POSTMORTEM CHANGES

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Overview

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Introduction

The changes and underlying biologic processes that a human body or its remains undergoes after death are complex and, as with other biologic phenomena, there is a broad range of variables influencing postmortem changes by altering the underlying progress of tissue destruction. As a general rule, changes in ambient (environmental) temperature tend to alter the rate but not the underlying biological mechanisms of postmortem changes. A summary of the main intrinsic and extrinsic factors accelerating or decelerating the onset and extent of postmortem changes is given in Table 1.

It is generally impossible to draw any definite conclusions concerning the time of death from the appearance of a single postmortem change, or to predict what postmortem changes are to be expected after a particular postmortem interval has elapsed. Despite this, in the early postmortem interval (approximately within 24 h of death), in some cases the presence of several postmortem changes may, when analyzed together with the deceased's rectal temperature, give the death investigator valuable hints concerning the timeframe in which the subject probably died. An experienced forensic entomologist can occasionally narrow the date-of-death window even in the very late postmortem period, whereas the forensic pathologist or medical examiner, respectively, working with the signs of putrefaction, adipocere, mummification, or skeletonization will only be able to give broad estimations in such cases. However, this article does not focus on detailed aspects of estimation of the time elapsed since death but rather concentrates on the morphology and conditions in which different postmortem changes may present.

While most postmortem changes discussed in this article are, at least to a certain degree, frequently observed in the death investigator's practice, some postmortem changes shown here may occur only occasionally and under specific intraindividual or environmental conditions. The inexperienced or unwary may interpret such unusual postmortem changes incorrectly, especially in curious death scene scenarios and when found in fatalities with additional signs of external violence preceding death. In such cases, hasty conclusions may lead the investigative enquiries in the wrong direction or, in the worst case, to a miscarriage of justice. Therefore, apart from giving a synopsis of common and uncommon postmortem changes seen in the death investigator's daily practice, it is also the aim of this article to draw the reader's attention to potential differential diagnoses between postmortem changes and vitally acquired body alterations and the pitfalls they may contain.

In the following context, death is defined as the irreversible cessation of blood circulation.

Table 1 Intrinsic and extrinsic factors influencing the onset and extent of postmortem changes

Acceleration of onset and extent of postmortem changes
Death occurred in a hot, moist environment/under high ambient temperatures
Subject is overweight/has a high fat content
Subject suffered/died from underlying infection or sepsis
Subject was intoxicated (e.g., with illicit drugs such as heroin)
Subject suffered/died from open wounds (perforating/penetrating traumatic injuries such as stab wounds, gunshot wounds, impalement injuries) or during surgical procedures
Body surface is insulated by warm clothing or other covering
Considerable time interval elapsed after death until artificial cooling of the body
Deceleration of onset and extent of postmortem changes[a]
Death occurred in a cool (cold), dry environment/under low ambient temperatures[b]
Subject was scantily dressed/naked/undressed shortly after death
Subject was stored in a cooling apparatus shortly after death

[a]All these factors slow the rate of postmortem changes but in general do not alter the underlying postmortem biologic processes.
[b]In addition to slowing the onset and extent of postmortem changes, low ambient temperature has a considerable impact on a delayed manifestation of odor of the body and thereby on the attraction of insects to the body, thus making the human remains less olfactorily absorbing for carnivores and rodents.

Livor Mortis

Livor mortis (synonyms: livores, postmortem lividity, postmortem hypostasis) is visible as a usually bluish-violaceous to purple coloration appearing on lower (dependent) parts of the body within 30 min to 3 h after death.

After the cardiovascular system has ceased to function, under the influence of gravity, movement of blood into the dependent parts of the body occurs (Figure 1). Livores correspond to hypostasis (gravitational pooling of blood) into the capillaries within the dermis (consisting of the papillary and reticular layer) in the dependent parts of the body. Therefore, when a subject has died in a prone position, livores will spread over the front of the body, while when death has taken place in a supine position, livores will spread over the back of the body (Figure 2).

Patterned Appearance and Contact Blanching of Livor Mortis

Livor mortis is frequently patterned since the appearance of livores is hindered when the vessels in dependent parts of the body are obstructed due to outer-surface compression, for example, when prominent parts of the body such as areas over bony structures firmly adhere to a rigid surface due to the weight of the body or when tight clothing compresses the involved vessel lumina (Figures 3 and 4). In such areas, livor mortis is absent: the involved outer-body surface appears pale to white, in contrast with the surrounding livor mortis. This contact blanching may image the exterior of objects that were in contact with the dependent parts of the body surface during livor mortis formation and occasionally the distinctive morphological appearance of contact blanching may give the death investigator valuable hints about the case in question (Figure 5).

Creasing of the skin or tight clothing may produce contact blanching on the neck which may resemble ligature marks (Figure 6). Therefore, knowledge of the position of the head and neck as well as the cloth worn at the time of death is important for the death investigator.

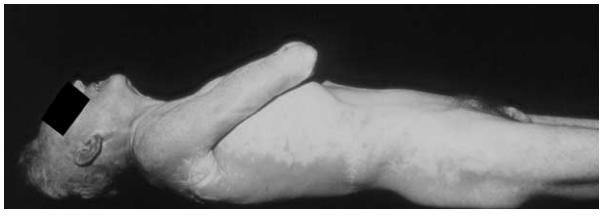

Figure 1 Livor mortis in dependent areas of the body.

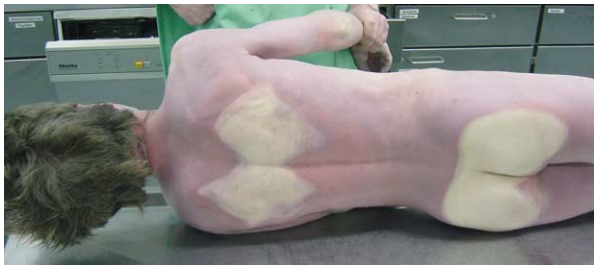

Figure 2 Intense livor mortis formation on the posterior parts of the body surface with typical contact blanching over the scapular region and buttocks.

Chronological Sequel of Livor Mortis Formation

After a first patchy development of livor mortis within 30 min to 3 h after death, livores become confluent. Under moderate to cool climatic conditions, livores are usually fully developed within 4–8 h postmortem, reaching their maximum intensity after an average of 10 h postmortem (Table 2). Livor mortis is most intense in cases of sudden death with a short agonal period and a great circulating blood volume.

In the early postmortem interval, roughly until 12–18 h after death, livor mortis is not yet fixed. Nonfixation of livor mortis means that it can be blanched when a blunt object such as a finger, the hand, or an instrument is pressed against the skin in areas of livores formation (Figure 7). This selective pressure forces the blood from the engorged capillaries, resulting in a pale to white blanching which quickly refills. A similar phenomenon results if the body is moved into a new position. Livor mortis will then shift to the dependent parts of the body as a result of body movement. The capability of livores to shift as a result of the gravitational movement of blood is assumed to depend on a prevailing number of intact erythrocytes within the vascular system: selective pressure moves the blood cells within the vessels. However, this assumption has been questioned more than once by different authorities.

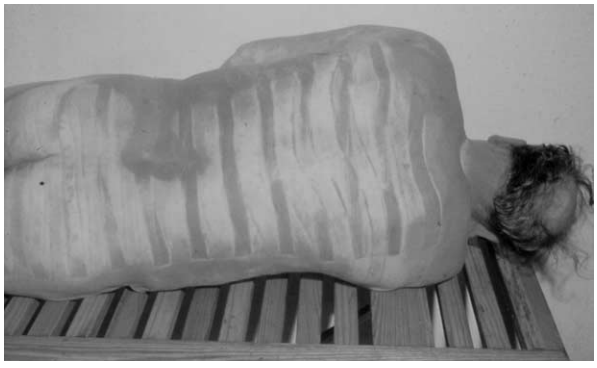

Figure 3 Patterned appearance of livor mortis on the back of the body.

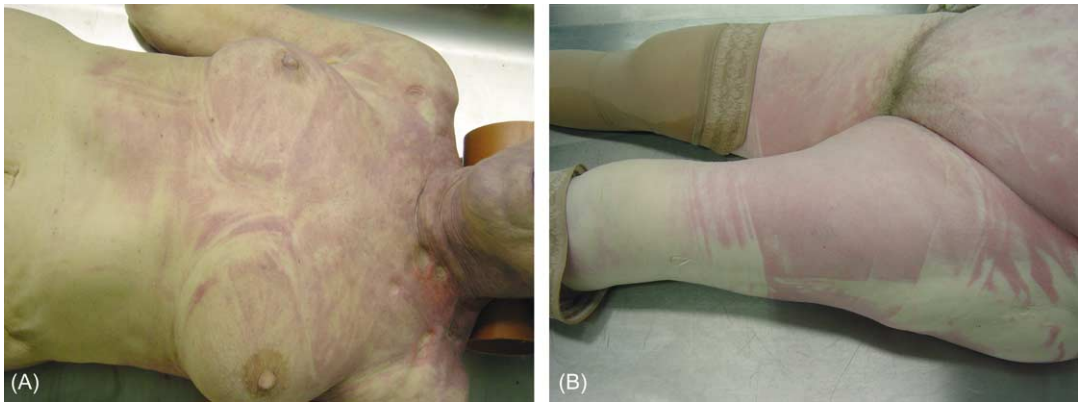

Figure 4 (A), (B) Contact blanching of livor mortis induced by tight clothing.

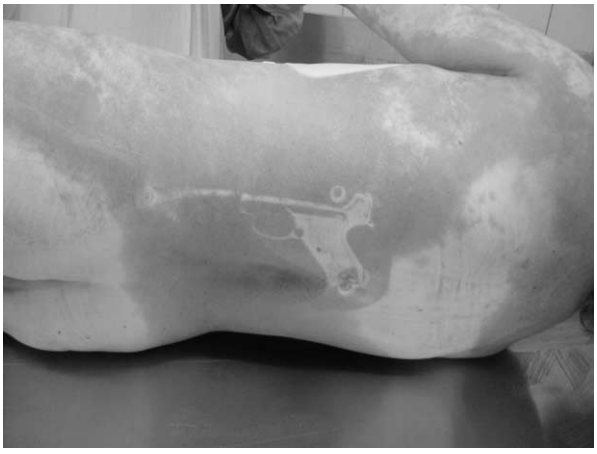

Figure 5 Contact blanching on the back of the body depicting a pistol.

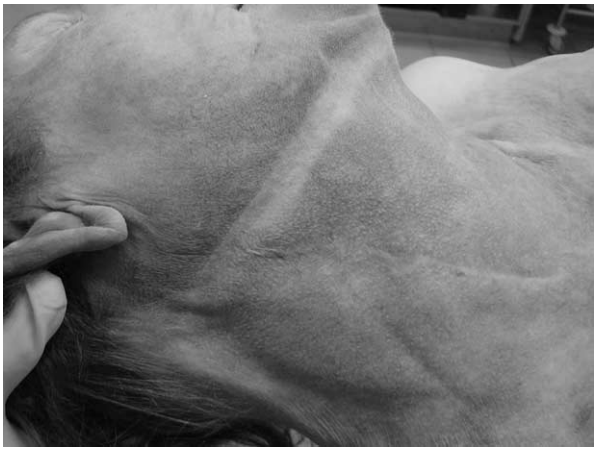

Figure 6 Contact blanching upon the skin of the anterior neck that should not be confused with a ligature mark.

After 18–24 h, livor mortis becomes fixed: livores cannot be blanched by selective pressure on the body surface and the effect of gravity does not move

Table 2 Sequential order of usual appearance of the different states of livor mortis under moderate to cool climatic conditions

Finding	*Earliest appearance*	*General appearance*	*Latest appearance*
Patchy beginning of development	0.5 h p.m.	2 h p.m.	3 h p.m.
Full development, confluence	4 h p.m.	6 h p.m.	8 h p.m.
Reaching maximum of intensity	6 h p.m.	10 h p.m.	16 h p.m.

p.m., postmortem.

livores. The time of onset of fixation of livores mostly depends on the ambient temperature to which the body has been exposed: high ambient temperatures are positively correlated with an early onset of fixation of livor mortis. When livor mortis is fixed, a change in body position will have no effect on the original pattern of livores. Fixation of livor mortis is considered a result of hemolysis of the blood serum: with the breakdown of erythrocyte membranes during autolysis, the erythrocytes become pervious for hemoglobin and its derivates: subsequent diffusion of hemolytic blood serum through the walls of the vessels is involved in livores formation. In this case, selective pressure over an area of livor mortis will have no noticeable effect on the movement of blood cells within the vessels or on the hemolytic coloration of the surrounding tissue. This theory has also been doubted, but the underlying pathophysiological mechanisms that are decisive for nonfixation or fixation of livores have so far shown no real practical value.

It should be mentioned that cases have been reported where shifting of livores has been observed, even after 48 h or more but this phenomenon is mostly restricted to cases with cold ambient temperatures.

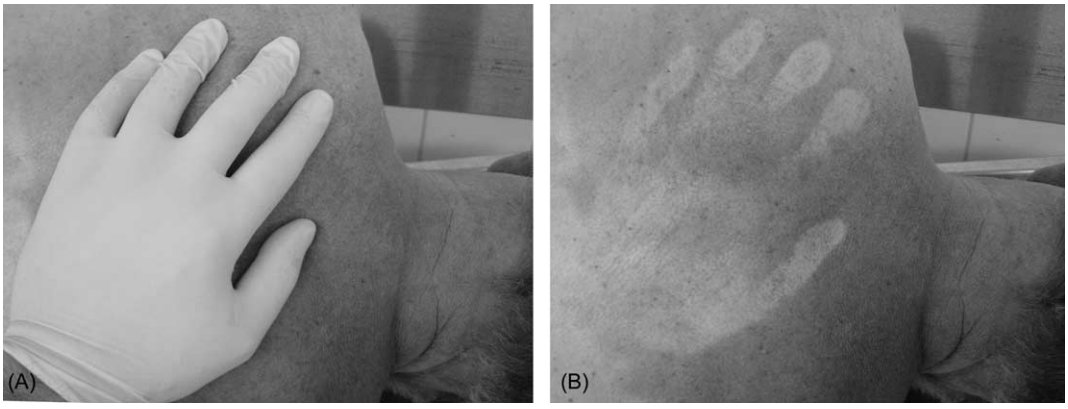

Figure 7 (A), (B) Contact blanching of livor mortis (within the state of nonfixation of postmortem lividity six hours after death of this individual). Selective pressure with the hand leads to a pale to white area of blanching which quickly refills.

Color of Livor Mortis

In the early phase of their formation, livores have a reddish color, due to the prevailing number of erythrocytes carrying oxygenated hemoglobin. With an increase in the length of the postmortem interval, livores become darker, and when fully developed, the normal color of livor mortis is bluish-violaceous to purple. This is a result of oxygen dissociation from both the postmortem hemoglobin of erythrocytes and continuous oxygen consumption from cells that initially survive the cessation of cardiovascular function (e.g., skeletal muscle cells survive cessation of the cardiovascular system for 2–8 h). The resulting product is deoxyhemoglobin, which is bluish-violaceous to purple in color.

Light reddish/pink livores Light reddish or pink livores are frequently seen in carbon monoxide poisoning, fatal hypothermia, cyanide poisoning, or in bodies deposited postmortem in cold ambient temperatures.

A light reddish or pink, sometimes described as "cherry-red," coloration of livores is classically seen in carbon monoxide poisoning as a result of carboxyhemoglobin formation (Figure 8). The assumption that a bluish-violaceous color of the matrix of the nails, when found together with a light reddish color of livores, refutes carbon monoxide poisoning as being responsible for the reddish coloration of livores must be vehemently contradicted. Tsokos and coworkers have seen cases of fatal carbon monoxide poisoning where the matrix of the nails showed a bluish-violaceous color despite carbon monoxide hemoglobin concentrations of 50% and more. In such doubtful cases, laboratory testing of heart blood samples should be carried out immediately to avoid exposing other individuals to danger at the scene of death.

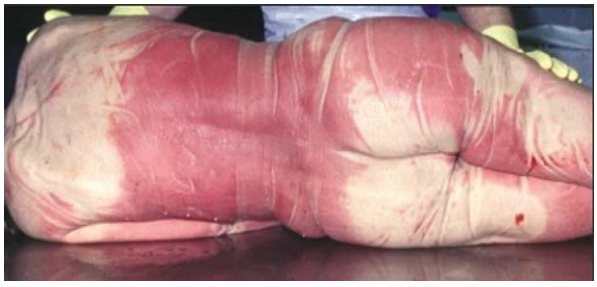

Figure 8 Pink (''cherry red'') coloration of livores in carbon monoxide poisoning.

