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

R.B. Dettmeyer et al., *Forensic Medicine*,
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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

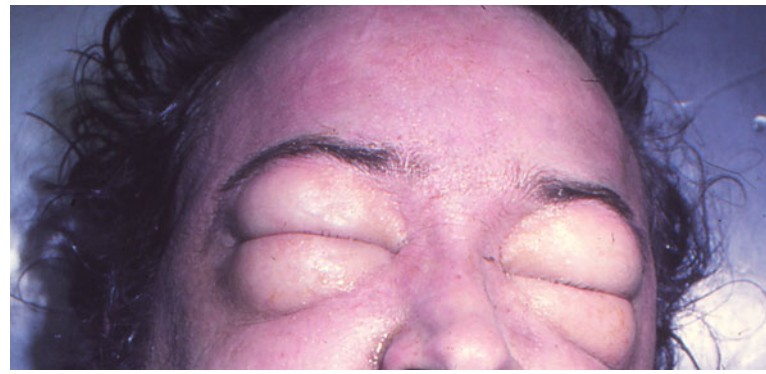

Fig. 7.1 Injury-related dispersion of air to soft tissues

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

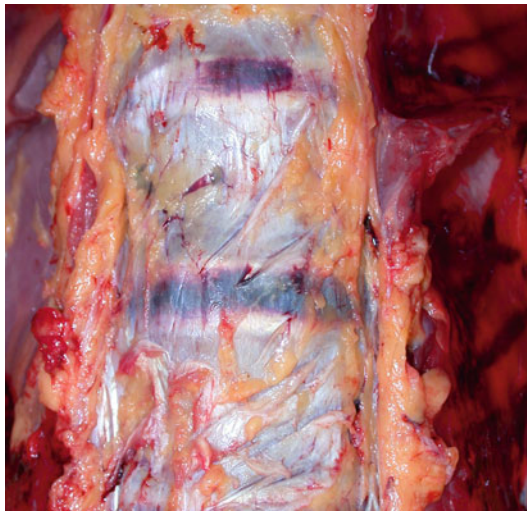

Fig. 7.2 "Simon's hemorrhage" into the intervertebral discs is a sign of vitality in death by hanging

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,

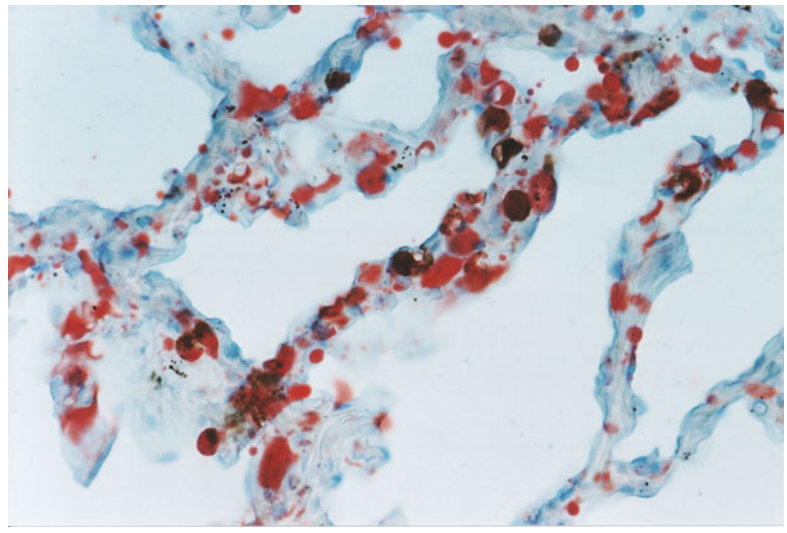

Fig. 7.3 Pulmonary fat embolism with fat droplets in the capillaries of the alveolar septa: a polytrauma patient after a traffic accident and a survival time of approximately 9 h (Sudan III ×400)

