Chapter II

TIME OF DEATH AND CHANGES AFTER DEATH

Part 1

ANATOMICAL CONSIDERATIONS

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DEFINITION OF DEATH

The definition of death is important to both the medical and legal professions. While it is true that for the clinician the demise of a patient signals the unfortunate end to the medical effort, the very determination of death may carry secondary therapeutic implications regarding transplantation of organs. Obviously, the occurrence of a death is the starting point in the professional involvement of the forensic pathologist. Similarly for attorneys, the death of an individual generates a great number of legal challenges related to inheritance rights, estate management, criminal liability and tortual injuries.

Until the 1960s, the cessation of circulation and respiration was the unchallenged definition of death. For example, the 1968 edition of Black's Law Dictionary defined death as: the cessation of life; the ceasing to exist; defined by physicians as the total stoppage of the circulation of the blood, and cessation of vital functions consequent thereon, such as respiration, pulsation, etc.

As a matter of fact, even today in most deaths, particularly in those which occur outside hospitals or are unwitnessed, the criteria used are still the cessation of circulation and respiration.

However, the classical definition of death has been challenged in recent times by two medical advances

1. Advanced resuscitation techniques (cardiopulmonary resuscitation [CPR], mouth to mouth, heart massage, electric shock) capable of effectively reviving many of the clinically dead.
2. Advanced life-sustaining equipment capable of maintaining blood pressure, circulation and respiration in individuals with severe brain injury.

Though the first instance of mouth-to-mouth resuscitation was recorded long ago in the biblical story of the Prophet Elijah resuscitating a child (II Kings 4:32-36), modern cardiac massage, electric shock and routine CPR came into use only three decades ago.

These developments necessitated, in many cases, the obvious revision of the definition of death from just cessation to irreversible cessation of respiratory and heart activity following modern resuscitation attempts. The reversibility of the death process is dependent on the capability of tissues to recover from the effects of ischemia/anoxia occurring between the advent of clinical death to the initiation of effective resuscitation. The resistance of various organs to ischemia/anoxia is variable, with the central nervous system displaying a particularly high sensitivity. The classic literature indicates that a four- to six-minute period of cerebral anoxia from a delay in effective resuscitation will commonly result in irreversible and extensive brain damage. However, more recent experimental and clinical evidence points to instances where the reversible interval may be as long as fifteen to sixteen minutes.

Young children and hypothermic individuals are known to resist cerebral hypoxia for thirty minutes or more with no ill effects. In a case reported by Kvittingen and Naess, a five-year-old boy fell into a partly frozen river and recovered fully following a presumed submersion time of twenty-two minutes.

The development of life-sustaining equip-
ment has also changed the definition of death by permitting a dissociation between a severely hypoxic or dead brain (incapable of sustaining spontaneous respiration and circulation) and the peripheral organs which can be kept alive artificially. Therefore, the definition of death in a person with severe and irreversible brain injury, incapable of sustaining spontaneous respiration and/or circulation, had to be revised to include what is now defined as brain death.

The clinical definition of brain death was first advanced in 1968 by the Ad Hoc Committee of the Harvard Medical School. The committee, which consisted of physicians, a theologian and a lawyer, defined the following conditions for determining irreversible brain death:

1. Unreceptivity and unresponsivity, including a total lack of response to the most intense painful stimuli.
2. No movement or spontaneous respiration, defined as no effort to breathe for three minutes off the respirator with the patient’s carbon dioxide tension normal and room air being breathed for ten minutes prior to the trial.
3. No reflexes fixed, non-reactive pupils, and a lack of cranial nerve reflexes (corneal, pharyngeal, ocular movements in response to head turning and irrigation of ears with ice water, etc.).
4. Isoelectric electroencephalogram.

The committee suggested that all tests be repeated in twenty-four hours and emphasized that the determination of the irreversibility of cerebral damage should be made only after the exclusion of potentially reversible conditions, such as hypothermia (temperature below 90° F [32.2° C]) and central nervous system depressants such as barbiturates.

In subsequent years, the list of reversible causes of coma has been expanded to include metabolic neuromuscular blockade, shock and young age (less than five years of age).

Also, additional objective tests besides electroencephalogram were added to the determination of brain death, such as cerebral angiography and radionuclide studies, in order to confirm the absence of cerebral blood flow.

In 1977, the National Institute of Neurological Diseases and Stroke conducted a collaborative study which somewhat refined the Harvard criteria. The criteria of brain death proposed by the collaborative study were as follows:

1. Coma and cerebral unresponsiveness.
2. Apnea.
3. Dilated pupils.
4. Absent cephalic (brainstem) reflexes.
5. Electrocerebral silence.

These criteria were to be present for thirty minutes at least six hours after the onset of coma and apnea, and all appropriate diagnostic therapeutic procedures were to be performed. Confirmatory tests of cerebral blood flow were necessary if one of the standards was doubtful or could not be tested.

Starting with Kansas in 1968, increasing numbers of states have adopted the definition of brain death. In 1980, representatives of the American Bar Association and the National Conference of Commissioners of Uniform State Laws agreed upon a model legislative definition of death: An individual who has sustained either, (1) irreversible cessation of circulatory and respiratory functions or, (2) irreversible cessation of all functions of the entire brain, including the brain stem, is dead. A determination of death shall be made in accordance with accepted medical standards.

It has been pointed out that in spite of some differences between the legal definitions of brain death in various states, physicians can meet therequirementsoffallofthemby:

1. Using the commonly accepted criteria of brain death.
2. Having two physicians, one of whom is a neurologist, make the brain death determination.
3. Avoiding a conflict of interest in having physicians separate from the transplant team, certifying the brain death of a potential donor.
4. Determining brain death before removing any organs or disconnecting life-support systems.

As a matter of fact, a number of states have formally incorporated such provisions into their laws.
The Pathology of Brain Death and Persistent Vegetative State (PVS)

The brain findings of brain death or so-called respirator brain are characteristic on gross examination. The brain has a dusky, grayish appearance, with marked swelling and evidence of transtentorial hippocampal and tonsillar herniation. Depending on survival time, the cerebral parenchyma shows a variable spectrum of anoxic/ischemic damage from minimal changes to severe encephalomalacia and liquefaction, and generally cannot be satisfactorily examined prior to fixation.

Microscopic findings in respirator brains are variable, non-specific, non-diagnostic and correlate poorly with gross findings.

Brain death changes generally become apparent approximately twelve to sixteen hours after cessation of the cerebral circulation, though in some cases they may be evident after only six hours, and in others they may be delayed for twenty-four hours longer.

Rapid respirator brain changes may be observed in cases with acute onset, such as severe head trauma, large parent hemorrhages or sudden cardiac arrest with delayed resuscitation. Delayed manifestations of brain death are seen in chronically ill persons with brain tumors or metabolic problems.

Sometimes identification of true antemortem hemorrhages or contusions may be substantially obscured or hampered by respirator brain related hemorrhages.

Agonal changes in the microcirculation, such as petechiae in leptomeninges and cerebral parenchyma, may be dramatically accentuated in cases of brain death. Stagnant or stasis thrombi in veins and arteries may also be present and should be distinguished whenever possible from non-agonal clots.

It is important to distinguish between brain death and the irreversible brain injury known as Persistent Vegetative State (PVS). Patients with either condition are clinically, irreversibly comatose and show severe brain injury with neuronal death. However, the diagnosis of brain death is based on brain stem death determination, while PVS involves only permanent and total destruction of frontal lobe function.

Disconnection of life-support equipment is permissible following a determination of brain death, while it is much more problematic in cases of PVS, where a living will or specific court approval is required.

One should emphasize that premature or illegal discontinuation of life-supporting systems for an irreversibly unconscious but not brain dead patient may result not only in civil liability but also in criminal charges.

The Medicolegal Implications of the Determination of Death

The practical major medicolegal implications related to the new definition of death are:

1. The earliest determination of death for prompt harvesting of organs for transplantation purposes.
2. The legality of discontinuation of life-supporting equipment.
3. The determination of the time of death in criminal and civil litigation.

In medicolegal cases the transplantation procedures require not only a prior authorizing donation by the deceased and/or his family but also the specific consent of the medical examiner or coroner.