Light reddish or pink livores are also frequently seen in fatal hypothermia cases since cold ambient temperature inhibits dissociation of oxygen from the hemoglobin. Oxygenated hemoglobin has a lighter red color than deoxyhemoglobin. Under cold ambient temperatures (roughly below 15 °C), reoxygenation of hemoglobin slowly occurs postmortem, and this is the explanation for the light red color of livores seen in bodies after storage in a cooling apparatus postmortem (Figure 9).

In cyanide poisoning, the cyanide inhibits dissociation of oxygen from the hemoglobin by blocking cytochromoxidase activity, leading to a light reddish or pink coloration of livor mortis.

Brownish color of livores A brownish, sometimes described as "chocolate," color of livor mortis is seen in poisoning with nitrates, nitrites, or sodium chloride (Figure 10). The reason for this brown coloration is the formation of methemoglobin.

Greenish color of livores Livores often turn partly green under the influence of putrefaction processes due to hemoglobin conversion into sulfhemoglobin.

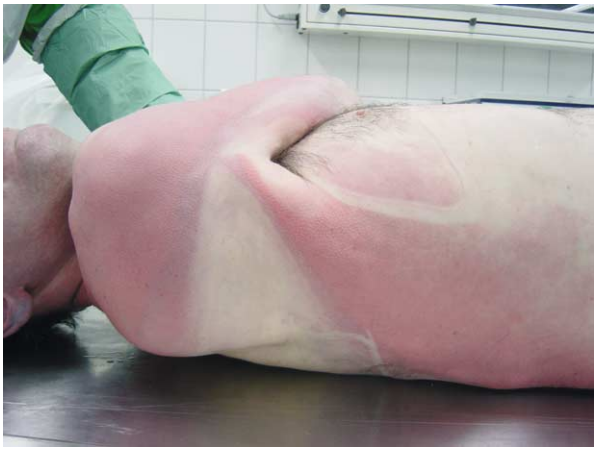

Figure 9 Light reddish/pink livores due to reoxygenation of hemoglobin after storage of the body in a cooling apparatus. Note the bluish-violaceous marginal zone next to the contact blanching over the scapular region which is a result of anewed oxygen dissociation from the hemoglobin of erythrocytes after the body was brought into the relatively warmer temperatures of the autopsy room.

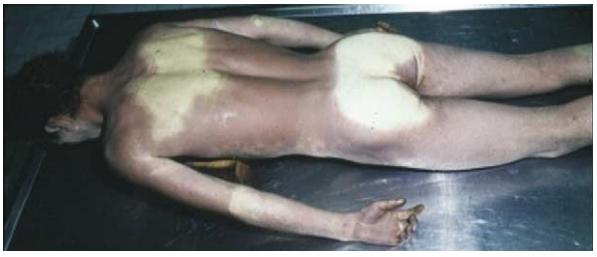

Figure 10 Brownish color of livor mortis in nitrate poisoning.

Livor Mortis in Internal Organs

Comparable with the development and their location of appearance on the outer body surface, livores are found in dependent parts of internal organs such as the lungs, heart, liver, and kidneys.

In the lungs, blood and transudation accumulation, the latter due to circumscribed hypostasis, may occasionally be confused with edema of the lungs or pneumonia.

In the heart, internal livores, demonstrated by a reddish discoloration zone in the myocardium, may mimic fresh infarction. The exact location of livores in the myocardium depends on the posture of the body after death.

In the liver, when livores have developed in a right-sided position of the body, contact blanching deriving from the ribs may be observed.

Difficulties arising from the presence of livor mortis in internal organs and their differentiation from underlying diseases are easily solved by microscopic examination.

Absence of Livor Mortis

Livores may be sparse or even absent in fatalities where there was considerable blood loss before death, whether from internal sources (e.g., gastrointestinal bleeding) or as a result of external hemorrhage (e.g., stab wounds, traumatic amputation of limbs). In most such cases, the external examination will explain inconspicuous or absent livores by revealing the source of bleeding (e.g., blood smears resulting from hematemesis or melena (Figure 11), external injuries). The total absence of livores necessitates a blood loss of at least 65% of the circulating blood volume in adolescents and 45% in infants. In cases with antemortem anemia (e.g., aplastic anemia, autoimmune hemolytic anemia, anemia secondary to malignancy, malnutrition, or infection) livor mortis will be unnoticeable, depending on the hemoglobin count in the circulation before death.

In sun-tanned or dark-skinned individuals, livores may be difficult to establish or unnoticeable.

In drowning deaths, depending on the depth and time the body was under water, livores may not develop since the vessels beneath the outer surface of the body that are normally involved in livor mortis formation are obstructed due to compression by surrounding hydrostatic water pressure. If the body is recovered from the water within roughly 24 h, livores may develop in the then dependent parts of the body, but this will depend on the water temperature. In bodies recovered from cold water, this phenomenon may be observed even after a postmortem interval of 48–72 h.

Criminalistic Aspects

When a subject has died in a supine position, livores will spread over the posterior side of the body, and when death took place in a prone position,

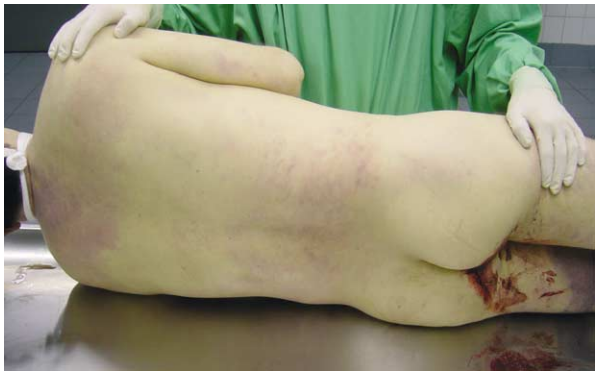

Figure 11 Sparse postmortem lividity due to fatal anal blood loss from a carcinoma of the rectum. Note blood smears around the anal orificium.

livores will spread over the anterior side of the body. However, the reverse assumption, that a deceased who has livores on the back of the body has died in a supine position or that livores found on the front of the body indicate that this person died in a prone position, may be misleading since, as mentioned above, livores have the ability to shift when the body is moved to a new position before livor mortis has become fixed.

In hanging deaths, livor mortis will be apparent in the dependent parts of the body (e.g., glove-like and stocking-like appearance of livores on the lower parts of the arms and legs, respectively). However, the finding of livores in the dependent parts of the body corresponding to hanging does not unequivocally exclude that this body has been transferred into this posture postmortem, for example, to conceal a homicide.

If a body is found in a supine position but livor mortis is seen on the anterior side of the body, this implies that the body has been moved a considerable time after death, after fixation of livor mortis.

Differential Diagnoses

Livor mortis may be confused with bruising by the inexperienced, although this is rare. Within the first 24 h after death, evidence of contact blanching caused by selective pressure to the outer body surface will help to differentiate between bruising and livores, since application of surface compression to an area of bruising will not cause blanching. In the later postmortem interval, the incision of a doubtful skin area will make a clear distinction since no hemorrhage will be apparent in the soft tissue beneath livores formation.

Frost erythema, hypothermia-induced red-purple spots seen over prominent bony parts of the body such as the shoulder, knee, or elbow joints, may occasionally be mistaken for livores by the unwary (Figure 12). Bearing in mind that frostbite is regularly found on nondependent parts of the body and often shows no confluence, its differentiation from livores should not present any difficulties.

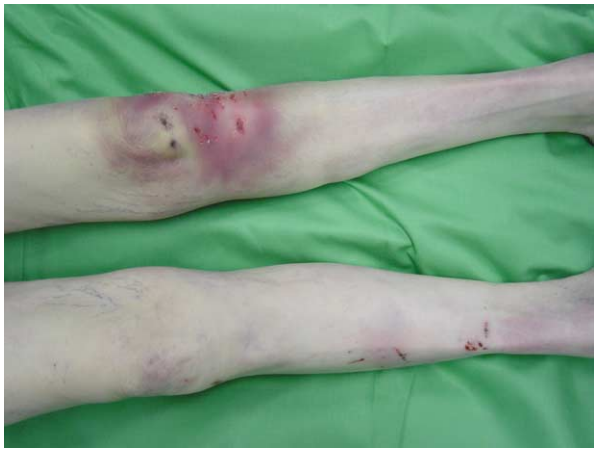

Figure 12 Frost erythemas over the left knee joint.

Vibices

Vibices (synonyms: death spots, postmortem ecchymoses) are tiny, often spot-like, sometimes confluent, oval to round, bluish-blackish hemorrhages of postmortem origin exclusively limited to areas of livor mortis (Figure 13). Vibices result from postmortem mechanical rupture of subcutaneous capillaries and smaller vessels (predominantly veins) due to an increase in intravascular pressure arising from pooling of erythrocytes in this vascular compartment under the influence of gravity during livor mortis formation. Histologically, erythrocytes appear intact within the vibices in the early postmortem interval (Figure 14). With increasing length of postmortem interval, under the influence of autolysis and putrefaction processes, vibices diminish in number and intensity due to the breakdown of erythrocyte membranes, with subsequent hemolysis and diffusion of hemoglobin and its derivates into the surrounding tissue.

When livores are sparse, vibices are usually absent. The formation of vibices depends on the total amount of circulating blood volume antemortem and is therefore more often seen in obese than in underweight decedents. The duration of the agonal period has no influence on whether vibices manifest or not.

Differential Diagnoses

Vibices should not be confused with petechial bleeding as a result of traumatic asphyxia or spot-like or more confluent cutaneous bleedings due to septic disseminated intravascular coagulation. As a general rule and helpful in the differential diagnosis is the fact that the appearance, intensity, and extent of vibices are positively correlated with that of livor mortis and therefore vibices are strictly limited to the body parts where livores are existent.

Rigor Mortis

Rigor mortis (synonym: postmortem rigidity) is the stiffening of muscles after death. Rigor mortis is preceded by a total (primary) relaxation of the musculature immediately after death. Shortly thereafter, rigor mortis begins to appear in the muscles of the eyelids and the jaw (at the earliest 20 min postmortem); the jaw tightens due to stiffening of the masticatory

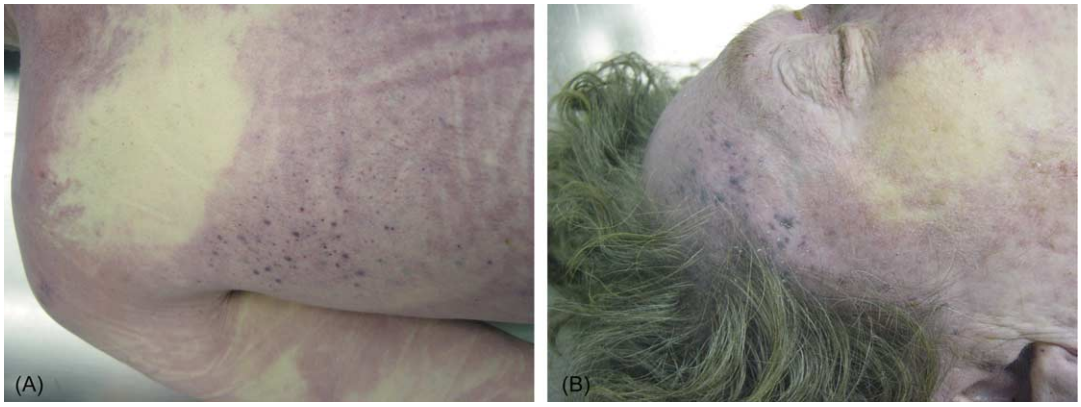

Figure 13 Vibices seen on the skin of the (A) back of the body and (B) temple.

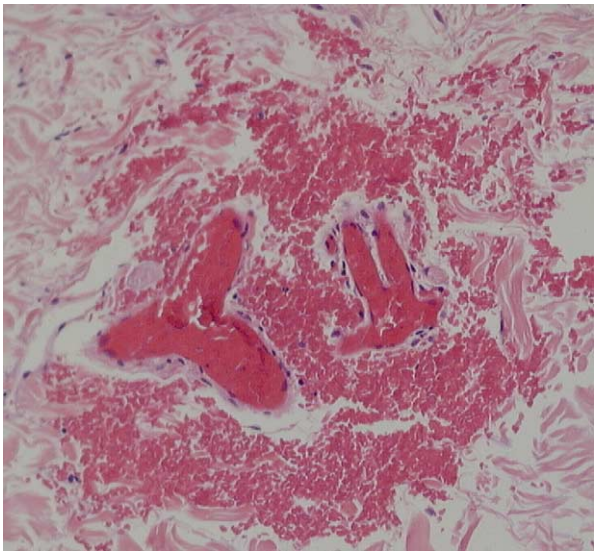

Figure 14 Histological appearance of vibices 8 hours postmortem. A zone of postmortem ''bleeding'' with intact erythrocytes lacking any surrounding inflammatory cellular response is seen next to two subcutaneous venules.

muscles. After that, postmortem rigidity begins to affect larger muscle groups with stiffening of elbow and knee joints 2–6 h after death. However, the rate of onset and time of full development of rigor mortis are highly variable and, as with all postmortem changes, for the most part are particularly dependent on the ambient temperature (high ambient temperatures accelerate the onset and intensity of rigor mortis, although extremes of cold have also been reported to produce a rapid onset of rigor mortis). In forensic pathological practice, the intensity of rigor mortis is assessed on a purely subjective basis by scrutinizing whether joints can be moved (flexion/extension) and if muscle stiffening offers resistance. As a result, determination of the state of rigor mortis varies greatly from one investigator to another. In addition, numerous intrinsic and extrinsic factors affect the development of rigor mortis and therefore using the state of rigor mortis to estimate the postmortem interval is of no real value.

Pathophysiology of Rigor Mortis Formation

Muscles are composed of myofibrils, which are again composed of myofilaments. Two types of myofilaments can be distinguished: actin and myosin. Under the influence of adenosine triphosphate (ATP), actin and myosin form a contractile compound, actomyosin, which is the basic source of energy for muscle contraction. After death, ATP formation terminates and ATP is continously consumed (to be precise, some ATP is still generated by anerobic glycolysis for a short period of time postmortem but this can be overlooked in the present context). With a decrease in ATP levels, actin and myosin enter into a nonshiftable and rigid state of adhesion until, under the influence of autolysis and putrefaction, protein disintegration of myofibrils leads to loosening of rigor mortis.

In mechanical terms, postmortem rigidity is characterized by a loss of muscle elasticity and plasticity, an increase in stiffness, and shortening of muscle length.

Experimental investigations have shown that the onset of rigor mortis is earlier and more rapidly progressive in red than white muscles, which has been attributed to ATP levels falling more rapidly after death in red than white muscles. However, this observation is highly academic and of no value for practical forensic casework.

Chronological Sequel of the Development and Disappearance of Rigor Mortis

In cool and temperate climate zones, rigor mortis is usually fully developed after 6–18 h (Figure 15). In high ambient temperatures the onset of rigor mortis is

accelerated and postmortem rigidity may even be fully developed as early as 1–2 h after death.

Any forceful physical exertion before death leads to a decrease in ATP levels within the musculature and therefore also accelerates the time of onset of rigor mortis (e.g., in death by drowning where there is a violent struggle during the drowning process). The onset of rigor mortis may also be rapid in babies and children or in deaths due to electrocution. When the onset of rigor mortis is rapid, its duration is usually shorter than in cases with delayed onset under equal ambient (environmental) temperatures.

Rigor mortis develops in all muscles at the same time and at the same speed. However, due to the different diameters of the muscles involved, postmortem rigidity becomes noticeable at first in smaller muscle groups. When fully developed, rigor mortis may lead to such a rigidity of the body that it may be capable of supporting the whole body weight (Figure 16). In such cases, even the most forceful efforts to break down rigor mortis may be fruitless.

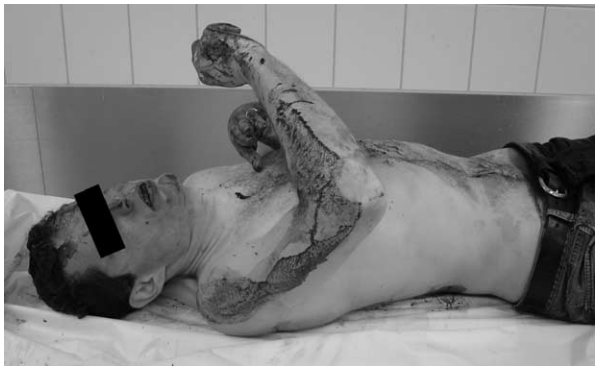

Figure 15 Fully developed rigor mortis in a case of homicide due to stabbing. At the death scene, the deceased was found in an upright to forward body posture leaning against a chair.

