Chapter 2

Postmortem Changes

The "Great Pretenders"

Summary

After death, natural degradation of a body is manifest as a sequence of changes. Certain postmortem changes, despite being affected by numerous variables, remain rooted as popular methods in the determination of the time of death. A pathologist can also be faced with confounding postmortem changes and artifacts, which either alter real injuries or mimic trauma.

Key Words: Postmortem changes; rigor mortis; embalming; entomology.

1. Introduction

Deaths under medicolegal investigation are frequently unwitnessed and remain undiscovered for a period of time. As a result, the pathologist encounters postmortem changes that alter and obscure pathological findings, hindering their assessment in the determination of the cause of death. These changes can mean that "no anatomic cause of death" is found. Postmortem changes also mimic injuries, potentially shifting the focus of a medicolegal investigation.

Certain bodily changes are assumed to occur at a constant rate after death ("rate parameters"); therefore, documentation of their extent at a specific time can be extrapolated back to the time of death. A review of standard textbooks of forensic medicine written in the last 50 yr shows that despite the myth fostered by popular literature, television, and movies that the time of death can be determined accurately within a narrow time frame, the reality is that the evolution of various postmortem changes is imprecise, and determination of the time of death is only an estimate *(1–8)*. The pathologist can still be asked during an investigation or in court to determine the time of death; however, the pathologist is at a disadvantage in this determination.

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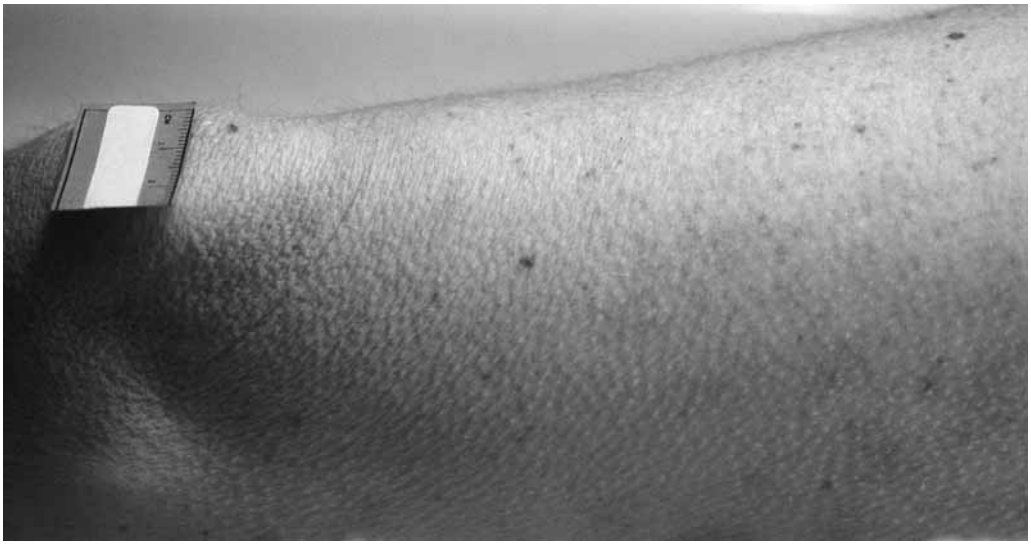

Fig. 1. Cutis anserina ("goose bumps") caused by cold exposure after death.

Postmortem changes observed during an autopsy are made after the body has been recovered from the scene. Various intervening events (e.g., manipulation of the body, storage in a morgue cooler) mean increased inaccuracy to an already imprecise determination of time of death. Examination at the scene is the best time to record these findings.

Rigor mortis, livor mortis, temperature change, decomposition, and gastric emptying are examples of postmortem changes that progress after death and are within the scope of a pathologist's observations during autopsy. Other measurable parameters, such as vitreous potassium, are affected by numerous variables (e.g., antemortem concentration, possible concentration differences between eyes, renal function; *see also* refs. *9–17*). Various biomarkers used to estimate the postmortem interval are beyond the scope of the usual forensic pathology practice *(18)*. A combination of observations can improve the accuracy of time-of-death estimation *(19)*.

2. Rigor Mortis

Rigor mortis is defined as postmortem muscle contraction owing to locking of actin–myosin filaments because of decreased ATP synthesis *(20)*.

2.1. Involvement of Involuntary Muscle

Contraction of arrectores pilorum (smooth muscle of hair follicles) manifests as "goose bumps" (cutis anserina; Fig. 1).

- Significance
  - Exposure to cold postmortem.

Seminal vesicles (smooth muscle) can contract *(4)*.

- Significance
  - Contraction leads to slight expulsion of seminal fluid at tip of penis suggesting sexual activity prior to death (Fig. 2).

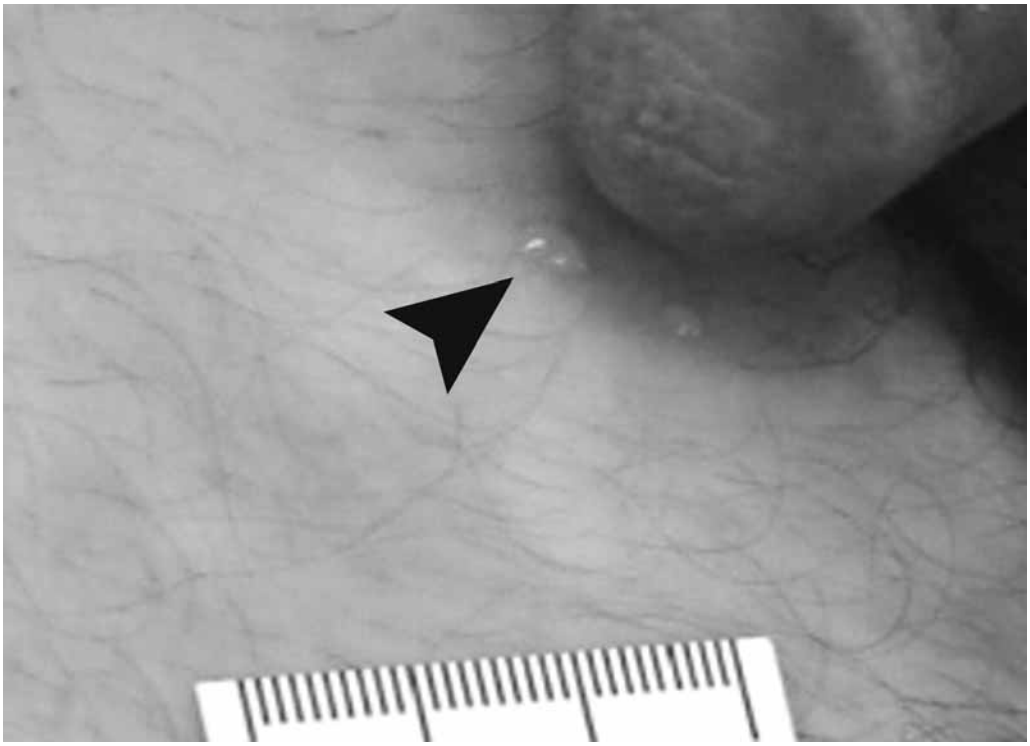

Fig. 2. Semen deposition (arrowhead) on inner thigh owing to postmortem contraction of seminal vesicles.

Ciliary muscles of the iris, in rigor, alter pupil size. Pupil diameters range from 0.2 to 0.9 cm (average size = 0.4 to 0.5 cm or about 0.25 in.). The pupil outline is not always circular. Pupils can change independently and be of unequal size (Fig. 3).

- Significance
  - Pupil size has no relation to the cause of death; inequality of pupil diameter does not indicate head injury.

If the myocardium of the left ventricle contracts, its wall appears thickened, and the chamber contains a small amount of blood.

- Significance
  - Apparent left ventricular hypertrophy.

2.2. Involvement of Voluntary Muscle (Skeletal Muscle)

Involvement of skeletal muscle leads to joint stiffening. The following sequence occurs:

- Initial flaccidity (exception: instantaneous rigor):
  - There is sufficient ATP in the immediate postmortem period to allow muscles to remain relaxed and joints limp. This phase ranges from 0.5 to 7 h (mean = 3 ± 2 h *[7]*).
- Onset and progression:
  - There is simultaneous development of rigor in all muscles, but it is evident sooner in smaller muscle groups *(20)*. The evolution of rigor mortis is not necessarily constant or symmetrical *(1,8)*. Rigor onsets in the jaws, progressing to the upper extremities and then to the lower extremities (Nysten's law; *see also* refs. *7* and *21*). Rigor is

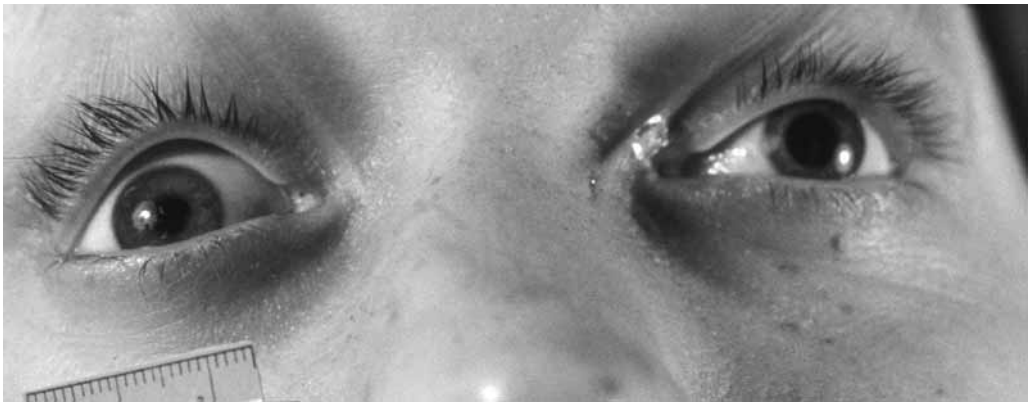

Fig. 3. Pupil inequality after death.

assessed as absent, partial, or complete by manipulation of joints during the external examination (i.e., opening of the mouth to assess the temporomandibular joints and flexion/extension of the elbow and knee joints). The time that rigor is fully established in all joints varies from 2 to 20 h (mean = 8 ± 1 h *[7]*). An individual who dies in the supine position shows slight flexion of the elbows and knees. Rigor persists for 24 to 96 h (mean = 57 ± 14 h *[7]*).

- Resolution (secondary flaccidity):
  - Rigor lessens and eventually disappears as denaturation of actin–myosin linkages occurs with early decomposition. The disappearance will follow the same pattern as the onset. The time range is 24 to 192 h (mean = 76 ± 32 h *[7]*).

Various intrinsic and extrinsic factors influence the development of rigor *(22,23)*:

1. Temperature: increased body or environmental temperature decreases the time of onset and resolution *(24)*. The former is likely the result of increased metabolism of ATP, and the latter from rapid denaturation of actin–myosin linkages. Hyperthermia, under various circumstances (e.g., sepsis, cocaine use), enhances the onset. Rigor persists for days under cold conditions *(25)*.
2. Muscle volume and body habitus: increased muscle bulk delays the onset of rigor, but the rigor will be better developed *(3,26)*. Time for resolution may or may not be increased *(26)*. The elderly, cachectic individuals, and infants have a rapid progression of rigor, but it is less developed and disappears more quickly. Rigor can be absent in emaciated and obese individuals *(1)*.

   Muscle volume is considered a factor that explains why rigor becomes established in small joints before larger ones *(21)*. Rigor mortis occurs more rapidly in small muscles (e.g., masseter muscle), assuming rigor is a physiochemical process occurring at the same time in all muscles *(20,21)*. Animal experimentation shows a greater decline of ATP in the masseter muscle than larger muscles *(20,27)*. The sequence of rigor could also be explained by the proportion of red and white fibers around the joint being manipulated *(21,27)*. Red muscle undergoes rigor more quickly than does white muscle *(21)*. The muscles of mastication in humans have a high proportional area of red muscle fibers compared with the leg muscles (e.g., gastrocnemius; *see also* ref. *21*).
3. Antemortem muscle contraction: increased muscle activity (e.g., exercise, seizure) prior to death hastens rigor *(1)*. Instantaneous rigor (cadaveric spasm, cataleptic rigidity) is characterized by the sudden development of rigor usually localized to muscles that have

Fig. 4. Instantaneous rigor. Hand gripping telephone in a case of sudden death.

forcefully contracted perimortem (Fig. 4; *see also* ref. *1*). Generalized instantaneous rigidity has been described in situations of considerable excitement and tension.

- Significance
  - Prior to the onset of rigor mortis, a body can be manipulated to any position.
  - Once rigor is established, it will not conform to a new position (Fig. 5).
  - Because many factors influence the progression of rigor, it is variable as a measure used to determine the time of death. The inaccuracy is enhanced if only one assessment is done *(28)*.
  - Rigor mortis is broken by manipulation of the body. If rigor is maximum, then it does not return. If rigidity is not fully established, then it returns to a lesser degree in a particular joint (range 2–8 h *[7]*). Forceful manipulation of a stiff joint can tear muscles and fracture a long bone weakened by disease (e.g., osteoporosis, metastasis; *see* Fig. 6). Manipulation of the body occurs at different times prior to the autopsy—i.e., at the scene, during body removal and transportation, and during removal of clothing (e.g., removal of pants from a shirtless individual selectively diminishes rigor in the legs). Rigid upper limbs are deliberately manipulated to allow appropriate autopsy incisions to be made on the torso. Failure to manipulate rigor and expose certain areas of the body results in injuries being missed (e.g., venipuncture in antecubital fossa of an intravenous drug user).
  - Resolution of rigor in the jaws allows the mouth to be examined for injuries, if the mouth could not be opened during the initial examination.
  - Flaccidity following resolution of rigor can relax muscles of the pelvic floor, leading to widening of the vaginal and anal orifices. Suspicion of sexual assault arises (Fig. 7).

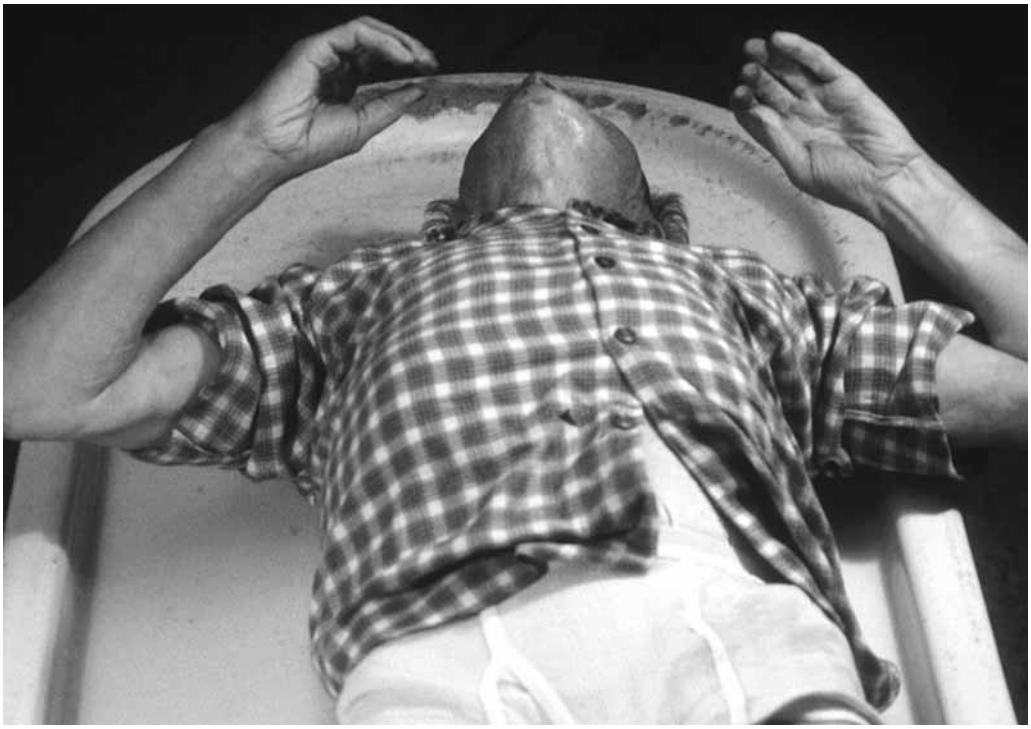

Fig. 5. Man found dead sitting at kitchen table, slumped forward and resting on arms. Moved from scene to autopsy facility.