Medicolegal offices are major source of organs for transplantation purposes, and their standing policies may substantially advance or impede the transplantation program of a particular community. The humanitarian value of transplantation procedures notwithstanding is incumbent on the forensic authority to insure not only that the harvesting of organs is duly authorized but also that it does not substantially interfere with the medicolegal autopsy.

We have recently seen, for example, two cases of shaken baby syndrome with no external injuries in which the abusing parents authorized tissue donation. It is unclear whether the motivation of the parents was purely altruistic or an attempt to cover up a possible homicide. Regardless of the motive, when the cause and manner of death are unclear, postmortem harvesting of or-
gans may substantially interfere with the medicolegal autopsy.

If one wishes to approve the harvesting of organs under such circumstances, it is strongly recommended that a forensic pathologist be present during the procedure. Such presence is required to insure proper documentation and interpretation of findings of medicolegal significance and to avoid the interference of confusing artifacts.

The Determination of Death and Survivorship

When two individuals who previously designate each other as mutual heirs die together in a single incident, the legal question of whether they died at the same time or not is of paramount importance in determining who will be the ultimate inheritor.

This question, known as survivorship, is often posed to the forensic pathologist who will have to weigh the specific circumstances of each case, including the age of the deceased, the state of health, the extent of injuries and the corresponding reactive changes, the level of alcohol and toxic substances, and the nature and state of postmortem changes.

One should critically evaluate the various parameters in order to ensure that they are comparable under similar conditions. For example, two individuals showing marked differences in the development of postmortem changes may still have died simultaneously if one was more prone to accelerated death (e.g. excessively obese, septicemic or close to a hot radiator).

The Death Certificate and the Determination of the Cause and Manner of Death

Though the laws of many states are unclear in regard to who should make the actual determination of death, all legislatures require that every deceased be issued a certificate which is to be signed by the last attending physician or by the coroner/medical examiner.

The central medicolegal requirement of the death certificate is the determination of the cause and manner of death. The cause of death is the medical finding or findings responsible for the death, and the manner of death is the legal classification of death, whether it be natural, suicide, homicide, accident or undeterminable.

The death certificate has two major groupings for the cause of death: the primary or immediate cause of death and the secondary cause of death. The primary cause of death is subdivided into a three-link sequential chain, for example:

1. Primary cause of death:
   — Hypoxemic necrosis of brain (brain death) due to
   — Exsanguination due to
   — Gunshot wound (GSW) of abdomen

The secondary cause of death includes conditions which are not related to the primary cause of death but are substantially contributory to the individual’s demise (e.g. emphysema of lungs, hypothermia, arteriosclerotic cardiovascular disease).

In cases of sudden unexpected death, suspicious death or unnatural death (suicide, accident or homicide) the coroner/medical examiner is the exclusive authority who has the jurisdiction to issue the death certificate. When the death follows shortly after a catastrophic event (e.g. traffic accident, suicide, accidental overdose, homicidal gunshot wound), the reporting is generally accurate and prompt.

Problems arise when the injured individual survives a longer period of time and dies of either predictable or unexpected complications or when death is a result of a combination of pre-existing natural disease and the initial injury. It is not uncommon that the last physician to treat the patient is unaware of, or forgets, the initial traumatic or toxic event which triggered the chain of complications. Furthermore, many physicians are unaware of the medicolegal approach to the determination of the manner of death when both natural and unnatural causes of death coexist. A simple rule of thumb states that if an unnatural cause of death (trauma, drug overdose, electrocution, drowning, etc.) plays a contributory role in the death, then the manner of death is unnatural (i.e., accidental, suicidal or homicidal), and, therefore, the case
falls under the jurisdiction of the coroner/medical examiner.

It is immaterial whether the unnatural factor (i.e., trauma) was a direct, indirect, complicating or aggravating element, or whether it was a major or minor factor in the causation of death. The same is true for the unnatural physical or chemical injury which occurs long before the patient's demise, providing that the death can be traced back to the initial physical or chemical injury. A continuous chain of symptoms and/or findings must be demonstrated, however, in order to link the initial injury to its later complications and the death.

For example, the case of a seventy-year-old woman who dies of pneumonia or pulmonary embolism after being confined to bed for several weeks following a hip fracture sustained in a fall is clearly a case to be reported to the coroner/medical examiner. The accidental hip fracture and the subsequent immobilization can be viewed as the triggering elements of the bronchopneumonia or embolism. This obviously colors the manner of death as accidental and implicitly makes it a reportable forensic case.

The lack of clinical continuity of symptoms between an injury (e.g. trauma) and a subsequent, apparently natural death does not necessarily classify the death as purely natural and unrelated to the trauma. A physical or chemical injury may trigger subclinical but nevertheless potentially fatal complications which may be substantiated only at autopsy.

A fifty-five-year-old man who dies four to five days following a symptom-free interval from the time of a physical assault is to be considered a forensic case if the autopsy demonstrates a recent infarction, subclinical cardiac contusions or other fatal complications related to the time of the incident.

Difficulties also occur when the death is intraoperative, peri-operative or related to the patient's care, and the question arises whether this is or is not a reportable case. When a substantial active medical misadventure occurs, the case is clearly a prima facie coroner/medical examiner case.

Such active misadventures include both physical and chemical injuries, such as perforation and/or laceration of blood vessels or other organs, disconnection of anesthetic equipment causing asphyxia, administration of the wrong type of blood, burns or electrocution by the medical equipment, overdose of anesthetics or other medications, administration of mistaken medication, and anaphylactic reactions.

The jurisdiction of the coroner/medical examiner in unexplained deaths may be exercised only when the manner of death is unexplained. If a diagnostic determination was reliably made that the death is natural, no jurisdiction exists. The precise cause or mechanism of death, in a natural death, may be medically tantalizing and may have public health significance, but its mystery cannot prompt the forensic jurisdiction unless specific authority is granted under state law. Therefore, the failure to know the precise cause of mechanism of death in an otherwise natural death does not activate the coroner/medical examiner's jurisdiction.

The same approach applies to suspicious deaths. It is only when reasonable and reliable information indicates that the death is suspicious that the forensic jurisdiction may be assumed. In such situations, the presence of a potentially fatal natural cause of death is not incompatible with the coroner's assumption of jurisdiction, as the natural condition may well be only the background on which a subtle unnatural fatal injury occurred. Obviously, only a forensic autopsy may confirm or exclude, in such cases, the possibility of poisoning, hidden time or other unnatural causes of death.

**Embalming and Exhumation**

Postmortem embalming is an extremely widespread custom on the North American continent of which most religious denominations approve. The embalming procedure usually includes the application of cosmetic cream to the face and hands of the deceased, covering of the eyes under the eyelids with plastic cups, fixation or wiring of jaws, injection of embalming fluids in the neck (carotid), axilla (axillary) and groin (iliac and femoral) arteries, and
Time of Death and Changes After Death

FIGURE II-1. Embalmed five-month-old child, (a) Cosmetic cream covering the face of embalmed child, successfully concealing extensive bruising of the face, (b) Face after removal of cream.

Injection through trocars of cavity fluid into the abdomen and thorax.

Autopsies performed after embalming and/or exhumation require special techniques. Embalming artifacts may conceal or mimic real injuries.

The sticky embalming cosmetics placed on the face and hands of the deceased may effectively mask substantial bruises and abrasions of the skin and, therefore, should be carefully removed with an alcoholic solution or scraped away (Fig. II-1). The injection of embalming fluids into the body cavities will obviously affect the composition, appearance and amount of any previously present fluid. Perforations of the internal organs by the embalmer's trocar may be difficult or impossible to differentiate from genuine lacerations.

Perhaps the most intrusive artifactual complication of embalming is the chemical alteration of the blood and tissues by the injected embalming fluid. Embalming fluids contain various mixtures of formaldehyde, glutaraldehyde, alcohols and other preservatives (e.g. hexylresorcinol, phenol, methylsalicylate, sodium benzoate, sodium and calcium oxalates, quaternary ammonium compounds, EDTA). The use of metallic salts (e.g. arsenic, mercury, lead, copper, silver, etc.) in embalming fluid is now prohibited to prevent concealing of heavy metal poisoning. However, embalming still interferes with the chemical analysis of many compounds. Some compounds, such as ethanol, opiates, carbon monoxide and cyanide, are destroyed or cannot be reliably tested.