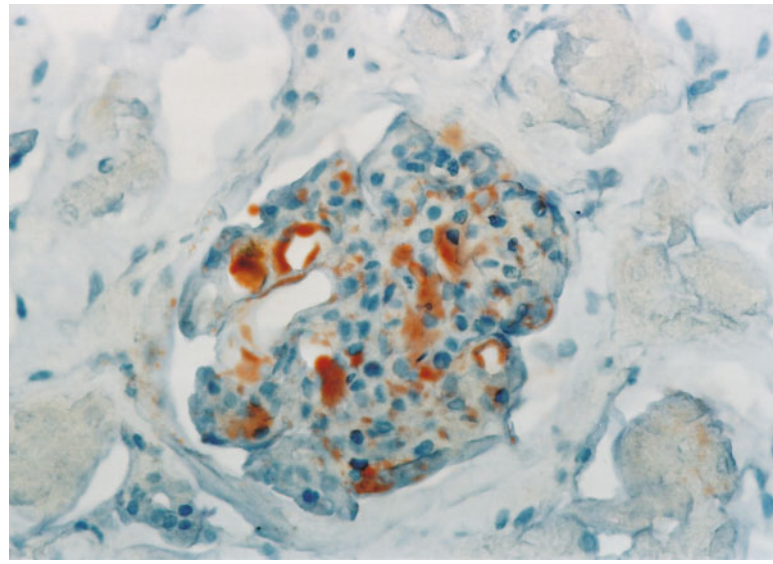

Fig. 7.4 Renal glomerular fat embolism in a polytrauma patient: a traffic accident victim with a survival time of approximately 6 h (Sudan III ×400)

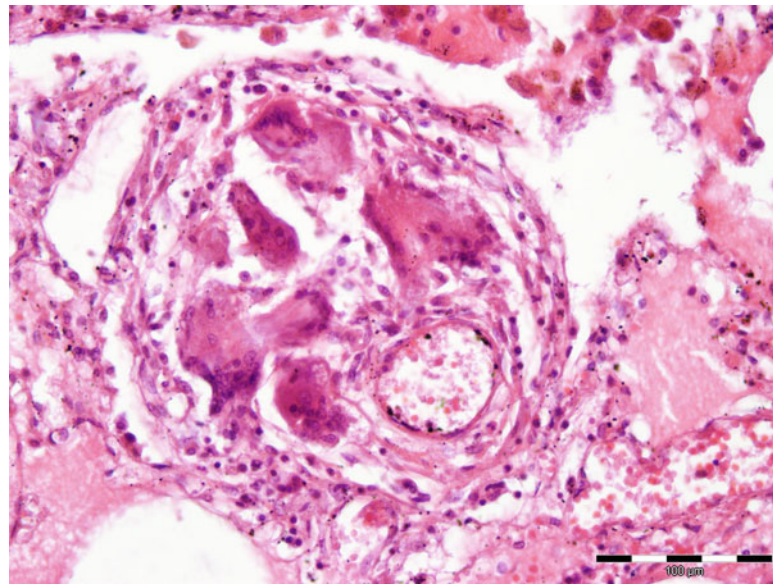

Fig. 7.5 Pulmonary granuloma in a case of junkie pneumopathy: embolically spread foreign material following intravenous injection and a foreign body reaction (H&E ×400)

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;

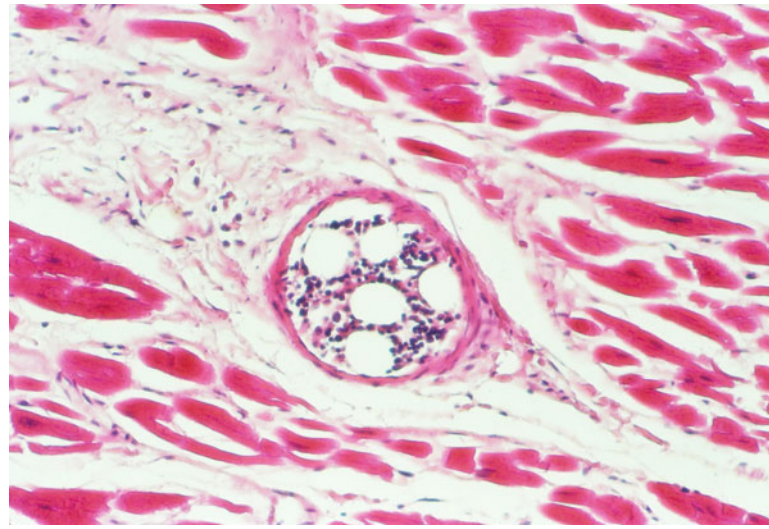

Fig. 7.6 Pulmonary bone marrow embolism: death on the operating table during implantation of a femoral head endoprosthesis following femoral neck fracture (H&E×100)

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.