- The rapid onset of rigor can be linked to certain causes of death associated with increased or abnormal muscle contraction (e.g., electrocution, strychnine poisoning, myotonic dystrophy; *see* Fig. 8).
- "Instantaneous" rigor may be consistent with the circumstances of the death.

3. *Livor Mortis (Livor, Lividity, Postmortem Hypostasis, Postmortem Suggilation)*

Because of the cessation of blood flow at death, there is gravitational settling of blood that distends capillaries and veins, resulting in discoloration of skin in the non-compressed dependent areas of the body (Fig. 9). Slight hypostasis has been described in living individuals dying from a prolonged illness and terminal circulatory failure *(4)*. Patchy lividity (1- to 3-cm or about 0.5- to 1-in. areas) has been described in nondependent areas of the body and attributed to venous blood being squeezed to the skin surface by muscles undergoing rigor mortis *(4)*. A sequence of changes has been described:

- Onset and progression: lividity first becomes apparent 20 min to 4 h after death *(2,7)*. Discoloration is at maximum intensity from 3 to 16 h *(7)*. At "fixation," lividity does not shift with a change of body position (time range = 6 to 12 h). Complete shifting occurs from 2 to 6 h and incomplete shifting (i.e., lividity partly persisting in the original location and changing according to a new position) is possible from 4 to 24 h *(7)*. Fixation can

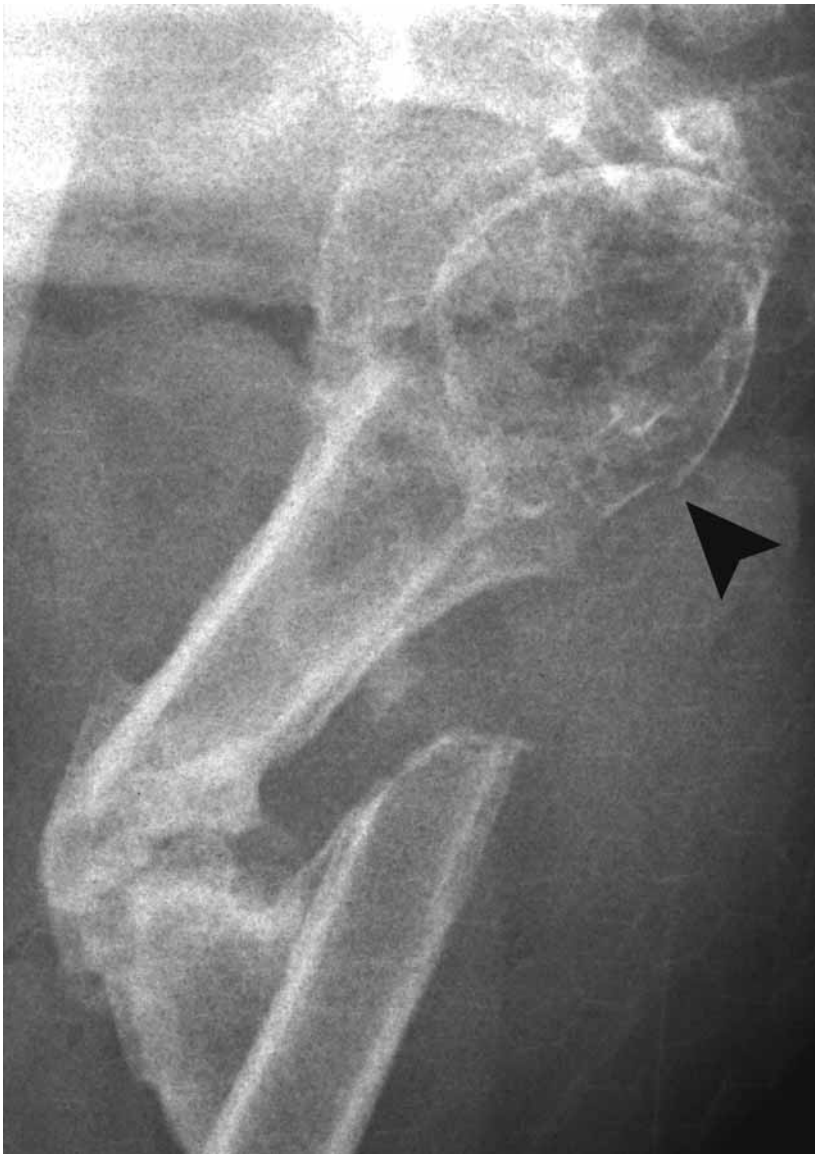

Fig. 6. Fracture of humeral head (arrowhead) caused by forceful manipulation of previously fractured arm in rigor mortis at the scene. (Courtesy of Dr. E. Tweedie, London Health Sciences Centre, London, Ontario, Canada.)

occur up to 3 d after death *(7,29,30)*. Blanching of livid areas by thumb pressure occurs from 1 to 20 h *(7,31)*.

Various mechanisms have been proposed to explain fixation. Blood, initially fluid, clots; however, the presence and time of occurrence of postmortem clotting are variable. Alternatively, there is diffuse extravasation of blood into soft tissues owing to vessel leakage, but this is also observed in nonfixed livor. Blood seepage is not consistently present in fixed lividity *(1,30)*. Blood cannot shift from engorged capillaries because adjacent venous pressure is high *(4)*. Another explanation is that unfaded lividity (fixed livor)

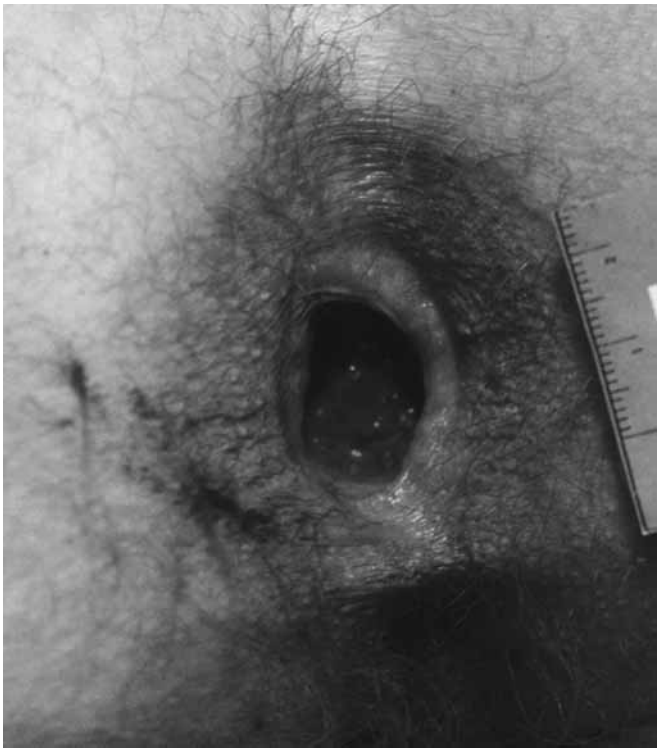

Fig. 7. Postmortem muscle flaccidity, following resolution of rigor, leads to widening of anal orifice.

Fig. 8. Electrocution. Pronounced flexor muscle contraction of the arms of a man holding an antenna, which contacted a high-voltage line. (Courtesy of Office of the Chief Medical Examiner, Chapel Hill, NC.)

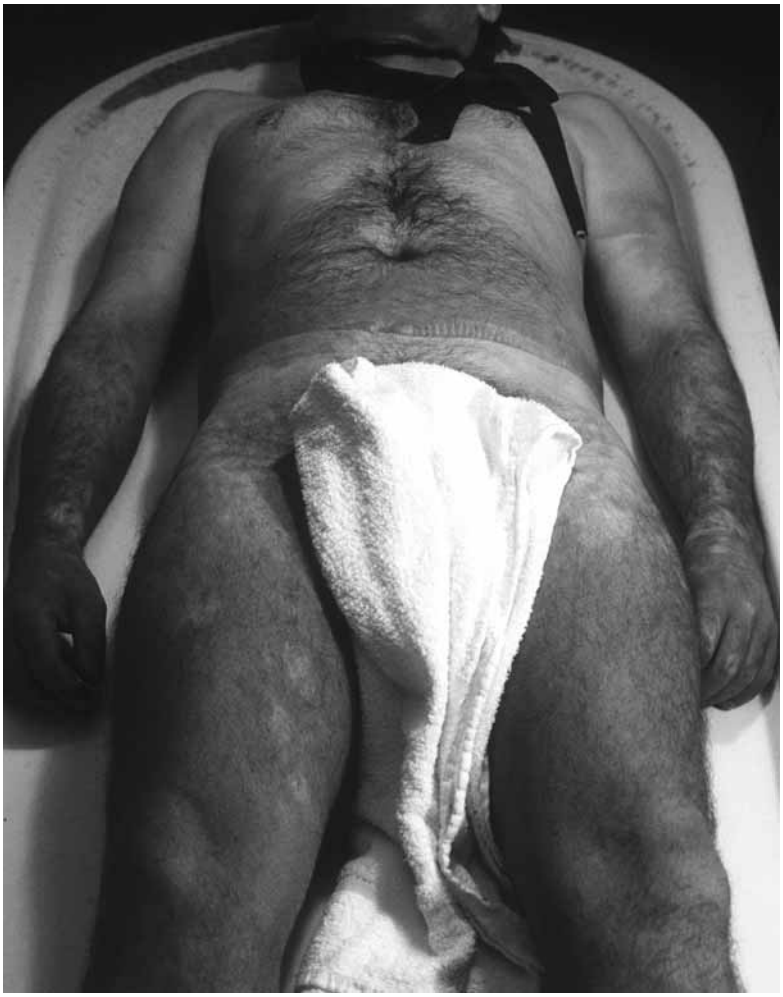

Fig. 9. Suicidal hanging. Suspension for unspecified period of time; lividity in dependent areas of arms and legs.

results from progressive hemoconcentration and red blood cell-clumping resulting from transudation of plasma from engorged vessels *(30,32)*.

- Resolution: livor persists until decomposition occurs.

The significance is as follows:

- The assessment of the degree of lividity can provide only an estimate of the time of death.
- The presence of livor, particularly if fixed, in the nondependent areas of a body at a death scene indicates that the body's position has been changed *(1)*.

3.1. Distribution and "Pressure Points"

Compressed parts of the body result in areas of normal-colored skin demarcated by lividity. If a deceased individual is supine, "pressure points" are observed on the occipital scalp, midback, buttocks, posterior thighs, calves, and heels (Fig. 10). If prone, pressure is applied to the forehead, nose, cheek (if the head is turned), chin, chest, lower abdomen, and anterior thighs. Pressure points also develop in areas of constriction or

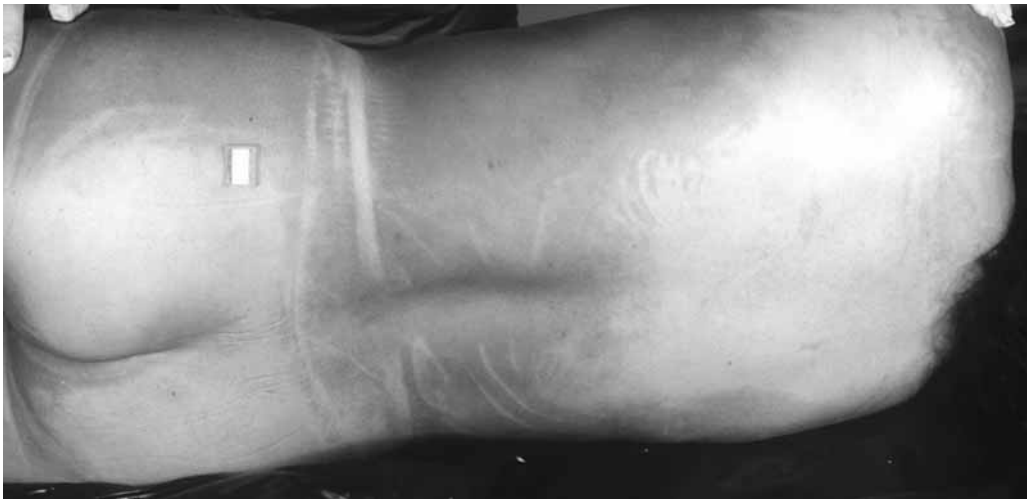

Fig. 10. Person found dead in supine position. Livor on back. Pallor on upper back and buttocks corresponds to typical pressure points. Note pale bands at waistline created by constriction of belt and underwear. Refrigeration had turned the lividity partly red. (*See* Companion CD for color version of this figure.)

localized compression (e.g., tight clothing; *see* Fig. 10). In a supine individual, the larynx pressing the esophagus against the spine can cause a transverse band of pallor across the cricoid esophagus *(4)*.

- Significance
  - Irregular pale areas in livor on the neck can suggest to an investigator the possibility of manual application of pressure—i.e., strangulation; however, this scenario is not realistic, as pressure would need to be sustained during the time livor is developing.
  - Horizontal pale lines on the skin of the neck suggest ligature compression, assuming a ligature had been present while livor was developing. Taping or binding an endotracheal tube around the cheeks and neck can create this artifact (*see* Chapter 3, Fig. 20). Similarly, prominent skin folds can squeeze an intervening crease (*see* Chapter 3, Fig. 20).
  - If part of the body is resting on a particular surface or object, then its negative image can be reproduced on the livid site. This observation can be linked to the scene and circumstances of the death (Fig. 11).

3.2. Discoloration

Livid skin is either purple/blue or red, depending on the concentration of deoxygenated blood. Red livor is seen if the body is cold (Fig. 10). This is indistinguishable from certain poisoning deaths (e.g., carbon monoxide poisoning *[33]*). Lividity is difficult to discern in a dark-skinned individual. Decomposition turns livid areas brown or green. Dependent organs and tissues appear "congested" or hemorrhagic (e.g., if supine, posterior lungs and retroperitoneum, including kidneys; if prone, anterior organs such as heart and intestines).

- Significance
  - Lividity is poorly developed in individuals who are anemic, have bled severely, or had organs harvested.

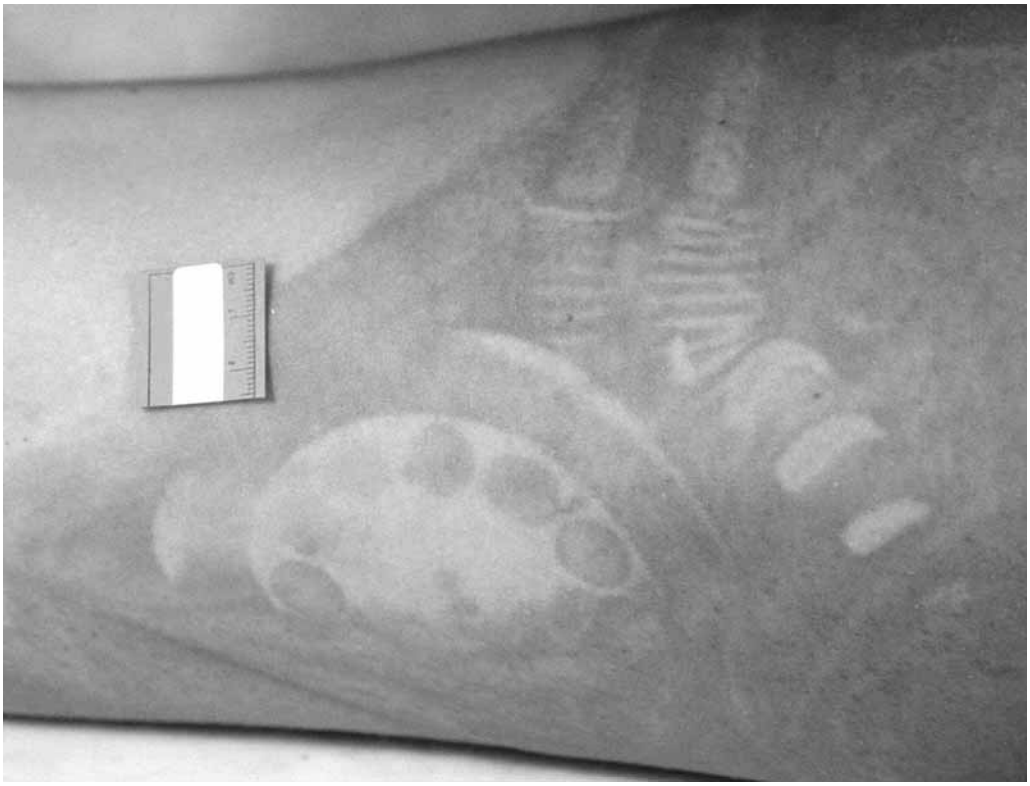

Fig. 11. Person found dead with thigh resting on rotary telephone. "Negative" impression of dial and extension cord in livid area.