Other chemicals (such as barbiturates, tricyclic compounds and benzodiazepines) can be qualitatively tested if only a few weeks or months have elapsed since embalming. However, a quantitative evaluation of these compounds is largely unreliable because of dilution and other factors. In some cases, analysis of more protected biological fluids (such as vitreous of the eye and cerebrospinal fluid) may give a fairly good quantitative estimate.

Metallic compounds and metalloids (e.g. arsenic) may be recovered from the embalmed body after many years. However, their elution into the
environment or diffusion from the environment if the soil has a higher concentration, makes one doubt the reliability of their quantitation.

When testing embalmed tissues for metals and metalloids, it is recommended that the embalming fluid be analyzed as well in order to exclude the possibility that it contains related contaminants.

The situation becomes more complex when there is a need to examine an embalmed body after exhumation. In cases where the death is due to trauma, it is advantageous that a forensic pathologist be present during the exhumation to ensure that the coffin did not collapse, and that the body was not otherwise physically damaged in the process.

Similarly, if poisoning is suspected, the forensic pathologist should collect samples of the soil around the coffin (above, below and sides) as well as any water which may have leaked into the coffin which may contain increased amounts of the chemical suspected in the poisoning. A typical poison which may be present in increased amounts in the soil is arsenic, but obviously, other toxic substances and metals may be present as well.

Following exhumation, most bodies show significant fungal growth on the face and exposed skin areas which may severely disfigure the deceased and practically obliterate bruised or abraded areas. Areas of bruising are especially difficult to evaluate because of the black, gray or greenish discoloration due to the combination of fungus and decomposition changes. Usually, fungal growth is maximal in areas of pre-mortem injury and bleeding. Aspergillus nigrans, which is black, and flaky white mildew, is especially common on the body surface (Fig. II-2). Interestingly, decomposition is usually reduced in areas with fungal growth because of the bacteriostatic effect of most strains of fungi.

Furthermore, if fluid is present in the coffin, the skin may become very soggy and slippery and develop adipocere (see below). The preservation of the internal organs varies considerably with the quality of embalming. In some cases, we have seen excellent preservation after ten or more years; in others we observed advance internal decomposition in a few weeks following burial.

**Incineration and Cremation**

Close to seventy percent of the human body is composed of water, twenty-five to twenty-six percent of combustible organic tissue and less than five percent of fireproof inorganic compounds. Most of the latter are present in the bones in the form of calcium salts, mainly as crystalline hydroxyapatite and partly as amorphous calcium phosphate. Upon exposure to temperatures in excess of 1000° C, the soft tissues of the body and the organic components of the bone literally go up in smoke, leaving only a minimal amount of ash.

In most fires, temperatures do not reach such levels, and variable amounts of soft tissue in dif-
ferent stages of carbonization are still present. In rare instances where temperatures are so high that the bones burn extensively, incinerated bones which may remain appear as white or white-gray, porous, friable, calcinated fragments of various size.

Careful sifting through incinerated bones reveals, in most cases, fragments which are large enough to be recognized as human by an experienced examiner, particularly by an anthropologist.

The funeral disposition of human remains by fire is known as cremation. In some parts of the world, such as India, this is the common funeral method. In the United States, the incidence of cremations has increased in recent years.

In the past, most cremations involved paupers and unclaimed bodies, fetuses and body parts. Recently, increased numbers of upper-middle-class professionals are opting for the method of postmortem disposal. In most states, cremation requires a special permit by the local department of health or the coroner/medical examiner and a mandatory twenty-four-hour postmortem waiting period.

Cremation is by open flame or oven heating (calcination) at temperatures between 1600° F and 2200° F. Cremation at these temperatures and subsequent grinding of the cremated bones results in a mixture of small calcinated fragments of various color (brown, light-brown, gray and blackish) which cannot be diagnosed by current methods as being specifically human. The total volume of these cremated remains depends not necessarily on the weight of the individual but on the mass of skeletal bones, as most soft tissue incinerates with very little trace.

In a personal study of the cremated remains of two hundred and forty-six males and one hundred and forty-eight females, the weight generally varied between 1,500 and 5,510 grams, with a mean in men of 3,035 grams and in women of 2,508.3 grams, and a standard deviation of 538.6 grams and 598.4 grams, respectively. If the total weight exceeds 6,000 grams, it is likely that the cremated remains consist of more than one person. A few legal suits have indeed alleged that negligent funeral directors have, on occasion, mixed the ashes of several people.

POSTMORTEM CHANGES AND THE DETERMINATION OF THE TIME OF DEATH

Following death, numerous physicochemical changes occur which ultimately lead to the dissolution of all soft tissues. The medicolegal importance of these postmortem changes is related primarily to their sequential nature which can be utilized in the determination of the time of death and the related destructive and/or artifactual changes which may simulate premortem injuries or modify toxicological findings.

The determination of the time of death is generally based on the principle of using sequential changes as a postmortem clock. The evaluation may include:

1. Physicochemical changes evident upon direct examination of the body, such as changes in body temperature, livor, rigor and decomposition. These changes are routinely reported in a protocols and are most commonly used in postmortem timing.
2. Changes in the chemical composition of body fluids or tissues (e.g. postmortem potassium concentration of vitreous fluid). These changes are not routinely evaluated and are generally recorded when the determination of the time of death is in doubt and is perceived as crucial in the medicolegal investigation.
3. Postmortem residual reactivity of muscles to electrical or chemical stimuli (e.g. electrical stimulation of the masseter muscle and reaction of the iris to chemicals.) The recording of these changes, primarily popular in European medicolegal center, is exceedingly uncommon, if at all practiced, in the United States.
4. Evaluation of physiological processes with established starting time or progress rate and cessation at death (e.g. presence of gastric contents as affected by time of digestion and the gastric emptying time). Recording of the
amount, nature and appearance of gastric contents is routine in any adequate autopsy.

5. Survival time after injuries, particularly when the time of infliction is known. The nature, extent and severity of injuries as well as the quantitation of associated complications (e.g. the amount of bleeding, early tissue reaction to injury) are often useful in determining the time of death.

The major problem encountered when relying upon the results of these methods is the variation in the environmental and individual factors on the magnitude and kinetics of postmortem phenomena.

For example, the physicochemical changes following death are greatly dependent on environmental conditions and the metabolic status of the individual prior to death. Therefore, the deceased must be considered in view of environmental factors (temperature, ventilation, humidity) and his characteristics (body build, pre-mortem exercise, state of health). Because of significant variation of kinetics of postmortem phenomena, the time of death cannot be pinpointed exactly but is estimated within a variable time frame. Furthermore, the longer the time interval since death, the wider the estimated range.

Because of inherent inaccuracies in timing of individual postmortem changes, the following approach is usually effective:

1. An initial determination of a wide window of death which is subsequently narrowed and refined by using variable parameters. The window of death is defined as the time interval prior to which one may assert with confidence that the individual was alive. The window of death should be established according to the most reliable testimony or evidence as to when the individual was last alive (e.g. witnesses, verified signed documents, last time newspapers were brought in the house, last time of withdrawal on bank accounts).

2. Conservative determinations of time of death as a range utilizing individual postmortem changes.

3. An algebraic integration of all postmortem timing changes.

Postmortem Cooling (Algor Mortis)

Postmortem body temperature declines progressively until it reaches the ambient temperature. Under average conditions, the body cools at a rate of 2.0° F to 2.5° F per hour during the first hours and slower thereafter, with an average loss of 1.5° F to 2° F during the first twelve hours, and 1° F for the next twelve to eighteen hours. Careful studies under controlled conditions have shown that the decrease in the postmortem body temperature is not rectilinear but sigmoid in shape with a plateau at the beginning and at the end of the cooling process.

