When the heart stops beating

1. Heart stops beating and/or lungs stop breathing.

2. Body cells no longer receive supplies of blood and oxygen.

- Blood drains from capillaries in the upper surfaces and collects in the blood vessels in the lower surfaces.
- Upper surfaces of the body become pale and the lower surfaces become dark.

3. Cells cease aerobic respiration, and are unable to generate the energy molecules needed to maintain normal muscle biochemistry.

- Calcium ions leak into muscle cells preventing muscle relaxation.
- Muscles stiffen and remain stiff (rigor mortis) until they begin to decompose.

4. Cells eventually die and the body loses its capacity to fight off bacteria.

5. The cells' own enzymes and bacterial activity cause the body to decompose - muscles lose their stiffness.

Timing?

- Brain cells can die if deprived of oxygen for more than three minutes. Muscle cells live on for several hours. Bone and skin cells can stay alive for several days.
- It takes around 12 hours for a human body to be cool to the touch and 24 hours to cool to the core.
- *Rigor mortis* commences after three hours and lasts until 36 hours after death.
- Forensic scientists use clues such as these for estimating the time of death.

What is grave wax?

Adipocere (grave wax) forms in fatty regions of the decomposing body including the cheeks.

Grave wax, or adipocere, is a crumbly white, waxy substance that accumulates on those parts of the body that contain fat - the cheeks, breasts, abdomen and buttocks. It is the product of a chemical reaction in which fats react with water and hydrogen in the presence of bacterial enzymes, breaking down into fatty acids and soaps. Adipocere is resistant to bacteria and can protect a corpse, slowing further decomposition. Adipocere starts to form within a month after death and has been recorded on bodies that have been exhumed after 100 years. If a body is readily accessible to insects, adipocere is unlikely to form.

What Causes Rigor Mortis?

Chemistry of Muscle Fibers

A few hours after a person or animal dies, the joints of the body stiffen and become locked in place. This stiffening is called *rigor mortis*. Depending on temperature and other conditions, rigor mortis lasts approximately 72 hours. The phenomenon is caused by the skeletal muscles partially contracting. The muscles are unable to relax, so the joints become fixed in place.

More specifically, what happens is that the membranes of muscle cells become more permeable to calcium ions. Living muscle cells expend energy to transport calcium ions to the outside of the cells. The calcium ions that flow into the muscle cells promote the cross-bridge attachment between actin and myosin, two types of fibers that work together in muscle contraction. The muscle fibers ratchet shorter and shorter until they are fully contracted or as long as the neurotransmitter acetylcholine and the energy molecule adenosine triphosphate (ATP) are present. However, muscles need ATP in order to release from a contracted state (it is used to pump the calcium out of the cells so the fibers can unlatch from each other). ATP reserves are quickly exhausted from the muscle contraction and other cellular processes. This means that the actin and myosin fibers will remain linked until the muscles themselves start to decompose.

Rigor mortis can be used to help estimate time of death. The onset of rigor mortis may range from 10 minutes to several hours, depending on factors including temperature (rapid cooling of a body can inhibit rigor mortis, but it occurs upon thawing). Maximum stiffness is reached around 12-24 hours post mortem. Facial muscles are affected first, with the rigor then spreading to other parts of the body. The joints are stiff for 1-3 days, but after this time general tissue decay and leaking of lysosomal intracellular digestive enzymes will cause the muscles to relax. It is interesting to note that meat is generally considered to be more tender if it is eaten after rigor mortis has passed.

What is *rigor mortis*?

Rigor mortis refers to the state of a body after death, in which the muscles become stiff. It commences after around 3 hours, reaching maximum stiffness after 12 hours, and gradually dissipates until approximately 72 hours after death. Rigor mortis occurs due to changes in the physiology of muscles when aerobic respiration ceases.

Muscles are made up of two types of fibre. These fibres have connections between them that lock and unlock during muscle contraction and relaxation. These connections are controlled by a biochemical pathway within the cell, which is partially driven by the presence of calcium ions. The concentration of calcium ions is higher in the fluid surrounding muscle cells than it is inside the cells, so calcium tends to diffuse into the cell. High calcium levels inside the cell drive the biochemical pathway in the direction that maintains muscle contraction. To relax, muscle cells must expel the calcium ions from the cell and this requires energy molecules to pump them across the cell membrane.

After a body has died, the chemical reaction producing these energy molecules is unable to proceed because of a lack of oxygen. The cells no longer have the energy to pump calcium out of the cell and so the calcium concentration rises, forcing the muscles to remain in a contracted state. This state of muscle stiffening is known as rigor mortis and it remains until the muscle proteins start to decompose.

TIME OF DEATH

A recurring problem in forensic medicine is the need to fix the time of death within the limits of probability. It is self-evident that the longer the interval of time between death and the examination of the body, the wider will be the limits of probability. The longer the post mortem interval, the more likely it is that associated or environmental evidence will furnish more reliable data on which to estimate the time of death than will anatomical changes.

It is necessary to be alert to the possibility that the post mortem interval (the time elapsed from death until discovery and medical examination of the body) may be preceded by a significant survival period (the time from injury or onset of the terminal illess to death). The survival interval is best established by evaluating the types,

severity and number of injuries present and the deceased's response to them, taking into account pre-existing natural disease. At autopsy it is necessary to assess the evolution of the inflammatory response and repair process in skin and viscera.

Establishing the times of an assault and death has a direct bearing on the legal questions of alibi and opportunity. If the suspect is able to prove that he was at some other place when the fatal injury was inflicted then he has an alibi and his innocence is implicit. Conversely, if the time of a lethal assault coincides with the time when the suspect was known to be in the vicinity of the victim, then the suspect clearly had an opportunity to commit the crime. In cases of infanticide, it is necessary for the prosecution to establish that the child was born alive and was killed afterwards. In the absence of proof that death occurred after a live birth, there can be no prosecution for infanticide. Similarly, in bodies recovered from fires, it is critical to establish whether death occurred before or during the fire and this necessitates correlating information relevant to establishing both the time of death and the cause of death. When a body is recovered from water, a critical question is whether the person was alive or dead when they entered the water. Determining whether specific injuries were inflicted before or after death is another important example of establishing temporal relationships.

Sources of Evidence

Evidence for estimating the time of death may come from three sources:

1. Corporal evidence, i.e. that present in the body.
2. Environmental and associated evidence, i.e. that present in the vicinity of the body,
3. Anamnestic evidence, i.e. that based on the deceased's ordinary habits, movements, and day to day activities.

All three sources of evidence should be explored and assessed before offering an opinion on when death or a fatal injury occurred.

There are two methods for estimating the time of death:

1. The rate method. Measuring the change produced by a process which takes place at a known rate which was either initiated or stopped by the event under investigation, i.e. death. Examples include the amount and distribution of rigor mortis, the change in body temperature, and the degree of putrefaction of the body.
2. The concurrence method. Comparing the occurrence of events which took place at known times with the time of occurrence of the event under investigation, i.e. death. For example, a wrist watch stopped by a blow during an assault, the extent of digestion of the last known meal.

Postmortem changes and time of death

Many physico-chemical changes begin to take place in the body immediately or shortly after death and progress in a fairly orderly fashion until the body disintegrates. Each change has its own time factor or rate. Unfortunately, these rates of development of post mortem changes are strongly influenced by unpredictable endogenous and environmental factors. Consequently, the longer the post mortem interval, the wider is the range of estimate as to when death probably occurred. In other words, the longer the post mortem interval, the less precise is the estimate of the time of death.