- Apparent cyanosis of the face could be lividity. The heart of a large individual is at a higher level than the more dependent head.
- Different colors of lividity are associated with certain poisoning deaths *(1)*:
  Red—carbon monoxide (*see* Chapter 3, Subheading 3.9.; *see also* Fig. 12); fluoroacetate; cyanide.
  Green—hydrogen sulfide (also generated during decomposition).
  Brown—methemoglobinemia (e.g., ingestion of nitrates; *see also* Fig. 13).
  Intense purple—propane *(34)*.
- During the initial external examination, livor can obscure an antemortem contusion (Fig. 14). Body repositioning during the autopsy can shift lividity from an apparent bruise, allowing better visualization. The suspicious area can be examined at the conclusion of the autopsy dissection, when blood has drained from the body and the intensity of lividity has diminished. Re-examination of a refrigerated autopsied body the next day may reveal bright red but diminished livor in contrast to a darker bruise (Fig. 14).
- Antemortem injuries are mimicked by lividity. Patchy red/blue skin discoloration suggests a contusion. If such an area is due to nonfixed lividity, then thumb blanching helps distinguish it from a bruise. Similarily, incision of an area of lividity is more likely to result in oozing of nonclotted blood from vessels in contrast to a true contusion, where blood has seeped into soft tissue.

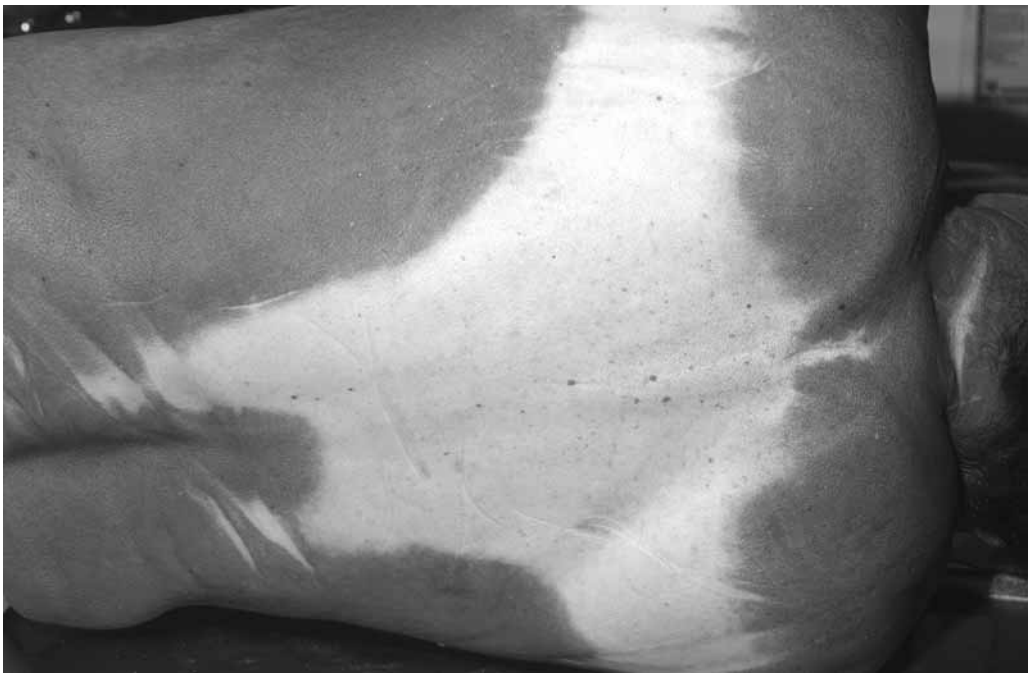

Fig. 12. Red livor mortis on back. Carbon monoxide poisoning. (*See* Companion CD for color version of this figure.)

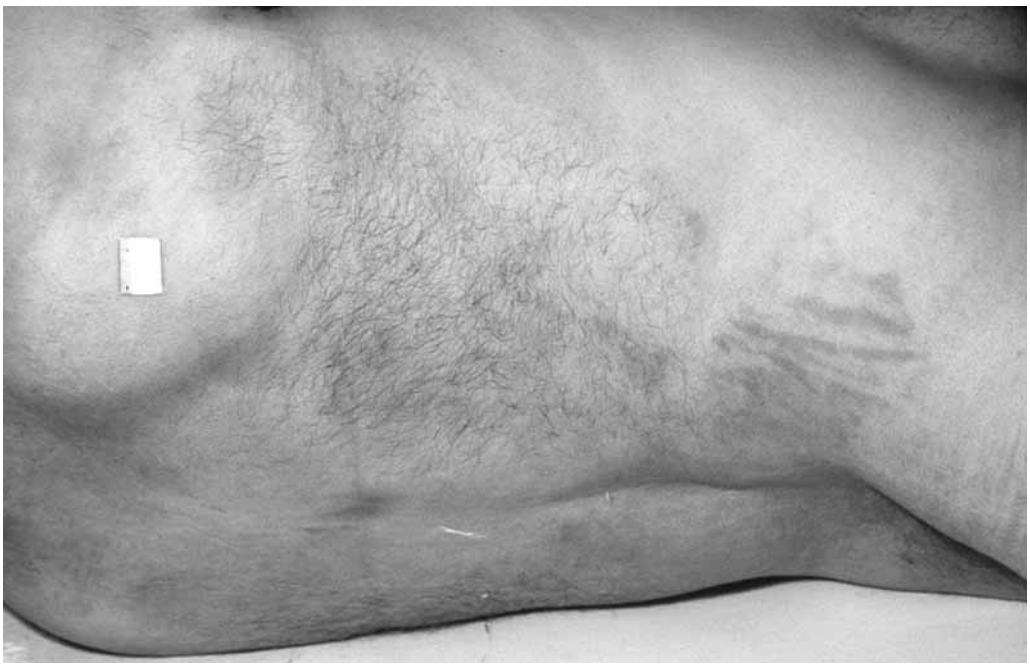

Fig. 13. Brown livor mortis on back. Methemoglobinemia owing to nitrate ingestion. (*See* Companion CD for color version of this figure.)

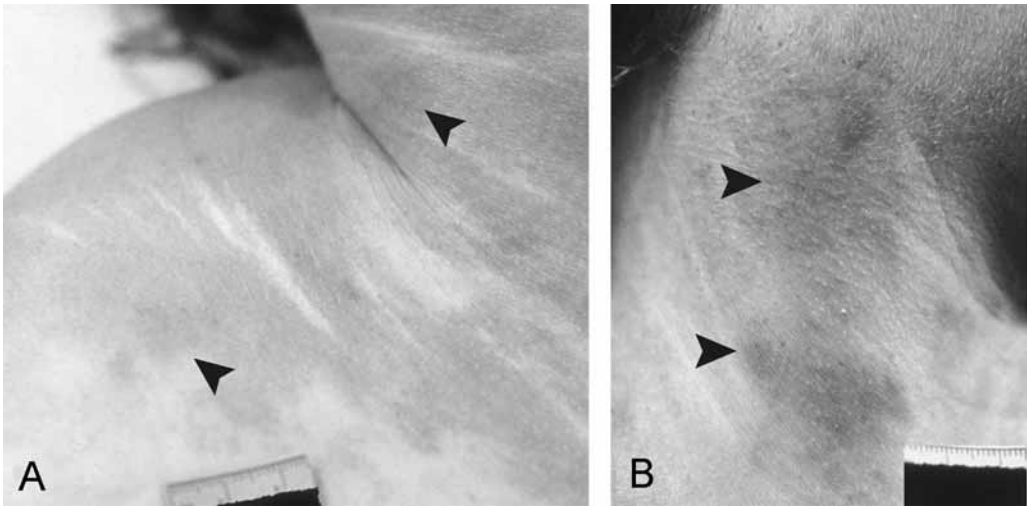

Fig. 14. Woman strangled using armhold. **(A)** Pale linear lines on lateral aspect of neck are caused by neck creases in area of livor mortis. Indistinct bruises (arrowheads) **(B)** Examination of the deceased next day after autopsy dissection. Lividity diminished allowing visualization of extensive bruising (arrowheads) on lateral neck. (*See* Companion CD for color version of this figure.)

3.3. Postmortem Hemorrhage

Bleeding occurs after death if blood vessels, engorged by blood postmortem, rupture. Contributory factors include increased pressure caused by body position, early decomposition, and trauma. Rupture of capillaries is manifest as petechiae (Tardieu spots). The development of petechiae is a harbinger of decomposition, but they can be seen as early as 2 to 4 h after death, if the body has been suspended *(6)*. Rupture of larger vessels (venules) causes purpura or larger ecchymoses. The distribution of postmortem cutaneous hemorrhage is in areas of livor and is not seen in areas uninvolved by lividity (Fig. 15).

- Significance
  - Postmortem hemorrhage (petechiae, purpura/ecchymoses or "pseudobruises," external bleeding) mimics true injuries occurring antemortem (*see* Chapter 8, Subheading 3.1.; *see also* Fig. 16).
  - Open antemortem wounds in dependent body sites can continue to ooze blood after death. Open postmortem injuries (e.g. abrasions, lacerations) can redden and "bleed" mimicking a vital reaction *(35,36)*.
  - A significant amount of bleeding can occur from major vessels in a dependent part of the body (e.g., aorta, ranging from 100 to 1300 mL [average 450 mL] into the pleural cavity *[37]*). The presence of a hemothorax from a traumatically ruptured aorta in the context of an instantaneously fatal brainstem injury implies that bleeding likely occurred postmortem *(37)*.
  - Microscopic examination cannot distinguish between the extravasation of blood seen in an early antemortem contusion and postmortem hemorrhage.
  - Loose supporting connective tissue (e.g., face, neck) in a dependent body site can promote the development of external and internal postmortem hemorrhage (*see* Chapter 5, Fig. 5).

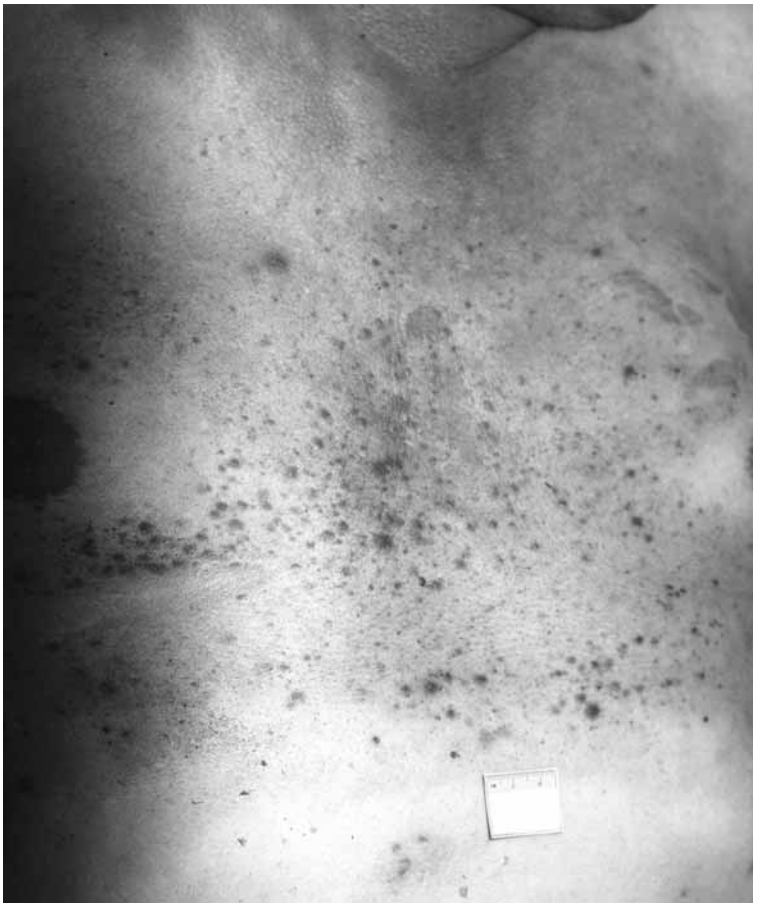

Fig. 15. Found prone. Lividity on chest associated with numerous petechiae, which did not involve the nonlivid abdomen.

Nosebleeds can occur *(3)*. "Pseudobruises" tend to be small; however, pronounced facial bruising has been described in an individual who was in a head-down position and sustained postmortem scalp injuries *(38)*. Periorbital hematomas ("black eyes") can be caused by direct trauma, tracking of blood from a scalp wound, and hemorrhagic extravasation from an orbital plate fracture *(39)*. Black eyes can occur around the time of infliction of a rapidly fatal injury *(39)*. They can also be observed postmortem (*see* Heading 11.). Removal of eyes for corneal transplantation can cause periorbital hemorrhage (Fig. 17; ref. *39*). Postmortem head trauma can result in periorbital hematoma and is enhanced by lividity *(39)*. A small hematoma of a single eyelid, compared with extensive hemorrhage in both eyelids, in the absence of direct trauma is not necessarily an antemortem event *(39)*. The presence of postmortem petechiae and larger confluent hemorrhages in the conjunctiva and sclera raises the suspicion of asphyxia from neck compression (Figs. 18 and 19, and refs. *40–42*). Information from investigators about the circumstances of the death and the exclusion of internal neck trauma by careful neck dissection help rule out foul play.

A head-dependent position is associated with temporalis muscle and galeal hemorrhages simulating blunt trauma (*see* Chapter 3, Fig. 2; *see also* ref. *40*). The

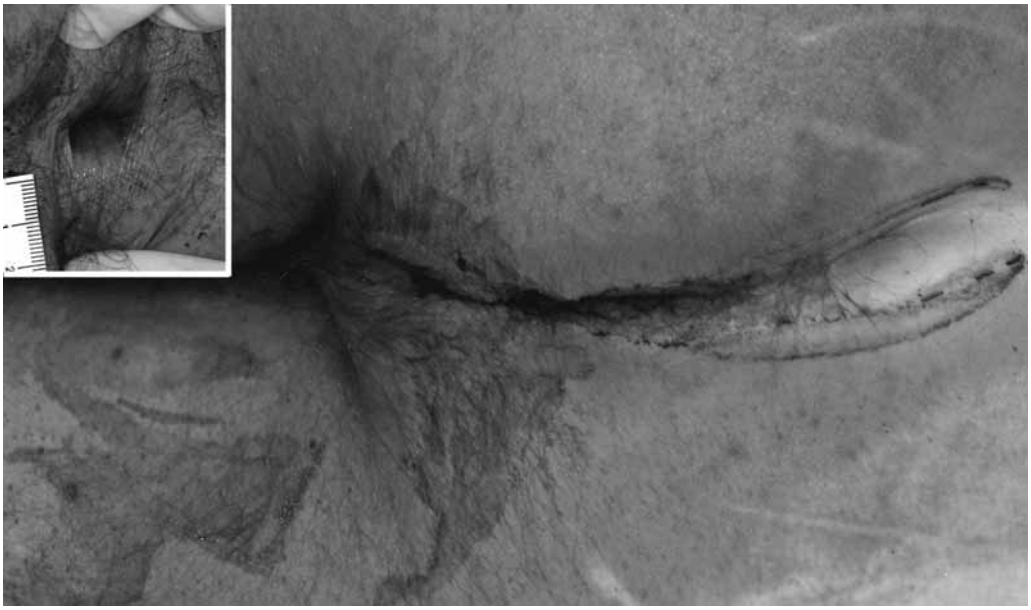

Fig. 16. Suicide by hanging. Victim had also bound his flexed legs with electrical cord (*see* Chapter 3, Fig. 11). Intense lividity of lower extremities and petechiae noted on posterior legs. Perianal "hemorrhage." No evidence of anal trauma (inset). Congested mucosal veins were seen.