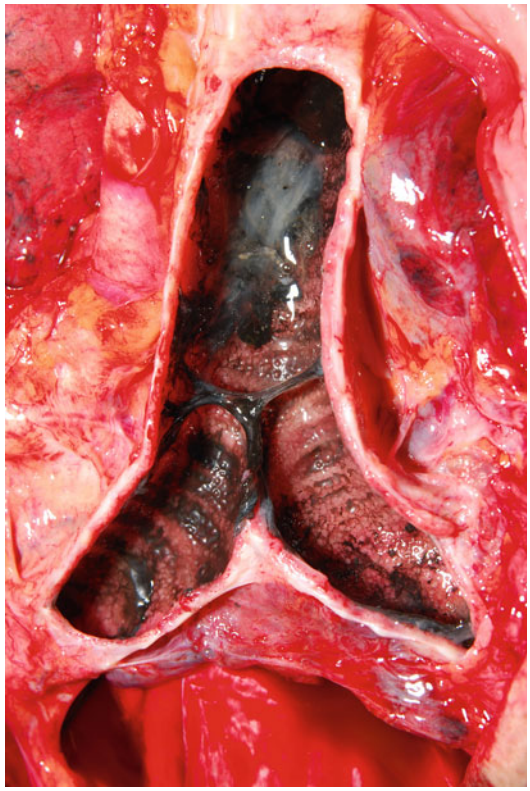

Fig. 7.7 Deep aspiration of soot particles as a sign of vitality at the time a fire broke out

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

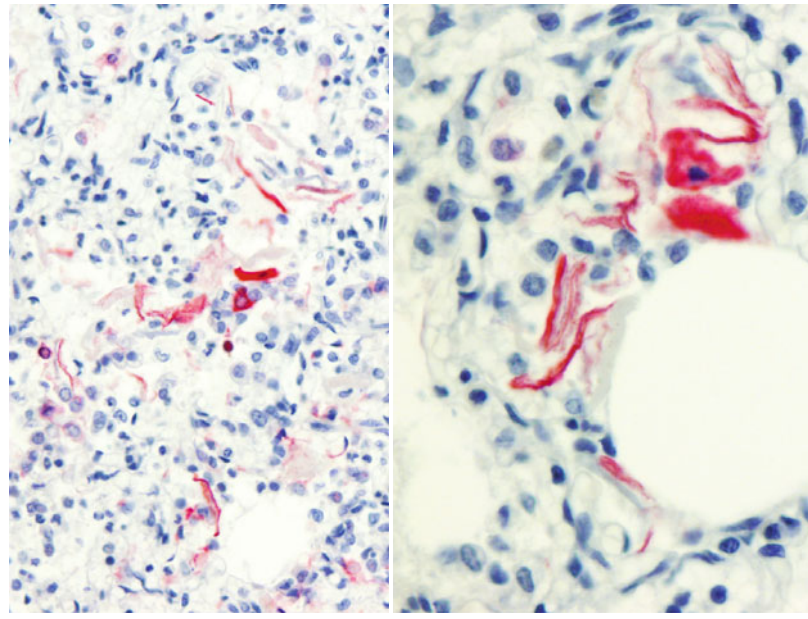

Fig. 7.8 Neonaticide: positive hydrostatic test (see Figs. 20.2 and 20.3). Lung tissue following amniotic fluid aspiration: intra-alveolar, partially anucleated keratin lamellae exfoliated from the fetal epidermis can be seen using immunohistochemistry (anti-cytokeratin ×250; ×400)

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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