The initial plateau, which rarely lasts more than three to four hours, is generally explained on the basis of heat generated by the residual metabolic process of dying tissues and by the metabolic activity of intestinal bacteria. A recent study by Hutchings reports elevations of the temperature rather than a plateau within the first hours following death, with a return to baseline within four hours. The final slowing of the rate of cooling is attributed to the reduced gradient between body temperature and ambient temperature.

The skin, as the closest organ to the environmental air, cools quite rapidly and is not useful for sequential temperature measurements. Temperature changes of the inner core are preferred, because the decline is slower and more regular. Many sites have been tried for taking body temperatures. The most convenient and commonly used procedure involves hourly measurements of the rectal temperature. Some prefer the liver and brain as more representative sites of the inner core temperatures.

The postmortem rate of cooling may be used for estimating the time interval since death. As a matter of fact, literature surveys indicate that more than a hundred and fifty years ago postmortem cooling was used for this purpose in medicolegal cases. Since then, numerous studies by forensic scientists have attempted to refine the use of cooling rate as a reliable postmortem clock. A thorough historical review of various methods of estimating the time of death from body temperature by Bernard Knight cont-
cluded that in spite of the extensive application of physical theory and a great deal of direct experimentation, the level of accuracy remains low, even in the artificial venue of a controlled experiment. This does not mean that measurements of postmortem temperatures are worthless in determining the postmortem interval, but that these data should be cautiously interpreted in view of variables affecting postmortem cooling.

Postmortem cooling of the human body at the skin surface (i.e. loss of heat to the environment) takes place by three major mechanisms:

1. Conduction: transferal of heat by direct contact to another object.
2. Radiation: transfer of heat to the surrounding air by infrared rays.
3. Convection: transfer of heat through moving air currents adjacent to the body.

Internal organs cool primarily by conduction. It follows that factors which affect these mechanisms are bound to affect the rate of cooling as well.

For example, body insulators such as clothing and increased body fat will decrease the rate of heat loss and, therefore, decrease the rate of cooling. Active air currents increase heat loss by convection and, therefore, accelerate the rate of cooling. Similarly, immersion in cold water will increase the heat loss by conduction and accelerate the rate of cooling. A larger body surface ratio to body mass, such as the case in children, will increase relative heat loss and therefore increase the rate of cooling. Furthermore, the rate of cooling is dependent on the temperature gradient between the body and the environment, and its calculation assumes that the environment is cooler than the body temperature; the higher the gradient, the faster be the loss of heat.

However, if the environment is warmer than the body temperature, the postmortem body temperature will be increased. In calculating back to the time of death, one should not necessarily assume that the body temperature at the time of death was normal (36.5° C to 37° C, or 98.6° F). People may die with hyperthermia at much higher than normal body temperature because of a variety of factors including sepsis, hyperthyroidism, physical exercise, heat stroke, seizures or drugs (cocaine, amphetamines, anticholinergic drugs, phencyclidine). Head injury, with damage of the hypothalamic area of the brain, may cause a terminal body temperature of 105° F or higher. Obviously, postmortem cooling would be significantly affected in such cases. On the other hand, individuals may die in a state of hypothermia caused by shock, environmental exposure or drugs (alcohol, sedative-hypnotics, opiates, phenothiazines).

Early Postmortem Ocular Changes

The eyes often exhibit some of the earliest postmortem changes. An immediate sign of death in the fundi of the eyes is the arrest of capillary circulation with settling of red blood cells, in a rouleaux or boxcar pattern.

When the eyes remain open, a thin film may be observed within minutes on the corneal surface, and within two to three hours corneal cloudiness develops. If the eyes are closed, the appearance of the corneal film may be delayed by hours and that of corneal cloudiness by twenty-four hours or longer.

If the eyes are partly open in a dry environment, the exposed areas between the lids may develop a blackish-brown discoloration known as tache noire (black spot). This phenomenon has been mistakenly interpreted as bruising. Absence of intraocular fluid suggests a time of death of at least four days. (Even in the absence of fluid within the eyeballs, the interior of the globes can be rinsed with water or saline and the fluid submitted for toxicological analysis.)

Postmortem Lividity (Livor Mortis)

Postmortem lividity (livor mortis) or postmortem hypostasis is a purplish-blue discoloration due to the settling of blood by gravitational forces within dilated, toneless capillaries of the deceased’s skin. Accordingly, livor is seen in the dependent areas, i.e. on the back if the body was in a supine position, and on the face and front if the body remained prone. Within the circumscribed sites of livor, one may see pale areas where the skin was pressed against a hard surface or object pre-
venting postmortem sedimentation (Fig. II-3). Postmortem lividity may be evident as early as twenty minutes after death or may become apparent after several hours. The development of lividity is a gradual process which progressively becomes more pronounced. However, even after a number of hours postmortem lividity may be difficult to discern in cases of severe anemia or following extensive blood loss. In a case of a ruptured aortic aneurysm or severed aorta, postmortem lividity may be so faint as to be practically indiscernible.

In individuals with dark skin pigmentation, lividity in the skin can go unnoticed. At autopsy, finding congestion of internal organs, such as the kidneys, may assist in determining the presence of lividity.

In the early stages, livor can be blanched by compression (Fig. II-4) and may shift if the position of the body is changed. After eight to twelve hours, the blood congeals in the capillaries or diffuses into the extravascular tissues and does not usually permit blanching or displacement. In advanced stages of livor, the skin capillaries often burst and cause pinpoint hemorrhages known as Tardieu spots (Figs. II-5 and II-6).

Unusual discoloration of postmortem lividity may serve as a diagnostic clue regarding the cause of death. The pathological mechanism responsible for the abnormal discoloration is usually the presence of an abnormal hemoglobin compound (e.g. carboxyhemoglobin, methemoglobin). In some instances cherry-red discoloration may be caused by the poisoning of cellular respiration (inhibition of cytochrome oxidase) resulting in excessive oxygen in the venous blood, as in cyanide and fluoroacetate poisoning (see Table II-1).

Cherry-pink livor is also seen in bodies recovered from water, wearing or covered with wet clothes, or lying on moist metal trays. Humidity prevents the escape of oxygen, allowing for an excess of bright red oxyhemoglobin in the skin.

In certain cases it may be difficult to distinguish between postmortem livor and antemortem bruises. Incision of the skin may be required. Postmortem lividity is entirely intravascular and in its early stages can be drained. In a bruise, blood diffusely infiltrates the interstitial tissue and cannot be removed by drainage. With the onset of decomposition blood vessels become permeable and permit the escape of livor-blood into the interstitial tissues. Differentiation of such areas from true bruises may be difficult or impossible.

Since scars are devoid of blood vessels, postmortem lividity does not affect scarred areas. Thus, a scar in an area of lividity is usually easily

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**TABLE II-1**

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<thead>
<tr>
<th>Etiology</th>
<th>Color of Liver</th>
<th>Mechanism</th>
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<tbody>
<tr>
<td>Normal</td>
<td>Blue-purplish</td>
<td>Venous blood</td>
</tr>
<tr>
<td>Carbon monoxide</td>
<td>Pink, cherry-red</td>
<td>Carboxyhemoglobin</td>
</tr>
<tr>
<td>Cyanide</td>
<td>Pink, cherry-red</td>
<td>Excessive oxygenated blood because of inhibition of cytochrome oxidase</td>
</tr>
<tr>
<td>Refrigeration/ hypothermia</td>
<td>Pink, cherry-red</td>
<td>Same as above Oxygen retention in cutaneous blood by cold air</td>
</tr>
<tr>
<td>Sodium chlorate</td>
<td>Brown</td>
<td>Methemoglobin</td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
<td>Green</td>
<td>Sulfhemoglobin</td>
</tr>
</tbody>
</table>
FIGURE II-4. Livor is blanched by the patterned glove compression seven-and-one-half hours after death.

FIGURE II-5. Tardieu's spots in the feet of a hanging victim. There is also considerable swelling of the ankles as a result of hanging for several hours.
noticeable. Also, the absence of blood retards decomposition of a scarred area. A cirrhotic liver, for instance, is likely to decompose at a significantly slower rate than a normal liver.