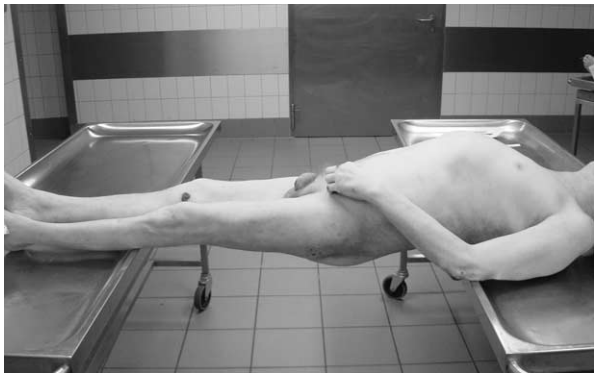

Figure 16 When fully developed, rigor mortis may lead to such a rigidity of the body that it may be capable of supporting the whole body weight.

The development of postmortem rigidity is usually progressive from the head down to the shoulder girdle and arms and then to the legs, a phenomenon that has been known since 1811 as "Nysten's rule." This finding can be explained by the greater diameter of the muscle groups located at joints that are farther down the body. But the exception proves the rule: after heavy physical exertion of the leg muscles, rigor mortis will develop earlier in the lower parts of the body than in the upper parts due to muscle activity in the legs with a resulting decrease in ATP levels.

When rigor mortis is broken, which means by forcefully stretching the joints against their fixation by rigor mortis, it will not return if it is fully developed. When only partly set, rigor mortis may redevelop again after having been broken.

In cool and temperate climates, loosening of rigor mortis reflected by a secondary relaxation of the muscles (in other words, a decrease in tension after full development of rigor mortis) begins 24–36 h postmortem, a finding that is again highly dependent on the ambient temperature (high ambient temperatures accelerate the time of onset of loosening of rigor mortis and therefore shorten its duration). Usually, rigor mortis disappears in the order in which it appeared but the finger joints usually remain stiff the longest.

In putrefied bodies, rigor mortis is absent. Rigor mortis may be weak or even unnoticeable in subjects who were suffering from a debilitating illness before death, in cachectic individuals, or those who died in an advanced state of multiple sclerosis, amyotrophic lateral sclerosis, or Duchenne muscular dystrophy.

Table 3 gives an overview of intrinsic and extrinsic factors that influence the onset of postmortem rigidity.

Table 3 Intrinsic and extrinsic factors influencing the onset of postmortem rigidity

Factors accelerating the time of onset of rigor mortis
Physical exhaustion before death (e.g., forceful muscular exertion during a fight or violent struggle during drowning)[a]
High body temperature/fever at the time of death[a]
Convulsions before death (e.g., due to underlying epilepsy or drug-induced)[a]
High ambient temperatures
Factors delaying the time of onset of rigor mortis
Debilitating diseases
Cachexia
Cool/cold ambient temperatures
Death following a short agonal period

[a] The accelerated onset of rigor mortis is mediated by a decrease of adenosine triphosphate concentration in such constellations.

Cadaveric Spasm

Reports of an instanteous appearance of full rigor mortis of the whole body immediately after death ("cadaveric spasm") occasionally appear in the literature. The true existence of cadaveric spasm is highly doubtful from an academic point of view since a satisfactory biological explanation for this phenomenon is lacking. When personal belongings or leaves are found grabbed in the hands (Figure 17), the logical explanation is that they were actually located under the palms when postmortem rigidity set in. Most reports of cases of cadaveric spasm derive from observations on the battlefields of World Wars I and II. In the overwhelming majority, the phenomenon of cadaveric spasm is easily explained by postmortem movement of the affected bodies due to blast waves from explosives hitting the battlefield. After blast wave-induced movement of the bodies, they were found in unreasonable positions that would not have been maintained during primary relaxation of the muscles after death. Another logical explanation in some cases is the fixation of tetanic convulsion in rigor mortis, which has been observed in rare cases.

Rigor Mortis in Internal Organs

Postmortem rigidity not only affects the skeletal muscles, it is also found in the myocardium as well as in internal organs such as the uterus, the gallbladder, or urinary bladder. As with the skeletal musculature, rigor mortis is preceded by a total primary relaxation of all muscles of internal organs. This muscle relaxation immediately after death explains leaking of urine or seminal fluid from the urethral orifice due to flaccidity of the urinary bladder and the pelvic diaphragm, respectively.

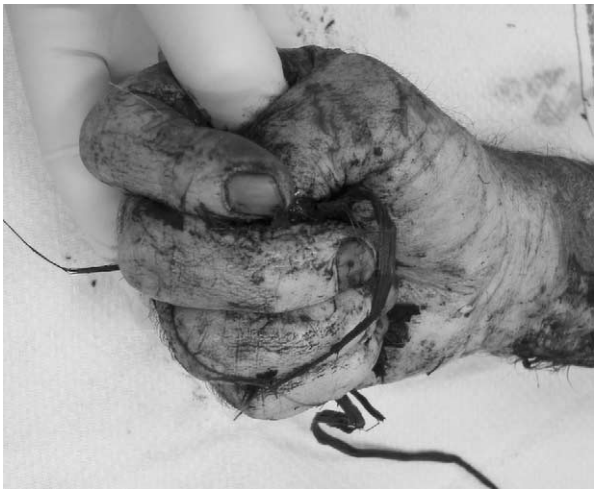

Figure 17 Leaves found grabed within the hands of a body recovered from water (no ''cadaveric spasm''!).

The finding of "gooseflesh" after death is the result of postmortem contraction of the erector pili of the skin.

Criminalistic Aspects

Rigor mortis is occasionally helpful in determining whether a body has been moved after death. If a body is found in an illogical posture, such as a body position that would not have been maintained under the influence of gravity during primary relaxation of the muscles after death, this position implies that the body has been moved after the development of rigor mortis.

Rigor mortis may make examination of the palms and the inside aspects of the fingers difficult so that current marks or defense injuries located here may be overlooked.

Particularly in children, a marked anal dilatation may be observed postmortem. As mentioned before, immediately as death occurs and preceding the onset of rigor mortis, the whole body musculature loses its tone. In children, rigor mortis may fix a dilated anal orifice and this finding may persist after rigor mortis has faded. Therefore, anal dilatation alone is not a sufficient marker for penetrative anal abuse before death.

Muscle relaxation immediately after death with opening of the eyes and the mouth and subsequent fixation in rigor mortis often occurs after death, giving the face the appearance of grimacing. However, despite common belief, the face of a deceased does not reflect whether the individual's last moments were of fear or fright.

Algor Mortis

Algor mortis (synonym: postmortem cooling) is the normal cooling of a body after death as the result of equilibration of the body with the ambient (environmental) temperature.

The normal rectal temperature in life is 36.9 °C (range 34.2–37.6 °C) but the assumption that a "healthy" person had a "normal" body (rectal) temperature at the time of death is often erroneous since many factors influence body temperature at the time of death (Table 4).

The heat exchange between the body and the surroundings is mediated by conduction, convection, radiation, and evaporation. The main factors influencing a drop in body temperature after death are conduction and convection. Radiation can usually be disregarded, but evaporation may be important if the body or clothing is wet.

The rate of cooling of a body after death depends on the following factors:

Table 4 Individual factors potentially influencing body temperature at the time of death

Rise in body temperature at the time of death due to:
Infectious diseases (e.g., pneumonia, sepsis)[a,b]
Psychic (''emotional'') stress
Physical activity (e.g., sports, fight, escape)
Central fever (e.g., stroke, intracranial hemorrhage)
Hyperthyroidism
Malignant hyperthermia
Exsiccosis
Administration of neuroleptic medication
Intoxication with illicit drugs (e.g., heroin, cocaine)
Lowering of body temperature at the time of death due to:
Hypothermia (e.g., prolonged exposure of the subject to a natural cold environment, artificial hibernation)
Hypothyroidism
Administration of muscle relaxants
Peripheral arterial occlusive disease

[a]Note that in elderly people infectious diseases may present without fever.
[b]When estimating the time since death using temperature-based methods, the error due to fever is greatest during the first hours postmortem and decreases with progression of the postmortem interval.

- body weight (the heavier the body, the slower the rate of cooling)
- temperature gradient between the core temperature of the deceased and the ambient temperature (the higher the temperature gradient, the faster the cooling; heat exchange between the body core and surface is exclusively mediated by conduction)
- gender (females have a higher fat content than males and thus males cool more rapidly postmortem when compared to females of identical weight)
- body mass index in relation to surface area (the lower the body mass index and surface area, the faster the cooling)
- environmental conditions and surrounding medium (e.g., still or flowing water, draft, wind, sun radiation on the body (with potential rewarming of the body days after death); water immersion cools a body much faster by convection than does exposure to air of the same temperature)
- surface insulation of the body by clothing or other covering such as blankets
- wet clothing
- body posture (the body cools faster in a stretched-out than in a crouched-down position).

After death, the body temperature stays relatively constant – this is referred to as the "postmortem temperature plateau." In moderate to cool climates, this temperature plateau lasts for 1–3 h and this is then followed by a linear rate of cooling (between 0.5 and 1.5 °C per hour) for the next 10–16 h. Then, as the body temperature approaches ambient temperature, the hourly cooling rate slows down. Estimating the time since death based only on body (rectal) temperature is often inaccurate since the length of the temperature plateau is generally unknown and such an assessment is useless when the body temperature has approached the ambient temperature.

Criminalistic Aspects

Temperature-based nomogram methods to estimate the time since death that are founded on: (1) measurements of rectal temperature and mean ambient temperature at the death scene; (2) determination of body weight; and (3) the use of an empirical corrective factor are considered the most reliable methods by leading authorities in the field. Such temperature-based methods and their related formulas are most useful in temperate and cool climates in industrialized countries where most people die indoors where there is heating; they are often useless in warm or tropical climate zones and outdoor deaths.

Autolysis

Autolysis is "self-digestion" of tissue resulting from the breakdown of cell function postmortem. When the continuous oxygen supply stops after death and the cytoplasmic pH decreases, loss of cell membrane integrity results. As a result, lysosomes and their digestive enzymes (mainly hydrolases) are released from the cells. Self-protective mechanisms of cells and tissues from endogenous noxae break down. Lysosome-rich organs express signs of autolysis earlier than do organs with less hydrolytic enzyme content. Autolysis develops faster in warm and hot ambient (environmental) temperatures than in cool or cold conditions and is accelerated by fever antemortem.

On external examination of the body, the earliest sign of autolysis is detectable as a whitish, cloudy appearance of the cornea. At autopsy, autolytic changes are manifest as a doughy consistency of the parenchyma of the pancreas with loss of its normal macroscopic architecture on cut surfaces. Liquefaction of the splenic pulp is another early phenomenon of autolysis that may be confused with softening of the spleen as a sequel of acute splenitis (septic spleen). The lung parenchyma contains a large number of macrophages whose lysosomes release hydrolytic enzymes, leading to a dim appearance of the outlines of cellular structures under the microscope. The adrenal glands normally retain their macroscopic appearance but appear flabby, with loss of cohesion of the medulla. In the stomach, where mucus secretion has stopped after death, gastric acid affects the mucosal surface with resultant loss

of relief of the gastric mucosa. Postmortem leakage of gastric juice within the peritoneal cavity due to autolytic self-digestion has been reported to take place in rare cases. As a result of breakdown of erythrocyte membranes, hemolysis of the blood serum occurs. The intima of larger and smaller arteries becomes reddish to light brownish, and this is referred to as "imbition." This imbition is a result of a hemolytic discoloration of the inner vessel layer.

By definition, autolysis is a pure result of endogenous enzyme activity and bacterial processes play no role. However, destruction of tissue by autolysis and by bacterial processes runs a parallel course and their products overlap. Therefore, it is more an academic question than a matter of practical relevance whether superficial skin slippage is a result of pure autolysis (as considered by some authors) or whether putrefaction processes play the major part in its development.

Maceration

Maceration is sterile autolysis of an unborn fetus who has died *in utero* enclosed within the amniotic cavity. The most prominent finding at the external examination is skin slippage with underlying brownish-blackish discoloration of tissue (Figure 18). If the amniotic cavity has been opened prior to the delivery of a stillborn fetus, bacterial putrefaction will alter the appearance of maceration. The presence of maceration without any putrefactive changes in a recently delivered child is indicative of stillbirth.

Putrefaction

Putrefaction is the bacterial degradation of soft tissue. After death, when homeostasis ceases, anaerobic bacteria (mostly *Clostridium* and *Proteus* species) migrate from the gut into blood vessels and into tissue where they multiply and spread through the whole body.

Figure 18 Manifestation of maceration in a stillbirth.

The terms "putrefaction" and "autolysis" are often used synonymously in the Anglo-American literature but these processes must be strictly differentiated since, by definition, bacterial processes play no role in the development of autolytic changes. In addition, autolysis paves the way for bacteria to spread through the body by the breakdown of cell integrity of the gut mucosa. The term "decomposition" is also often used synonymously with putrefaction. By strict definition, decomposition is the product of soft-tissue degradation by aerobic bacteria (originating from outside environmental sources), but for practical purposes this differentiation seems far too academic since both processes cannot be distinguished adequately.

Factors Accelerating the Onset of Putrefactive Tissue Changes

Many variables affect the onset, extent, and time course of putrefaction but temperature is the most decisive factor. Putrefactive tissue changes develop faster under warm and hot ambient (environmental) temperatures than under cool or cold conditions. Putrefaction is accelerated in individuals who die of systemic infections (e.g., gas gangrene, sepsis) because blood and organs have already been invaded by bacteria before death on the one hand and on the other hand, body temperature is usually raised in such fatalities at the time of death. The administration of antibiotics before death often slows down putrefaction processes. Since open wounds are a portal of entry for microorganisms from the outside environment, those who die with or from wounds that are wide open and extending far down within the subcutaneous tissue show accelerated rates of putrefaction. Obesity also accelerates the onset of putrefaction. Putrefaction is delayed in individuals with a considerable loss of blood before death since hemoglobin as well as other proteins from blood cells are a main source of energy for the bacteria involved in putrefactive processes.

Underlying Mechanisms of Putrefaction

The process of putrefaction is catalyzed by autolysis-induced breakdown products of proteins, carbohydrates, and lipids that serve the bacteria as a source of energy.

Hydrogen sulfide is the main product of reductive catalysis by endogenous bacteria. The compound of hydrogen sulfide with hemoglobin released from autolytic erythrocytes leads to the formation of sulfhemoglobin, which is responsible for the characteristic greenish discoloration of putrefied human skin and tissue.

Venous marbling, the outlining of superficial epidermal blood vessels, is the result of a combination

of autolysis of erythrocytes (postmortem hemolysis) and intravascular multiplication and growth of intestinal bacteria that use blood vessels as "through roads" to spread over the entire body. Whether marbling manifests with a greenish or a more violaceous to muddy-brownish color depends on the total amount of sulfhemoglobin formation within the affected vessels.

The characteristic bloating of a putrefied body as reflected by the swelling of the face, distension of the abdomen, and, in males, scrotal swelling is a result of bacterial gas formation. On palpation, crepitus is noticed. Putrefactive gas has an offensive foul odor and is the volatile final product of bacterial reductive catalysis. It is mainly composed of methane, hydrogen sulfide, carbon dioxide, ammonia, mercaptanes, and primary amines. The purging of putrefactive fluid from mouth and nostrils as well as eversion of the lips and protrusion of the tongue are also a result of bacterial gas formation with subsequent increase in intrathoracic pressure.

Morphology of Putrefaction

The exact chronological order of the appearance of putrefactive changes is highly variable and depends on a broad variety of individual as well as environmental conditions. Therefore, it is beyond the scope of this article to give a satisfactory overview of all morphological findings and the timeframes in which they may have developed in different seasons and climatic zones. However, putrefactive body changes usually follow a sequential order.

The earliest sign of putrefaction is a greenish skin discoloration of the abdomen, usually visible first in the right lower abdominal quadrant (Figure 19). While this greenish skin discoloration becomes more prominent and spreads over the whole body, skin slippage with glove-like peeling of the horny skin layer of the hands, formation of gaseous or putrefactive fluid-filled skin blisters, marbling (Figure 20), purging of putrefactive fluid from mouth and nostrils (Figure 21), swelling of the face with bulging of the eyes, eversion of the lips, and protrusion of the tongue between the teeth and lips (Figure 22), bloating of the abdomen under tension, and, in males, scrotal swelling develop. Hair and nails become loose and can be easily pulled out. In advanced states, the skin has a brownish-blackish appearance.