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1. ALGOR MORTIS (BODY COOLING)

This is the most useful single indicator of the time of death during the first 24 hours post mortem. Some writers would regard it as the only worthwhile corporal method.

It is of some importance to note that the use of body temperature estimations to assess time of death applies only to cool and temperate climates since in tropical regions there may be a minimal fall in body temperature post mortem and in some extreme climates, (e.g. central Australia) the body temperature may even rise after death.

The assessment is made on the basis of measurement of the body core temperature which, post mortem, requires a direct measurement of the intra-abdominal temperature. In practice either the temperature is measured per rectum or the intra-hepatic/sub-hepatic temperature is measured via an abdominal stab. Oral and axillary temperatures should not be used. An ordinary clinical thermometer is useless because its range is too small and the thermometer is too short. A chemical thermometer 10-12" long with a range from 0-50o Celsius is ideal. Alternatively a thermo-couple probe may be used and this has the advantage of a digital readout or a printed record.

Whether the temperature is measured via an abdominal stab or per rectum is a matter of professional judgement in each case. If there is easy access to the rectum without the need to seriously disturb the position of the body and if there is no reason to suspect sexual assault, then the temperature can be measured per rectum. It may be necessary to make small slits in the clothing to gain access to the rectum, if the body is clothed and the garments cannot be pushed to one side. The chemical thermometer must be inserted about 3-4" into the rectum and read in situ. The alternative is to make an abdominal stab wound after displacing or slitting any overlying clothing. The stab may be over the lower ribs and the thermometer inserted within the substance of the liver or alternatively a subcostal stab will allow insertion of the thermometer onto the undersurface of the liver.

The body temperature should be recorded as early as conveniently possible. The environmental temperature should also be recorded and a note made of the environmental conditions (see below) at the time the body was first discovered and any subsequent variation in these conditions. If a method of sequential measurement of body temperature is use then the thermometer should be left in situ during this time period. This latter method is much easier to undertake when using a thermo-couple with an attached print-out device.

Temperature readings of the body and observations made at the scene by one physician are always available for evaluation by an expert at a later time.

The normal oral temperature fluctuates between 35.9°C (96.7°F) and 37.2°C (99°F). The rectal temperature is from 0.3-0.4°C (0.5-0.75°F) higher. Since heat production ceases soon after death but loss of heat continues, the body cools.

During life the human body loses heat by radiation, convection, and evaporation. Heat loss by conduction is not an important factor during life, but after death it may be considerable if the body is lying on a cold surface. The fall in body temperature after death mainly depends upon a loss of heat through radiation and convection, but evaporation may be a significant factor if the body or clothing is wet. The cooling of a body is a predominantly physical process which, therefore, is predominantly determined by physical rules.

Newton's law of cooling states that the rate of cooling of a body is determined by the difference between the temperature of the body and that of its environment. Consequently, a plot of temperature against time gives a curve which is exponential. However, Newton's law applies to small inorganic bodies and does not accurately describe cooling of the human body which has a large mass, irregular shape, and is composed of tissues of different physical properties. Practical observations indicate that the cooling of a human body is best represented by a sigmoid curve when temperature is plotted against time. Thus, there is an initial maintenance of body temperature which may last for some hours - the so-called "temperature plateau" - followed by a relatively linear rate of cooling which subsequently slows rapidly as the body approaches the environmental temperature. The initial lag in cooling was first described by Rainy, Regius Professor of Forensic Medicine in Glasgow, in 1869. (It was independently described in the German literature in the same year by Seydeler). The post mortem temperature plateau is physically determined and not a special feature of the dead human body. Post mortem heat production is said to contribute approximately one-sixth to the plateau. Any inert body which has a low thermal conductivity has such a plateau during its first cooling phase. It is this plateau which produces the sigmoid shape of the resultant cooling curve. The post mortem temperature plateau generally lasts 1/2 to one hour but may persist as long as three hours and some authorities claim that it may persist as long as five hours.

It is usually assumed that the body temperature at the time of death is normal, but in individual cases it may be subnormal or markedly raised. As well as in deaths from hypothermia, the body temperature at death may be sub-normal in cases of congestive cardiac failure, massive haemorrhage, and shock. However, the claim that severe agonal bleeding lowers the body temperature is said to be without foundation. The body temperature may

be raised at the time of death in heat stroke, some infections, and pontine haemorrhage. Simpson (Ref. 11 at p. 7) cites a personal observation of a case of pontine haemorrhage with an initial temperature at death of 42.8oC (109oF) and another instance of a temperature of 37.4°C (99.4°F) about three hours after death in a case of manual strangulation. However, another author claims that there is no convincing proof that asphyxia by strangulation leads to a raised agonal temperature. Where there is a fulminating infection, e.g. septicaemia, the body temperature may continue to rise for some hours after death.

Thus the two important unknowns in assessing time of death from body temperature are

(1) the actual body temperature at the time of death; and
(2) the actual length of the post mortem temperature plateau.

For this reason assessment of time of death from body temperature clearly cannot be accurate, (even approximately), in the first four to five hours after death when these two unknown factors have a dominant influence. Similarly, body temperature cannot be a useful guide to time of death when the cadaveric temperature approaches that of the environment. However, in the intervening period, over the linear part of the sigmoid cooling curve, any formula which involves an averaging of the temperature decline per hour may well give a reasonably reliable approximation of the time of death. It is in this limited way that the cadaveric temperature may assist in estimating the time of death in the early post mortem interval, provided the sigmoid nature of the relationship between the temperature of the cooling body and that of its environment is kept in mind.

The linear rate of post mortem cooling is affected by environmental factors and cadaveric factors other than the environmental temperature and the body temperature at the time of death. These include:

1. The "size" of the body. The greater the surface area of the body relative to its mass, the more rapid will be its cooling. Consequently, the heavier the physique and the greater the obesity of the body, the slower will be the heat loss. Some authors claim that in obese individuals the fat acts as an insulator, but for practical purposes body mass, whether from muscle mass or adipose tissue, is the most important factor. Children lose heat more quickly than adults because their surface area/mass ratio is much greater. Prominent oedema in individuals with congestive cardiac failure is said to retard cooling because of the large volume of water present with a high specific heat whilst dehydration has the opposite effect. The effect of oedema fluid is said to be more potent than body fat. The exposed surface area of the body radiating heat to the environment will vary with the body position. If the body is supine and extended, only 80% of the total surface area effectively loses heat, and in the foetal position the proportion is only 60%.
2. Clothing and coverings. These insulate the body from the environment and therefore cooling is slower. Simpson states that cooling of a naked body is half again as fast as when clothed (Ref. 11 at p. 9). Henssge (see back of nomogram) has graded the effect of clothing by the number of layers and thickness. He states that only the clothing or covering of the lower trunk is relevant.
3. Movement and humidity of the air. Air movement accelerates cooling by promoting convection and even the slightest sustained air movement is significant. Cooling is said to be more rapid in a humid rather than dry atmosphere because moist air is a better conductor of heat. The humidity of the atmosphere will affect cooling by evaporation where the body or its clothing is wet.
4. Immersion in water. A cadaver cools more rapidly in water than in air because water is a far better conductor of heat. For a given environmental temperature, cooling in still water is about twice as fast as in air, and in flowing water, about three times as fast. Clearly the body will cool more rapidly in cold water than warm water. It has been said that bodies will cool more slowly in water containing sewage effluent or other putrefying organic matter than in fresh water or sea water.