Postmortem Rigidity (Rigor Mortis)

Following death, the muscles become initially flaccid, and the lower jaw and extremities can be passively moved. The flaccidity is followed by an increasing stiffness or rigidity of the muscular mass, which *freezes* the joints and is known as postmortem rigidity or rigor. The rigidity then gradually subsides, and the body becomes flaccid again.

In temperate climates, under average conditions, rigor becomes apparent within half an hour to an hour, increases progressively to a maximum within twelve hours, remains for about twelve hours and then progressively disappears within the following twelve hours (Fig. II-

Rigor mortis develops and disappears at a similar rate in all muscles. However, because of a lesser volume, small muscles (e.g. masseters, hands) become totally involved by rigor before the large volume muscles (e.g. thigh muscles), a phenomenon which formerly led to the misleading belief that rigor progresses from the head downwards. Once fully established, the breaking of rigor in joints is irreversible and it will not reappear. However, if the rigor is broken before it is totally completed, a variable extent of rigidity will reappear.

The occurrence of postmortem rigor is a phys-
The chemical process following somatic death where the muscles continue their metabolic activity of glycolysis for a short time. During this process, ATP is hydrolyzed to ADP, and lactic acid is produced, lowering the cellular pH. The lack of ATP regeneration after death and the increased acidity result in the formation of locking chemical bridges between the two major muscle proteins, actin and myosin. This interlocking connection is fixed and produces rigor, without shortening of the muscle. In physiologic contraction, in contrast, the actin molecules slide inwards over the myosin, and the muscle shortens. Animal experiments indicate that in addition to the declining postmortem levels of SATP, a certain concentration of free calcium ions is also required for the development of rigor mortis, and that rigor is inhibited by calcium binding agents.

With decomposition of body proteins, the chemical bridges between actin and myosin of the muscle in rigor break down, and the muscle becomes flaccid again.

As with other sequential postmortem changes, rigor mortis can assist in the determination of the postmortem interval. However, one should remember that the progression of rigor may be substantially modified by a variety of factors which affect the underlying chemical process. Rigor mortis appearance and disappearance is accelerated by prior exercise, convulsions, electrocution, hyperpyrexia or hot environmental temperature. In a hot environment, for example, the rigor mortis may disappear in only nine to twelve hours. Similarly, metabolic states associated with acidosis and uremia hasten the process. Hypothermia and cold environmental temperatures slow the chemical reactions and, therefore, delay the rigor process. Rigor mortis development is also affected by total body muscle mass and has been shown to develop poorly in young children, the elderly and debilitated. Drugs affect postmortem rigor according to their physiological actions. Strychnine poisoning, which is associated with strong tetanic convulsions, accelerates rigor while car-
bon monoxide poisoning, associated with shock or hypothermia, delays it.

The variability of postmortem rigor makes its use as a postmortem clock rather tenuous, to be considered only in conjunction with other timing indices. When the appearance of rigid limbs is inconsistent with gravitational forces, rigor is a reliable indicator of a postmortem shift in the position of the body. For example, an individual who is found in rigor with arms raised, defying gravity, was obviously moved from his original position after the initiation of rigor (Figs. II-8 and II-9).

**Rigor Mortis of Involuntary Muscles**

Rigor mortis affects not only the voluntary muscles but the involuntary muscles as well, producing misleading artifacts. Rigor mortis, for example, may, to a different extent, affect the iris of each eye and produce an artifactual difference between the pupil size which may simulate a significant premortem pupillary disparity. The arrectores pilorum, the tiny muscles of the hair follicles, may be strikingly affected by rigor, resulting in cutis anserina or gooseflesh (Fig. II-10). Some believe erroneously that gooseflesh is somehow associated with drowning or death in water. Also, some believe mistakenly that hair grows after death because rigidity of the arrectores pilorum muscles causes hair to erect and appear longer.

Another manifestation of postmortem rigidity is the finding of semen at or near the tip of the penis. This expulsion of semen is the result of contraction due to postmortem rigidity of the layer of muscle in the wall of the seminal vesicles, which function as semen reservoirs.

The heart in rigor mortis may simulate hypertrophy, while secondary flaccidity may mimic pathologic dilation. It is interesting to note that following open heart surgery cardiac patients may develop an ischemia-related, irreversible contraction of the heart resembling rigor, which has been graphically described as stone heart.11-13

**Cadaveric Spasm**

In rare instances, a forceful agonal contraction or seizure is converted almost immediately into tight rigor without preceding primary flaccidity. In such cases, labeled as cadaveric spasm, the clenched fist may be seen tightly holding a cigarette, blades of grass, clothing or some other object. Cadaveric spasm usually occurs in deaths preceded by great excitement or tension. It is usually seen in cases of drowning, with the deceased grasping weeds or other aquatic vegetation, and in cases of homicide where the victim clutches some of the assailant's hair or clothing (Fig. II-11).

**Stomach Contents**

The presence, appearance and amount of stomach contents may be helpful in determining the time of death. This determination is based on the assumption that the stomach emp-
FIGURE II-9. Rigidity maintaining the legs against the brick wall. The flexed position of the legs led to the immediate conclusion that she had died elsewhere and had been moved after being dead at least six hours. Search of the area disclosed bloodstains in the home where she had been beaten. Her male friend confessed to having moved her body from the house hours later during darkness, concealing it in the court of an adjacent house.

ties at a known rate. However, the emptying rate may be only approximated, because it changes according to various factors, including the amount and type of food, drug or medication intake, prior medical and emotional condition of the deceased and other individual variables.

An ideal postmortem evaluation protocol of the rate of gastric emptying should include:
1. A description of the nature, amount, size and condition of the stomach contents.
2. A microscopic examination of the contents if the contents are difficult to identify or are partially liquefied by the digestive process.
3. An examination of the small intestine for undigestible markers (e.g. corn kernels, tomato peels) to see how far ahead certain digested foods traveled.
4. A toxicological examination of both blood and stomach contents for drugs and alcohol.
5. An evaluation of the prior medical and psychological status and related medications and drugs.

Gastric emptying is a complex process which depends on signals originating not only in the stomach but in the intestines and brain as well. The stomach's distension by the meal affects the emptying process through reflex relaxation of the gastric fundus. Additionally, the meal's presence stimulates the gastric mucosa to secrete hormonal substances of a peptide nature (e.g. gastrin) which delay gastric emptying. Osmotic and calcium binding receptors in the duodenal mucosa respond to the composition of the incoming food and trigger the release of additional hormonal peptides (e.g. cholecystokinin) which have both a direct and indirect neural effect on post-prandial gastric emptying. Furthermore, additional chemical receptors in the distal small intestines and colon trigger the release of additional factors, such as peptide VV,
which also affect the rate of gastric emptying. Finally, the central nervous system also exerts a substantial control over gastric emptying.

This complicated array of monitoring stations is affected by many factors. The rate of emptying, for example, is substantially influenced by the physical state of the food. Solid foods empty slower than liquid foods.

While the half-emptying time for one hundred and fifty grams of orange juice is reported to average about half an hour, the amount of time required to digest and empty fifty grams of solid food may require two hours.\textsuperscript{15,16} This, however, depends on the type of food and its nutritive density (isocaloric value). The greater the nutritive density and osmolarity of a meal, the slower the meal is transferred from the stomach into the duodenum. Starchy and fatty foods may delay both the digestive process and emptying of the stomach. Light meals are usually present in the stomach for up to one-and-a-half to two hours, medium meals up to three to four hours and heavy meals four to six hours or more.\textsuperscript{17,18} The head of the meal usually reaches the cecum within six to eight hours.

The stomach does not empty instantaneously; neither are large amounts of food expelled periodically. Only a small amount of food (a few grams) is expelled per minute, only after having been ground to small particles. Therefore, the size of food particles and the extent of mastication also affect the emptying rate of the stomach. Individuals who gulp their food without adequate mastication, whether because of lack of dentition or poor habits, have prolonged gastric retention of the meal prior to emptying to allow for its digestion.

An increased volume of ingested food accelerates only moderately the rate of gastric emptying when the energy density of the meal remains the same.