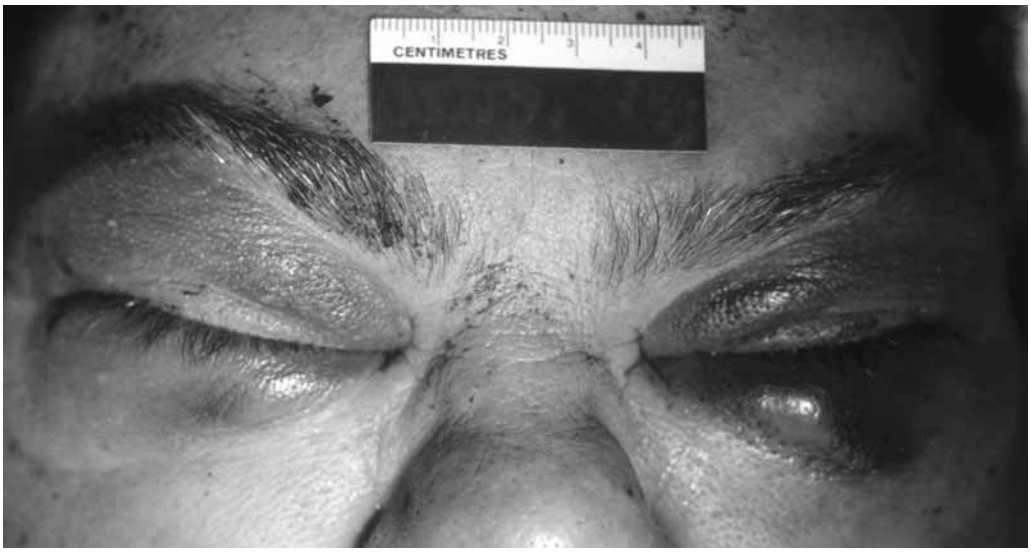

Fig. 17. "Black" eyes. Eyes removed postmortem for the purpose of donation. Sudden death owing to cardiomyopathy in an obese individual. (Courtesy of Dr. D. King, Regional Forensic Pathology Unit, Hamilton, Ontario, Canada.)

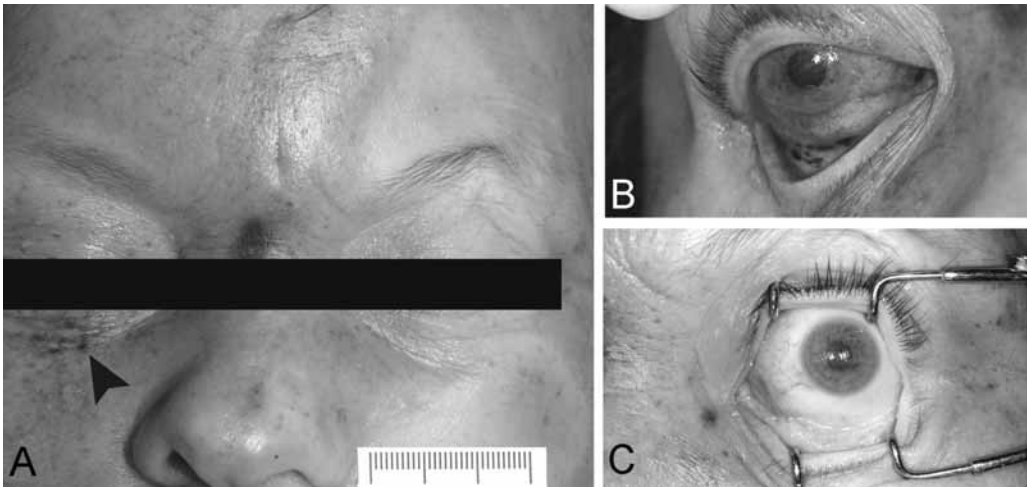

Fig. 18. Postmortem orbital petechiae, resulting from the prone head-down position. **(A)** Lividity of face with sparing of left side which was pressed against the floor. Note petechiae below right eyelid (arrowhead). **(B)** Right lower eyelid retracted to show conjunctival petechiae. **(C)** Left eye showed no petechiae.

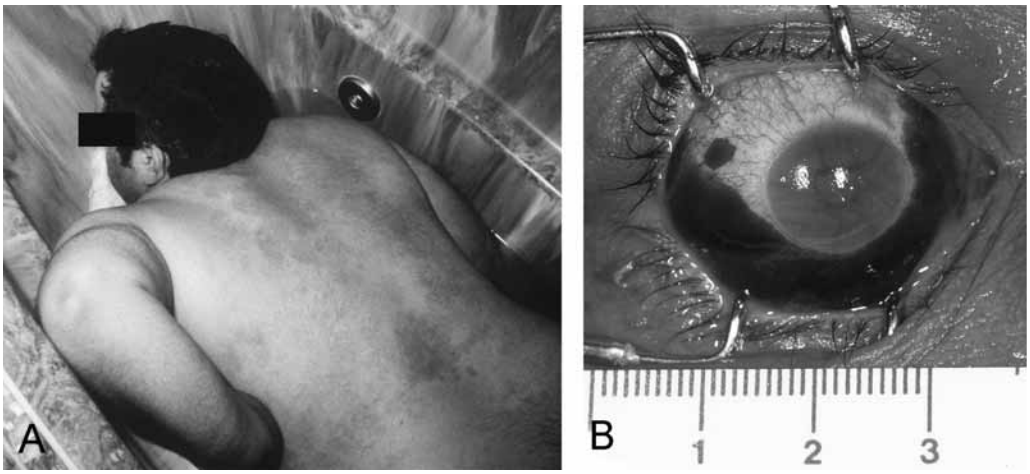

Fig. 19. Postmortem orbital hemorrhage. **(A)** Found prone in tub. **(B)** Confluent ocular hemorrhage owing to pronounced facial lividity.

head-down position has been recognized as a predisposing factor for the development of soft-tissue neck hemorrhage in fatal drownings *(6)*. These hemorrhages can be extensive (Fig. 20 and ref. *43*). Focal hemorrhages are found in the anterior, middle, and posterior compartments of the neck and are, at least, partly attributable to hypostasis (*see* Chapter 5, Subheading 10.6. *[43]*). The finding of neck hemorrhage is not exclusive to drowning and is found in any situation in which lividity occurs in a body that is prone or head-down (*see* Chapter 3, Subheading 1.4.; Chapter 5, Heading 12.). The effect of the prone or head-down position is enhanced if the buttocks are elevated.

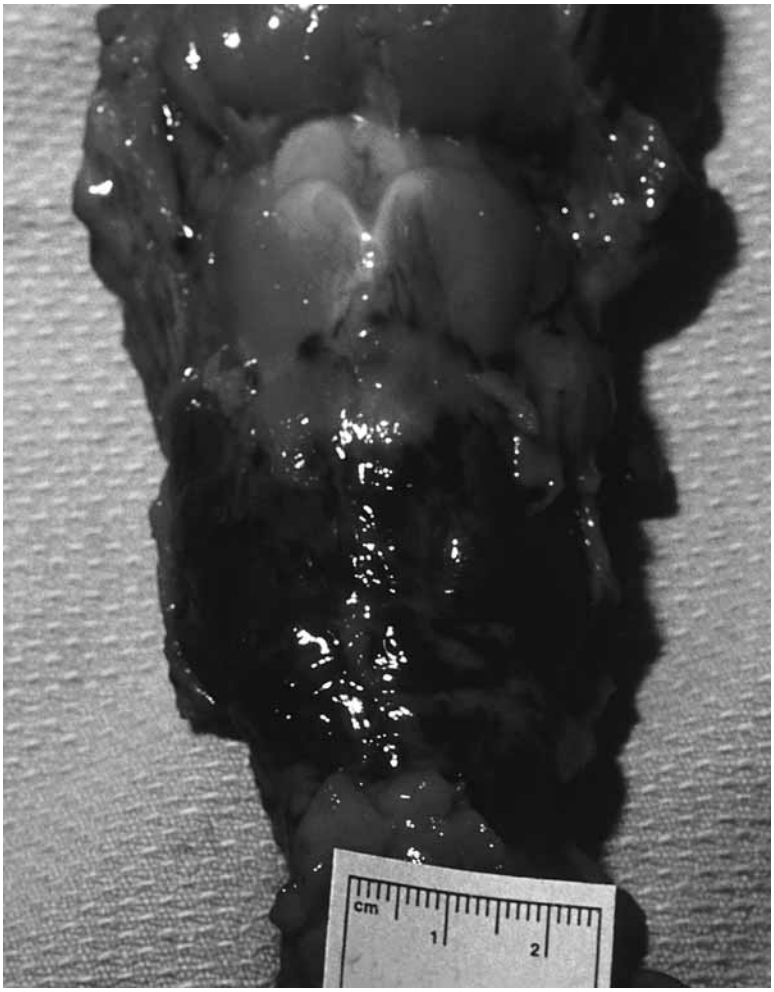

Fig. 20. Extensive soft-tissue hemorrhage in anterior neck in individual found prone. No fractures of the hyoid–larynx were observed.

4. Body Cooling (Algor Mortis)

Postmortem heat loss occurs by four mechanisms, depending on the scene and circumstances *(4,6,7)*:

- Evaporation—humidity and precipitation vary the amount of evaporation from skin and clothing.
- Radiation—as a factor, it decreases as the body cools (e.g., sun shining on a body, cooling system in a dwelling).
- Conduction—heat exchange occurs between contact surfaces owing to temperature differences.
- Convection—heat loss is from air exchange from noncontact surfaces.

Loss of body heat postmortem has been described as a sigmoidal decline over time *(4,7)*. The initial plateau phase assumes that the body is a solid cylinder that

loses heat from its surface according to a slowly developing temperature gradient. During this time, body metabolism continues to generate heat. If this phase is not considered, then a calculation based on rectal temperature, used to estimate the time of death, is affected. A linear decrease in rectal temperature follows and is proportional to the temperature difference between the warm body surface and cooler environment. The final plateau phase marks the decreased effect of the temperature gradient.

Various equations, algorithms, and nomograms using rectal temperature have been developed *(44)*. As stated by Henssge et al., "even with the increased complexity of the algorithms, this does not guarantee any greater accuracy of the calculated times" *(7)*. Simple rule-of-thumb formulae are comparable to more complex methods *(6,7)*.

Examples include:

1. Time since death = (Rectal temperature at time of death – measured rectal temperature [°F]) ÷ 1.5
2. Time since death = (Rectal temperature at time of death – measured rectal temperature [°C]) + 3*

Models developed to determine the postmortem interval based on body temperature rely on assumptions and a number of factors, some incalculable *(1,7)*. The rectal temperature reflects the core body temperature (98.6°F or 37°C). Normal rectal temperature varies in living individuals (in adults, 34.2–37.6°C or 93.6°–99.7°F) depending on many factors (e.g., diurnal variation, exercise *[7]*). Because of less developed thermoregulatory control, newborns and premature infants can have an elevated rectal temperature. Body temperature increases under certain conditions (e.g., sepsis, use of certain drugs [cocaine, neuroleptics], nonfebrile diseases [hyperthyroidism]). Depending on environmental conditions, rectal temperature can be raised or lowered. Hypothermia increases inaccuracy. A prolonged agonal period may follow injury *(6)*. During this time, body temperature regulation can be affected and cooling may occur before death.

The greater the difference between body and environmental temperature, the faster is the rate of cooling *(4)*. Submersion in cold water cools a body more quickly *(4)*. Uncertainty arises if there have been changes in environmental temperature between the time of death and temperature recording. The investigator must realize that the body recovery site is not necessarily the place of death. Increased body size, body fat, and clothing/coverings decrease the rate of cooling *(4,7)*. A thin unclothed body in a cold environment may not exhibit an initial temperature plateau *(4)*. Body position (extended or crouched) can influence body heat loss *(7)*.

5. Decomposition

Decomposition is the process of progressive postmortem dissolution. Decomposition is the result of autolysis and putrefaction *(45)*. Autolysis is tissue softening and liquefaction caused by the release of intracellular enzymes. Putrefaction is liquid and gaseous transformation of tissues and organs by local and hematogeneous spread of intestinal bacteria and

* Compensatory number to account for possible initial delay in cooling of body.

other microorganisms. Insect and animal predation (anthropophagy) contribute. Putrefaction is enhanced if there are open skin wounds. Decomposition is delayed in newborns because meconium is sterile.

- Significance
  - Decomposition appears after other postmortem changes (rigor mortis, livor mortis, decrease in body temperature) have occurred. As a determinant of the time of death, decomposition is subject to considerable variability.
  - A decomposed body poses an identification challenge.
  - A decomposed body found in a dwelling can reflect social isolation *(46)*. A claim by next of kin for burial may be slow in developing.

5.1. Factors Promoting Decomposition

Factors that promote the onset and progression of decomposition increase autolysis and bacterial growth, enhancing putrefaction *(45)*.

- Heat: a warm environment promotes decomposition *(47)*. An elevated body temperature hastens the onset—e.g., sepsis can accelerate decomposition even in patients who die in the hospital. Extreme heat retards decomposition by enzyme deactivation and reduction in the number of bacteria. Refrigeration does not necessarily delay decomposition. A frozen body can decompose faster when it thaws, as bacteria multiply.
- Environmental medium: the type of environment in which a body is found affects the rate of decomposition. Casper's rule is that 1 wk of open exposure to air is equivalent to 2 wk of water submersion and 8 wk of burial in soil *(45)*. The deeper a body is buried, the better its preservation during an elapsed period of time *(48)*. This is because the body is cooler, slowing decomposition, and is less subject to insect and animal activity *(45,48)*. Corpses are spared this activity when buried at a depth of about 1 m (3 to 4 ft) *(45, 47, 48)*. Bodies buried in peat have been well preserved for centuries *(45)*.

  Increased conductive heat loss in a body exposed to a certain medium slows decomposition. For example, a body lying on a conductive (metal) surface can decompose more slowly.
- Body habitus: infants and children cool more quickly, delaying decomposition. The insulative properties of fat decrease heat loss and increase the rate of decomposition in an obese person, although some have not observed this *(47)*.
- Clothing and other coverings: heavy clothing and thick coverings retain body heat and accelerate decomposition, but increased external pressure by tight clothing can delay decomposition by preventing gaseous expansion of tissues and bacterial spread in the bloodstream *(3)*.
- Body position—postmortem congestion: increased intravascular blood volume (congestion) promotes bacterial growth and dissemination. Blood loss decreases putrefaction. Anterior lividity resulting from a prone position congests intestinal vessels, accelerating putrefaction. Decomposition is enhanced in the head, neck, and upper chest in an individual in the head-down position.
- Trauma: injured sites provide an entry site for bacteria and faster decomposition *(45,49)*.

5.2. Decomposition Changes (The "Four Ds")

The onset and progression of decomposition are variable. In temperate conditions, changes begin 24 to 48 h after death.

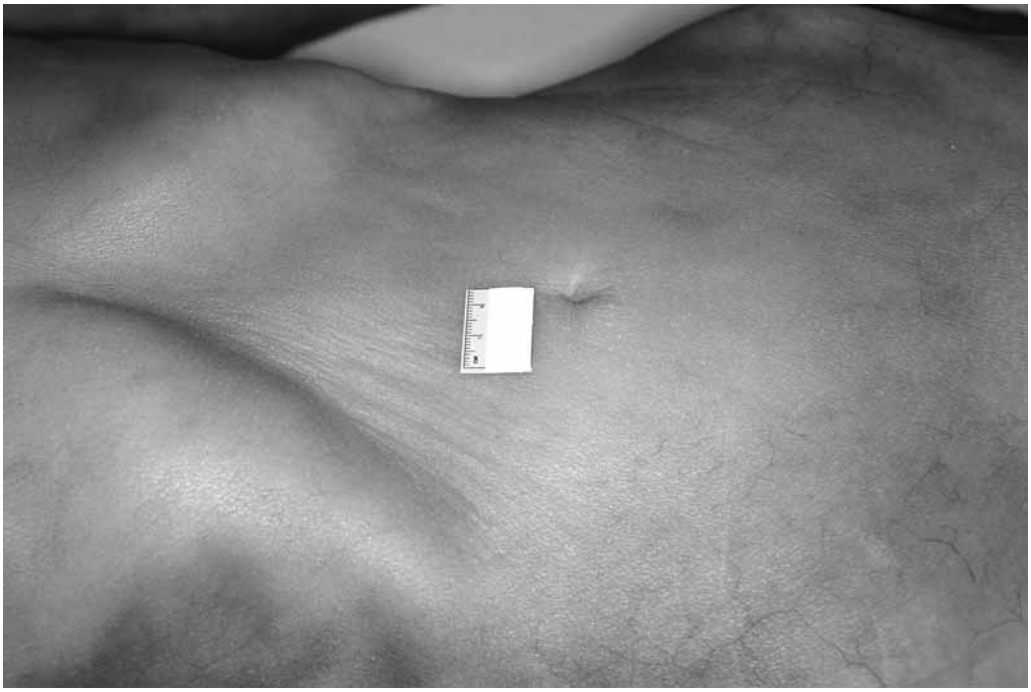

Fig. 21. Early decomposition. Green discoloration of abdomen. (*See* Companion CD for color version of this figure.)