Simple formulae for estimating the time of death are now regarded as naive. These include the formula of Simpson - "under average conditions the clothed body will cool in air at the rate of about 1.5°C an hour for the first 6 hours and average a loss of some 1°C for the first 12". Also the formula of Camps - "probably the best rough estimate is afforded by the formula 98.4 minus To/1.5 = number of hours dead up to six hours, based upon skin and rectal readings, whilst corrections must be made for readings taken under the liver". Knight devised a formula in which the fall in temperature in degrees Celsius was multiplied by a factor of 1, 11/4, 11/2, 1 3/4 or 2 for air temperatures of zero, 5, 10, 15, or 20°C respectively. His own experience with this formula has shown serious errors and he now no longer recommends it.

The best researched and documented method for assessing time of death from body temperature is that of Henssge. This is a nomogram method rather than a formula. The nomogram corrects for any given

environmental temperature. It requires the measurement of deep rectal temperature and assumes a normal temperature at death of 37.2°C.

Henssge's nomogram is based upon a formula which approximates the sigmoid shaped cooling curve. This formula has two exponential terms within it. The first constant describes the post mortem plateau and the second constant expresses the exponential drop of the temperature after the plateau according to Newton's law of cooling. Introducing more than two exponential terms complicates the theoretical model without producing better results in practice. In an individual case, the constant expressing the exponential drop of the temperature after the plateau is simply calculated from the body weight. The first constant which describes the post mortem temperature plateau was found to be significantly related to the second constant in that bodies with a low rate of cooling, (i.e. having a high body weight) also had a longer plateau phase than bodies with a high rate of cooling, (i.e. a low body weight). Using previously published data which establishes that the relative length of the post mortem temperature plateau depends upon the environmental temperature but is non-linear and pronounced in environmental temperatures above 23°C, Henssge evolved two nomograms, the one for ambient temperatures above 23°C and the other for ambient temperatures below 23°C. Within each of these two nomograms there is a differing allowance for the effect of environmental temperature on the rate of cooling as well as an allowance for the effect of body weight.

It is well recognised that the presence of layers of clothing, wetting of the clothing, and air movement, all influence the rate of body cooling. Similarly, bodies in still and flowing water cool more rapidly than in air. Henssge conducted experiments and derived empiric corrective factors to allow for the effect of these variables (see back of nomogram sheet which reproduces the data in his articles).

In using the nomogram, Henssge emphasised "You can use right rules, but get wrong results if the points of contact are wrong. The most important thing, and - certainly - often the most difficult one, is to analyse carefully the points of contact at the scene of crime. By using the nomogram you can quickly calculate some different times since death by taking some different points of contact as a basis. This is recommended if the points of contact are not closely defined and a range of any point of contact must be taken into account". By "point of contact" Henssge means one of the variable elements for which he has derived corrective factors. He specifically recommends "It is a good strategy to evaluate an upper and a lower limit of the mean ambient temperature which might be possible on the basis of both the ambient temperature actually measured and the probable changes of it". And, "The choice of a corrective factor of the body weight of any case is really only an approximation. It requires personal experience. ... Again it is a recommended strategy to select an upper and a lower corrective factor which might be possible". "It must be emphasised that this method cannot be used in every case. Under some circumstances (reproduced on the back of the nomogram provided) this method must not be used because the points of contact are really unknown."

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2. RIGOR MORTIS

Ordinarily, death is followed immediately by total muscular relaxation - primary muscular flaccidity - succeeded in turn by generalised muscular stiffening - rigor mortis. After a variable period of time rigor mortis passes off spontaneously to be followed by secondary muscular flaccidity. The first investigation of rigor mortis is attributed to Nysten in 1811.

No measurable shortening of muscle occurs during rigor mortis unless the muscles are subjected to tension. When rigor is fully developed, the joints of the body become fixed, and the state of flexion or extension of these joints depends upon the position of the trunk and limbs at the time of death. If the body is supine then the large joints of the limbs become slightly flexed during the development of rigor. The joints of the fingers and toes are often markedly flexed due to the shortening of the muscles of the forearms and legs. Since significant muscle shortening is not a normal concomitant of rigor, it is unlikely that rigor mortis would cause any significant change in the attitude adopted by the corpse at death. The view that the development of rigor mortis could produce significant movements of the body was promoted by Sommer, in about 1833, and the postulated movements became known as "Sommer's movements".

It is now accepted that movements of a corpse due to the development of rigor mortis can only occur in special circumstances, such as an extreme position of the body at the moment of death. If a body is moved before the onset of rigor then the joints will become fixed in the new position in which the body is placed. For this reason,

when a body is found in a certain position with rigor mortis fully developed, it cannot be assumed that the deceased necessarily died in that position. Conversely, if the body is maintained by rigor in a position not obviously associated with support of the body, then it can be concluded that the body was moved after rigor mortis had developed.

Rigor involves voluntary and involuntary muscles. Rigor of the myocardium should not be mistaken for myocardial hypertrophy. Likewise secondary muscular flaccidity of the atria and ventricles should not be mistaken for ante-mortem dilatation or interpreted as evidence of myocardial dysfunction. Involvement of the iris muscles means that the state of the pupils after death is not an indication of their ante-mortem appearance. Different degrees of rigor development may give rise to irregularity and inequality of the pupils. Contraction of the arrectores pilorum muscles during rigor may result in "goose-flesh" or "cutis anserina". The phenomenon is commonly seen in cases of drowning where it is thought to result from an agonal contraction of the muscles. Involvement of the walls of the seminal vesicles by rigor may lead to discharge of seminal fluid at the glans penis.

Rigor mortis results from a physico-chemical change in muscle protein, the precise nature of which is unknown. When the muscle tissue becomes anoxic and all oxygen dependent processes cease to function, then the level of ATP is maintained by anaerobic glycolysis which results in increasing levels of pyruvic and lactic acids. Eventually, the muscle glycogen is depleted, the cellular pH falls to around 6, and the level of ATP falls below a critical level beyond which rigor rapidly develops. Normally ATP inhibits the activation of the linkages between actin and myosin; a fall in the level of ATP allows the irreversible development of these linkages. In individuals who have been exhausted or starved before death, the glycogen stores in muscle are low, so that rigor may develop rapidly. Some authors have simplified the concept of the development of rigor mortis by taking the view that a fall in the muscle pH to around 6.6 - 6.3 results in coagulation of the actinomyosin.

Classically, rigor is said to develop sequentially, but this is by no means constant, symmetrical or regular. Ante-mortem exertion usually causes rigor to develop first in the muscles used in the activity. Typically, rigor is first apparent in the small muscles of the eyelids, lower jaw and neck, followed by the limbs, involving first the small distal joints of the hands and feet and then the larger proximal joints of the elbows, knees and the shoulders and hips. Shapiro has suggested that this apparent progression through the muscles of the body reflects the fact that although rigor begins to develop simultaneously in all muscles, it completely involves small masses of muscle much more rapidly than large masses. Consequently, differences in the sizes of the joints, and in the muscles which control them, determine the development of joint fixation by rigor and produce the observed pattern of progression in the body. It is generally accepted that rigor mortis passes off in the same order in which it develops. The forcible bending of a joint against the force of rigor results in tearing of the muscles and the rigor is said to have been "broken". Provided the rigor had been fully established, it will not reappear once broken down by force. In temperate climates rigor will typically start to disappear at about 36-48 hours after death. However, if the environmental temperature is high then the development of putrefaction may completely displace rigor within 9-12 hours of death. Accelerated putrefaction resulting from ante-mortem septicaemia may also lead to a rapid displacement of rigor.