Changes of internal organs as a result of putrefactive processes are dilatation of the renal pelvis and the ventricles and vestibules of the heart as a result of bacterial gas formation. Muddy-brownish putrefactive fluid is found within the pleural and peritoneal cavity. So-called "putrefaction crystals," yellowish particles composed of tyrosine and leucine, are found

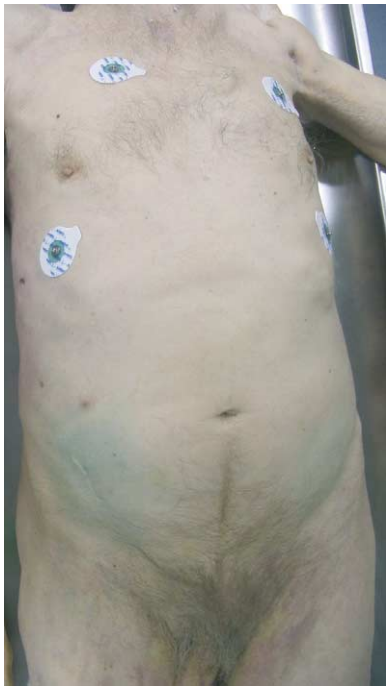

Figure 19 Greenish skin discoloration more prominent in the right than the left lower abdominal quadrant.

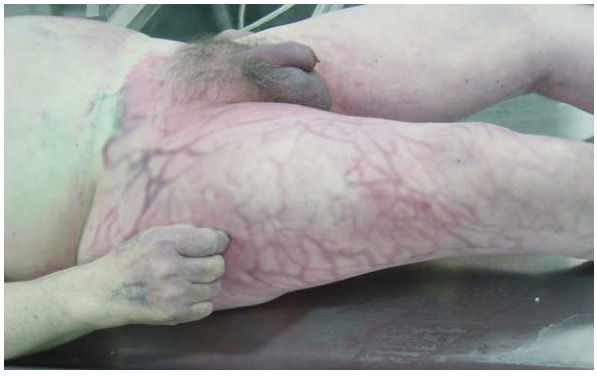

Figure 20 Marbling.

on the surface of the internal organs, especially on the surface and bottom side of the liver, adhering loosened to Glisson's capsule, as well as on the capsule of the spleen. The liver shows a spongy consistency, giving both cut sections and histologic sections in more advanced stages a Swiss-cheese-like appearance (Figure 23). The intestinal loops are distended due to gas formation. The myocardium appears muddy-brownish to blackish and hence, myocardial infarction easily escapes macroscopic detection. The brain appears soft to liquefied with loss of the cortical-surface structures and a dark grayish to green discoloration of cortex, caudate nucleus, and putamen. Gaseous bubble formation is seen under the mucosal surfaces of internal organs. In advanced stages of

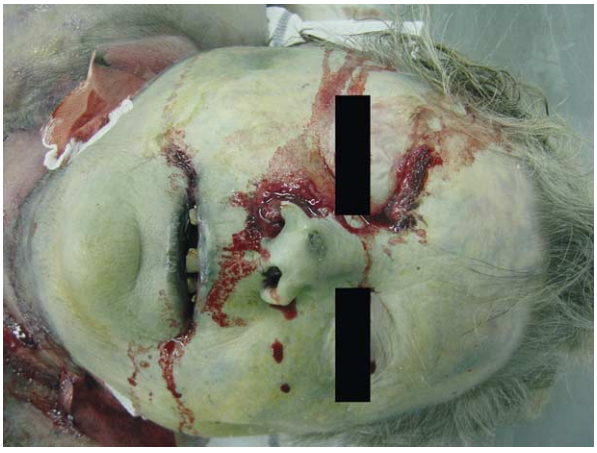

Figure 21 Purging of putrefactive fluid from mouth and nostrils.

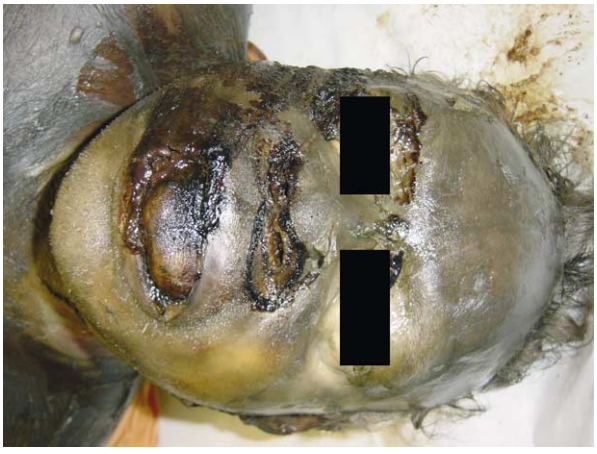

Figure 22 Marked swelling of the face with bulging of the eyes, eversion of the lips, and protrusion of the tongue. Note also purging of putrefactive fluid. It is easily understood from this photograph that visual identification of a deceased is rendered difficult in an advanced state of putrefaction.

putrefaction, the volume of bacterial gases produced is usually enough to float solid organs such as liver, kidneys, or spleen when brought into a water bowl at autopsy. The prostate gland is usually the organ offering most resistance to putrefaction and may occasionally be used to determine the gender of otherwise totally putrefied human remains.

Criminalistic Aspects

The manifestation of putrefaction may cause problems of interpretation of autopsy findings, and accordingly, death may seem suspicious. Putrefaction may mask the traumatic injuries an individual sustained before death. In addition, purging of putrefaction fluid from the mouth and nostrils is frequently confused with blood deriving from

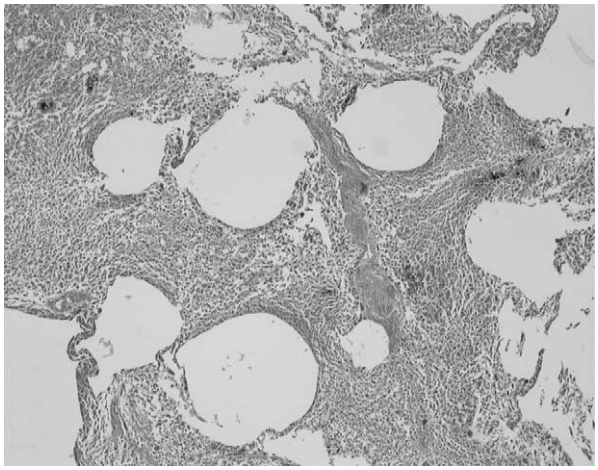

Figure 23 Swiss cheese-like appearance of the liver at histological examination (original magnification × 10, hematoxylin-eosin).

antemortem facial injuries by individuals unfamiliar with the phenomenon.

Putrefactive tissue changes may also render visual identification difficult.

Differential Diagnoses

A major problem in forensic autopsy practice is the broad variety of a number of artifacts produced in more advanced states of putrefaction as well as the possible overlapping of putrefactive tissue changes with underlying diseases, the latter being lost at gross inspection of the affected organ. Examples are the presence of froth in the heart in a putrefied body that must not be misinterpreted as air embolism, a flabby appearance of the heart due to bacterial gas production that may mimic dilatation of the ventricles and vestibules of the heart, venous marbling seen under the mucosa of the esophagus that may be mistaken for esophageal varicosis, or putrefactive liquefaction of the lung parenchyma that may be misdiagnosed as edema of the lungs. Putrefactive fluid within the pleural cavity may be mistaken for hemothorax.

Coma blisters, bullous lesions occasionally found in the setting of coma, for example, due to intoxication with barbiturates, benzodiazepines, theophylline, or in carbon monoxide poisoning, may be mistaken for skin blistering as a result of putrefaction and, vice versa, coma blisters may be overlooked when putrefactive changes of the body have begun. Concerning the differential diagnosis, coma blisters are most often seen at sites of dermal compression and appear histologically as subepidermal blister formation without any epidermal necrosis but with eosinophilic necrosis of the eccrine sweat gland coils.

Adipocere

Adipocere (synonyms: grave wax, corpse wax) is the product of the decomposition of adipose tissue formed by the hydrolysis of triglycerides into glycerine and free fatty acids. Adipocere is a grayish-white, at first waxy mass that over time becomes a crumbly to solid consistency when fatty acids crystallize, leading to the solidification of the affected body parts.

Adipocere forms in both surface and subsurface conditions as well as in embalmed and unembalmed bodies.

Various bacteria, especially *Clostridium perfringens*, seem to be involved in the formation of adipocere by playing a key role in the formation of fatty acids postmortem. The presence of water is essential for the bacterial and enzymatic processes involved in adipocere formation. However, it is now well apprehended that the formation of adipocere does not necessarily depend on the presence and persistence of moisture since the water content of adipose tissue is sufficient for the bacterial and enzymatic activity involved in adipocere formation. The presence of oxygen (e.g., exposure of the body or parts of it to air) in general inhibits adipocere formation.

Since its formation requires lipids, adipocere is more frequently seen in females, the well nourished, and the obese than in individuals with a lower adipose tissue content such as individuals who are underweight or cachectic. Apart from the external manifestation of adipocere, internal organs may be involved in adipocere formation: this finding is independent of the lipid content of the affected organs since, through the hydrolysis of triglycerides into glycerine and free fatty acids, neutral fats liquefy and penetrate into the surrounding tissue. The finding of an involvement of skeletal muscles in adipocere formation can be attributed to the same underlying mechanism.

Depending on the environmental conditions, adipocere formation may be observed as early as 1 month after death. However, the presence of adipocere usually indicates a postmortem interval of at least several months. Once formed, adipocere may remain unchanged for hundreds of years.

Adipocere formation is most often seen in corpses that have been submerged in water for a long period of time, for example, bodies recovered from shipwrecks. The rate of development of adipocere in immersed corpses has been related to the water temperature and it was long assumed that adipocere formation is accelerated by higher water temperature. However, recent investigations do not regard the temperature of the water as essential for speed of adipocere development.

Apart from the resting time, the manifestation of adipocere in buried human remains depends on a variety of individual factors such as the geographical location of the burial site, season of burial, vegetation of the burial site, depth of the grave, insect colonization of the corpse before burial, and other anthropogenic influences (for example, if a body is easily accessible to insects, adipocere is unlikely to form), the composition of the coffin used, and the texture of the soil (chemical and physical soil properties).

Mummification and skeletonization may accompany adipocere formation but usually adipocere formation is accompanied by an increase in the volume of the affected body. A preferential formation of adipocere has been observed in body parts with open wounds, thus paving the way for clostridial colonization.

Mummification

Mummification is the product of desiccation, the drying-up process of soft tissue. Mummification may affect the entire body or only parts of it when only distinct portions of the body have been exposed to the proper environmental conditions. Natural mummification occurs in dry, usually hot climatological conditions. However, mummification also occurs in bodies located in frozen environments; mummified human bodies found in polar regions or in glaciers after hundreds of years are of special interest from the archeological and anthropological points of view.

Artificial mummification for the purpose of soft-tissue preservation has been practiced throughout time in prehistoric cultures, especially in regions in which climatological conditions favor natural mummification.

During the process of mummification, soft tissue undergoes considerable shrinkage by losing body fluids via evaporation, resulting in considerable loss of body weight (up to 60–70%). The skin turns hard and has a brown to black leathery appearance (**Figure 24**), forming a thick shell over the body. Eventually all the hair disappears. In mummified bodies, the arms are often found abducted in the shoulder joints, flexed in the elbow joints, and the hands are clenched into fists in most cases. This flexion is also often recognizable in the lower limbs. The reason for this phenomenon is the shrinkage of muscles and tendons, causing flexion in the joints of the extremities due to predominance of the flexor muscles.

Despite dehydration of the body surface, the internal organs become dark, viscous, and paste-like. With the increase in the postmortem interval and under the

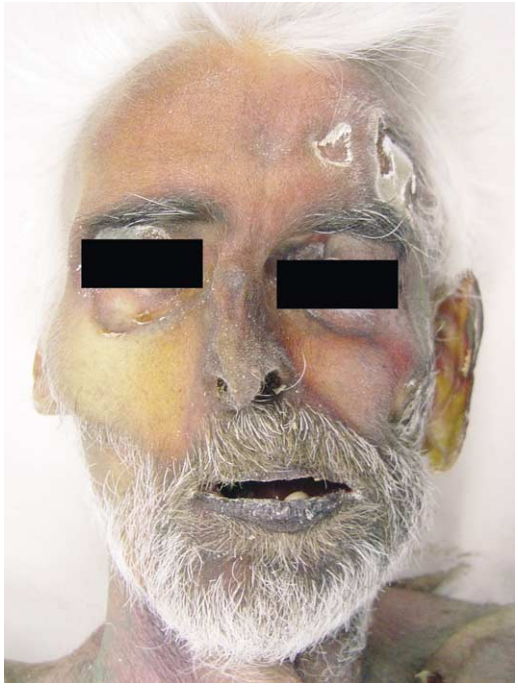

Figure 24 Natural mummification. Brown to black leathery appearance of the skin.

Figure 25 Cut sections through a forearm of a mummified body with total loss of muscles and soft tissue.

influence of putrefaction and maggot activity, the internal organs undergo considerable shrinkage and may vanish totally. On cut sections through the limbs, a total loss of muscles and soft tissue may be seen in advanced stages of mummification (Figure 25). Although some authors consider that microscopic examination of remnants of internal organs in bodies presenting in advanced states of mummification is unrewarding, this author has more than once seen cases where a precise histologic diagnosis relating to underlying diseases (e.g., bronchopneumonia, liver cirrhosis, carcinoma) could be established in mummified human remains even after postmortem intervals of up to several years.

Artifacts caused by insects on mummified corpses (e.g., holes made by maggots) may appear greater than they were before mummification due to the shrinkage of tissue; they should not be confused with stab wounds or shotgun pellet holes.

The appearance of mummified corpses may be modified by scavenger activity and bones may be lost due to animal activity. Occasionally, rodents nests may be found inside body cavities in mummified corpses.

Primary and Secondary Mummification

Two types of mummification are of general forensic interest: primary and secondary mummification. Primary mummification is generally not accompanied by relevant putrefaction processes of the affected body and will therefore predominantly occur in environmental conditions that favor a rapid drying of soft tissues, preventing enteric bacteria and microorganisms from outside causing relevant putrefaction. Secondary mummification by definition follows considerable putrefaction of the body. Secondary mummification of human bodies found in open spaces is seen more often than in bodies in indoor settings.

Skeletonization

Skeletonization (synonym: skeletalization) is the total or partial loss of soft tissue (complete or incomplete skeletonization, respectively) with resultant exposure of bones. Incomplete skeletonization may proceed to complete skeletonization but this does not necessarily take place since only discrete portions of the body may be exposed to the proper environmental conditions (Figure 26).

The length of the postmortem interval needed for skeletonization of a body or parts of it is highly variable and mainly depends on the ambient temperature, insect colonization of the body, and scavenger activity. Under warm to hot environmental conditions, and increasingly under the influence of moisture, aerobic bacterial activity from the outside is accelerated with resultant advanced manifestation of odor of the body, attracting insects and scavengers to the body. Skeletonization and mummification often occur together in different or identical parts of the body (Figure 27).

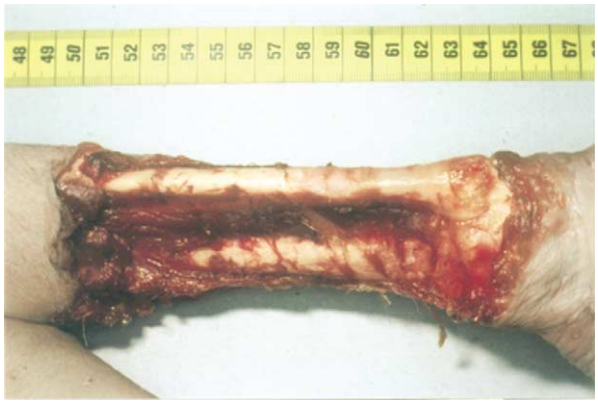

Figure 26 Incomplete skeletonization of a forearm due to postmortem animal scavenging by a domestic German Shepherd.

Figure 27 Co-occurrence of skeletonization and mummification.

Usually all skin, soft tissue, and muscles are lost before a skeleton becomes disarticulated. Disarticulation of bones in skeletonized bodies is more often seen to take place from the head downward and from central to peripheral than in the reverse way.

Postmortem Preservation by Freezing

Artificial Postmortem Preservation by Freezing

If a body has been frozen immediately after death, for instance in a freezer with the aim of hiding the corpse, the rate of postmortem changes slows virtually to zero. When a corpse is first frozen, for example, before dumping the body to conceal the time of death and confuse the investigating authorities, and then exposed to warm ambient temperatures, more advanced putrefaction is usually observed on the outer body surface than internally. The reason for this phenomenon is that in such cases the enteric flow has most often been put to death before any relevant putrefaction of the viscera could occur. Accordingly, anaerobic bacteria from the outside will have a greater effect on putrefaction of the exterior surface of the body than on the inside within the same time span after the corpse is defrosted. In addition, ice crystal artifacts may be seen in histological sections of the myocardium, corroborating such an assumption.

Freeze-Drying

Freeze-drying, the process of body preservation mediated by sublimation, is predominantly seen in bodies recovered from polar regions and permafrost zones in Siberia or the Middle East. Such bodies are usually well preserved externally and internally, or may show mummification. When freeze-dried bodies are mummified, the internal organs are usually better preserved than in cases where mummification occurred under hot environmental conditions.