5.2.1. Discoloration

Hemolysis and generation of hydrogen sulfide gas from anaerobic bacteria such as *Clostridium welchii* result in the earliest decomposition change, i.e., blue-dark green discoloration in the lower abdominal quadrants (right to left owing to proximity of cecum to skin; *see also* Fig. 21 and ref. *50*). Eventually the entire abdomen becomes discolored, followed by the rest of the body. Seepage of hemoglobin pigments from hemolysed blood and gaseous rupture of blood vessels leads to localized skin discoloration in areas of lividity (green, purple, black), mimicking contusions (Fig. 22). "Marbling" refers to an arborescent pattern of discoloration due to hemolysis following a vascular distribution (Fig. 23).

- Significance
  - Localized cutaneous discoloration from decomposition mimics antemortem bruises (Fig. 22 and ref. *51*). One study tried to resolve this problem. Although hemoglobin pigments filter easily through blood vessels, erythrocyte membranes diffuse less readily because of their molecular size *(51)*. Because bruises are caused by actual disruption of blood vessels, they contain a large amount of erythrocyte membrane *(51)*. Immunohistochemical studies of contusions and postmortem discolorations showed that a positive reaction for glycophorin A, a component of red blood cell membrane, indicated an antemortem injury *(51)*. Glycophorin A was not found in postmortem bruises and was present in only a minority of contusions occurring during life.
  - Pre-existent contusions and external hemorrhage are accentuated by hemolysis and diffusion of blood.
  - Diffuse extravasation of blood in deeper soft tissues and organs raises the possibility of disease and injury, e.g., diffuse "hemorrhage" in posterior scalp of a supine

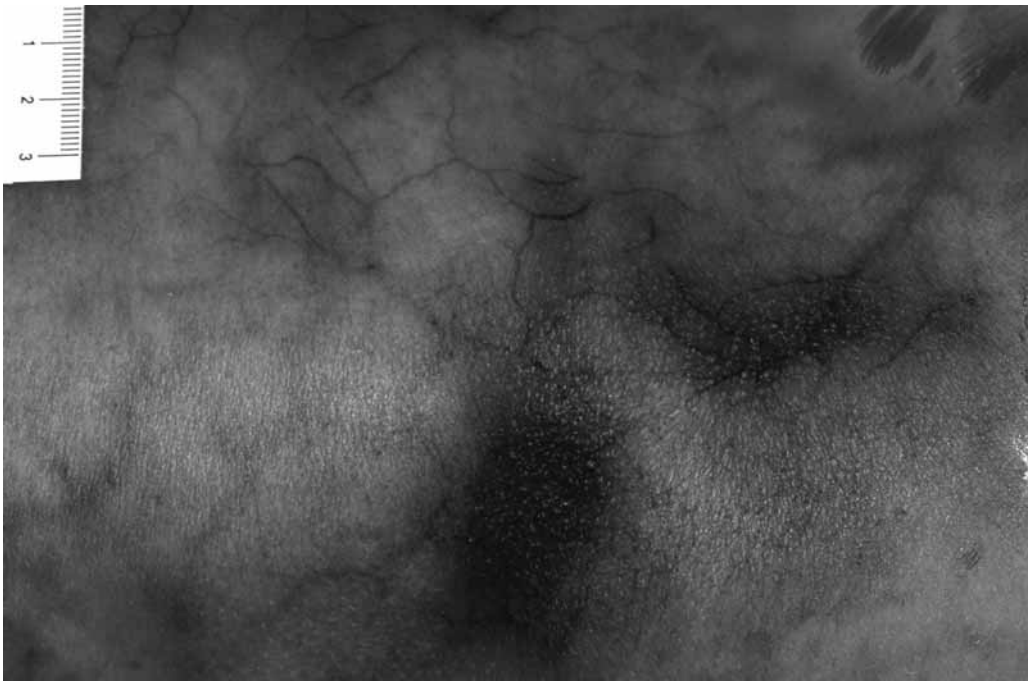

Fig. 22. Focal discoloration from decomposition mimics antemortem bruising. Note prominent blood vessels on the livid back. (Courtesy of Dr. M. Moussa, London Health Sciences Centre, London, Ontario, Canada.)

individual suggests trauma; "congested" hemorrhagic lungs (microscopy can even show apparent intra-alveolar edema); pancreatic "hemorrhages" simulating pancreatitis.

- Seepage of sanguinous fluid into body spaces and cavities suggests antemortem injury, e.g., "Hemothoraces" of less than a few 100 mL of sanguinous fluid in each chest cavity are consistent with decomposition; markedly "congested" vessels and thin films of blood in the subdural/subarachnoid spaces, particularly in the occipital lobe area in a supine individual, mimic subarachnoid hemorrhage (Fig. 24). Inspection of the vertex of the brain, after the calvarium is removed, is advantageous because softening from decomposition hinders orientation and examination once the brain is removed.
- Pink teeth occur during decomposition and result from hemolysis of extravasated blood in the dentinal tubules (Fig. 25; refs. *52–57*). Postmortem production of carboxyhemoglobin is not a factor *(55,57)*. Cephalic congestion, when it results from a head-down position, and a moist environment (e.g., drowning victim) are factors that promote the development of pink teeth *(55–57)*. Fingernails can also be discolored pink *(57)*. Pink discoloration of teeth is independent of the cause of death *(53,56,57)*. This change is observed within 1 to 2 wk following death *(54,56)*.

5.2.2. Distension

Various gases are produced (e.g., hydrogen sulfide, methane, ammonia, carbon dioxide) during putrefaction. Gaseous permeation into skin/soft tissue and organs manifests as crepitus and distension. Localized swelling of the genitalia, anus, and face (bulging eyes, tongue protrusion) is seen. The deceased is unrecognizable. Generalized body distension

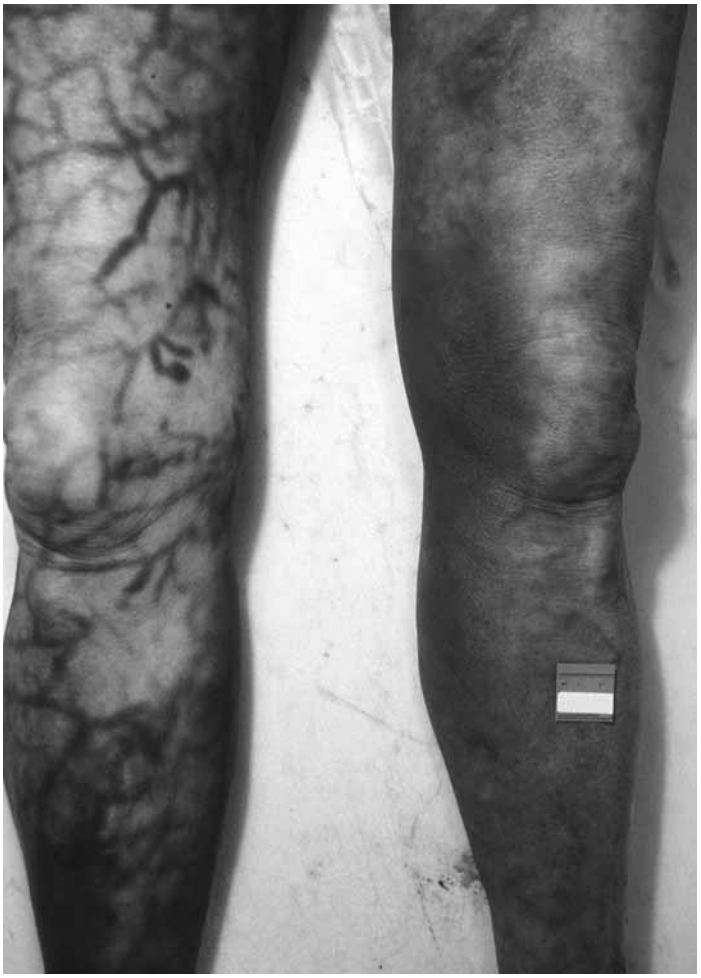

Fig. 23. "Marbling" of right leg in contrast to lividity of left leg. The blood vessels have been accentuated by decomposition.

can suggest an apparently large individual. Gas accumulates in body cavities, and their opening is marked by an audible expulsion and visible deflation of the body.

- Significance
  - Apparent "epistaxis" caused by rupture of nasal vessels occurs.
  - Gaseous distension of lungs leads to expulsion of sanguinous fluid and gastric content from the mouth and nose ("purge"), simulating a facial injury (Fig. 26). Gas can force feces from the rectum. Postmortem expulsion of a fetus has been described *(3)*.
  - Postmortem surgical wound dehiscence mimics sharp-force injury. A history of prior surgery and the finding of sutures in the wound solve this problem *(58,59)*. Dehiscence occurs even in wounds that are months old *(58)*.

5.2.3. Degradation

Decomposition causes a loss of anatomic integrity of skin and other tissues. Localized areas of skin peel ("skin slippage") occur (Fig. 27). Gas- or fluid-filled cutaneous blisters form (Fig. 28). Broken blisters and areas of skin slippage dry out, leaving yellow/brown/red

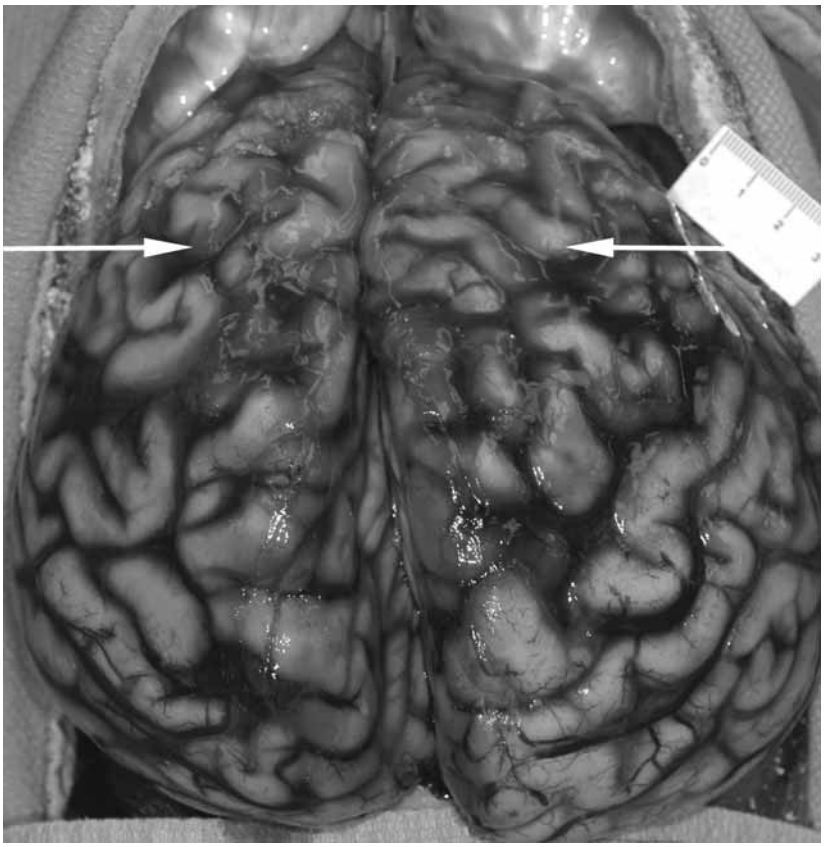

Fig. 24. Decomposed body. Note "congestion" of subarachnoid blood vessels on the posterior aspect of the brain (below level of arrows).

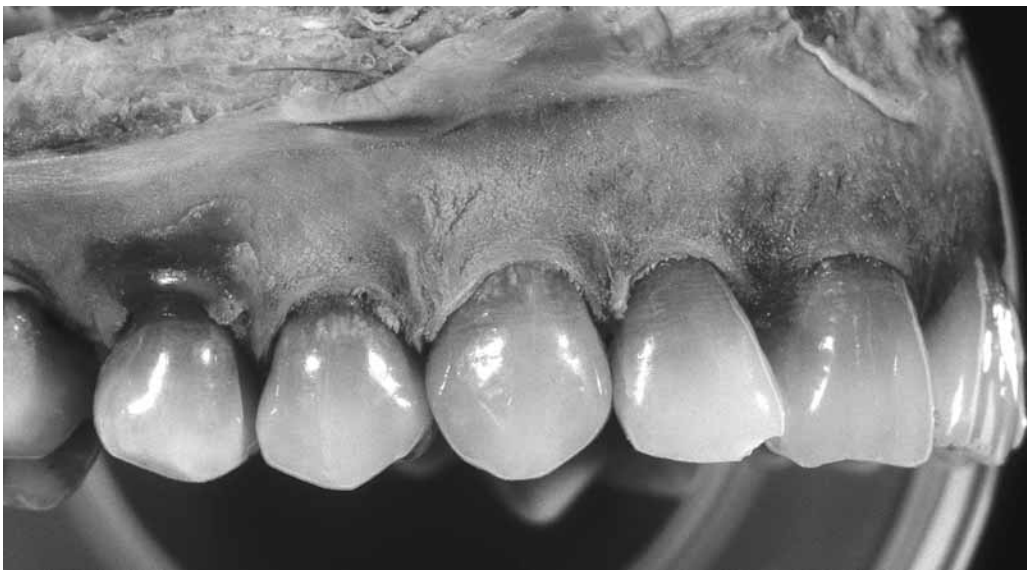

Fig. 25. Pink teeth caused by decomposition. (*See* Companion CD for color version of this figure.)

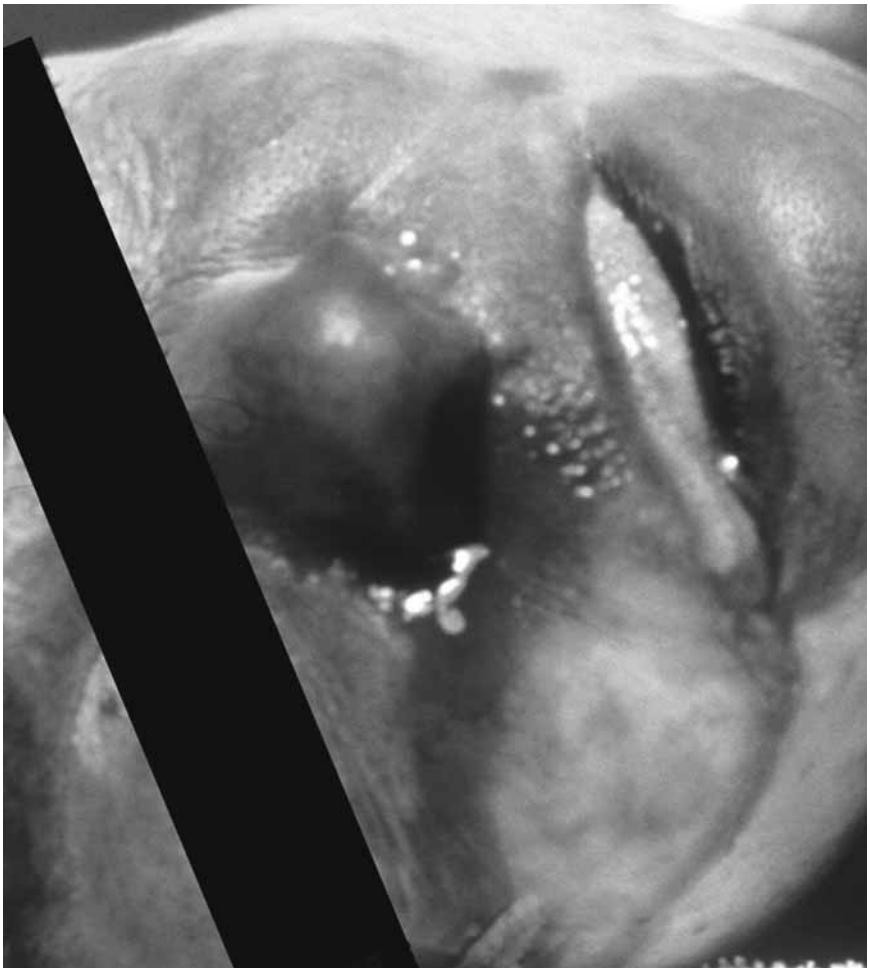

Fig. 26. Decomposition. "Purge" of sanguinous fluid from nostrils and mouth.

areas with a parchment-like texture (Fig. 29). The attachment of hair and nails loosens, allowing for their easy removal. Scalp hair remains as a mass in warm weather *(47)*. Loosening of skin of hands and feet leads to "degloving" (Fig. 30). Apparent ruptures of the upper gastrointestinal tract (esophagus, esophagomalacia; stomach, gastromalacia) are observed. If microscopic examination shows an inflammatory reaction, then the rupture is antemortem. Cystic change in the brain (encephalomalacia) mimics infarcts.