There is great variation in the rate of onset and the duration of rigor mortis.

Niderkorn's (1872) observations on 113 bodies provides the main reference database for the development of rigor mortis and is commonly cited in textbooks. His data was as follows:

Number of Cases	Hours Post Mortem at which Rigor was Complete
2	2
14	3
31	4
14	5
20	6
11	7
7	8
4	9
7	10
1	11
1	12
2	13

In this series, rigor was complete in 14% of cases at 3 hours post mortem and this percentage had risen to 72% at 6 hours and to 90% at 9 hours. By 12 hours post mortem rigor was complete in 98% of cases. (Note that this data is presented in a somewhat confusing way in. Against the background of this data it can be readily appreciated that the generally quoted rule of thumb that rigor commences in 6 hours, takes another 6 to become fully established, remains for 12 hours and passes off during the succeeding 12 hours, is quite misleading.

The intensity of rigor mortis depends upon the decedent's muscular development; consequently, the intensity of rigor should not be confused with its degree of development. In examining a body both the degree (complete, partial, or absent) and distribution of rigor should be assessed after establishing that no artefact has been introduced by previous manipulation of the body by other observers. Attempted flexion of the different joints will indicate the amount and location of rigor.

As a general rule when the onset of rigor is rapid, then its duration is relatively short. The two main factors which influence the onset and duration of rigor are

(a) the environmental temperature, and

(b) the degree of muscular activity before death. Onset of rigor is accelerated and its duration shortened when the environmental temperature is high.

If the temperature is below 10°C it is said to be exceptional for rigor mortis to develop, but if the environmental temperature is then raised, rigor mortis is said to develop in a normal manner. Rigor mortis is rapid in onset and of short duration after prolonged muscular activity, e.g. after exhaustion in battle, and following convulsions. Conversely, a late onset of rigor in many sudden deaths might be explained by the lack of muscular activity immediately prior to death.

In addition to these two principal factors, other endogenous and environmental factors are claimed to influence the onset of rigor. Onset is relatively more rapid in children and the aged than in muscular young adults. It develops early and passes quickly in deaths from septicaemia or from wasting diseases. It is delayed in asphyxial deaths, notably by hanging or carbon monoxide poisoning, and also when death has been immediately preceded by severe haemorrhage.

The opinion of Knight that "it is extremely unsafe to use rigor at all in the estimation of time since death" is somewhat extreme. However, the rule of thumb offered by Camps is overly simplistic - "corpses can usually be divided into those, still warm, in which no rigor is present, indicating death within about the previous three hours. Those in which rigor is progressing, where death probably occurred between 2 and 9 hours previously; and those in which rigor is fully established, showing that death took place more than 9 hours previously". Knight states that "the only possible use is in the period around the second day, when body temperature may have dropped to environmental but putrefaction has not yet occurred. If full rigor is present, then one might assume that this is about the second day following death, depending upon the environmental conditions".

Exposure of a body to intense heat results in heat stiffening due to coagulation of the muscle proteins. Unlike rigor mortis, heat stiffening is associated with muscle shortening resulting in the characteristic pugilistic posture of burned bodies. Heat stiffening obscures rigor mortis with which it should not be confused. Freezing of a body will cause stiffening of the muscles, postponing the development of rigor which is said to develop as soon as thawing of the body permits.

Cadaveric spasm (synonyms: instantaneous rigor, instantaneous rigidity, cataleptic rigidity) is a form of muscular stiffening which occurs at the moment of death and which persists into the period of rigor mortis. Its cause is unknown but it is usually associated with violent deaths in circumstances of intense emotion. It has medico-legal importance because it records the last act of life. Cadaveric spasm may affect all the muscles of the body but it most commonly involves groups of muscles only, such as the muscles of the forearms and hands. Should an object be held in the hand, then cadaveric spasm should only be diagnosed if the object is firmly held and considerable force is required to break the grip. Cadaveric spasm involving all the muscles of the body is exceedingly rare and most often described in battle situations.

Cadaveric spasm is seen in a small proportion of suicidal deaths from firearms, incised wounds, and stab wounds, when the weapon is firmly grasped in the hand at the moment of death. In such circumstances the gripping of the weapon creates a presumption of self-infliction of the injuries. This state cannot be reproduced after death by placing a weapon in the hands. It is also seen in cases of drowning when grass, weeds, or other materials are clutched by the deceased. In this circumstance, it provides proof of life at the time of entry into the water. Similarly, in mountain fatalities, branches of shrubs or trees may be seized. In some homicides, hair or clothing of the assailant may be found in the hands of the deceased.

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3. LIVOR MORTIS (HYPOSTASIS, POST MORTEM LIVIDITY, POSTMORTEM SUGGILLATIONS)

Lividity is a dark purple discolouration of the skin resulting from the gravitational pooling of blood in the veins and capillary beds of the dependent parts of the body following cessation of the circulation. The process begins immediately after the circulation stops, and in a person dying slowly with circulatory failure, it may be pronounced very shortly after death. Lividity is present in all bodies, although it may be inconspicuous in some and thus escape notice.

Lividity is able to develop post mortem under the influence of gravity because the blood remains liquid rather than coagulating throughout the vascular system. Within about 30-60 minutes of death the blood in most corpses, dead from natural or non-natural causes, becomes permanently incoagulable. This is due to the release of fibrinolysins, especially from small calibre vessels, e.g. capillaries, and from serous surfaces, e.g. the pleura. Clots may persist when the mass of clot is too large to be liquified by the fibrinolysin available at the site of clot formation. In some deaths associated with infection and cachexia, this fibrinolytic effect may fail to develop, explaining the presence of abundant clot in the heart and large calibre vessels. Thus, in cases of sudden death the blood remains spontaneously coagulable only during a brief period immediately following death; it then becomes completely free from fibrinogen and will never again clot. This incoagulability of the blood is a

commonplace observation at autopsy. The fluidity of the blood is not characteristic of any special cause or mechanism of death although many texts state that the blood remains liquid longer in asphyxial deaths.

The bluish colour of post mortem lividity does not have the same connotation as cyanosis produced during life. The term "cyanosis", which means a bluish discolouration of the skin or mucous membranes, should be confined to clinical descriptions and not used for corpses. In the living, the cyanotic colour of the blood requires the presence of at least 5 g of reduced haemoglobin per cent in the capillary blood. However, in the corpse, oxygen dissociation continues and there may be reflux of deoxygenated venous blood into the capillaries. For these reasons, the blood of a cadaver becomes purplish-blue, but this is not the result of a pathophysiological change occurring during life, e.g. strangulation. The normal colour of areas of post mortem lividity is a cyanotic hue, but this description should not be used since it is misleading.

The medico-legal importance of post mortem lividity lies in its colour and in its distribution. The development of lividity is too variable to serve as a useful indicator of the time of death.