Miscellaneous

Postmortem Changes and Injuries to the Skin

Injuries due to handling, transportation, and storage of the body Abrasions and lacerations on the skin may be produced by manipulation of the body during postmortem handling, transportation, and storage. When arising in the early postmortem interval (but only in cases where the epidermis is still intact and no postmortem skin slippage has occurred as a result of autolysis and/or putrefaction), these postmortem injuries are relatively easily distinguished from vitally acquired injuries by their light yellowish-brownish to golden, shiny appearance (Figure 28). They result from loss of the barrier function of the epidermal layer with subsequent evaporation of tissue fluid. In doubtful cases, incision of cutaneous injuries of postmortem origin will reveal a hardened, slightly flattened area on cut sections without any hemorrhage in the underlying soft tissue.

In skin areas where the epidermis is very thin (e.g., the tip of the nose and the scrotum), drying as a result of postmortem evaporation of tissue fluids occurs.

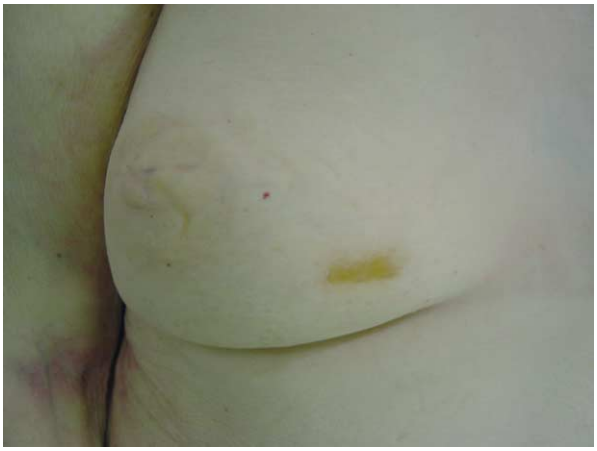

Figure 28 Superficial postmortem injury upon the skin with a light yellowish-brownish to golden, shiny appearance.

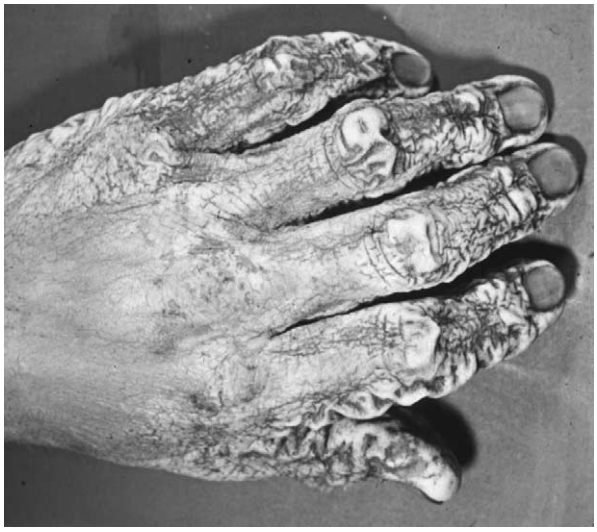

Figure 29 Washer-woman's skin in a drowning victim.

The result is a hardened, light-brownish appearance of the affected epidermal surfaces (sometimes referred to as the earliest stage of mummification).

Skin changes produced by corrosives Corrosives such as acids or alkalis may lead to loss of the epidermal layer of the skin. In particular, gastric juice running out during handling or transportation of the body may produce pale, sometimes band-like, cutaneous alterations that should not be confused with ligature marks when located on the neck.

Skin changes produced by postmortem urine leaking Postmortem urine leaking may cause extensive skin damage to a child on the perigenital skin areas that were in contact with a urine-soaked diaper postmortem. Such postmortem skin changes should be differentiated from vitally acquired alterations and not interpreted uncritically as signs of child neglect before death.

No vital reaction will be detected in any of the aforementioned skin alterations by microscopic examination.

Washerwoman's skin In bodies recovered from water or moist environments, the skin of the palms of the hands and the feet has whitish discoloration of the epidermis associated with swelling, wrinkling, and vesicular detachment up to glove-like peeling, mainly as a result of soaking of the horny layer of the epidermis. This finding, referred to as washer-woman's skin, that is especially seen after prolonged exposure to water in drowning deaths (Figure 29), should not be confused with another form of skin change seen on the palms of the hands and the soles of the feet and caused by heat. In the latter case, histological examination shows fluid-filled blisters in the stratum germinativum, hyperchromasia, and palisade arrangement of the nuclei as well as clumping of the erythrocytes corresponding to a morphological variation of a second-degree burn due to the special anatomy of friction skin.

Drying of Mucosal Surfaces

Postmortem evaporation of tissue fluids and hypostasis after cessation of circulation leads to drying of mucosal surfaces, for example, of the lips, the tip of the tongue, the glans of the penis, the glans of the clitoris, or the pudendal lips, resulting in a hardened, light to dark brownish appearance of the affected mucosal surfaces.

External Changes of the Eye after Death

As with most postmortem changes, alterations of the eye after death are accelerated in their onset and intensity under warm ambient temperatures and in dry climates.

Under moderate to cool ambient temperatures, 3–9 h after death, the cornea has a whitish, cloudy appearance as a result of autolysis. With further increase in postmortem interval, the cornea loses its turgor.

If the eyes remain open after death, the areas of the sclera exposed to air dry out; this results first in a yellowish discoloration that subsequently turns into a brownish-blackish band-like zone called "tache noire" (Figure 30).

The conjunctivae soften and become a light grayish color. In states of advanced putrefaction, conjunctival petechiae, for example, as a result of asphyxia, may not be distinguished due to hemolysis and

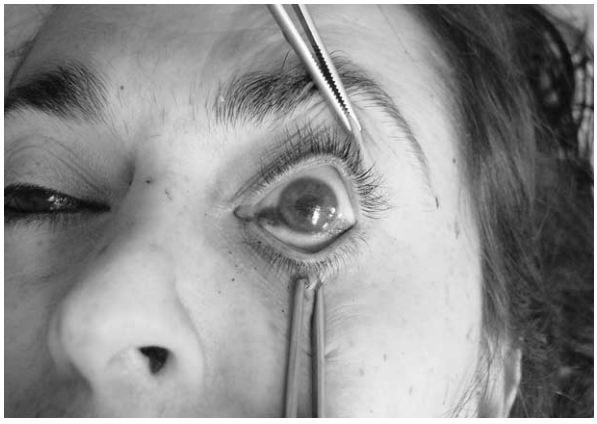

Figure 30 Tache noir.

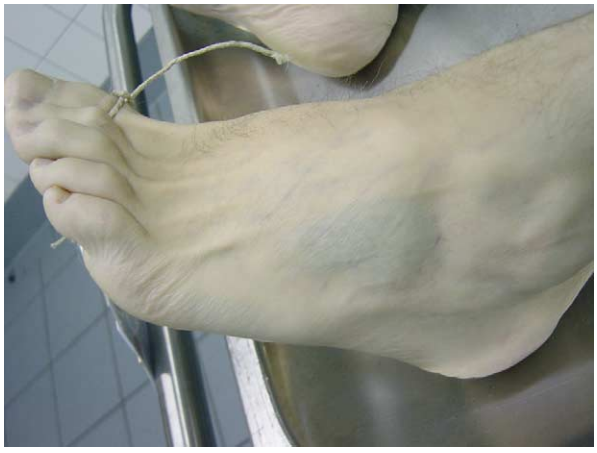

Figure 31 Postmortem hypostasis in the lateral sub-malleolar muscles.

sulfhemoglobin production, giving the conjunctivae a homogeneous grayish to light greenish appearance.

Pink Teeth and Nails Phenomenon

Pink discoloration of teeth and nails is a rare postmortem finding that is thought to derive from hemolysis after exudation of hemoglobin and hemoglobin derivates. This phenomenon seems to depend on the anatomical presence of porous structures, an anatomic feature that is found in the dentine tubules of the crowns and roots (but not in the enamel, which is more compact and therefore not colored) of the teeth as well as in the fingernails and toenails. The overwhelming majority of observations of this phenomenon have been described in association with premortem cranial blood congestion, particularly in asphyxial deaths such as strangulation, death in a head-down position, and drowning. Pink discoloration of teeth and nails has been reported even after postmortem intervals of several months.

Submalleolar and Thenar Eminence Hypostasis

Postmortem hypostasis in the muscles located in the lateral submalleolar region (Figure 31) and the thenar eminence may mimic antemortem bruising. An incision will show lack of hemorrhage within the muscle tissue.

Fungi

Fungi may colonize the body in every possible location and at all times during the postmortem interval. However, the eyes are more often affected by fungal colonization under dry conditions and the mouth and nose are more often colonized with fungi in moist to wet environments (Figure 32). Fungi may mimic antemortem pouring of chemical substances over the body.

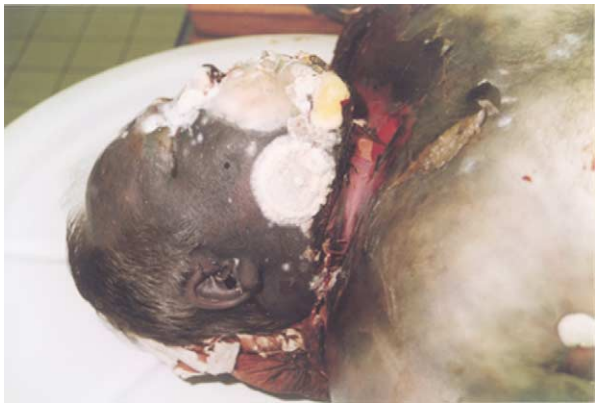

Figure 32 Fungi colonization most prominent around nose and mouth in a body exhumed 4 months after death.

Arthropods

A large number of arthropod species are attracted to human bodies after death, primarily flies (Diptera), beetles (Coleoptera), and their larvae. The arthropods feed, live, or breed in and on the corpse, depending on their biological preferences, and on the state of body decomposition. Most arthropod species colonize a corpse for only a limited period of time ("faunal succession").

Criminalistic aspects By calculating their developmental stages, arthropods are useful in estimating the time that a corpse has been inhabited by animals ("colonization interval"), and this opens up a wide range of applications for forensic entomology embedded in a criminalistic context. Apart from useful information concerning the estimation of time since

death or the time a body was stored in particular environmental conditions at a specific place, additional information can be obtained from arthropods, that is, found on or in corpses, at a scene of crime, at the place where a body had been dumped, or on the clothing of a suspect. In specific cases, a suspect may be linked to the scene of crime as a result of arthropods found on the soles of the shoes. Arthropods that live in restricted areas and are found on a corpse in a different area may prove that the body was moved after death, while blowfly larvae can give information on how long children or elderly people were subjected to neglectful care. An experienced forensic entomologist can make such conclusions, but regularly requires at least baseline data concerning the local area such as the time of appearance of arthropods and data on temperature ranges.

Cutaneous holes and soft-tissue defects, for example, made by maggots, especially when overlapped by tissue shrinkage due to mummification with resulting enlargement of the defects, can mimic gunshot wounds or other mechanical tissue defects sustained antemortem, for example, as a result of stabbing with pointed instruments such as knives or scissors.

Animal Depredation

The phenomenon of postmortem animal interference with human bodies or their remains is a substantial part of the taphonomic processes a body undergoes after death and animal depredation occurring after death is routinely encountered in forensic pathology. Postmortem injuries can be inflicted by all kinds of animals, irrespective of their size or environmental origin, whether from land, sea, or air. The discrimination between antemortem injury versus postmortem artifacts generally presents no difficulties because of the total absence of hemorrhages and reddening in the tissue adjacent to the wound margins (Figure 33) and the lack of any vital reaction under the microscope.

The most effective tissue removers are insects and rodents. Skin and soft-tissue artifacts caused by rodents may occur as early as within the first hour postmortem. In the majority of injuries inflicted postmortem by rodents, the wounds have a circular appearance (Figure 34) and the wound margins are finely serrated, showing irregular edges. Distinct parallel series of cutaneous lacerations deriving from the biting action of the upper and lower pairs of the rodent's incisors are diagnostic of rodent activity (Figure 35). However, the determination of a distinct rodent species (e.g., rats, mice) by the morphological appearance of the damage to skin and soft tissue is often unconvincing.

A broad range of carnivores may be involved in the postmortem destruction of corpses located in open spaces or indoors (e.g., wild animals such as foxes and big cats or domestic animals such as dogs or cats). The wound margins caused by carnivores often appear more regular than those caused by rodents (Figure 36). V-shaped to rhomboid punctured wounds are often seen in the intact skin in the immediate vicinity of the actual wound margins (Figure 37). Such stab-wound-like defects represent canine tooth marks of carnivore origin. An additional criterion of animal depredation by carnivores is

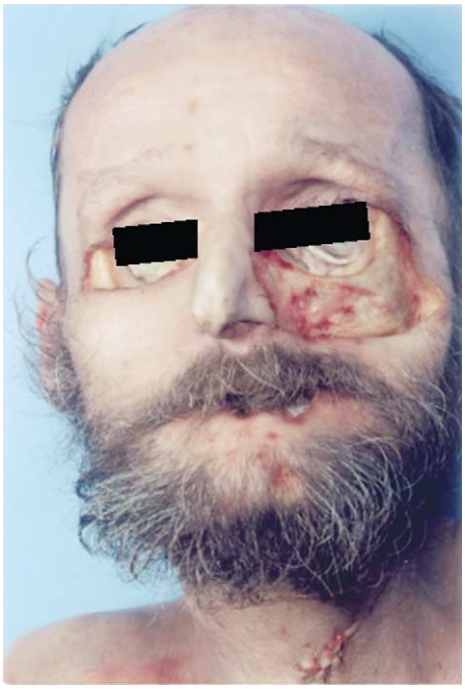

Figure 33 Circular defects around both eyes due to mixed rodent activity. Note the total absence of hemorrhages and reddening in the tissue adjacent to the wound margins indicative of postmortem origin of the wounds.

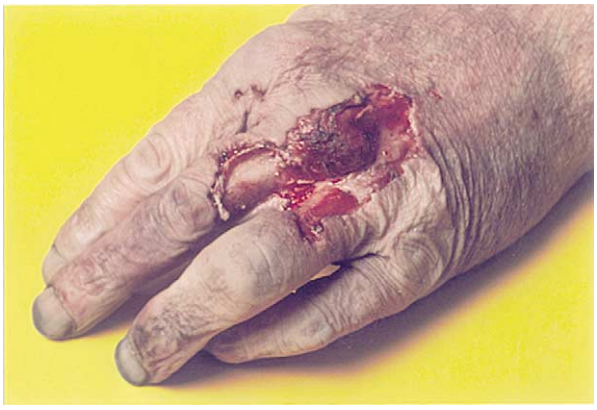

Figure 34 Circular skin and soft tissue defect on the back of the hand caused postmortem by mice.

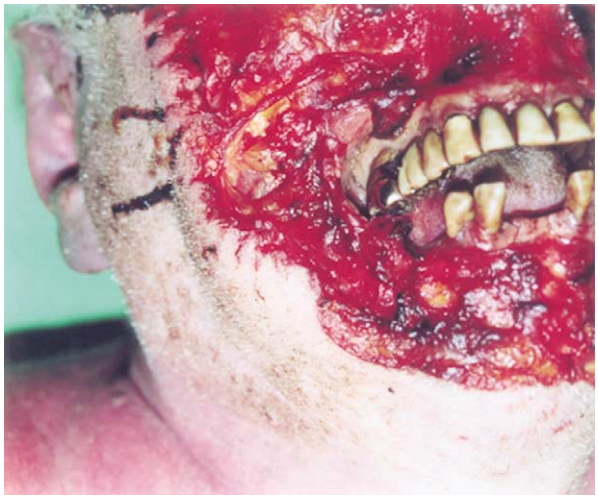

Figure 35 Irregular and finely serrated wound margins with a series of parallel cutaneous lacerations deriving from the biting action of the upper and lower pairs of rodents. This large tissue defect in the face was caused postmortem by rats.

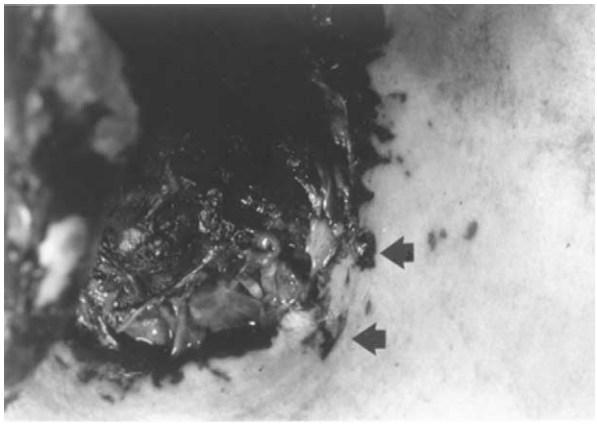

Figure 37 V-shaped to rhomboid punctured wounds are seen in the intact skin next to the wound margin. Such stab wound like defects represent canine tooth marks of carnivore origin (same case as **Figure 36**).