Drugs and alcohol also affect the rate of gas-
tric passage. The presence of concentrated alcoholic beverages (more than thirty percent) in the stomach causes constriction of the pyloric muscle and delays gastric emptying. A variety of compounds including narcotics (heroin, meperidine, etc.), phenothiazines, atropine, beta-adrenergic drugs, potassium salts and synthetic progestins also substantially inhibit gastric emptying, while others such as diazepam (Valium®), metoclopramide and bulk laxatives accelerate it.

Natural diseases may also affect the rate of gastric emptying. For example, diabetes, bulimia and pyloric diseases (e.g. pyloric stenosis or peptic ulcers) are associated with delayed gastric emptying. The final emptying time for an idiopathic functional dyspeptic patient, for example, was found to be delayed by more than forty percent as compared to normal.  

Emotional stress (fear, excitement, etc.) also affects the time of gastric emptying by delaying it for many hours. Similarly, individuals in shock may retain gastric contents for days. Age and body build also affect the rate of gastric emptying, the elderly and the obese shown to have a slower emptying gastric rate. Finally, environmental factors such as extreme cold or very hot weather may also retard gastric emptying.

Subtotal gastrectomy with gastroenterostomy and certain types of moderate exercise, such as running, have been shown to accelerate the gastric emptying rate. On the other hand, exhaustive exercise, such as a marathonic run, substantially slows the rate of gastric emptying.

In conclusion, the emptying of the stomach is a complex multifactorial process, and its evaluation for determining time of death requires caution and careful review of all limiting factors. Consideration must also be given to the possibility of one or more close consecutive meals.

It has been found that stomach contents which are readily identifiable by naked-eye inspection were usually ingested within a two-hour period.

Decomposition

The disintegration of body tissues after death is known as decomposition. Decomposition follows the arrest of the biochemical processes which preserve the integrity of the cellular and subcellular membranes and organelles. During decomposition, the tissue components leak and break up, hydrolytic enzymes are released from
the intracellular lysosomal sacs, and bacteria and other microorganisms thrive on the unprotected organic components of the body.

Accordingly, two parallel processes of decomposition have been distinguished:
1. **Autolysis**: self-dissolution by body enzymes released for the disintegrating cells.
2. **Putrefaction**: decomposition changes produced by the action of bacteria and microorganisms.

A third kind of postmortem destruction of the body occurs as a result of anthropophagy (i.e. attacks by various types of predators) from small insects to larger animals, particularly rodents.

### Autolytic Changes

The earlier autolytic changes occur in organs rich in enzymes such as the pancreas, gastric mucosa and the liver. Focal autolytic changes of the pancreas are almost invariably seen at autopsy.

Gastromalacia (autodigestion of the gastric mucosa with perforation) has been described to occur following injuries in the last stages of coma or shortly before or after death. We have observed it more often in cases of closed head injury, possibly related to stimulation of the heat regulatory center in the brain and a terminal surge of body temperature, promoting autolysis. It usually occurs in the area of the fundus of the stomach and is devoid of any vital reaction. Esophagomalacia is a similar process which involves the lower portion of the esophagus and allows esophageal and gastric contents to burst into the left chest cavity.

### Putrefaction

Putrefactive changes are dependent primarily on environmental temperatures and the prior state of health of the individual. Changes which in temperate climates take days to develop may develop within hours in a warm environment.

Furthermore, individuals dying in the same area may show very different stages of decomposition, according to their individual degree of exposure to the sun or proximity to a source of heat (stove, radiator, etc.) (Fig. II-12).

Individuals with sepsis usually undergo rapid decomposition with putrefaction. In some cases of gas-producing *Clostridia* sepsis, one may witness an amazingly rapid progression of putrefactive changes in the liver, from a seemingly normal appearance at the beginning of the autopsy, to a mushy, decomposing mass an hour or so later. The putrefaction gases include methane, carbon dioxide, hydrogen and particularly malodorous ammonia, hydrogen sulfide and mercaptans. This gas burns readily when ignited (Fig. II-13).

Fever prior to death, such as encountered in sepsis, rhabdomyolysis and cocaine overdose, also substantially accelerates decomposition and putrefaction. In such cases advanced putrefaction may be observed in less than twelve hours. Putrefaction is also more rapid in obese individuals. The putrefaction process is accelerated in edematous or exudative areas of the body and delayed in dehydrated tissues or following massive blood loss. On the other hand, in infants and thin individuals, putrefaction proceeds at a significantly slower pace.

The rate of putrefaction also depends on the physical environment in which the body lies. It is generally accepted that putrefaction in air is more rapid than in water, which is more rapid than in soil. One week in air equals two weeks in water and eight weeks in soil.

Exposure to cold also substantially delays the decomposition process. In evaluating postmortem changes, it is, therefore, important to consider any intermittent period of exposure to cold, refrigeration or freezing. A further consideration is postmortem rewarming or thawing of the body. Experiments have shown that previously frozen and thawed animal tissues decompose significantly faster than freshly killed animals. Tissues which are damaged by trauma show accelerated rates of decomposition.

Decomposition gases may cause tissue artifacts mimicking softening cysts in the brain (encephalomalacia) and elsewhere, although the Swiss cheese pattern of the cavities easily indicates their postmortem character.

Similarly, decomposition gases may make difficult the diagnosis of air and fat embolism and cause the lungs of stillborns to float, leading to
The influence of environmental temperature on postmortem decomposition. This couple was killed at the same time by a mentally deranged son. The body of the mother was found in the cool basement, while the body of the father was discovered in a warm upstairs room. Outside temperature was 90°F, postmortem interval about forty-eight hours.

erroneous determination of spontaneous breathing at birth.

Under condition that promote putrefaction, especially in hot and humid environments, one may occasionally see a peculiar red discoloration of the teeth (pink teeth). The red discoloration is due to diffusion of hemoglobin from hemolyzed red blood cells into the dentin canaliculi. Some studies have reported a frequency as high as twenty percent of pink teeth in sequential autopsies.21

A rare change caused by decomposition is the presence of white-gray, pinpoint foci, called miliaria, which are scattered below the endocardium and below the capsules of the liver, kidneys and spleen. The miliaria are easily distinguished from granulomas, fungi or fatty necrosis and are presumably due to autolytic changes resulting from precipitation of calcium and other salts.

In temperate climates, early decomposition becomes manifest within twenty-four to thirty hours with greenish discoloration of the abdomen, due to denaturation by colonic bacteria, ofhemoglobin to biliverdin and its reaction with hydrogen sulfide. Such discoloration is more prominent in the right lower abdominal area because of the close proximity of the cecum to the abdominal wall.

This is followed by gaseous bloating, dark greenish to purple discoloration of the face and purging of bloody decomposition fluids from the nose and mouth. The tongue swells and progressively protrudes from the mouth, and the eyes
FIGURE II-14. Prominent marbling two days after death. Note swelling and discoloration of face. Marbling is limited to areas of livor mortis.

The greenish and purplish discoloration rapidly spreads within thirty-six to forty-eight hours to the chest and extremities, displaying a marbling pattern which delineates the decomposition of the blood and formation of sulfhemoglobin and hematin within dilated subcutaneous blood vessels (Fig. II-14).

Postmortem discoloration of the skin may be so dark that white individuals may be easily mistaken as black (Fig. II-15).

As decomposition progresses, the skin becomes slippery with vesicles and slippage of the epidermis, and generally, after three days, the entire body becomes markedly bloated. Swelling is particularly dramatic in areas of loose skin (eyelids, scrotum and penis). The skin of the hands often sheds, together with nails, in glove-like fashion, and the skin of the legs in a stocking-like pattern, a phenomenon which is also seen following prolonged immersion in water and in cases of second-degree burns (Figs. II-16 and II-17).

Additional destruction of the body is caused by maggots. Fly eggs initially deposited at the corners of the eyes, mouth and other mucocutaneous junctions (Fig II-18) develop into innumerable crawling maggots which rapidly destroy soft tissues. The maggots concentrate primarily in areas of body openings and perforations where they seek shelter and feed on blood and tissues.

Anytime a decomposed body is found with an unusually large concentration of maggots in a particular area, it is probable that a wound pre-existed in that location. In the case of a close-range gunshot wound, maggots may remove tis-
sue at the edges of the wound but leave soot deposited on the bone and gunpowder undisturbed.