Typically, lividity has a purple or reddish-purple colouration. Lividity in bodies exposed to the air may acquire a pink colour at the sides, but not, as rule, at the back or other areas which are close to the ground. In deaths from carbon monoxide poisoning, it is classically described as "cherry red"; in cases where methaemoglobin is formed in the blood during life (e.g. potassium chlorate, nitrates, and aniline poisoning) it appears chocolate brown; in deaths from exposure to cold, it is bright pink, and a similar colouration is seen in bodies refrigerated very soon after death. Refrigeration of a body already displaying typical purple lividity will cause it to turn pink. Similarly, lividity in parts of the body covered with moist clothes appears pink, whereas it is the usual purple colour in other areas. Cyanide poisoning results in lividity which is described by different authors as pink, bright scarlet, and violet.

Lividity is first apparent about 20-30 minutes after death as dull red patches or blotches which deepen in intensity and coalesce over the succeeding hours to form extensive areas of reddish-purple discolouration. Slight lividity may appear shortly before death in individuals with terminal circulatory failure. Conversely, the development of lividity may be delayed in persons with chronic anaemia or massive terminal haemorrhage. After about 10-12 hours the lividity becomes "fixed" and repositioning the body, e.g. from the prone to the supine position, will result in a dual pattern of lividity since the primary distribution will not fade completely. Fixation of lividity is a relative, rather than an absolute, phenomenon, but nevertheless, well developed lividity fades very slowly and only incompletely. Fading of the primary pattern of lividity and development of a secondary pattern of lividity will be quicker and more complete if the body is moved within, say, the first six hours after death, than at a later period. Even after 24 hours, moving the body will result in a secondary pattern of lividity developing. Duality of the distibution of lividity is important because it shows that the body had been moved after death. However, the timing of this movement of the body is inexact. Polson claims "it shows that the body had been moved ... within 8 to 12 hours". Camps states more convincingly that "for the hypostasis of have value in this way, the body must have first remained in one position for a length of time, perhaps about 10 hours, sufficient for the lividity to have become well developed and it must then be examined early enough after being moved before much of the hypostasis has become redistributed". The blanching of post mortem lividity by thumb pressure indicates that the lividity is not fully fixed.

Pressure of even a mild degree is sufficient to prevent gravitational filling of the vessels and this is so in the compressed areas of skin in contact with the underlying supporting surface. The result is that these compressed areas of "contact flattening" also show "contact pallor" (or "pressure pallor"). A supine corpse will display contact pallor over the shoulderblades, buttocks, calves and heels. Other areas of contact pallor will correspond with the location of firm fitting clothing, e.g. elasticated underwear, belts and collars, and any firm object lying beneath the body, e.g. the arm of the decedent. Thus, the distribution of lividity depends upon the position of the body after death.

Within intense areas of lividity, the accumulated blood may rupture small vessels to produce a scattering of punctate purple-black haemorrhages between one and several millimetres in diameter. These haemorrhages are seen most commonly over the lower legs of victims of suicidal hanging with complete suspension. These haemorrhagic loci should be distinguished from ante-mortem petechial haemorrhages.

Lividity is usually well marked in the earlobes and in the fingernail beds. In a supine corpse there may be isolated areas of lividity over the front and sides of the neck resulting from incomplete emptying of superficial veins. If the head is slightly flexed on the neck, then lividity may have a linear distribution corresponding to the

skin folds. Isolated patches of hypostasis may be due to blood in the deeper veins being squeezed, against gravity, to the skin surface by the action of muscles developing rigor mortis.

Differentiation of lividity from bruising can be made by incising the skin. In areas of lividity the blood is confined to the dilated blood vessels whilst, in areas of bruising, the blood infiltrates the tissues and cannot be readily washed away under running tap water. Microscopic examination will resolve any doubts and provide a permanent record. In a decomposing body it may be impossible to definitively distinguish between livid staining of the tissues and a putrefying area of bruising. Areas of lividity are overtaken early in the putrefactive process. The red cells haemolyse and the haemoglobin diffuses into the surrounding tissues where it may undergo secondary changes such as sulphaemoglobin formation. In bruised areas similar putrefactive changes occur and it may be impossible to determine whether the pigment in the stained putrefied area originated from an originally intravascular or extravascular collection of blood, i.e. from a patch of congestion or from a bruise.

Lividity occurs in the viscera as well as the skin and this provides some confirmation of the external observations. In the myocardium lividity may be mistaken for an acute myocardial infarction, and in the lungs may be misdiagnosed as pneumonia. Livid coils of intestine may falsely suggest haemorrhagic infarction. Lividity developing in the viscera of a body lying prone and resulting in a purplish congestion of organs usually found pale at autopsy can be disconcerting to those unaccustomed to these changes.

Most texts agree that lividity attains its maximum intensity at around 12 hours post mortem, but there is some variation in descriptions of when it first appears, and when it is well developed. Adelson states that lividity "ordinarily becomes perceptible within 1/2 to 4 hours after death, is well developed within the next 3 or 4 hours, and attains its maximum degree between 8 and 12 hours post mortem". Polson states that "it varies in its time of onset, is ordinarily apparent within 1/2 to 2 hours after death, and its complete development is attained in from 6 to 12 hours". Camps states that it "first appears about 20-30 minutes after death as dull red patches which deepen, increase in intensity, and coalesce to form, within 6 to 10 hours, an extensive area of reddish-purple colour". Spitz and Fisher state that its "formation begins immediately after death, but it may not be perceptible for as much as two hours. It is usually well developed within 4 hours and reaches a maximum beween 8 and 12 hours. ... After 8 to 12 hours lividity becomes "fixed" and will remain where it originally formed". Simpson states that "it commences to develop within an hour or so of death, becoming marked in 5 or 6 hours".

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4. POSTMORTEM DECOMPOSITION (PUTREFACTION)

Putrefaction is the post mortem destruction of the soft tissues of the body by the action of bacteria and enzymes (both bacterial and endogenous). Tissue breakdown resulting from the action of endogenous enzymes alone is known as autolysis. Putrefaction results in the gradual dissolution of the tissues into gases, liquids and salts. The main changes which can be recognised in the tissues undergoing putrefaction are changes in colour, the evolution of gases, and liquefaction.

Bacteria are essential to putrefaction and commensal bacteria soon invade the tissues after death. The organisms most commonly found are those normally present in the respiratory and intestinal tracts, namely anaerobic spore-bearing bacilli, coliform organisms, micrococci, diphtheroids and proteus organisms. The marked increase in hydrogen-ion concentration and the rapid loss of oxygen in the tissues after death favour the growth of anaerobic organisms. The majority of the bacteria come from the bowel and Clostridium welchii predominates. Any ante-mortem bacterial infection of the body, particularly scepticaemia, will hasten the onset and evolution of putrefaction. Environmental temperature has a very great influence on the rate of development of putrefaction so that rapid cooling of the body following a sudden death will markedly delay its onset. In the temperate climate of the United Kingdom the degree of putrefaction reached after 24 hours in the height of summer may require 10 to 14 days in the depth of winter. A high environmental humidity will enhance putrefaction. Putrefaction is optimal at temperatures ranging between 70-100°F (21-38°C) and is retarded when the temperature falls below 50°F (10°C) or when it exceeds 100°F (38oC).