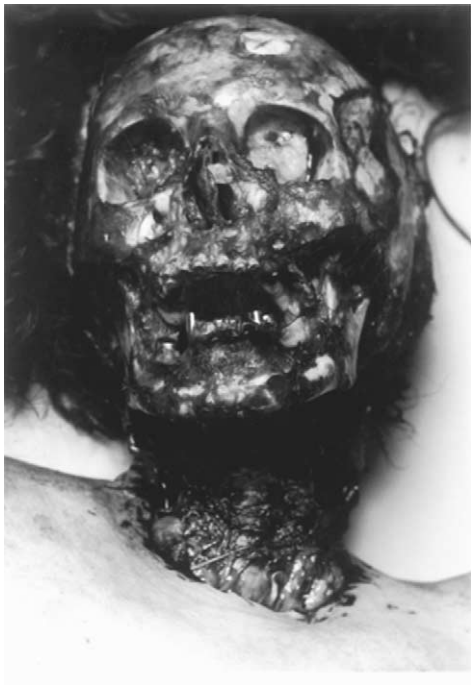

Figure 36 Postmortem injuries caused by a pit bull terrier.

Figure 38 Oval to round cutaneous defects caused by crustaceans postmortem on the neck and upper trunk in a drowning victim. Such injuries should not be confused with stab wounds or gunshot holes.

the presence of claw-induced linear scratch-type abrasions in the vicinity of the damaged skin areas.

In drowning victims or persons whose death led to their deposition in water or when a body has been dumped in an aquatic environment, postmortem artifacts on the body surface due to aquatic living structures are often observed, too, and should not be confused with vitally acquired injuries (Figure 38).

See Also

Autopsy: Procedures and Standards; **Autopsy, Findings:** Postmortem Drug Sampling and Redistribution; Organic Toxins; Drowning; **Carbon Monoxide Poisoning:** Incidence and Findings at Postmortem; **Postmortem Changes:** Postmortem Interval; **Toxicology:** Methods of Analysis, Postmortem

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Electrolyte Disturbances

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Introduction

Brief Historical Consideration

The postmortem chemistry of the blood and some other body fluids has been the subject of extensive and often elaborate and tedious investigations for a century or so. As early as the mid-1920s, Paul as well as Pucher and Burd had published on the "postmortem chemical determinations" of the blood and the cerebrospinal fluid (CSF). Among the earliest substances studied were electrolytes such as sodium (Na^+), chloride (Cl^-), and potassium (K^+) ions. Primarily, these research works had been clinically orientated especially in relation to the diagnosis of renal diseases. But forensic applications of the postmortem chemistry had soon become an established area for large numbers of forensically tailored studies. In the 1940s and 1950s, Jetter and McLean as well as Naumann, amongst other researchers, had published their classical works on the postmortem analysis of electrolytes in various body fluids such as the blood, CSF, and the vitreous humor. In 1963, Evans published his book "The Chemistry of Death" in which he discussed the chemistry of various tissues in the early and late postmortem periods. Since the late 1960s and early 1970s, Coe had published several papers rightly considered by many as landmarks in this field. This topic does still attract considerable interest from forensic scientists as is evident by the large volume of recent literature that exists today. A number of useful reviews are provided in this article.

Physiological Aspects and Definitions of Terms

An electrolyte is a substance that when dissolved in water separates into ions and is able to conduct electricity by movements of ions. Thus, electrolyte disturbances cannot be discussed without an understanding of the body fluid balance. Electrolytes are measured in milliequivalents per liter ($mEq\,l^{-1}$). For ions having a single charge, e.g., Na^+, K^+, Cl^-, or HCO_3^-, the number of $mEq\,l^{-1}$ equals the number of $mmol\,l^{-1}$ and for ions having two charges such as Ca^{2+}, Mg^{2+}, or phosphate HPO_4^{2-}, the number of $mEq\,l^{-1}$ is twice the number of $mmol\,l^{-1}$. Two-thirds of body fluid is within the cells and called intracellular fluid (ICF) and the rest is outside the cells and called extracellular fluid (ECF). Interstitial fluid (ISF), i.e., fluid in between the cells, constitutes about 80% of the ECF and plasma (blood) forms the other 20% of the ECF, which also includes many other body fluids, such as the CSF, aqueous and vitreous humors, and lymph and pleural, pericardial, peritoneal, and synovial fluids. During life, cell membranes separate the ICF from the ISF and blood vessel walls divide ISF from plasma although at the level of capillaries, plasma and ISF may exchange with each other, as the capillary wall is thin and permeable. In life, the amounts of water and solutes (electrolytes and inorganic ions) in various body fluids are continuously corrected so that the volume of each fluid type remains fairly stable. This is called fluid balance,

which depends on electrolyte balance and vice versa. By the same token, acid–base balance means that the amounts of acids and bases, determined by H^+ and HCO_3^- (i.e., another form of electrolyte balance), are continuously corrected during life so as to keep the pH of each body fluid within a fairly stable range; the pH of arterial blood in a healthy adult is usually 7.35–7.45. This is regulated by various buffer systems, which means the mechanisms that enable the body to convert strong acids and bases to weak acids and bases. During life, water constitutes about 45–75% of total body mass depending on age and gender. Under normal conditions, water and electrolyte gain and loss are regulated hand in hand, i.e., "water follows salt." The ultimate source of water and electrolyte gain is via ingestion (about 2300 ml of water daily) although metabolism produces a small amount called "metabolic water" (about 200 ml daily). On the other hand the body loses about 2500 ml of water daily by kidneys (1500 ml), skin (600 ml), lungs (300 ml), and via feces (100 ml). Most electrolytes are absorbed through the small intestines by active transportation, passive diffusion, or both. For example, sodium is absorbed first by diffusion into the intestinal epithelium then transported out by active sodium pumps (Na^+/K^+ ATPase). Chloride and most anions can passively follow sodium, whereas potassium, calcium, phosphate, and many other electrolytes are absorbed by active transportation. The electrolyte contents of ECF and ICF are different: sodium and chloride are most abundant in the former and potassium, magnesium, and phosphate are most abundant in the latter. During life, this is maintained via active Na^+/K^+ pumps at the cellular membrane. Homeostasis is the condition in which the contents of the body fluid compartments are maintained relatively constant within physiological limits.

Disease, Death, and Electrolytes

During life, when electrolyte contents of body fluids are disturbed beyond homeostatic limits, pathological conditions (with or without signs and symptoms), diseases, or even death may follow (Table 1). In fact, many think that the mechanism of almost any death is basically biochemical in origin, which is largely due to electrolyte disturbances. This effectively means that every death is associated with at least some electrolyte disturbances. This complicates the interpretation of postmortem results and restricts the usefulness of electrolyte analysis after death. At and after the somatic death, i.e., when the body ceases to function as a whole entity, body cells are confronted with three

Table 1 Blood electrolyte disturbances and associated diseases

	Pathological condition	
Electrolyte (Normal adult values)	*Deficiency*	*Excess*
Sodium (Na^+) 136–148 mEq l^{-1}	*Hyponatremia* E.g., starvation, neglect, sun stroke, vomiting, diarrhea, burns, alcohol and/or drug intoxication, diuretic abuse, renal disease, water intoxication (a form of child abuse)	*Hypernatremia* E.g., high sodium diet and salt intoxication (a form of child abuse); decreased sodium loss due to renal impairment, hyperthermia, drug and alcohol intoxication, low water intake in dehydration and starvation
Chloride (Cl^-) 95–105 mEq l^{-1}	*Hypochloremia* E.g., excessive vomiting, overhydration, aldosterone deficiency, congestive heart failure and therapy with some diuretics (e.g., furosemide (frusemide)).	*Hyperchloremia* E.g., dehydration, starvation, neglect, severe renal failure, hyperaldosteronism, and excessive chloride intake
Potassium (K^+) 3.5–5.0 mEq l^{-1}	*Hypokalemia* E.g., vomiting or diarrhea, hyperaldosteronism, kidney disease or some diuretics therapy	*Hyperkalemia* E.g., renal failure, crushing injuries to body tissues, severe burns, or hemolyzed blood transfusion
Calcium (Ca^{2+}) Total = 9–10.5 mEq l^{-1}	*Hypocalcemia* E.g., hypoparathyroidism, elevated phosphate levels or low calcium intake	*Hypercalcemia* E.g., hyperparathyroidism, some cancers, excessive vitamin D, and Paget's disease of bone
Magnesium (Mg^{2+}) 1.3–2.1 mEq l^{-1}	*Hypomagnesemia* E.g., alcoholism, congestive cardiac failure, diabetes mellitus, and some diuretics therapy	*Hypermagnesemia* E.g., some antacids high intake, aldosterone deficiency, and hypothyroidism
Phosphate (HPO_4^{2-}) 1.7–2.6 mEq l^{-1}	*Hypophosphatemia* E.g., disturbances in gastrointestinal and renal functions and aldosterone	*Hyperphosphatemia* E.g., renal failure and hemolytic anemias

Data from Tortora GI and Grobowski SR (2000) *Principles of Anatomy and Physiology*, 9th edn., pp. 613, 856, and 965–973. New York: John Wiley & Sons, Inc.

major events or phenomena, which have profound effect on the ability of the body to maintain its homeostatic contents. First, hypoxia or anoxia, i.e., lack or deficiency of oxygen (O_2) at the tissue or cell level, increases the permeability of the cell membrane and blood vessel wall leading to leaking of the ICF, ECF, ISF, and the plasma to each other. Also, the lack of O_2 causes changes in the pH of the blood and other body fluids leading to further disturbances in fluid contents including electrolytes. Second, lack of energy due to the death process leads to the abrupt or gradual cessation of the active electrolyte pumps across the cell membrane including that of the alimentary system and vascular wall cells. Therefore, electrolyte movements through various body compartments are not maintained according to the physiological requirements but rather follow physical laws governed principally by gravity and simple physical diffusion leading to drastic pH and electrolyte disturbances. Third, the processes of autolysis and cell disintegration in general cause breakdown of the cell membrane leading to virtually complete mix-up of various body fluids. The end result is a dramatic and unpredictable change of electrolyte contents of body fluids.

Limitations of Postmortem Electrolyte Interpretations

The analysis of electrolytes (and in fact, other chemicals) of body fluids after death has been used for the following purposes: estimation of the postmortem interval (PMI); diagnosis of the cause of death; and evaluation of certain anatomical disorders. Practically, it is virtually unviable to find a meaningful way for the analysis of postmortem electrolyte contents of ICF and ISF. Therefore, most or all studies of electrolytes after death are conducted on a small ECF fraction, namely, the vitreous humor, CSF, and the blood. However, because of the hemolysis of the red cells, a postmortem blood sample is, in reality, a mixture of intravascular ICF and ECF. Moreover, due to general breakdown of the cell membrane after death, body fluid compartments ooze into each other and fluids are mixed up together to extents that vary with tissue temperature and pH, environmental temperature, time, cause and mode of death, and other factors. Also, as electrolyte movements after death follow almost pure physical laws such as the gravity laws, the electrolyte contents of a postmortem sample are understandably site-related. For example, in recent years, several workers such as Balasooriya and Hill, Madea and coworkers, and Pounder and coworkers have pointed out significant quantitative between-eye electrolyte differences. Regardless of the type of body fluid and the location of sampling, individual variations in electrolyte contents are known to exist during life and continue to exist or even widen after death. Besides these individual variations, there are other factors that tend to add uncertainties to the already highly erratic and unpredictable postmortem electrolyte levels. Examples of these factors and uncertainties are the subtle biochemical conditions that exist in life (Table 1) and pass unnoticed or undiagnosed after death so that the subject or the sample is marked as normal or a control. Other examples include the mysterious effect of the agony of the moment of death and the uncontrolled influence of the conditions and length of sample storage.

Forensic Applications

It follows from the above discussion that a postmortem electrolyte level found in a given body fluid roughly indicates the value of that electrolyte in that body fluid during life $\pm$ the net effect of various factors such as:

- the length of the interval of death or the PMI
- cellular pH and temperature
- environmental temperature
- mode and cause of death
- individual variations
- effects of the agony of death
- presence or absence of undiagnosed minor biochemical conditions during life or at the moment of death
- the pathological conditions that existed antemortem.

Therefore, the overall picture is rather complex, which is reflected in the many inconsistencies and irregularities found in the results of most postmortem electrolyte studies. This remark together with the observation that most chemical methods are cumbersome, expensive, time-consuming, require special machinery, and yet, in most instances yield difficult results to interpret or practically unreliable conclusions have made many leading field workers consider that chemical methods are of limited practical value and the postmortem electrolyte analysis is not an exception to this.

Nevertheless, the study of electrolyte changes after death is, at least from theoretical and academic points of view, of some value in the following situations.

Assessment of certain antemortem conditions or disorders In renal disease, Coe has used hypernatremia accompanied with mild degrees of urea retention

as "good" evidence of dehydration. Most pathologists, however, would be reluctant to use such an inference in medicolegal reports prepared for a court of law. In cases of electrolyte imbalance, Coe has studied a large number of cases and suggested four patterns:

1. "Dehydration pattern" is characterized by increased vitreous sodium and chloride (>155 and >135 mEq l^{-1} respectively) and moderate increase of urea nitrogen (40–100 mEq l^{-1}). This pattern may be found in cases of neglect, salt intoxication, and starvation (all can be rare forms of child abuse).
2. "Uremia pattern" is characterized by marked increases of vitreous urea and creatinine without significant increases in sodium or chloride. This can be seen in renal disease.
3. "Low-salt pattern" is characterized by low vitreous sodium, chloride, and potassium (<130, <105, <15 mEq l^{-1} respectively) associated with some increase of serum bilirubin and urea nitrogen <5 mg 100 ml^{-1}. This pattern is most likely seen in chronic alcoholics.
4. "Decomposition pattern" is characterized by low vitreous sodium and chloride but high potassium indicating a long postmortem interval (<130, <105, and >20 mEq l^{-1} respectively).

In cases of asphyxia vs. cardiac arrhythmia, studies of blood gases are not consistent and must be viewed with caution. Although many modern neuroresearchers would like to link epilepsy with abnormalities of $Na^+/K^+/Ca^{2+}$ channels (channelopathies), this is largely a molecular genetic problem and, therefore, the implication on forensic postmortem chemistry is of no or little consequence. Recently, Delva has described trends to link various forms of congestive heart failure and some cardiomyopathies to magnesium deficiency. He also emphasized the important role played by magnesium in the operation of the sodium/potassium pumps (Na^+/K^+ ATPase).

Confirmation of the cause of death The only example in forensic medicine is that of drowning in salt water and river water. "Biochemical tests" of the blood obtained from the right and left sides of the heart have been postulated to differentiate between these two types of drowning, but, unfortunately, most results of the so-called "biochemical tests" have been shown to be unreliable. Recently, Zhu and coworkers described new serum markers for the differentiation between fresh and salt water drowning. They reported that the characteristic feature of salt water drowning was a low left–right blood urea nitrogen (BUN) ratio and marked increase in the serum chloride, magnesium, and calcium levels of the left heart blood. Azparren and coworkers studied the left–right ventricular blood strontium (Sr) concentration as a marker for drowning and claimed that this test had yielded a highly significant positive result.

Estimation of the time of death Chemical approaches for the timing of death form the second most commonly investigated techniques after the temperature-based methods. However, many of these studies are made on substances other than electrolytes. Nonetheless, one of the electrolytes, i.e., the vitreous K^+, is correctly considered as the most reliable of all chemical methods. It is worth noting, that, in general, the chemical methods for estimating the time since death are widely considered as the least reliable and the least practicable of all other methods. Almost all types of electrolytes have been studied for the purpose of the estimation of PMI. The most notable of these are Na^+, Cl^-, K^+, Ca^{2+}, P^{3-} or P^{5-}, S^{2-}, and Mg^{2+}.

***Sodium* (Na^+)**

Blood Blood or serum sodium was shown to decrease quickly after death but the rate of decrease involved large individual variations and although it was estimated at 0.9 mEq l^{-1} h^{-1}, visualizing the plot of the scattered Na^+ values indicates that the least-square regression used was meaningless. Recently, Singh and coworkers claimed a highly significant relationship between logarithms of serum Na^+ and K^+ concentrations, Na^+/K^+ ratio, and PMI within the first 3–58 h after death. Yet they noted that these variables were significantly affected by other factors, such as environmental temperature, cause of death, age, and gender.