Ultimately, decomposition ends in complete skeletonization. In temperate areas, under average conditions, the minimum period for full skeletonization is about one-and-a-half years.

The rate of putrefaction is significantly faster in arid environments. Galloway et al., in reviewing the earliest time of postmortem change in the hot, dry climate of Arizona, reported bloating of bodies as early as two days, gases at three days, advanced sagging of tissue and advanced intra-thoracic and intra-abdominal activity of maggots at four days, partial mummification with leathery change of skin at four days, and skeletonization after six to nine months.\(^{22}\)

Under most favorable conditions, particularly with necrophagous insect activity, skeletonization may occur even earlier. Stewart\(^{23}\) reports the case of a thirteen-year-old Mississippi girl, victim of a homicide, whose body became almost completely skeletonized within ten days during late summer.

**Post-Skeletonization Weathering Changes and the Time of Death**

Once the body is fully skeletonized, the bones undergo a slow process of weathering and breaking down, lasting decades or centuries. Typical weathering of bones includes bleaching, exfoliation (desquamation) of cortical bone and demineralization. The rate and severity of these changes depends on environmental conditions, whether the bones were buried or exposed, the acidity of the soil and extent of humidity. Soil staining, which is a brown or sometimes tan discoloration of the bone surface, is variable but may occur in as little as one to two years after complete skeletonization. Green discoloration of the bone surface is often caused by contact with copper or brass and may be seen as early as six months after exposure.

In hot, arid climates such as Arizona, bleaching of bones has been reported to occur as early as two months and exfoliation as early as four months, though usually the former takes six months and the latter as long as twelve to eighteen months. Demineralization is a late process, commonly seen in old bones or those found in archaeological excavations. It results in very light, porous and friable bonds. Such bones may turn to dust on touching. Contact with certain roots may significantly accelerate bone demineralization.

[FIGURE II-16. Skin stockings and left glove. Bloating of body, especially of the breasts and marked discoloration of the face (three-and-a-half days after death).]
Mummification and Adipocere

Two types of postmortem changes, mummification and adipocere, may counter substantially the process of tissue destruction by decomposition. Mummification results from drying of tissues under conditions of high environmental temperature, low humidity and good ventilation.

The conjunctivae of the eyes dry along the opening between the lids, causing a dark-brown horizontal band across the corneal surface sometimes referred to as tache noire (Fig. II-19). The scrotum dries at the sides where exposed and not in contact with the moist skin of the thighs (Fig. II-20). Tightened mummified skin displays a brownish discoloration and a parchment-like appearance, which preserves facial contour and dries and discolors bent knees (Fig. II-21). Similar drying may be observed in fingers and toes exposed to hot, dry air. Mummified fingers and toes are shriveled with wrinkled, firm, brown skin (Fig. II-22). The process begins at the fingertips which become spindly. Fingers in this condition are unsuitable for fingerprint-
FIGURE II-18. Eggs laid by flies in the moist areas of the corners of the eyes, nares and angles of the mouth.

ing unless first soaked in warm water to stretch and unfold the skin for the return of its natural texture. Shrinkage of the nail beds has occasionally misled investigators and mystery book writers to conclude that fingernails and toenails grow after death.

The skin around the fingernails and toenails shrinks as a result of drying and may give the erroneous impression that the nails have grown after death. Drying of certain parts of the body may cause shrinkage of the skin to the extent of causing large splits that resemble actual injury.

FIGURE II-19. Postmortem dark discoloration of sclera (tache noire) along the exposed palpebral fissure of the

Such splits are especially common in the groins, neck and armpits.

In mummified bodies in temperate areas, the internal organs are usually poorly preserved or may have totally disappeared due to decomposi-

FIGURE II-20. Drying of the scrotal skin is sometimes mistaken for bruising.

FIGURE II-21. Leathery, shrunken face of mummified body found two months after death. Deceased was found covered by some clothing in a basement.
tion. Once mummification is fully developed, the body remains preserved as a shell for long periods of time, even years (Fig. II-23). The rate of mummification and its extent depend on the humidity of the air and the intensity of the environmental heat, and its full development in temperate areas generally requires at least three months of postmortem interval.

Adipocere (waxy fat) (Fig. II-24) develops under conditions of high humidity and high environmental temperature and especially involves the subcutaneous tissues of the face, extremities, buttocks and female breasts. The chemical process underlying adipocere consists of hydration and dehydrogenation of body fats, a process which imparts a grayish-white color and soft, greasy, clay-like, plastic consistency to the soft tissues of the body.

Recent research has demonstrated that bacterial enzymes of both intestinal and environmental sources, particularly *Clostridia*, are primarily responsible for adipocere, by converting unsaturated liquid fats (oleic acid) to saturated solid fats (hydroxystearic acid and oxostearic acid). The time for the development of adipocere is estimated to be at least three months and usually is not observed before six months.

**Stillbirths**

Human fetuses are generally considered viable after twenty-four weeks of pregnancy, at which time they reach a weight of six hundred to eight hundred grams and measure twenty-one to twenty-two centimeters from crown to rump. (Foot length is the most reliable external measurement parameter for gestational age.) A stillbirth is the delivery of a viable fetus which is not breathing and shows no sign of life (Apgar score 0). The term *stillbirth* is synonymous with *dead birth* and the word *still* describes the absence of fetal respiration or other movements.

The determination of stillbirth has important medicolegal implications, particularly in instances when the death of a newborn is concealed. In such instances, it must be determined whether the delivery was indeed that of a dead fetus, i.e. a stillborn, whether the fetus was born alive and died as a result of failure to provide required care, or whether there was an intentional infanticide.

In such cases, the questions which the forensic pathologist must address are:
1. What is the gestational age of the fetus (by weight and dimensions)?
2. Was the fetus viable by estimated age, weight and size, and alive at birth? (Did the child breathe air?)

3. Were there traumatic injuries present in the fetus, and what was their significance?

4. What was the cause and manner of death?

To fully answer these questions, a thorough autopsy, including careful examination of the umbilical cord and placenta, should be performed. Common histological abnormalities of the placenta in stillbirth cases include placental infarcts, hemorrhagic endovasculitis, retroplacental hematomas, acute chorioamnionitis and hydrops.25

Similarly, the examination of the umbilical cord of the stillborn may reveal significant abnormalities such as true knots, torsion, arterial agenesis, thrombosis and funisitis.

When intrauterine death has occurred days or months prior to the delivery, the body of the fetus shows postmortem changes defined as maceration. Maceration is an autolytic process, i.e. a decomposition due to self-disintegration of the body by released cellular enzymes. The fetus and the bathing amniotic fluid are sterile and, therefore, will not undergo putrefaction if the membranes are intact. The macerated fetus initially shows a reddish, dusky discoloration and an easily peeling skin with fluid accumulation beneath the epidermis and formation of large bullae. The reddish discoloration is due to diffuse hemolysis and involves both the skin and the internal organs. Easy separation of the epidermis does not occur until the last trimester of pregnancy. Before this period, the epidermis is less differentiated and is tightly adherent to the dermis.

This is followed, within one to three days, by further darkening of the skin, flaccidity, separation and overlapping of the cranial bones, dislo-
FIGURE II-24. Adipocere formation in the face and head of a seaman who had drowned six months before recovery of his body in April in the Chesapeake Bay. During winter the bay was covered with ice for two or three weeks. Water temperature when body was recovered was near 60° F. A key with the Norwegian word for cook was found in the pocket of the seaman and helped to establish identification.

cation of the temporomandibular joints and accumulation of hemolyzed blood and fluid in the body cavities. Rigor is almost never observed in macerated fetuses. In fetuses which are retained for more than seven to ten days, the reddish dark color starts to change into a light greenish-brown. Within a few days, the brain liquifies and the abdominal tissues decompose, often with disintegration of the intestinal wall, releasing meconium into the abdominal cavity. In cases where pregnancy continues, the dead fetus may be gradually resorbed or mummified (papyraceous appearance). In rare cases, usually associated with ectopic pregnancy, the dead fetus may calcify (lithopedion).