- Significance
  - Loss of skin integrity mimics trauma (e.g., abrasion).
  - Blister formation simulates thermal injury.
  - Ocular petechiae can disappear with decomposition, although drying can preserve them *(42)*.

5.2.4. Dissolution

Certain organs and tissues (e.g., pancreas) decompose quickly, and others (e.g., bone, uterus, prostate) are slower to decay. Progressive decomposition leads to disappearance of

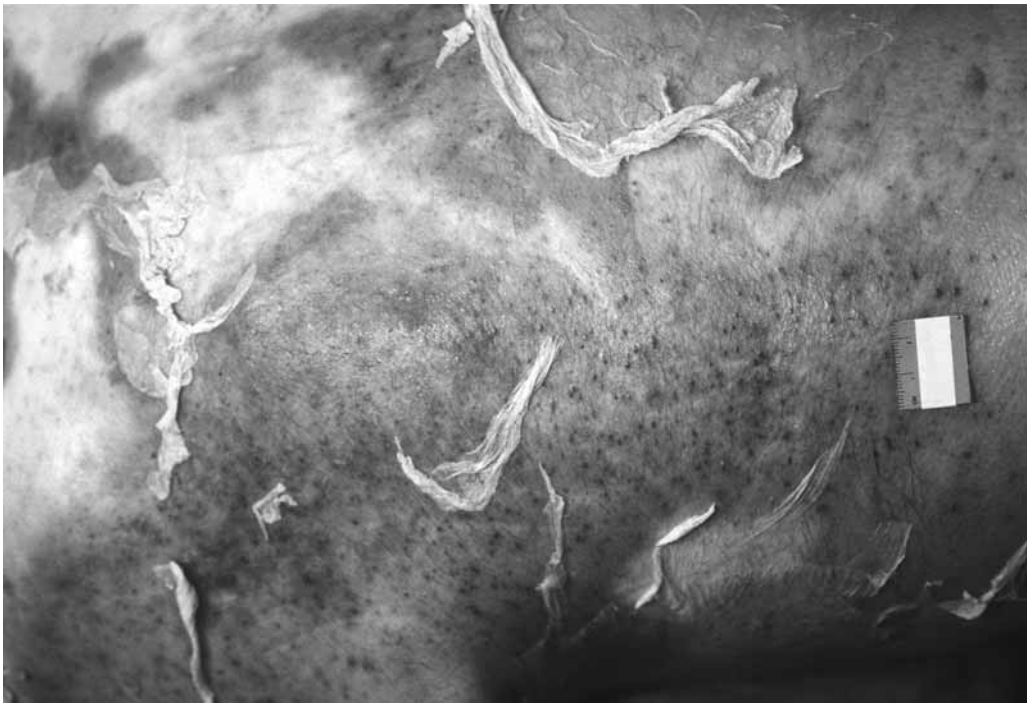

Fig. 27. Decomposition. Skin slippage.

Fig. 28. Blisters from decomposition.

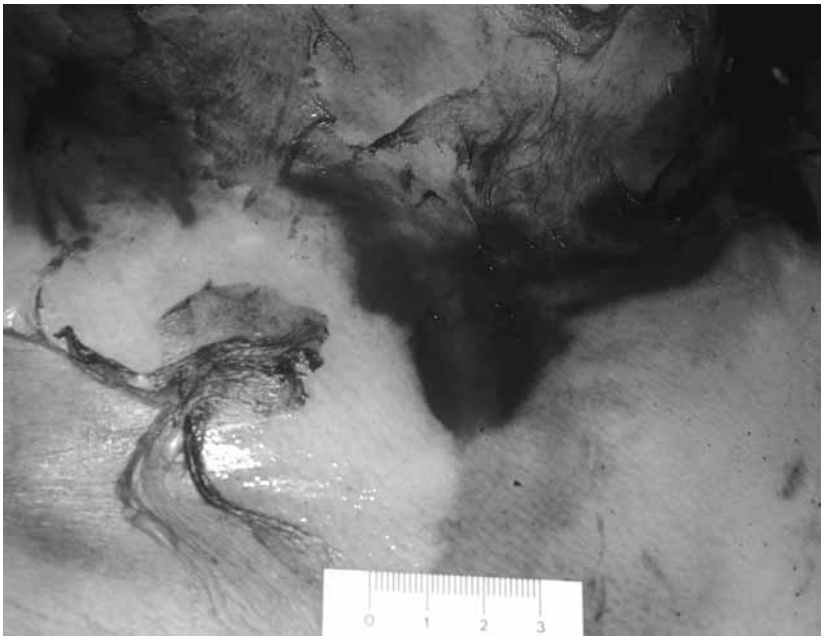

Fig. 29. Decomposition. Neck and upper chest show skin slippage. Band of yellow-brown discoloration following distribution of collar.

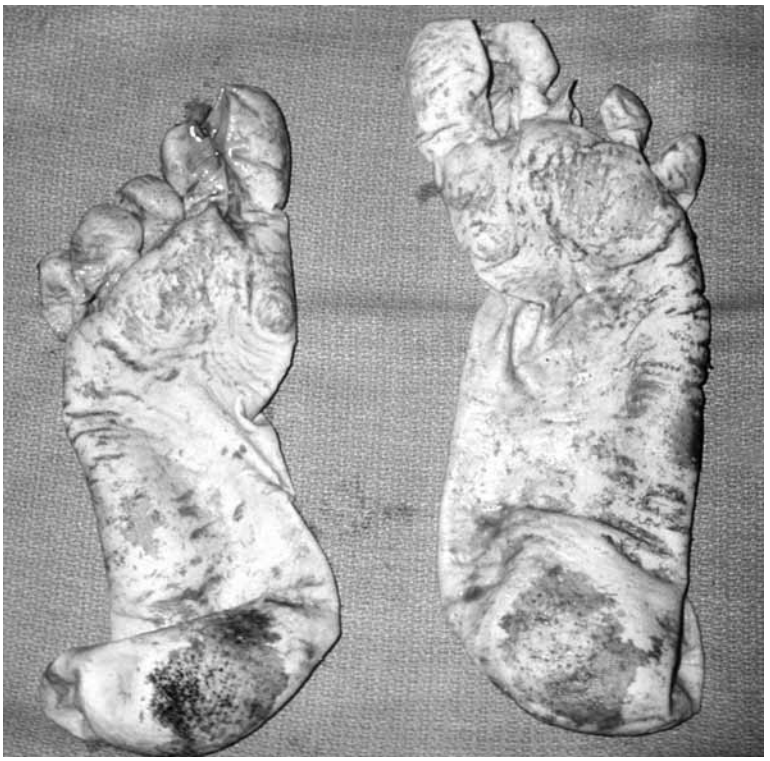

Fig. 30. Decomposition. Degloving of skin of feet. (Courtesy of the Office of the Chief Medical Examiner, Chapel Hill, NC.)

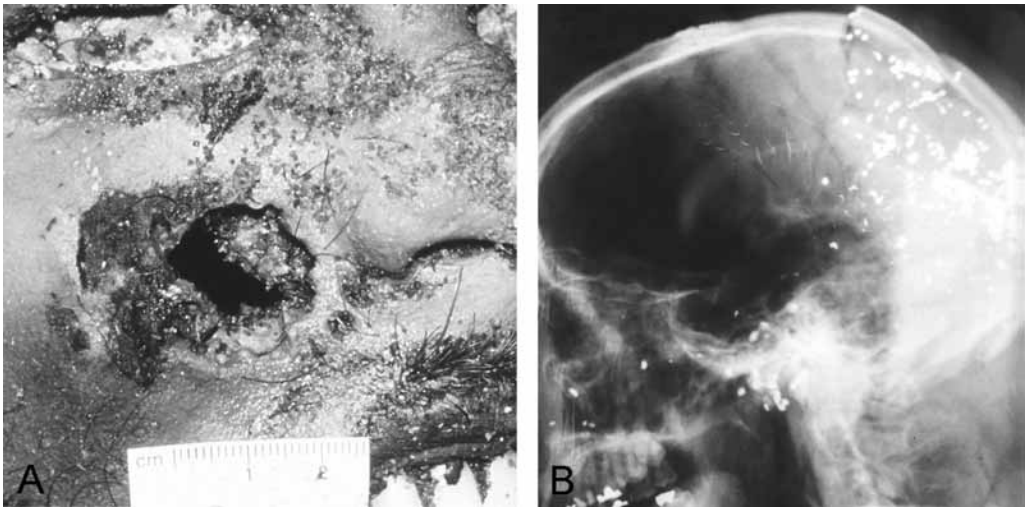

Fig. 31. Homicide. Autopsy not done during initial investigation because of severe decomposition. **(A)** Shotgun wound of right cheek. Wound was obscured by maggot infestation. Death was originally attributed to natural causes.**(B)** Radiograph of skull showing shotgun pellets. (Courtesy of the Office of the Chief Medical Examiner, Chapel Hill, NC.)

tissues and organs and eventual skeletonization (2 to 4 wk in warm to hot weather) *(47)*. Disarticulation of skeletal remains eventually occurs *(60)*. Issues may arise as to whether the recovered remains are human *(61)*.

5.2.5. "Unfit for Examination"

Discoloration, distension, degradation, and dissolution, as components of decomposition, can disorient a pathologist already reeling from malodorous gases eminating from a body seething with maggots. Failure to approach these cases systematically with a complete autopsy can result in a traumatic cause of death being missed (Fig. 31; ref. *62*). The more advanced the decomposition, the greater the likelihood of not finding a cause of death ("no anatomic cause of death," "undetermined"; *see also* ref. *62*).

5.3. Variants of Decomposition

Drying (mummification) is a variant of decomposition. Localized drying can affect the tip of the partly exposed tongue, the lips, the tips of fingers and toes, and the scrotum (Figs. 32–34). If the eyelids are not closed after death, then scleral drying appears as a horizontal brown line (*taches noires sclérotiques*; *see* Fig. 35). Another eye change is corneal clouding. It occurs 2 to 3 h after death if the eyes are open, and by 24 h if they are shut *(6,8)*. Generalized transformation of skin to a brown-black leathery consistency happens under dry conditions (Fig. 36). An "empty" body—i.e., empty body cavities—is sometimes seen *(63)*. Although the estimated time for generalized mummification in temperate climates is months, the author has observed it 1 mo after death in his locale (southwestern Ontario in the summer). Mummification is possible in the snow *(64)*.

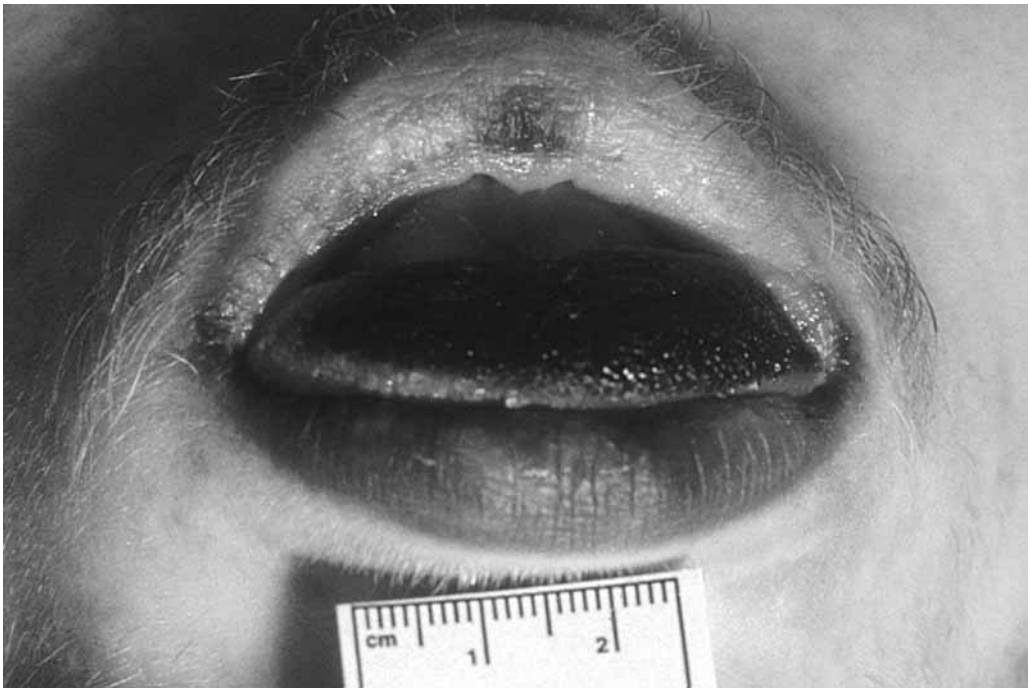

Fig. 32. Postmortem drying of tip of exposed tongue and lower lip.

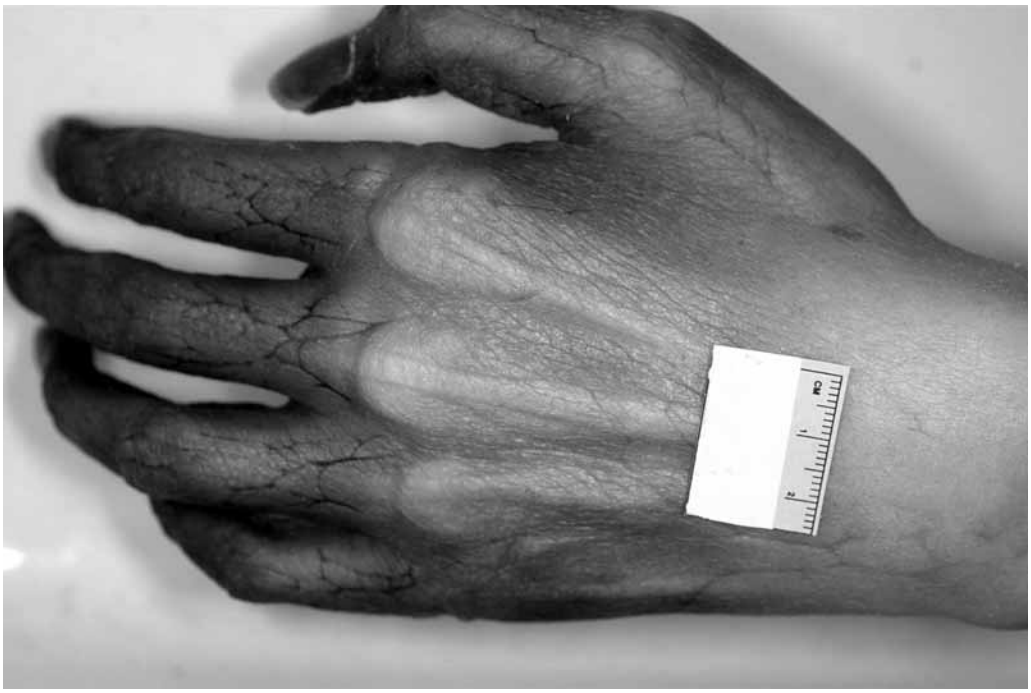

Fig. 33. Postmortem drying of fingertips: a challenge for police officers trying to obtain fingerprints.