Cerebrospinal fluid (CSF) Naumann showed that the concentrations of many of the CSF electrolytes including sodium, chloride, potassium, calcium, magnesium, and phosphorus generally decrease after death. However, this decrease was noticeably erratic to the extent that it could not be used for any sensible timing of death. Fekete and Brunsden made similar observations.

Vitreous humor (VH) Jaffe and Coe demonstrated that vitreous sodium is stable for the first 30 h after death and in a study of 145 normal adults, Coe found that vitreous sodium concentrations ranged from 135 to 151 mEq l^{-1} with an average value at 143 mEq l^{-1}.

Synovial fluid Madea and coworkers compared the sodium contents of synovial fluid (SF) with that of VH in 74 normal adults and found that the concentrations in both fluids were comparable. In the SF, they found that Na^+ values ranged from 86 to 164 mmol l^{-1} (= mEq l^{-1}) with the median at 145 mmol l^{-1}.

Chloride **(Cl^-)**

Blood Jetter, Schyler and Coe have all reported a decrease in the plasma or blood chloride after death with average concentrations ranging roughly between 80 and 90 mEq l^{-1} and a postmortem decrease of 0.25–0.97 mEq l^{-1} h^{-1}. However, looking at the scatter plot, the so-called least-squares correlation appears meaningless. Recently, Singh and coworkers claimed a highly significant relationship between logarithms of plasma chloride and PMI within the first 3–58 h after death, yielding a standard error of ± 2.1 h. However, they stated that this was significantly influenced by other factors, e.g., environmental temperature, cause of death, age, and gender.

Vitreous humor (VH) Coe reported that vitreous chloride, like sodium, remained constant for the first 18 h after death with a concentration ranging between 104 and 132 mEq l^{-1} and an average of 120 mEq l^{-1}.

Synovial fluid The levels of synovial chloride were reported by Madea and coworkers in 65 cases to range between 49 and 116 with a median value at 100 mEq l^{-1}.

Potassium **(K^+)**

Blood Blood or plasma potassium increases so rapidly after death that its use for the estimation of the time of death is virtually impossible. Jetter reported that within the first hour or so after death, serum K^+ increases to 18 mEq l^{-1} followed by further albeit gradual increase.

Vitreous humor (VH) Many investigators have noted that the rise in the levels of vitreous potassium after death is the most reliable chemical marker for estimating the PMI. Jaffe was the first to note that vitreous K^+ increases after death at a regular rate. Subsequently, many workers including Adelson and coworkers, Coe, and others had reinforced this observation. Although there was a general agreement that the rise in the vitreous potassium was roughly arithmetic, there were wide variations in the degree of correlation between the vitreous potassium concentration and the PMI. These discrepancies were reflected in the large differences of the standard deviations (SD) of the time estimates found by various investigators. For instance, while Sturner and Lie reported ±5 h, Adelson, Hughes, Hanson, and Coe had all reported more than ±10 h. In fact, Hanson and coworkers, in a series of 203 cases found ±20 h (1 SD). Marchenko (cited by Madea) in a series of 300 cases found vast individual variations and he concluded that vitreous potassium was only useful within 3–6 h. Krause and coworkers (cited by Madea) demonstrated even worse correlation than the above and showed a range of scatter between 9 and 107 h for potassium values between 5 and 28 mEq l^{-1}. In view of these wide variations, many researchers attempted to refine the potassium method. Thus, Adjutantis and Coutselinis claimed to have achieved ±1.1 h within the first 12 h of death by analyzing bilateral samples withdrawn at 3 hourly intervals and extrapolating back to the "normal" vitreous K^+ value at the moment of death at 3.4 mEq l^{-1}. Madea and coworkers used selective methods by analyzing the vitreous humor for both potassium and urea (as an internal standard) so as to exclude the cases where there was, in their view, antemortem K^+ imbalance. They carried out extensive mathematical calculations and attempted several maneuvers only to narrow the confidence limit by <4 h (from ±25.51 to ±21.78 h). Recently, Muñoz and coworkers claimed considerable improvement in the potassium to postmortem (K^+/PMI) correlation results simply by repositioning the variables so that the PMI is used (correctly) as the dependent variable and the K^+ as independent, instead of the other way round that, according to them, seemed to have been used (incorrectly) so far. The same workers claimed improved estimations of the PMI by combined analysis of vitreous potassium and hypoxanthine.

Synovial fluid Madea and coworkers found K^+ concentrations of the synovial fluid to range from 2.6 to 23.6 mEq l^{-1} with a median value of 9.5 mEq l^{-1}.

Calcium **(Ca^{2+})**

Blood According to Jetter and Naumann, serum calcium remains constant in the early PMI. Hodgkinson and Hamblton found that the serum total calcium concentrations elevated in eight samples obtained between 11 and 50 h after death. In two other samples, they found that total serum calcium remained constant for the first 2 h postmortem. However, Fekete and Brundson were not able to establish normal postmortem values, as the scatter they found was unduly wide.

Cerebrospinal fluid (CSF) Naumann found that antemortem and postmortem calcium concentrations in the CSF were comparable.

Vitreous humor (VH) Coe found that in adults, calcium concentrations remain constant in the early postmortem interval and vary from 6 to 8.4 mg $100\,ml^{-1}$ with an average value at 6.8 mg $100\,ml^{-1}$. Naumann studied 211 cases and found an average calcium concentration of 7.2 mg $100\,ml^{-1}$ within an average PMI of 9 h.

Synovial fluid Madea compared total calcium concentrations of vitreous humor and synovial fluid and found the values to range from 1.05 to 2.33 mmol l^{-1} with a median of 1.68 mmol l^{-1} for vitreous calcium and from 1.51 to 3.58 mmol l^{-1} with a median value of 2.36 mmol l^{-1} for synovial calcium.

Sulfur **(S^{2-})**

Blood Jensen found that the level of postmortem sulfate was directly proportional to the level of urea or creatinine in the serum. Coe reported that inorganic sulfate in the serum remained unchanged for the first 24 h after death and then decreased by approximately 20% in the following 2 days.

There are no available reports on the sulfur in the CSF and VH.

Phosphorus **(P^{3-} or P^{5-})**

Blood Jetter showed that inorganic phosphorus increased in the serum as early as 1 h after death and reached a level of 20 mEq l^{-1} at about 18 h postmortem. Both Jetter and Schyler reported an increase in postmortem serum organic phosphorus.

Cerebrospinal fluid (CSF) Naumann reported a significant increase in the CSF postmortem phosphorus (being 5.2 mEq l^{-1}) compared to its antemortem value (being 0.8 mEq l^{-1}), but no correlation exists between this and the time of death.

Vitreous humor (VH) Naumann also reported that inorganic phosphorus of the VH varied from 0.1 to 3.3 mEq l^{-1} with an average value of 1.2 mEq l^{-1}. No correlation with postmortem interval was mentioned.

Magnesium **(Mg^{2+})**

Blood In five of eight cases, where blood or serum samples were obtained from 11 to 38 h after death, Hodgkinson and Hamblton found increases in postmortem serum magnesium concentrations varying from 2.4 to 5 mEq l^{-1}. Coe's investigations for determining calcium by cresolphthaline complexone demonstrated that magnesium probably begins to move from the intracellular fluid to the plasma well before significant hemolysis occurs. A similar inference was substantiated by Hodgkinson and Hamblton. According to Naumann, when hemolysis starts plasma magnesium increases rapidly to levels up to 20–30 mEq l^{-1}.

Cerebrospinal fluid (CSF) According to Naumann, who studied 131 cases for about 10.5 h after death, the average CSF magnesium increases slightly during the postmortem interval (being on average 2.9 mEq l^{-1}) from that of the antemortem level (being 2 mEq l^{-1}). This appears not to have challenged.

Vitreous humor (VH) Swift and coworkers examined 38 cases of children in relation to postmortem interval and found that two-thirds of these cases showed a slow increase in magnesium concentration of VH varying from 0.003 to 0.09 mg $100\,ml^{-1}\,h^{-1}$. They also showed that postmortem vitreous magnesium varies with the age of the child. Blumenfled and associates substantiated this last finding.

Hydrogen (pH)

Blood Jetter demonstrated that acidity of the blood after death increases as a factor of time (PMI). He found that pH values averaged 6.73 for the first 12 h after death while for the next 12 h period it averaged 6.43. Straumfjord and Butler reported a similar observation and added that there were variations in the pH depending on the source of the blood sample being highest in the blood obtained from the upper extremities and lowest in the right ventricular blood.

Conclusion

In conclusion, the electrolytes of various body compartments undergo profound redistribution after death resulting in significant quantitative changes, which have been used in forensic pathology for three main purposes as described earlier. However, unfortunately, the interpretation of the postmortem electrolyte analysis is very difficult and often futile. Nevertheless, postmortem chemistry of body fluids and tissues still attracts considerable research attention.

See Also

Autopsy: Adult; **Autopsy, Findings:** Postmortem Drug Sampling and Redistribution; Fire; Drowning; **Children:** Emotional Abuse; **Postmortem Changes:** Overview; Postmortem Interval; **Starvation**

Further Reading

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Postmortem Interval

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Introduction

Evidence of the time elapsed since death, the postmortem interval, may come from three sources: (1) the body of the deceased; (2) the environment in the vicinity of the body; and (3) information on the deceased's habits, movements, and day-to-day activities. All three sources of evidence (corporal, environmental, and anamnestic) should be explored and assessed before offering an opinion on when death occurred. The longer the postmortem interval, the less accurate is the estimate of it based upon corporal changes. As a consequence, the longer the postmortem interval, the more likely it is that associated or environmental evidence will provide the most reliable estimates of the time elapsed.

No problem in forensic medicine has been investigated as thoroughly as the determination of the postmortem interval on the basis of postmortem changes to the body. Many physicochemical changes begin to take place in the body immediately or shortly after death and progress in a fairly orderly fashion until the body disintegrates. Each change progresses at its own rate which, unfortunately, is strongly influenced by largely unpredictable endogenous and environmental factors. Consequently, using the evolution of postmortem changes to estimate the postmortem interval is invariably difficult, and always of limited accuracy.

Body Cooling

Body cooling (algor mortis or "the chill of death") is the most useful single indicator of the postmortem interval during the first 24 h after death. Some authorities would regard it as the only worthwhile corporal method. The use of this method is only possible in cool and temperate climates, because in tropical regions there may be a minimal fall in body temperature postmortem, and in some extreme climates, such as desert regions, the body temperature may even rise after death.

Since body heat production ceases soon after death but loss of heat continues, the body cools. After death, as during life, the human body loses heat by radiation, convection, and evaporation. The fall in body temperature after death is mainly the result of

radiation and convection. Evaporation may be a significant factor if the body or clothing is wet. Heat loss by conduction is not an important factor during life, but after death it may be considerable if the body is lying on a cold surface.

Newton's law of cooling states that the rate of cooling of an object is determined by the difference between the temperature of the object and the temperature of its environment. A plot of temperature against time gives an exponential curve. However, Newton's law applies to small inorganic objects and does not accurately describe the cooling of a corpse which has a large mass, an irregular shape, and is composed of tissues of different physical properties. The cooling of a human body is best represented by a sigmoid curve when temperature is plotted against time. Thus, there is an initial maintenance of body temperature which may last for some hours – the so-called "temperature plateau" – followed by a relatively linear rate of cooling, which subsequently slows rapidly as the body approaches the environmental temperature. The postmortem temperature plateau is physically determined and is not a special feature of the dead human body. Any inert body with a low thermal conductivity has such a plateau during its first cooling phase. The postmortem temperature plateau generally lasts between 30 min and 1 h, but may persist for as long as 3 h, and some authorities claim that it may persist for as long as 5 h.

It is usually assumed that the body temperature at the time of death was normal. However, in individual cases the body temperature at death may be subnormal or markedly raised. As well as in deaths from hypothermia, the body temperature at death may be subnormal in cases of congestive cardiac failure, massive hemorrhage, and shock. The body temperature may be raised at the time of death following an intense struggle, in heat stroke, in some infections, and in cases of pontine hemorrhage. The English forensic pathologist Simpson recorded a personal observation of a case of pontine hemorrhage with a temperature at death of 42.8 °C (109 °F). Where there is a fulminating infection, e.g., septicemia, the body temperature may continue to rise for some hours after death.

Thus the two important unknowns in assessing time of death from body temperature are: (1) the body temperature at the time of death, and (2) the length of the postmortem temperature plateau. For this reason assessment of time of death from body temperature cannot be accurate in the first 4–5 h after death when these two unknown factors have a dominant influence. Similarly, body temperature cannot be a useful guide to time of death when the cadaveric temperature approaches that of the environment. However, in the intervening period, over the linear part of the sigmoid cooling curve, any formula which involves an averaging of the temperature decline per hour may well give a reasonably reliable approximation of the time elapsed since death. It is in this limited way that the cadaveric temperature may assist in estimating the time of death in the early postmortem period.

Unfortunately the linear rate of postmortem cooling is affected by environmental factors other than the environmental temperature and by cadaveric factors other than the body temperature at the time of death. The most important of these factors are body size, body clothing or coverings, air movement and humidity, and wetting or immersion in water.

Body size is a factor because the greater the surface area of the body relative to its mass, the more rapid will be its cooling. Consequently, the heavier the physique and the greater the obesity of the body, the slower will be the heat loss. Children lose heat more quickly because their surface area to mass ratio is much greater than that of adults. The exposed surface area of the body radiating heat to the environment will vary with the body position. If the body is supine and extended, only 80% of the total surface area effectively loses heat, and in the fetal position the proportion is only 60%.

Clothing and coverings insulate the body from the environment and therefore slow body cooling. The effect of clothing has a greater impact on corpses of low body weight. A bedspread covering may at least halve the rate of cooling. For practical purposes, only the clothing or covering of the lower trunk is relevant.

Air movement accelerates cooling by promoting convection, and even the slightest sustained air movement is significant if the body is naked, thinly clothed, or wet. Cooling is more rapid in a humid rather than a dry atmosphere because moist air is a better conductor of heat. In addition the humidity of the atmosphere will affect cooling by evaporation where the body or its clothing is wet. A cadaver cools more rapidly in water than in air because water is a far better conductor of heat. For a given environmental temperature, cooling in still water is about twice as fast as in air, and in flowing water, it is about three times as fast.

Simple formulae for estimating the time of death from body temperature are now regarded as naive. The literature is replete with formulae that were enthusiastically recommended at first and later disavowed. The best tested and most sophisticated current method for estimating the postmortem interval from body temperature is that of the German researcher Henssge. Even so, it is acknowledged that the method may produce occasional anomalous results. It uses a nomogram based upon a complex formula, which approximates the sigmoid-shaped cooling curve. To make the estimate of postmortem interval, using this method requires: (1) the body

weight; (2) the measured environmental temperature; and (3) the measured deep rectal temperature, and assumes a normal body temperature at death of 37.2 °C. Empiric corrective factors allow for the effect of important variables such as clothing, wetting, and air movement. The use of these corrective factors requires an element of personal experience. At its most accurate this sophisticated methodology provides an estimate of the time of death within a time span of 5.6 h with 95% probability. One of the most useful aspects of the nomogram is the ease with which the effect of changes in the variables can be tested. As a result it is an educational as well as a practical investigative tool.

The assessment of body cooling is made on the basis of measurement of the body core temperature, and, postmortem, this requires a direct measurement of the intraabdominal temperature. In practice either the temperature is measured rectally, or the intrahepatic/subhepatic temperature is measured through an abdominal wall stab. Oral, aural, and axillary temperatures cannot be used because after death these are not reflective of the body core temperature. For the measurement, an ordinary clinical thermometer is useless because its temperature range is too narrow, and the thermometer is too short for insertion deep into the rectum or liver. A chemical thermometer 25–30 cm (10–12 in.) long with a range from 0 to 50 °C is ideal. Alternatively, a thermocouple probe can be used, and this has the advantage of a digital readout or a programmable printed record.

Whether the temperature is measured via an abdominal stab or per rectum is a matter of professional judgment in each case. If there is easy access to the rectum without the need to disturb the position of the body seriously and if there is no reason to suspect sexual assault, then the temperature can be measured per rectum. It may be necessary to make small slits in the clothing to gain access to the rectum, if the body is clothed and the garments cannot be pushed to one side. The chemical thermometer must be inserted about 8–10 cm (3–4 in.) into the rectum and read *in situ*. The alternative is to make an abdominal stab wound after displacing or slitting any overlying clothing. The stab can be made over the right lower ribs and the thermometer pushed into the substance of the liver, or alternatively a subcostal stab will allow insertion of the thermometer on to the undersurface of the liver. If a method of sequential measurements of body temperature is to be used, then the thermometer should be left *in situ* during this time period. Taking sequential readings is much easier with a thermocouple and an attached printout device.

The core body temperature should be recorded as early as conveniently possible at the scene of death. The prevailing environmental temperature should also be recorded and a note made of the environmental conditions at the time the body was first discovered, and any subsequent variation in these conditions. Temperature readings of the body represent data, which, if not collected, are irretrievably lost. Therefore, the decision not to take such readings should always be a considered one.

