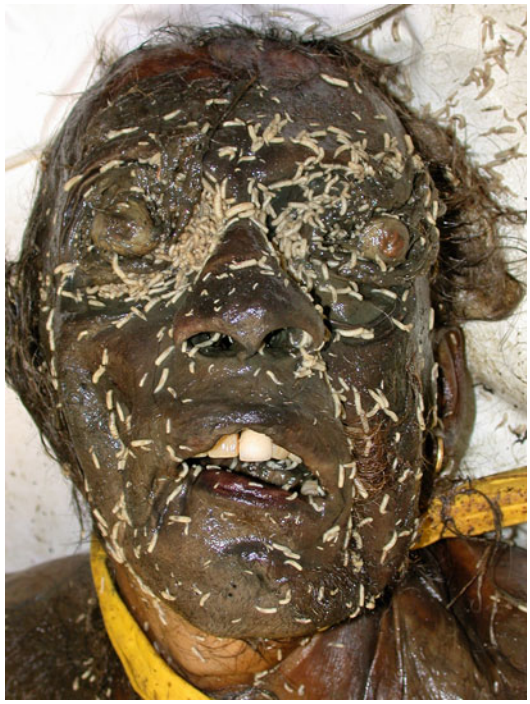

Fig. 3.13 Advanced putrefaction and extensive maggot colonization

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

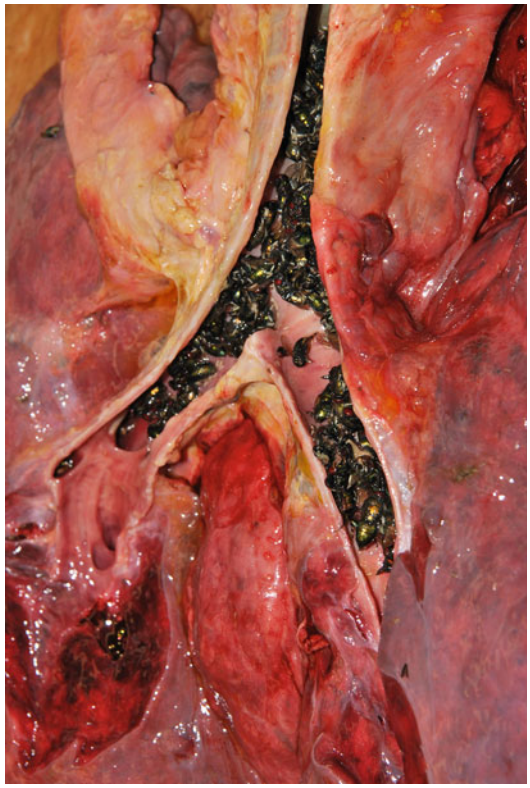

Fig. 3.15 Flies found at autopsy in the entire upper respiratory tract and extending to the main bronchi

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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