It is important to realize that some body mea-
Time of Death and Changes After Death

Measurements, including weight of a macerated fetus, may be altered by postmortem changes and be unsuitable for estimating the gestational age. This makes it difficult, from the pathologist's point of view, to correlate the gestational age of the fetus, to a dire traumatic event to the mother.

Decomposition certainly complicates the autopsy of a macerated fetus, although major developmental anomalies can still be detected. One should be careful not to interpret as traumatic the subarachnoid or intraventricular hemorrhages often observed in stillborns as a result of hypoxemia.

Microscopic examination of lungs of stillborns frequently reveals some evidence of amniotic fluid aspiration with keratin squames in the alveoli, a finding which has been related to fetal distress. Other histological findings seen in fetal distress are tubulocystic change in the adult cortex of the adrenal gland, involution (starry sky appearance) of the thymus and meconium staining of the skin and placental membranes.

**POSTMORTEM ARTIFACTS**

Distinction of antemortem injuries from postmortem artifacts is of obvious importance. Problems in differential diagnosis may occur as a result of faulty autopsy technique, distorting postmortem changes, and destructive environmental factors such as high environmental temperatures, postmortem mechanical trauma and anthropophagy, i.e. destructive changes by scavengers.

Even examination of fresh bodies may give rise to diagnostic difficulties when fatal injuries are accompanied by minimal hemorrhage or when immediate postmortem trauma opens blood vessels and causes artifactual bleeding.

I. Faulty Autopsy Technique

Faulty autopsy techniques can create confusing postmortem artifacts. For example, careless removal of congested neck organs before extracting the chest and abdominal organs may produce artifactual hemorrhages in the strap muscles of the neck or laryngeal fractures, mimicking strangulation. Similarly, prying the skull with a chisel to break any remaining bridge of bone missed by sawing may produce linear basilar fractures simulating fractures sustained during life. Removing the chest plate at the autopsy involves tearing large arteries and veins and may result not only in bleeding within the chest and abdominal cavities but also in drawing air into blood vessels, mimicking air embolism.

II. Errors in Interpretation of Decomposition Changes

The following is a non-inclusive list of common situations in which distortions due to postmortem changes may be subject to misinterpretation:

1. Postmortem bloating of the body may create a misleading appearance of obesity.
2. Bloody decomposition fluid purging from the mouth and nose may be misinterpreted as premortem bleeding due to trauma.
3. The presence of decomposed bloody fluid in the chest cavity may be misconstrued as hemothorax.
4. Agonal or postmortem autolysis and perforation of the stomach may be misinterpreted as a perforated ulcer.
5. Postmortem dilatation and flaccidity of the vagina and anus may produce the appearance of a sexual attack or sodomy.
6. Pinpoint foci of extravasated blood from burst capillaries in areas of intense livor may simulate premortem petechial hemorrhages.
7. Diffusion of hemolyzed blood into tissues in areas of livor may be difficult to distinguish from genuine bruising in cases of moderately advanced decomposition.
8. Focal autolytic changes in the pancreas may be misinterpreted as focal necrosis or focal hemorrhagic pancreatitis.

Failure to carefully examine areas where decomposition is particularly advanced may result in missing of significant trauma, particularly since premortem injuries undergo more rapid decomposition.

Alterations in blood and tissue levels of premortem toxic substances as a result of postmor-
FIGURE II-25. Anthropophagy: Irregular, bloodless defect under the eye, caused by dog (?) postmortem nibbling.

tern hydrolysis or decomposition may make a diagnosis of poisoning or overdose difficult if not impossible. For example, cocaine disappears rapidly from postmortem blood and tissue as a result of hydrolysis, while postmortem levels of alcohol can substantially increase due to tissue decomposition.

A less known toxicological artifact results from postmortem redistribution of drugs. Postmortem diffusion of certain drugs, such as pentodiazepines, barbiturates and digoxin, has been reported to occur from areas of high tissue concentration into the blood.27

III. Destructive Environmental Factors

Exposure of the body to environmental forces may result in pathological changes which may obscure, modify or mimic genuine premortem injuries.

Some of the artifactual injuries clearly point to their etiology, while others are less specific and more questionable. Postmortem thermal ar-
FIGURE II-29. Postmortem artifact produced by ants. The girl was dumped face-down in a wooded area after being raped and strangled. The injuries of the face were first mistaken for fingernail marks. The fine linear scratches are due to twigs and undergrowth.

FIGURE II-30. Postmortem artifact. Skin lesions of arm caused by roaches. These are easily confused with antemortem abrasions if the roaches are not observed when the body is discovered. Note the similarity with the postmortem injuries produced by ants. The latter are somewhat smaller and more discreet.
Time of Death and Changes After Death

Artifacts often seen in fire victims include fractures of the skull and epidural hemorrhage due to intracranially generated steam, fractures of the extremities due to thermal contractions of tendons, and wide splitting of skin and muscles, simulating lacerations, cuts and stab wounds. It is interesting to note that subdural and subarachnoid hemorrhages are not artifically produced in conflagrations.

Frozen bodies of infants may show artifactual folding of the skin of the neck, simulating ligature strangulation. Drowning fatalities recovered from larger bodies of water (rivers, lakes, oceans) may show extensive postmortem mutilation due to boat propellers, simulating injuries sustained during life.

Similarly, postmortem injuries caused by various scavengers (such as flies, ants, beetles, roaches, dogs, rodents, aquatic animals) may cause injuries (anthropophagy) simulating premortem trauma (Figs. II-25 to II-36). Superficial (epidermal) skin defects produced by insect feeding are sometimes mistaken for cigarette burns. When in doubt, microscopic examination of such areas readily reveals the true nature of such lesions. We have seen cases where dogs removed the head from a decomposing body.
and gnawed it at some distance away. Outdoors animals often take body parts a long distance away and sometimes such parts are never recovered. It is often possible to determine the size of an animal involved in a particular case of postmortem mutilation by examining the edges of the injuries on the body.

**Anthropophagy and Postmortem Vegetal Growth**

Anthropophagy (Greek: *eating of man*) is the postmortem assault of the body by various scavengers. Identification of the specific scavenger and the stage of its development may assist in estimating the minimal period of time which elapsed since death.

For example, identification of maggots as being those of a particular fly (e.g. the bluebottle fly) permits determination of the minimum time elapsing from the deposition of the eggs to the hatching of larvae. Most flies require at least twenty-four hours for hatching, but knowledge of the life cycle of a particular fly permits a more precise determination of the hatching time.

Maggot size (length) may permit further evaluation of the time which elapsed from hatching to the time of recovery of the larvae. Pupae, the next stage of larval development, will obviously

**FIGURE II-33.** Confluent superficial (epidermal) defects produced by roaches and ants.

**FIGURE II-34.** Superficial skin injuries produced after death by ants and roaches. Injuries resemble abrasions and possibly assault.
indicate an even longer postmortem interval. It is recommended that larvae by measured, preserved in a fixative (seventy percent alcohol or four percent formalin solution) and submitted for examination by an entomologist.

Similarly, botanists may be helpful in determining the minimal time interval since death by identifying the type and stage of postmortem growth of fungi and vegetation on bodies which were buried or left exposed to soil.29

Recent Developments for Determination of the Time of Death

In recent years a number of new methods have been suggested for determining the time of death. Some of the methods are based on progressive postmortem increases in levels of 3 methoxytyramine (3MT) in the basal ganglia of the brain and on the circadian variations in the

Figure II-35. Superficial skin defects between the fingers produced by ants. Moisture and shelter attracted the ants.

Figure II-36. Body recovered from water after several months during the winter. The group of small defects near the area of the web of the thumb were caused by fly maggots which burrow in and out through the skin.
levels of betareceptors and melatonin in the pineal gland.

Unfortunately, these methods involve quite intricate and time-consuming chemical or radioimmune techniques and dedicated expertise, and are as yet insufficiently confirmed and largely in early experimental stages.

CONCLUSION

In conclusion, none of the methods used in establishing the time of death are totally reliable and mathematically precise. Dogmatic and pinpoint accuracy in this matter is clearly not achievable.

However, careful consideration of environmental and individual influences, and the concomitant use of as many postmortem clocking devices as possible, frequently permit determination of a realistic range of the postmortem interval.

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