Rigor Mortis

Ordinarily, death is followed immediately by total muscular relaxation, primary muscular flaccidity, succeeded in turn by generalized muscular stiffening, rigor mortis. After a variable period of time, as a result of the development of putrefaction, rigor mortis passes off spontaneously to be followed by secondary muscular flaccidity. There is great variation in the rate of onset and the duration of rigor mortis, so that using the state of rigor mortis to estimate the postmortem interval is of very little value. In general, if the body has cooled to the environmental temperature and rigor is well developed, then death occurred more than 1 day previously and less than the time anticipated for the onset of putrefaction, which is about 3–4 days in a temperate climate.

As a general rule, when the onset of rigor is rapid, then its duration is relatively short. The two main factors which influence the onset and duration of rigor are the environmental temperature and the degree of muscular activity before death. Onset of rigor is accelerated and its duration shortened when the environmental temperature is high, so that putrefaction may completely displace rigor within 9–12 h of death. Rigor mortis is rapid in onset, and of short duration, after prolonged muscular activity, e.g., after exhaustion in battle, and following convulsions. Conversely, a late onset of rigor in many sudden deaths can be explained by the lack of muscular activity immediately before death.

Classically, rigor is said to develop sequentially, but this is by no means constant, symmetrical, or regular. Antemortem exertion usually causes rigor to develop first in the muscles used in the activity. Otherwise, rigor is typically first apparent in the small muscles of the eyelids, lower jaw, and neck, followed by the limbs. It involves first the small distal joints of the hands and feet and then the larger proximal joints of the elbows, knees, and then the shoulders and hips. It is generally accepted that rigor mortis passes off in the same order in which it develops. The forcible bending of a joint against the fixation of rigor results in tearing of the muscles and the rigor is said to have been "broken." Provided the rigor had been fully established, it will not reappear once broken down

by force. Reestablishment of rigor, albeit of lesser degree, after breaking it suggests that death occurred less than about 8 h before rigor was broken.

The intensity of rigor mortis depends upon the decedent's muscular development, and should not be confused with its degree of development. In examining a body both the degree (complete, partial, or absent joint fixation) and the distribution of rigor should be assessed, after establishing that no artifact has been introduced by previous manipulation of the body by other observers. Attempted flexion of the different joints will indicate the degree and location of rigor. Typically, slight rigor can be detected within a minimum of 30 min after death but may be delayed for up to 7 h. The average time of first appearance is 3 h. It reaches a maximum, i.e., complete development, after an average 8 h, but sometimes as early as 2 h postmortem or as late as 20 h.

The biochemical basis of rigor mortis is not fully understood. Postmortem loss of integrity of the muscle cell sarcoplasmic reticulum allows calcium ions to flood the contractile units (sarcomeres), initiating the binding of actin and myosin molecules and mimicking the normal contraction process. Normal relaxation in life is achieved by energy-dependent (adenosine triphosphate (ATP)-driven) pumping of calcium back across the membrane of the sarcoplasmic reticulum but this fails postmortem because of membrane disruption and lack of ATP. The actin–myosin complex is trapped in a state of contraction until it is physically disrupted by the autolysis which heralds the onset of putrefaction. This process is characterized by proteolytic detachment of actin molecules from the ends of the sarcomeres, and consequent loss of the structural integrity of the contractile units. Although the biochemical basis of rigor mimics that of muscle contraction in life, it does not cause any movement of the body in death.

Livor Mortis

Lividity is a dark purple discoloration of the skin resulting from the gravitational pooling of blood in the veins and capillary beds of the dependent parts of the corpse. Synonyms include livor mortis, hypostasis, postmortem lividity, and, in the older literature, postmortem suggillations. Lividity involves the skin and the internal organs such as lungs, myocardium, and skeletal muscles. Pressure of even a mild degree prevents the formation of lividity in that area of skin, so that a supine body shows contact pallor over the shoulder blades, elbows, buttocks, thighs, and calves. Similarly, tight areas of clothing or jewelry, as well as skin folds, leave marks of contact pallor. Lividity is present in all corpses, although it may be inconspicuous in some, such as following death from exsanguination. Intense lividity may be associated with postmortem hemorrhagic spots (punctate hemorrhages), that are best not referred to as petechial hemorrhages since they have nothing to do with asphyxia or agonal phenomena. They are easily recognized, occurring only in association with intense lividity and sparing adjacent areas of contact pallor.

The medicolegal importance of lividity lies in its color, as an indicator of the cause of death, and in its distribution, as an indicator of body position. The purple color of lividity reflects the presence of deoxyhemoglobin in the increasingly oxygen-depleted postmortem blood. Death from hypothermia or cyanide poisoning imparts the pink hue of oxyhemoglobin, carbon monoxide poisoning the cherry-red of carboxyhemoglobin, and poisoning from sodium chlorate, nitrates, and aniline derivatives impart the gray to brown color of methemoglobin.

The development of livor is too variable to serve as a useful indicator of the postmortem interval, but the tradition of evaluating it remains entrenched in forensic practice. Most authorities agree that lividity attains its maximum intensity, on average, at around 12 h postmortem, but there is some variation in descriptions of when it first appears, and when it is well developed, i.e., confluent. Hypostasis begins to form immediately after death, but it may not be visible for some time. Ordinarily its earliest appearance, as dull red patches, is 20–30 min after death, but this may be delayed for up to 2 h, or rarely 3 h. The patches of livor then deepen, increase in intensity, and become confluent within 1–4 h postmortem, to reach a maximum extent and intensity within about 6–10 h, but sometimes as early as 3 h or as late as 16 h. Faint lividity may appear shortly before death in individuals with terminal circulatory failure. Conversely, the development of lividity may be delayed in persons with chronic anemia or massive terminal hemorrhage.

After about 10–12 h the lividity becomes "fixed" and repositioning the body, e.g. from the prone to the supine position, will result in a dual pattern of lividity since the primary distribution will not fade completely but a secondary distribution will develop in the newly dependent parts. The blanching of livor by thumb pressure is a simple indicator that it is not fixed. Fixation of lividity is a relative, but not an absolute, phenomenon. Well-developed lividity fades very slowly and only incompletely. Fading of the primary pattern and development of a secondary pattern of lividity will be quicker and more complete if the body is moved early during the first day. However, even after a postmortem interval of 24 h, moving the body may result in a secondary pattern of lividity

developing. Duality of the distribution of lividity is important because it shows that the body has been moved after death. However, it is not possible to estimate with any precision, from the dual pattern of livor, when it was that the corpse was moved.

Areas of lividity are overtaken early in the putrefactive process, becoming green at first and later black. The red cells are hemolyzed and the hemoglobin stains the intima of large blood vessels and diffuses into the surrounding tissues, highlighting the superficial veins of the skin, a process referred to as "marbling."

Putrefaction

Putrefaction is the postmortem destruction of the soft tissues of the body by the action of bacteria and endogenous enzymes and is entirely capable of skeletonizing a body. The main changes recognizable in the tissues undergoing putrefaction are the evolution of gases, changes in color, and liquefaction. The same changes seen on the surface of the body occur simultaneously in the internal organs.

Bacteria are essential to putrefaction and commensal bacteria, mainly from the large bowel, soon invade the tissues after death. Colonic anaerobes, such as *Bacteroides* spp., anaerobic lactobacilli, clostridia, and anaerobic streptococci, thrive in the oxygen-depleted tissues of the corpse. Typically, the first visible sign of putrefaction is a greenish discoloration of the skin of the anterior abdominal wall. This most commonly begins in the right iliac fossa, i.e., over the area of the cecum, where the contents of the bowel are more fluid and full of bacteria (about 10^8 to 10^{10} organisms per gram of solid). Any antemortem bacterial infection of the body, particularly septicemia, will hasten the onset and evolution of putrefaction. Injuries to the body surface promote putrefaction by providing portals of entry for bacteria. Putrefaction is delayed in deaths from exsanguination because blood usually provides a channel for the spread of putrefactive organisms within the body. Although putrefaction tends to be more rapid in children than in adults, the onset is relatively slow in unfed newborn infants because of the lack of commensal bacteria in the gut.

Environmental temperature has a great influence on the rate of development of putrefaction, so that rapid cooling of the body following a sudden death will markedly delay its onset. In a temperate climate, the degree of putrefaction reached after 24 h in the height of summer may require 10–14 days in the depth of winter. Putrefaction is optimal at temperatures ranging between 21 and 38 °C (70 and 100 °F), and is retarded when the temperature falls below 10 °C (50 °F) or when it exceeds 38 °C (100 °F). A high environmental humidity will enhance putrefaction. Heavy clothing and other coverings, by retaining body heat, will speed up putrefaction. The rate of putrefaction is influenced by body build because this affects body cooling. Obese individuals putrefy more rapidly than those who are lean. Whereas warm temperatures enhance putrefaction, intense heat produces "heat fixation" of tissues and inactivates autolytic enzymes, with a resultant delay in the onset and course of decomposition.

There is considerable variation in the time of onset and the rate of progression of putrefaction. As a result, the time taken to reach a given state of putrefaction cannot be judged with accuracy. An observer should not assert too readily that the decomposed state of a body is inconsistent with an alleged time interval. As a general rule, when the onset of putrefaction is rapid, then the progress is accelerated. Under average conditions in a temperate climate, the earliest putrefactive changes involving the anterior abdominal wall occur between 36 and 72 h after death. Progression to gas formation, and bloating of the body, occurs after about 1 week. The temperature of the body after death is the most important factor generally determining the rate of putrefaction. If it is maintained above 26 °C (80 °F), then the putrefactive changes become obvious within 24 h and gas formation is seen in about 2–3 days.

According to an old rule of thumb (Casper's dictum), 1 week of putrefaction in air is equivalent to 2 weeks in water, which is equivalent to 8 weeks buried in soil, given the same environmental temperature. After normal burial, the rate at which the body decomposes will depend to a large extent on the depth of the grave, the warmth of the soil, the efficiency of the drainage, and the permeability of the coffin.

Gases produced by putrefaction include methane, hydrogen, hydrogen sulfide, and carbon dioxide. The sulfur-containing amino acids, cysteine, cystine, and methionine yield hydrogen sulfide, which combines with hemoglobin and ferrous iron to produce green sulfhemoglobin and black ferrous sulfide, respectively. Decarboxylation of the amino acids, ornithine and lysine, yields carbon dioxide and the foul-smelling ptomaines, putrescine (1,4-butanediamine) and cadaverine (1,5-pentanediamine), respectively. These ptomaines are detectable by cadaver dogs. Deamination of L-phenylanaline yields ammonia and phenylpyruvic acid, which forms a green complex with ferric iron. Bacterial and fungal fermentation yields ethyl alcohol, confounding the interpretation of postmortem alcohol concentrations.

Early putrefaction is heralded by the waning of rigor, green abdominal discoloration, a doughy consistency to the tissues, and hemolytic staining of

vessels. Localized drying of the lips, tip of the nose, and fingers may be seen. The face swells and discolors and the swollen lips are everted, making facial recognition unreliable. The epidermis separates from the dermis, giving rise to "skin-slip." Distension of the abdominal cavity by putrefactive gases characterizes the bloating stage of decomposition. It is these gases that cause a submerged body to rise to the surface and float. In males, gas is forced from the peritoneal space down the inguinal canals and into the scrotum, resulting in massive scrotal swelling. Gaseous pressure expels dark malodorous fluid, "purge fluid," from the nose and mouth, mimicking antemortem hemorrhage or injury. Similar fluid flows from the vagina; there is emptying of feces from the rectum and prolapse of rectum and uterus may occur. The doughy consistency of the tissues of early putrefaction is replaced by the crepitant effect resulting from gaseous infiltration along necrotic tissue planes beneath the skin and in deeper tissues. Large subepidermal bullae fill with gas, sanguineous fluid, or clear fluid. Gas bubbles appear within solid organs such as liver and brain, giving a "Swiss-cheese" appearance, and the blood vessels and heart are filled with gas. These putrefactive changes are relatively rapid when contrasted with the terminal decay of the body. When the putrefactive juices have drained away and the soft tissues have shrunk, the speed of decay is appreciably reduced.

The progression of putrefaction may be modified by vertebrate or invertebrate animal activity. Wild animals, domestic pets, livestock, fish, and crustaceans may be involved; most commonly it is insects, particularly fly larvae (maggots). In a hot humid environment with heavy insect activity, a corpse may be skeletonized in as little as 3 days. Although this insect activity is destructive of physical evidence, the insects themselves can provide useful information for estimation of time of death. All soft tissues are generally lost before the skeleton becomes disarticulated, typically from the head downward (with the mandible separating from the skull and the head from the vertebral column) and from central to peripheral (from vertebral column to limbs).

Adipocere

Saponification (making soap) or adipocere formation is a modification of putrefaction characterized by the transformation of fatty tissues into a yellowish-white, greasy (but friable when dry), wax-like substance, which has a sweetish rancid odor when its formation is complete. During the early stages of its production, a penetrating and very persistent ammoniacal smell is emitted. Adipocere, also known as "grave wax" or "corpse wax," develops as the result of hydrolysis of fat with the release of fatty acids which, being acidic, inhibit putrefactive bacteria. Fatty acids take on sodium or potassium to form hard soap (sapo durus) or soft soap (sapo domesticus) respectively. Replacement of sodium and potassium by calcium gives an insoluble soap, which contributes a more brittle quality to the adipocere. However, fat and water alone do not produce adipocere. Putrefactive organisms, of which *Clostridium welchii* is most active, are important, and adipocere formation is facilitated by postmortem invasion of the tissues by commensal bacteria. A warm, moist, anaerobic environment thus favors adipocere formation.

Adipocere develops first in the subcutaneous tissues, most commonly involving the cheeks, breasts, and buttocks. Rarely, it may involve the viscera such as the liver. The adipocere is admixed with the mummified remains of muscles, fibrous tissues, and nerves. The primary medicolegal importance of adipocere lies not in establishing the postmortem interval but rather in the preservation of the body, which aids in personal identification and the recognition of injuries.

The presence of any adipocere indicates that the postmortem interval is at least weeks and probably several months. Under ideal warm, damp conditions, adipocere may be apparent to the naked eye after 3–4 weeks. Ordinarily, this requires some months and extensive adipocere is usually not seen before 5 or 6 months after death. Some authorities suggest that extensive changes require not less than a year after submersion, or upwards of 3 years after burial. Once formed, adipocere will ordinarily remain unchanged for years.

Mummification

Mummification is a modification of putrefaction characterized by the dehydration or desiccation of the tissues. The body shrivels and is converted into a leathery or parchment-like mass of skin and tendons surrounding the bone. Mummification develops in conditions of dry heat, especially when there are air currents, e.g., in a desert. Newborn infants, being small and sterile, commonly mummify. Mummification of bodies of adults in temperate climates is unusual unless associated with forced hot-air heating in buildings or other artificial favorable conditions. The forensic importance of mummification lies primarily in the preservation of tissues, and this aids in personal identification and the recognition of injuries. The time required for complete mummification of a body cannot be precisely stated, but in ideal conditions mummification may be well advanced by the end of a few weeks.

Maceration

Maceration is the aseptic autolysis of a fetus, which has died *in utero* and remained enclosed within the amniotic sac. Bacterial putrefaction plays no role in the process. The changes of maceration are only seen when a stillborn fetus has been dead for several days before delivery. Examination of the body needs to be prompt since bacterial putrefaction will begin following delivery. The body is extremely flaccid with a flattened head and undue mobility of the skull. The limbs may be readily separated from the body. There are large moist skin bullae, which rupture to disclose a reddish-brown surface denuded of epidermis. Skin-slip discloses similar underlying discoloration. The body has a rancid odor but there is no gas formation. Establishing maceration of the fetus provides proof of a postmortem interval *in utero*, and therefore proof of stillbirth and conclusive evidence against infanticide.

Vitreous Potassium

Several researchers have studied the relationship between the potassium concentration of the vitreous humor of the eye and the postmortem interval. However, within 100 h postmortem, the 95% confidence limits of the different researchers vary from $\pm$ 9.5 h up to $\pm$ 40 h. Cases with possible confounding antemortem electrolyte disturbances can be excluded by eliminating all cases with a vitreous urea above an arbitrary level of 100 mg dl^{-1}. (High urea values in vitreous humor always reflect antemortem retention and are not due to postmortem changes.) Having eliminated these cases, there is a linear relationship between vitreous potassium concentration and time elapsed after death up to 120 h. However, the 95% confidence limits are $\pm$22 h, so that the method has no real practical application. There are also sampling problems in that the potassium concentration may differ significantly between the left and right eye at the same moment in time.

See Also

Decomposition, Patterns and Rates; **Postmortem Changes:** Overview

Further Reading

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