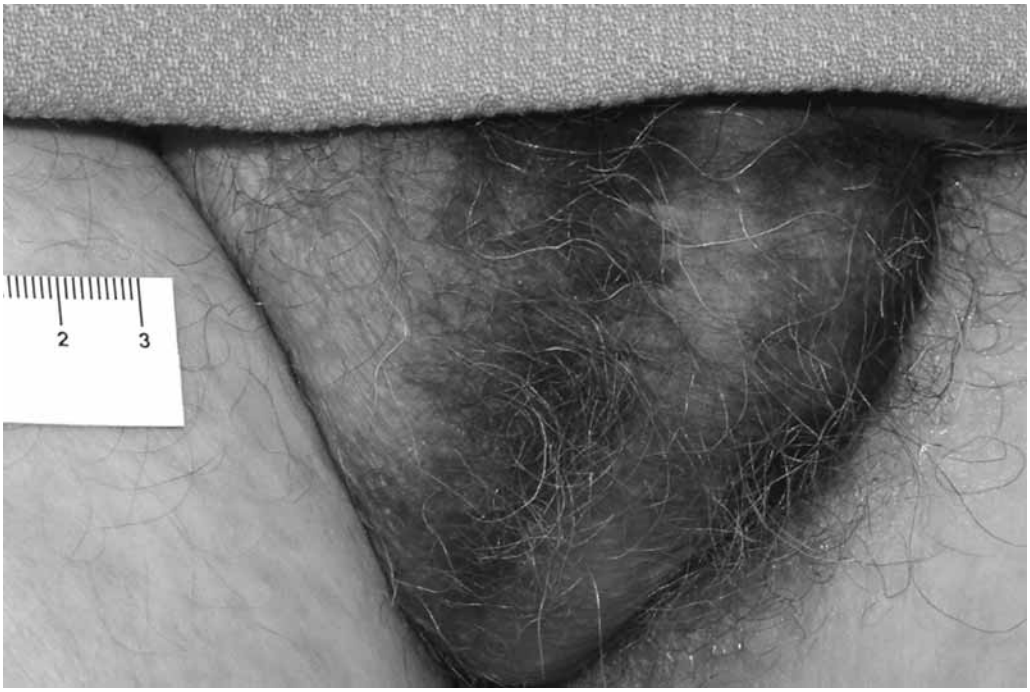

Fig. 34. Postmortem drying of scrotum, mimicking bruising.

Fig. 35. *"Taches noires."* Scleral drying artifact.

- Significance
  - Drying of fingertips makes fingerprinting a challenge (Fig. 33; refs. *65* and *66*).
  - The localized dark discoloration seen in a dried scrotum or tongue mimics injury (Figs. 32 and 34). Incision into the site reveals no hemorrhage. Examination of the removed testes shows no abnormality.
  - Scleral drying artifact suggests antemortem hemorrhage (Fig. 35). The artifact does not extend elsewhere on the sclera.

Fig. 36. Mummification. Leather-like skin in pelvic area of partly skeletonized remains.

Another variant of decomposition is adipocere (*adipo* = fat, *cere* = wax), a dirty yellow, greasy, or clay-like material formed by alteration of subcutaneous fat by endogenous lipases and bacterial enzymes (Fig. 37; refs. *45* and *67*). Hydrolysis of triglycerides leads to liquified neutral fats (storage fats), which penetrate adjacent soft tissue (e.g., muscle) and viscera. Bacterial enzymes transform unsaturated fatty acids into saturated forms, mainly palmitic and stearic acid. These fatty acids have higher melting points (palmitic acid = 63°C or 142°F, stearic acid = 81°C or 176°F) than the temperature of a gravesite (3–16°C or 37–59°F). As a result, the fatty acids crystallize, leading to the formation of a firm solid. Adipocere is more likely to form in an obese body or a female because of a higher fat content *(45)*. Numerous other factors have been proposed in the development of adipocere (e.g., moist or water-logged soils) *(45)*. Adipocere is variably distributed on the body surface and encases bones; it has also been noted in a fatty liver. Adipocere can remain unchanged for years *(45)*. The reduced water solubility of adipocere means that a corpse can retain its shape. The estimated time of occurrence of adipocere in temperate climates is several months postmortem *(45)*.

6. Gastric Contents

The description of stomach contents has been used to estimate the time of death, if the time, volume, and character of the individual's last meal are known *(68–70)*. On occasion, if pills are not digested, stomach contents provide a clue to a toxicological cause of death (Fig. 38; ref. *68*). The investigator may request that the pathologist examine the stomach, particularly if other postmortem parameters were not recorded during the initial examination of the body at the scene.

Documentation of the type of food, its volume, and state of digestion is done at autopsy. One assumption is that if the contents are recognizable and distinctive, then a

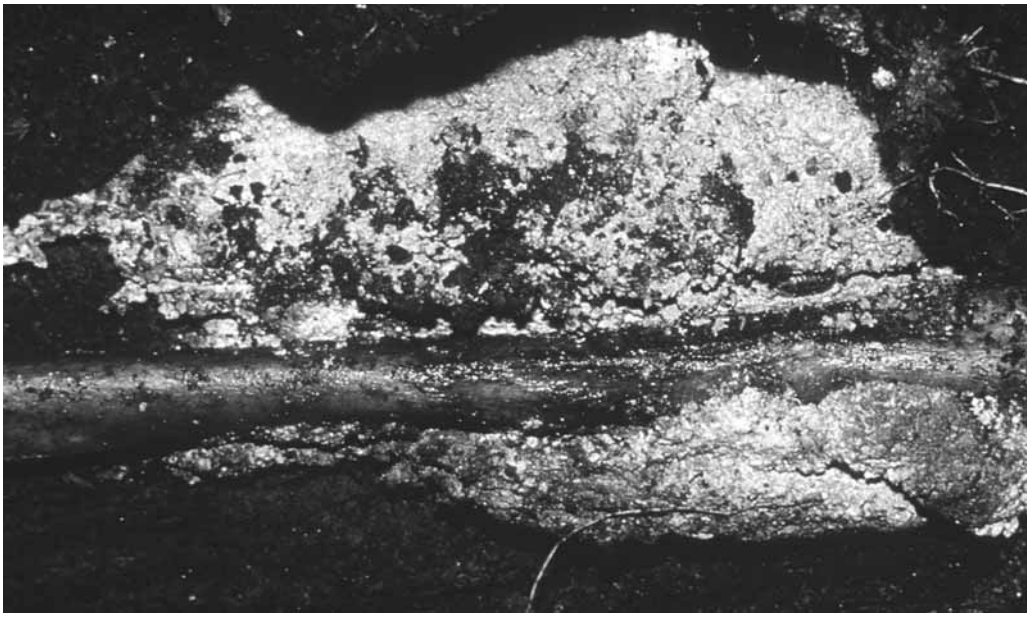

Fig. 37. Adipocere encasing long bone.

Fig. 38. Stomach opened at autopsy. In most cases, medications will have dissolved in the stomach by the time of death. The finding of an excess number of pills supports intentional ingestion.

similar meal has not been eaten in the interim. Simultaneously ingested liquid, digestible solid, and nondigestible solid foods are emptied from the stomach at different rates *(69)*. Liquids are emptied rapidly *(68–70)*. Another assumption is that digestion and gastric emptying proceed at regular rates. Meals of high volume and caloric content are emptied more slowly (e.g., light meal, 2 h; medium-sized meals, 3–4 h; heavy meals, 4–6 h), but digestion of various foods is variable and continues after death *(5,68,69,71,72)*. Some

foodstuffs (e.g., fibrous vegetables) are more resistant to digestion and persist in the stomach *(72)*. Larger pieces of less-chewed food are digested more slowly and remain in the stomach longer *(71–73)*. Gastric emptying varies in the same individual and between individuals under similar circumstances eating the same meal *(69)*. Gastric emptying is affected by many factors. Some factors delaying emptying include trauma, shock, disease, increased intracranial pressure owing to head injury, emotional upset, ethanol, and other drugs (e.g., narcotic analgesics; refs. *68,70–72*). Severe trauma can delay emptying up to several days *(70)*. Stomach contents are rarely useful in estimating the time of death because of the many variables involved *(68,69,71,74)*.

7. Embalming Artifacts

Embalming is a funeral home procedure that prepares and preserves a body for an open-casket funeral and attempts to reduce the effects of various postmortem changes *(75,76)*. Embalming leads to drying and hardening of soft tissues *(75)*. Embalmed bodies eventually decay, beginning at pressure points such as the buttocks and legs, the areas least penetrated by embalming fluid *(47,77)*.

After clothing is removed, the hair is shampooed and a man's face is shaved *(76)*. The body is cleaned *(76)*. An incision, usually in the subclavicular area, allows blood to be drained from the venous system and embalming fluid to be perfused, under pressure, into an artery (Fig. 39; refs. *75* and *77*). Embalming fluid is a mixture of formaldehyde, anticoagulants, perfumes, surfactants to reduce surface tension and increase permeation of fluid, coloring agents, modifying agents (e.g., moisturizers or dehydrating agents), and solvents (alcohols, water, glycerine compounds) carrying the various embalming chemicals. Femoral and brachial arteries are also used *(75,76)*. More than one access site has been seen *(77)*. The incisions are sutured and absorbent powder or cotton is put into the incision base to prevent leakage *(77)*.

To reduce gaseous distension, the abdomen is punctured near the umbilicus by a trocar that perforates the intestine *(77)*. Other viscera are also punctured (Fig. 39; ref. *76*). Injection of the scrotum may be done *(77)*. Trocared hollow organs (e.g., heart, urinary bladder) are aspirated of their contents, and embalming fluid is injected into the body cavities *(75–77)*. The trocar cutaneous wound is plugged with a button or sutured (Fig. 39; ref. *77*). Sometimes a trocar is inserted directly into an extremity *(77)*.

Cosmetics are applied to the face. Caps cover the eyes and the jaws are wired or sutured *(77)*. Injection of "tissue builders" into the orbits and face, to lessen a gaunt appearance, may be done *(76,77)*.

The body is reclothed. The backs of garments are cut to allow ease of dressing. Underlying plastic garments may be necessary to prevent leakage *(77)*.

If there has been an autopsy, stabilizing metallic clamps are screwed into the skull and joined by wires *(77)*. Cavities may be filled with material (e.g., cloth, paper towels) soaked in embalming fluid *(77)*. A viscera bag may be present *(77)*. Granular material inside the cavities consists of hardening or dehydrating compounds and embalming powders.

- Significance
  - The blood is unsuitable for toxicological analysis. Vitreous is an alternative *(76)*.
  - Blood coagulates, forming "pseudothrombi," and mimics pulmonary thromboembolism (Fig. 39; refs. *75* and *77*).

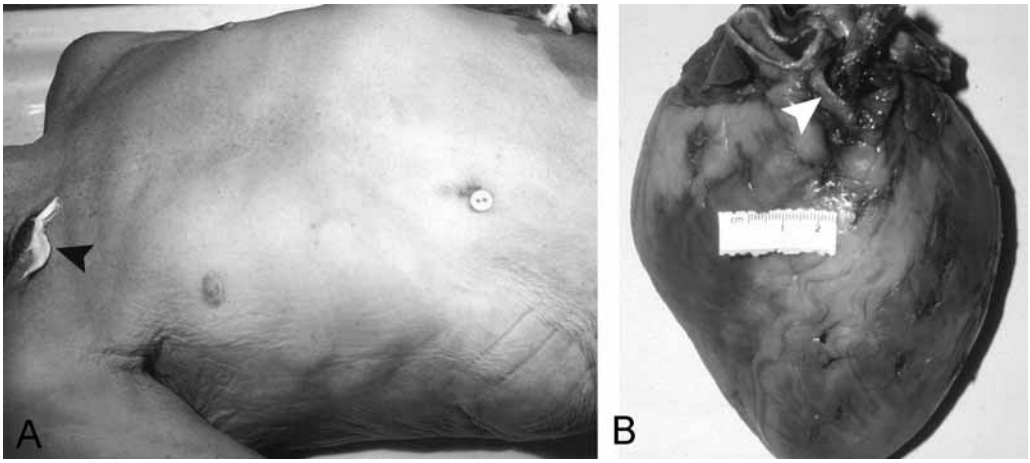

Fig. 39. Embalmed body. **(A)** Button on abdomen concealing trocar insertion site. Subclavian incision site covered by cotton dressing (arrowhead). **(B)** Embalmed heart showing artifactual trocar wounds. Note clotted blood in main pulmonary artery (arrowhead), mimicking a thromboembolus.

- Injuries are simulated *(75,76,78,79)*.
- Perfusion of embalming fluid can accentuate preexisting contusions or form postmortem bruises or hematomas (e.g., in the neck area) owing to vessel rupture (Fig. 40; refs. *75–77*). The decrease in lividity from drainage of blood further enhances the appearance of real or artifactual bruises, which can be a source of consternation for next of kin and investigators *(76)*.
- Trocar punctures affect not only intra-abdominal organs but also thoracic and pelvic structures (Fig. 39; ref. *75*). Cases have been described of antemortem bullet wounds being covered by trocar buttons *(75,76)*.
- Facial makeup can obscure trauma (Fig. 41; refs. *75* and *76*).
- Eye caps need to be removed to observe ocular hemorrhage *(76)*.
- Wounds can be altered (i.e., sutured *[75,77,79]*).
- Shaving can cause neck abrasions, simulating strangulation *(76,77)*.
- Coloring agents impart a bright red appearance, mimicking carbon monoxide poisoning *(76)*.

8. Forensic Entomology

Forensic entomology is defined as the use of insects and other arthropods in medicolegal death investigations *(45,80)*. The pathologist must be aware of the significance of insects on the body and can assist in their collection *(49)*. The pathologist also documents findings relevant to the forensic entomologist (e.g., clothing, apparent injuries).

Flies (order Diptera; Calliphoridae, or blowflies) are among the first colonizers of a deceased individual *(45)*. The maturation of these insects (egg–larva–pupa–adult insect) serves as a biological clock, 1 to 2 wk following death *(45,49,80,81)*. Data regarding fly lifecycles in one area cannot necessarily be used in another region *(45,80)*. The presence of ammonia-rich compounds and hydrogen sulfide are important stimulants for egg deposition *(45)*. Diptera do not oviposit in mummified tissue, favoring moist tissue *(45)*. Development during the larval stages is dependent on the temperature at the death

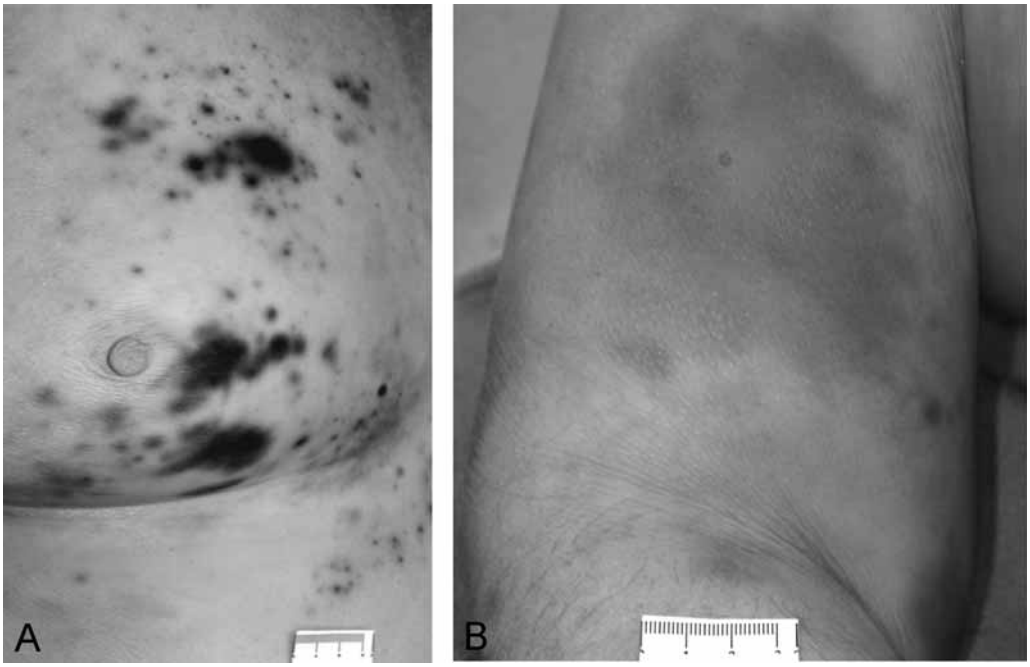

Fig. 40. Embalmed body. The deceased was described as "unsteady" on her feet. She was prescribed warfarin because of a previous diagnosis of pulmonary thromboembolism. She needed assistance walking when visiting a doctor a few days prior to her death. No contusions were visible on external examination by the coroner prior to embalming. The embalmer remarked that some bruises started to appear while perfusing the body. **(A)** At autopsy, numerous bruises were visible on the chest. **(B)** Large bruise, inner right arm.