The rate of putrefaction is influenced by the bodily habitus of the decedent; obese individuals putrefy more rapidly than those who are lean. Putrefaction will be delayed in deaths from exsanguination because blood provides a channel for the spread of putrefactive organisms within the body. Conversely, putrefaction is more rapid in persons dying with widespread infection, congestive cardiac failure or anasarca. Putrefaction is accelerated when the tissues are oedematous, e.g. in deaths from congestive cardiac failure, and delayed when

the tissues are dehydrated. It tends to be more rapid in children than in adults, but the onset is relatively slow in unfed new-born infants because of the lack of commensal bacteria. Whereas warm temperatures enhance putrefaction, intense heat produces "heat fixation" of tissues and inactivates autolytic enzymes with a resultant delay in the onset and course of decomposition. Heavy clothing and other coverings, by retaining body heat, will speed up putrefaction. Rapid putrefactive changes may been seen in corpses left in a room which is well heated, or in a bed with an electric blanket. Injuries to the body surface promote putrefaction by providing portals of entry for bacteria and the associated blood provides an excellent medium for bacterial growth.

After normal burial, the rate at which the body decomposes will depend to a large extent on the depth of the grave, the warmth of the soil, the efficiency of the drainage, and the permeability of the coffin. The restriction of air, in deep burials, particularly in clay soil, will retard decomposition, but never prevent it altogether. Buried in well drained soil, an adult body is reduced to a skeleton in about 10 years, and a child's body in about 5 years. Immersion of the body in faeces-contaminated water, such as sewage effluent will enhance putrefaction; however, it is generally accepted that in the first 48 hours after death changes are in the main due to organisms already present in the body. According to an old rule of thumb (Casper's dictum) one week of putrefaction in air is equivalent to two weeks in water, which is equivalent to eight weeks buried in soil, given the same environmental temperature.

Typically, the first visible sign of putrefaction is a greenish discolouration of the skin of the anterior abdominal wall. This most commonly begins in the right iliac fossa, i.e. over the area of the caecum, (where the contents of the bowel are more fluid and full of bacteria), but occasionally, the first changes are peri-umbilical, or in the left iliac fossa. The discolouration, due to sulph-haemoglobin formation, spreads to involve the entire anterior abdominal wall, and then the flanks, chest, limbs and face. As this colour change evolves, the superficial veins of the skin become visible as a purple-brown network of arborescent markings, which tend to be most prominent around the shoulders and upper chest, abdomen and groins. This change, owing to its characteristic appearance, is often described as "marbling". The skin, which now has a glistening, dusky, reddish-green to purple-black appearance, displays slippage of large sheets of epidermis after any light contact with the body, e.g. during its removal from the scene of death. Beneath the shed epidermis is a shiny, moist, pink base which dries, if environmental conditions permit, to give a yellow parchmented appearance. This putrefactive "skin-slip" superficially resembles ante-mortem abrasions and scalds. Indeed, post mortem scalding of a body with water at 65°C (149°F) produces skin slip of the same type as in putrefaction.

Subsequently, skin blisters varying in size from less than 1 cm to between 10 and 20 cm in diameter develop. These blisters are filled with dusky, sanguinous fluid and putrid gases. They burst on the slightest contact leaving the same slippery, pink base which underlies skin-slip. Putrid gas formation also occurs in the stomach and intestines causing the abdomen to distend and become tense. The increased pressure within the torso causes a purge of putrid, blood-stained fluid from the nose, mouth and vagina, and expulsion from the rectum of similar fluid admixed with faeces. Gas formation within the tissues causes generalised swelling of the body which is crepitant on palpation. The distention is greatest where the tissues are loose, particularly involving scrotum, penis, labia majora, breasts, and face. The gases produced include hydrogen sulphide, methane, carbon dioxide, ammonia and hydrogen. The offensive odour is caused by some of these gases and by small quantities of mercaptans.

The dusky, greenish-purple face appears bloated with the eyelids swollen and tightly closed, the lips swollen and pouting, the cheeks puffed out, and the distended tongue protruding from the mouth. The head hair and other body hair is loose at its roots and can be easily pulled out in large clumps. The finger and toenails detach, often with large sheets of contiguous epidermis forming complete "gloves" or "socks" - a process described as "degloving". The neck, trunk and limbs are massively swollen, giving a false impression of gross obesity. Finally, the putrid gases, which are under considerable pressure, find an escape and the whole mass of decomposing soft tissues collapses.

Putrefaction progresses internally beginning with the stomach and intestine. The gastric mucosa and the intestines are discoloured a brownish-purple. The mucosa of the airways is a deep red and there is haemolytic plum-coloured staining of the endocardium and the vascular intima which is most readily appreciated in the aorta and its major branches. Small white granules - so-called "miliary plaques" - are seen rarely over the endocardium and epicardium. The heart becomes flabby, the wall thinned, and the myocardium a deep dirty red. A similar discolouration is seen in the liver and kidneys. The spleen becomes mushy and friable. The liver develops a honey-comb pattern resulting from gas formation and similar changes may be seen in the brain, most readily if it is fixed in formaldehyde prior to cutting. Subsequently the brain becomes semi-liquid. The lungs, loaded with sanguinous fluid, appear dark red and are friable. Gradually a great part of this sanguinous fluid is lost by diffusion into the pleural cavities. Diffusion of bile pigments from the gall bladder discolours the adjacent liver,

duodenum and transverse colon. The capsules of the liver, spleen and kidneys resist putrefaction longer than their parenchymatous tissues with the result that these organs are often converted into bags of thick, turbid, diffluent material. Progression of decomposition is associated with organ shrinkage. The more dense fibro-muscular organs such as the prostate and uterus remain recognisable until late in the process, thus aiding in the identification of sex.

Perforation of the fundus of the stomach or lower oesophagus into the left pleural cavity or the abdomen may occur within a few hours of death. This is the result of autolysis rather than bacterial putrefaction. An uncommon finding, it is most frequently associated with cerebral injuries and terminal pyrexias. It is occasionally characterised as "neurogenic perforation of the oesophagus".

There is considerable variation in the time of onset and the rate of progression of putrefaction. As a general rule, when the onset of putrefaction is rapid then the progress is accelerated. Under average conditions in a temperate climate the earliest putrefactive changes involving the anterior abdominal wall occur between 36 and 72 hours after death. Progression to gas formation occurs after about one week. The temperature of the body after death is the most important factor generally determining the rate of putrefaction. If it is maintained above 26°C (80°F) after death then putrefactive changes become obvious within 24 hours and gas formation will be seen in about 2-3 days.

The putrefactive changes which have taken place up to this time are relatively rapid when contrasted with the terminal decay of the body. When the putrefactive juices have drained away and the soft tissues have shrunk, the speed of decay is appreciably reduced.

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5. ADIPOCERE

Saponification or adipocere formation is a modification of putrefaction characterised by the transformation of fatty tissues into a yellowish-white, greasy, (but friable when dry), wax-like substance, with a sweetish rancid odour. Mant states that when its formation is complete it has a sweetish smell, but during the early stages of its production a penetrating ammoniacal odour is emitted and the smell is very persistent. It floats on water, and dissolves in hot alcohol and ether. When heated it melts and then burns with a yellow flame. Ordinarily it will remain unchanged for years.

Adipocere develops as the result of hydrolysis of fat with the release of fatty acids which, being acidic, then inhibit putrefactive bacteria. The low (0.5%) level of free fatty acids in fat at the time of death may rise to 70% or more by the time adipocere is obvious to the naked eye. However, fat and water alone do not produce adipocere. Putrefactive organisms, of which Clostridium welchii is most active, are important, and aipocere formation is facilitated by post mortem invasion of the tissues by endogenous bacteria. A warm, moist, anaerobic environment thus favours adipocere formation. It was once thought that adipocere required immersion in water or damp conditions for its development. However, the water content of a body may be sufficient in itself to induce adipocere formation in corpses buried in well sealed coffins.

Adipocere develops first in the subcutaneous tissues, most commonly involving the cheeks, breasts and buttocks. Rarely, it may involve the viscera such as the liver. The adipocere is admixed with the mummified remains of muscles, fibrous tissues and nerves. The final product is of a larger bulk than the original fat with the result that external wounds may become closed and the pattern of clothing or ligatures may be imprinted on the body surface.

Under ideal warm, damp conditions, adipocere may be apparent to the naked eye after 3-4 weeks.Ordinarily, adipocere formation requires some months and extensive adipocere is usually not seen before 5 or 6 months after death. Other authors suggest that extensive changes require not less than a year after submersion, or upwards of three years after burial.

The medico-legal importance of adipocere lies not in establishing time of death but rather in its ability to preserve the body to an extent which can aid in personal identification and the recognition of injuries. The presence of adipocere indicates that the post mortem interval is at least weeks and probably several months.

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6. MUMMIFICATION

Mummification is a modification of putrefaction characterised by the dehydration or dessication of the tissues. The body shrivels and is converted into a leathery or parchment-like mass of skin and tendons surrounding the bone. The internal organs are often decomposed but may be preserved. Skin shrinkage may produce large artefactual splits mimmicking injuries. These are particularly seen in the groins, around the neck, and the armpits.

Mummification develops in conditions of dry heat, especially when there are air currents, e.g. in a desert or inside a chimney. New-born infants, being small and sterile, commonly mummify. Mummification of bodies of adults in temperate climates is unusual unless associated with forced air heating in buildings or other man-made favourable conditions.

The forensic importance of mummification lies primarily in the preservation of tissues which aids in personal identification and the recognition of injuries. The time required for complete mummification of a body cannot be precisely stated, but in ideal conditions mummification may be well advanced by the end of a few weeks.

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7. MACERATION

Maceration is the aseptic autolysis of a foetus which has died in utero and remained enclosed within the amniotic sac. Bacterial putrefaction plays no role in the process. The changes of maceration are only seen when a still-born foetus has been dead for several days before delivery. Normally the changes take about one week to develop.

Examination of the body needs to be prompt since bacterial putrefaction will begin following delivery. The body is extremely flaccid with a flattened head and undue mobility of the skull. The limbs may be readily separated from the body. There are large moist skin bullae which rupture to disclose a reddish-brown surface denuded of epidermis. Skin slip discloses similar underlying discolouration. The body has a rancid odour but there is no gas formation.

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8. VITREOUS HUMOUR POTASSIUM

The relationship between the rise of potassium concentration in the vitreous humour and the time since death has been studied by several workers and recently reviewed by Madea et al., An obstacle to using potassium concentration in vitreous humour as an aid in estimating the time since death are the different 95% confidence limits given by different authors. Up to 100 hours post mortem, the 95% confidence limits of different authors vary between ± 9.5 hours up to ± 40 hours; in the early post mortem interval up to 24 hours, the 95% confidence limits of different authors varies from ± 6 hours up to ± 12 hours. There are also sampling problems in that the potassium concentration may differ significantly between the left and right eye at the same moment in time. Simultaneous sampling of both eyes has shown that the potassium concentration in one eye can deviate by up to 10% from the mean value of both eyes. In order to improve the accuracy of the method cases with possible ante-mortem electrolyte disturbances can be excluded by eliminating all cases with a vitreous urea above an arbitrary level of 100 mg/dl. (High urea values in vitreous humour always reflect ante-mortem retention and are not due to post mortem changes). Having eliminated these cases with possible ante-mortem electrolyte imbalance, there is a linear relationship between potassium concentration and time after death up to 120 hours, but the 95% confidence limits are ± 22 hours

What is rigor mortis?

The initial flaccidity of the body that occurs after death is replaced by rigor mortis, which commences in the first few hours, and is more noticeable initially in the smaller muscles such as those around the jaws and fingers. It is caused by the overlapping actin and myosin filaments in the muscle fusing as the energy supplies in the muscle run out, leading to muscle stiffness and loss of elasticity. Rigor mortis becomes established by nine to twelve hours, and starts to wear off after twenty four to thirty six hours as the protein in the muscle starts to break down.

Why does rigor mortis progress downwards?

Abstract

Determinants of the downward sequence of rigor mortis are not clear, although rigor mortis is an important postmortem change referred to by forensic pathologists in estimating the postmortem interval. We determined postmortem changes in the tension of some muscles mounted in liquid paraffin at 37°C or 25°C, and found that the course of rigor mortis was affected by the proportion of muscle fiber types in the muscles and by temperature. These factors could be causes of the downward progress of rigor mortis.

Introduction

Rigor mortis is one of the important postmortem changes which forensic pathologists refer to in estimating postmortem interval. Nysten first reported the downward progress of rigor mortis in human cadavers in 1812. Many forensic pathologists have verified this tendency, although it may not be applicable to all cadavers. Some hypotheses about the determinants of the downward progress of rigor mortis have been proposed. The most famous hypothesis among these was Shapiro's theory in 1950. He suggested that rigor mortis would occur much more rapidly in small muscles than in large muscles because rigor mortis progressed similarly in all muscles. Therefore, he thought that a relatively small joint, such as the temporomandibular joint, surrounded by a moderate amount of muscle would become immobilized sooner than a large joint surrounded by a relatively large mass of muscle. When he proposed this hypothesis, little was known about muscle. Therefore his persuasive hypothesis was completely based on his own experiences and imagination. Since Shapiro's proposal, mechanisms of muscle contraction have been progressively clarified. For example, depletion of adenosine triphosphate (ATP) is now regarded as the main cause of rigor mortis. However, no one has experimentally tested Shapiro's hypothesis. We studied the sequence of rigor mortis. In this paper, we review our findings and propose possible determinants of the sequence.

First, we examined levels of adenosine nucleotides and lactic acid in rat five kinds of muscles removed immediately after death from one side of rats, and 2 hours after death from the other side of the same rats, to investigate whether the postmortem biochemical changes in the muscle would progress similarly in all the muscles as Shapiro's theory suggested. The results indicated that the postmortem changes in rat muscle were different between muscles. Next, we investigated determinants of the difference in postmortem changes between muscles, thinking that the proportion of muscle fiber types might influence rigor mortis.

Muscle fibers are divided into several types, mainly by enzyme histochemistry. Each type has its own metabolic, morphological, and physiological features as Table 1 shows. Muscles consist of a mosaic of these types of fibers as shown in Fig. 1 (above and to the left). In rats, red muscles consist mainly of Type I or IIA muscle fibers. Type IIB fibers predominate in white muscles. The proportion of fiber types differs according to the kind or part of the muscle. However, the proportion in a kind or part of muscle does not differ very much between individuals. For example, soleus is a representative of red muscles, in which Type I muscle fibers predominate, for almost all individuals.