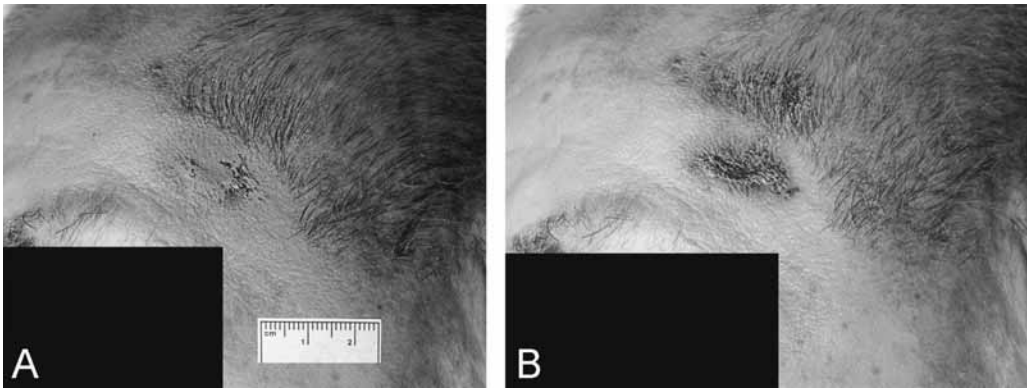

Fig. 41. Embalmed body. **(A)** Facial cosmetic had been applied. **(B)** Face wiped to reveal an abrasion on the left temple.

scene before the body was found *(45,47,49,80)*. Eggs are first laid in the orifices or wounds of the corpse within seconds of death (Figs. 31, 42; refs. *45, 47,* and *82*).

Eggs, larvae, pupae, and adult flies are collected *(80)*. Larvae are preserved in 10% formalin. Eggs and larvae are also placed in a specimen jar with a food source (e.g., liver, muscle from body), covered with gauze, and kept at ambient temperature and humidity.

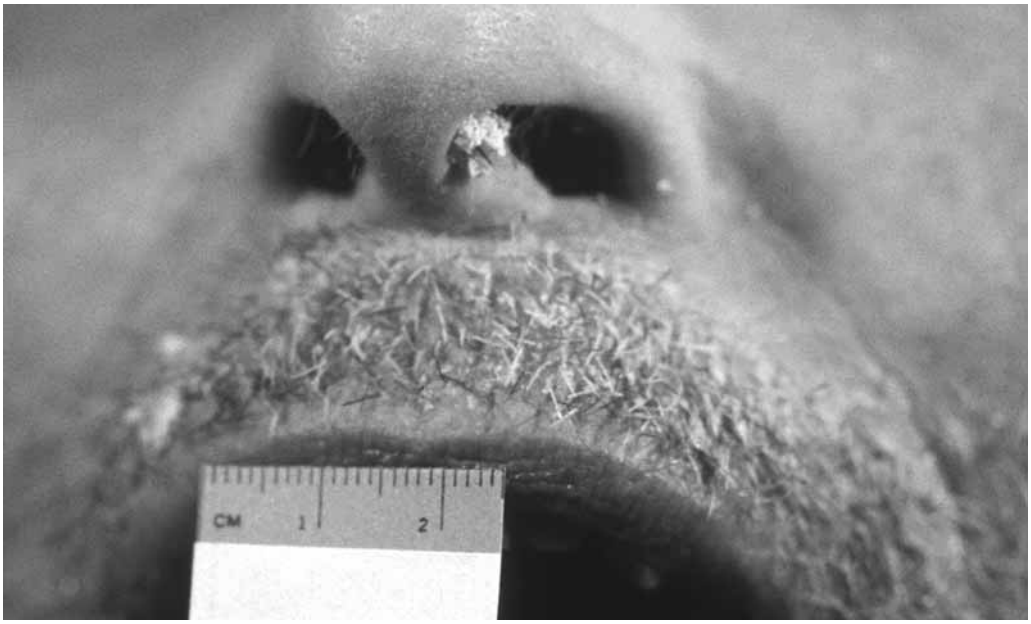

Fig. 42. Fly eggs deposited in left nostril.

- Significance
  - Larvae alter wounds *(36,49,62)*. Masses of larvae on the skin can be a clue to a site of bleeding *(49,82)*.
  - Live maggots, in the absence of a dead body at a location, suggest that a corpse was present *(49)*. The finding of human DNA in larvae indicates feeding on a human body *(50)*.
  - Toxicological analysis can be done on fly larvae and reflects use of drugs in the deceased *(49,50)*.
  - Refrigeration does not dampen feeding activity by larvae if maggot masses form *(47,82)*. Temperatures within these masses range from 27 to 35°C (81 to 95°F *[50]*).
  - Other insects (e.g., ants, cockroaches, beetles) can create artifactual injuries (Fig. 43; refs. *36, 78, 81, 83,* and *84*).

9. Postmortem Animal Predation

Postmortem predation creates injuries that arouse suspicions of homicide *(35,36,48,85–89)*. Animal activity alters antemortem injuries *(36)*. The typical case is an individual living alone who dies of natural causes *(36,89)*; a free-roaming house pet (dog or cat) is trapped inside the house, and its usual food is unavailable. A pet can also be motivated by other reasons when food is present *(85)*. A frantic pet may start to lick or nudge an unconscious owner and can become frantic, biting the body *(85,89)*. The confined space of a dwelling can also lead to aggressive behavior toward the owner *(89)*. The postmortem interval for predation is estimated to be less than 1 d *(88)*. One case report, involving a dog, indicated predation within 45 min *(89)*.

Other mammals (e.g., rodents) can gnaw on the body *(85,90,91)*. Rodent excrement or fur at the scene is a clue to the nature of the injuries *(85,87)*. Rodent activity

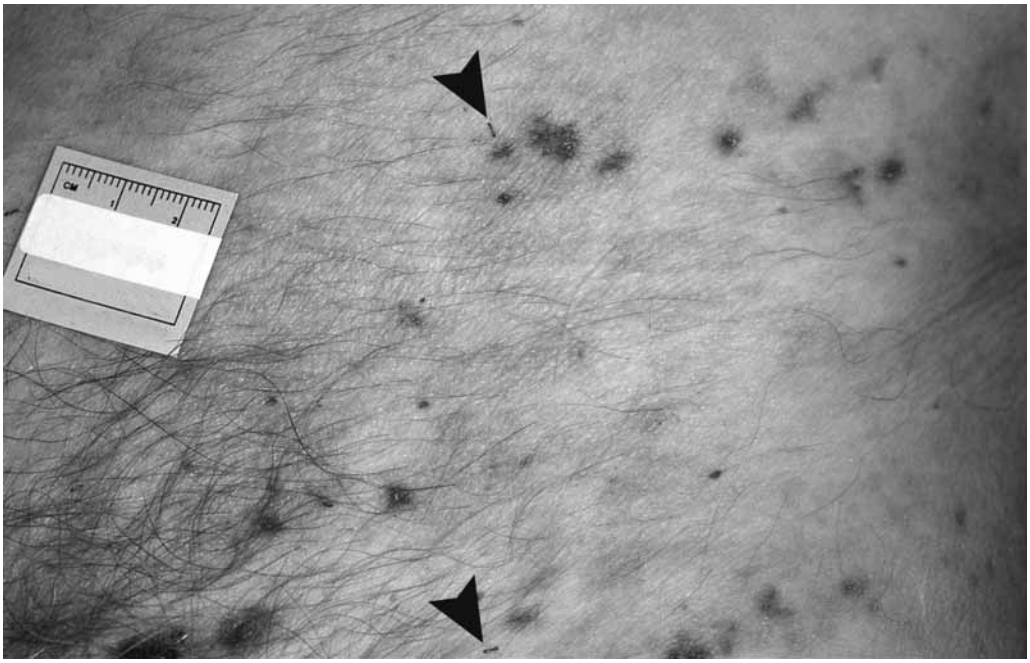

Fig. 43. Postmortem predation by ants (arrowheads) causing artifactual "abrasions."

occurs indoors in low socioeconomic settings and outdoors among homeless people *(87)*. Outdoors, various animals can carry away bones *(36,47)*.

9.1. Autopsy Examination

There is a predilection for the exposed soft parts of the body (e.g., mouth and nose *[36,87–89]*). Large defects on the face, neck, and torso with variable loss of viscera and bony injury are observed following predation by large pets (e.g., dog; *see* Fig. 44 and refs. *36, 85, 86,* and *88*). Body parts (e.g., ears) can be missing (Fig. 44; ref. *91*). Rodents can gnaw on areas of the body covered by clothes *(87)*. The wounds are associated with minimal bleeding or bruising *(85–87,89)*. Animal hair can be found in the wounds *(89)*. No self-defense injuries are evident *(85,87)*. Bite marks caused by canine teeth (dogs and large cats) and claw-induced linear scratch marks may be seen at the edge of the defect (Fig. 44; refs. *85, 88,* and *89*). Stab wound-like punctures are characteristic of canine dentition of carnivore origin *(85)*. Rodent predation is characterized by layered damage of tissue *(90)*. Rodents continue to gnaw in one area until all skin and soft tissue is chewed, exposing tendons, ligaments, and bone *(87)*. Compared with the large irregular defect edges of canine predation, rodent wounds are smoother, finely serrated, and scalloped (Fig. 45; refs. *36, 85, 87,* and *90*). Parallel cutaneous lacerations indicative of tooth marks can be seen *(36,87)*. Microscopic examination of the wounds does not show evidence of an inflammatory reaction *(85,86)*.

The stomach of an animal may contain human remains, which can be confirmed by microscopic and DNA analysis *(89)*.

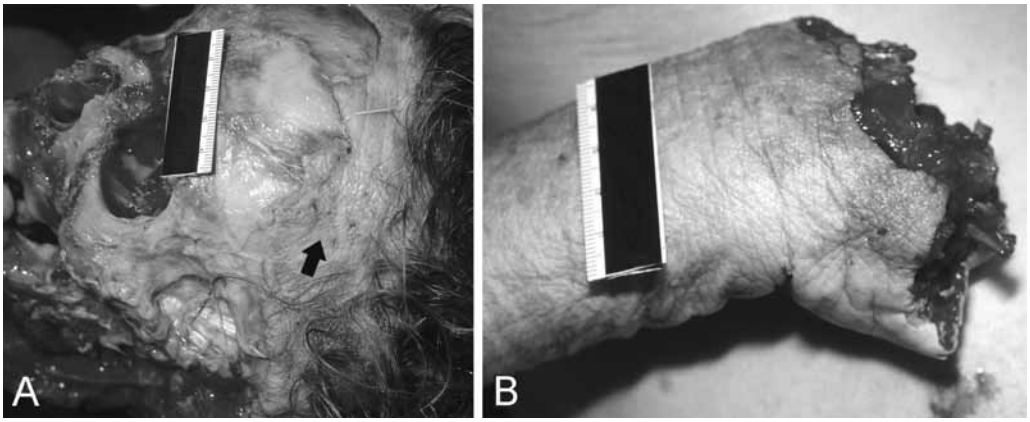

Fig. 44. Postmortem predation by a dog. **(A)** Large irregular soft tissue defect of face. Note puncture wounds possibly from canine teeth (arrow). **(B)** Loss of hand owing to predation. (Courtesy of Dr. C. Rao, Regional Forensic Pathology Unit, Hamilton, Ontario, Canada.)

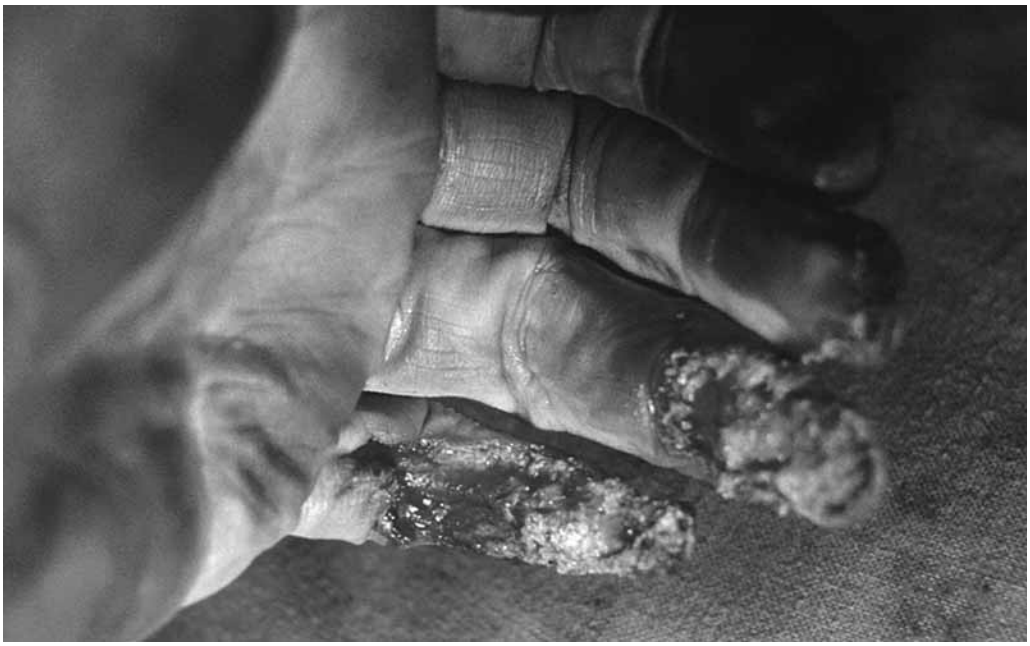

Fig. 45. Rodent predation of fingers after death. (Courtesy of Dr. D. King, Regional Forensic Pathology Unit, Hamilton, Ontario, Canada.)

10. Concurrence (Association) Observations

The time of death of an individual can be linked to the scene (Fig. 46). For example, a person who is found dead in a house and has 3 d worth of newspapers accumulated on the front porch has likely been dead for that period of time.

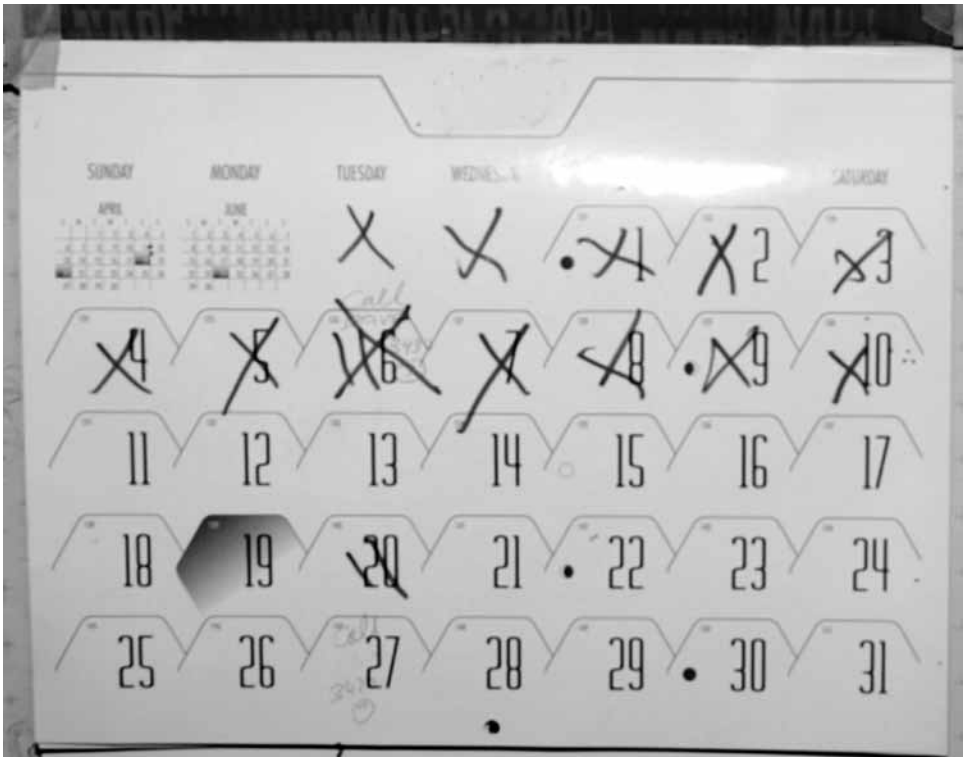

Fig. 46. Kitchen calendar crossed off to the 10th day of the month. The lone occupant was found dead 1 wk later in a decomposed state.

11. Postmortem Procedures

Postmortem procedure artifacts can create "injuries." Some examples include:

- Scleral hemorrhage from needle aspiration of eye to obtain vitreous humor; periorbital hematoma owing to enucleation of eyes for corneal harvesting (*see* Subheading 3.3.; Fig. 17; and refs. *92* and *93*).
- Basal skull fracture caused by forced removal of the calvarium when the skull has not been completely sawed.

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