In order to compare postmortem changes of several muscles, we examined muscles from rat cadavers. To do this we developed an experimental model of postmortem skeletal muscle which was soaked in liquid paraffin to prevent drying and diffusion across the muscle surface. Postmortem changes in tension, one index of the dynamic changes in rigor mortis, of the muscle samples were determined at 37°C or 25°C.

Materials and methods

Muscle samples

Nine-month-old male Sprague-Dawley rats were used in the experiments. They were injected with a total of 200-250 mg/kg mephenesin intraperitoneally and movement of all skeletal muscles except those used in breathing was stopped for 30 min to exclude the influence of antemortem muscle exercise. The rats were then killed by deep anaesthesia with diethyl ether. Red gastrocnemius (RG), white gastrocnemius (WG), soleus (SO), and erector spinae (ES) muscles were used. RG and WG are the deepest and most superficial portions of the medial head of the gastrocnemius muscle, respectively. The proportions of muscle fiber types in the muscles were determined with ATPase staining.

The muscles were removed immediately after the rats were killed. Strip sections 1.0-1.2 cm long and 0.4 cm thick were cut parallel to the muscle fibers and mounted in liquid paraffin.

Measurement of rigor mortis

The preparations were mounted vertically in a 20 ml water-jacketed tissue bath, containing liquid paraffin (Wako, Osaka, Japan), and the temperature was kept at 37°C or 25°C. ES was tested only at 37°C. The lower end of the preparation was attached to the bottom of the bath, the other end was connected to an isometric sensor (Star Medical, IM-20BS, Tokyo, Japan) by silk threads. The isometric tension was recorded (Graphtec, Thermal arraycorder WR 7300, Yokohama, Japan) using a preamplifier (Star Medical PA-001, Tokyo, Japan). The first 10 min after death were used to prepare the samples and to set up the apparatus. No measurements were made during this time. At the start of the measurements, the muscle was stretched by 50 mN to tense the thread and enable sensitive measurement of the tension. Change of tension was measured and recorded for 8 h for each sample.

Statistical analysis

There were four samples of each muscle at each temperature. For each sample the measured tension was expressed relative to the maximum tension found during the 8 h of measurement . Factorial analysis of variance (ANOVA) was used to determine significant differences in the postmortem interval at which the tension reached its peak. In addition, we analyzed the times at which the tension increased to 10%, 50% and 100% of the maximum and the times in which the tension decreased from 100% to 70% and 50% at 37°C. Spearman's correlation coefficients (two-tailed) between the times and the average proportion of Type IIB muscle fibers in the muscles were calculated.

Results

RG and SO were regarded as red muscles in which Type I or IIA red muscle fibers predominated, and WG and ES were white muscles, as shown in Table 2. The tension was recorded as demonstrated in Fig. 2. In all muscle samples, tension increased and then decreased over the 8 h postmortem period.

The tension reached a peak sooner in RG and SO than in ES, and earlier in ES than in WG. The time course of the progress of rigor mortis was faster in red muscles than in white muscles at 37°C as the significant positive correlation between the postmortem intervals in which rigor tension reached 10%, 50% and 100% of the maximum and the areal proportion of Type IIB white muscle fiber demonstrated. However, resolution of rigor mortis was faster in white muscles than in red muscles as the significant negative correlation indicated.

The progress of rigor mortis was slower at 25°C than at 37°C. Tension reached a peak sooner in RG than in SO, and earlier in SO than in WG at 25°C, and the resolution was fast in WG at 25°C. Thus, at 25°C the tendency that rigor mortis in red muscles progressed fast and resolved slowly was also seen at 37°C.

Discussion

The effect of muscle fiber types and temperature on rigor mortis

Rigor mortis progressed more rapidly in red muscles than in white muscles as the positive correlation between time for rigor mortis to progress and areal proportion of Type IIB white fibers demonstrates. Differences in the rigor mortis between these muscles are reflected by differences in rigor mortis between the predominant muscle fibers, but the cause of rapid progress of rigor mortis in red muscle fiber is unknown. Depletion of ATP, which facilitates rigor mortis, would be faster in red muscle than in white muscle. It is possible that postmortem production of ATP would be less in red muscle than in white muscle because red muscle fibers contain less glycogen than white muscle fibers.

Resolution of rigor mortis was faster in white muscles, in which rigor mortis progressed slowly, than in red muscles. Rigor mortis could be resolved by proteases which would be activated by high levels of intracellular Ca^{2+}. However, it is not known whether postmortem levels of Ca^{2+} are different between red and white muscle. More experiments are needed to investigate this. It is well-known that temperature influences the progress of rigor mortis. This was also demonstrated in our experiments where rigor mortis progressed and resolved much faster at 37°C than at 25°C.

Rigor mortis in human cadavers

Type I red muscle fibers in human masticatory muscles are unique and different from ones in other muscles. Type I muscle fibers have about twice the diameter of Type II white fibers in human masticatory muscles. This feature is not seen in the other human muscles or rat masticatory muscles. The areal proportion of Type I muscle fibers in human masticatory muscles is high in spite of their low proportion numerically. Therefore, rigor mortis in human masticatory muscles would progress faster than in the other muscles. This would lead to early fixing of the temporomandibular joint in human cadavers.

Distal extremities cool fast after death. This would result in the relatively slow progress of rigor mortis in the distal extremities. Therefore, the effect of temperature might be one of the determinants in the sequence of rigor mortis.

Moreover, we think that differences in morphological and dynamic features between joints might strongly affect the speed of progress of rigor mortis. In particular, the temporomandibular joint has a special form. The distance between the joint and the point in the mandible where the masseter or temporalis muscle is attached is relatively long compared with the length of the bone. This characteristic of the temporomandibular joint will result in early fixing of the joint by rigor mortis. However, we have not examined in detail the morphological differences between joints, and this must be analyzed to know whether this is one of the determinants of the sequence of rigor mortis.

We know that Nysten's law is not applicable to all human cadavers, but it is possible to say that rigor mortis tends to progress downwards. Especially, the onset of rigor mortis in the temporomandibular joint is usually earlier than in the other joints of the extremities. The determinants of this tendency would be the differences in the morphological characteristics in the joints, the proportion of muscle fiber types, and postmortem changes in temperature between muscles. Through scientific evidence we must clearly demonstrate these factors as being the significant determinants. Moreover, we investigated only postmortem changes in the tension of muscles, but this is only one of the indexes of rigor mortis. Synthetic analysis of rigor mortis is under investigation through determination of postmortem changes in stiffness, another index, and phosphates including ATP and creatine phosphate.

Conclusion

Rigor mortis progressed more rapidly in red muscles than in white muscles, and more rapidly at 37°C than at 25°C. The proportion of muscle fiber types and temperature might be determinants of the downward progress of rigor mortis in the human cadaver but more scientific evidence or experiments are needed for further clarification.

Establishing Time of Death (within the first day)

As a general rule body temperature decreases by 1 1/2 to 2 degree (f) per hour for the first 12 hours, then the temperature slows its cooling down to only 1 degree per hour. The ambient temperature is also taken, in cooler weather or water the temperature decreases rapidly, in warmer weather the body temperature decreases slower. Clothing on the body and any other coverings are also taken into account.