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# Time since Death from Rigor Mortis: Forensic Prospective



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#### Abstract

Time since death is extremely important topic of study in Forensic medicine. Time of death is a question almost invariably asked by investigating authorities to link the criminals with the crimes. Determining the time since death has always been a topic of keen interest amongst the forensic pathologists from its inception till date. Many workers in forensic medicine have tried to investigate to determine the time of death on the basis of post mortem findings. From the ancient era, the triad: Algor mortis, Rigor mortis and Liver mortis has been the basis for ascertaining the time since death collectively. Till date it is still the important and most fascinating criteria to ascertain the time since death. This assessment covers the importance of the time since death from rigor mortis in forensic science.

Keywords: Rigor Mortis; Postmortem; Death; Forensic Science; Criminals

## Introduction

Among this triad, Algor mortis is the cooling of body after death. The body temperature at the time of death is generally 37°C which falls to the temperature of surrounding by 12 to 18 hours after death. The liver mortis or postmortem staining generally appears over dependent parts of the body within ½ - 1 hour of death and gets fixed within 6 to 8 hrs after death. Similarly, the rigor mortis, which is cadaveric rigidity, starts developing within 1 to 2 hours after death and takes around 12 hours after death for complete development and remains in the developed stage for further 12 hours and disappears in the next 12 hours generally. This can give the approximate time since death till 36 hours after death. Many other sophisticated techniques like biochemical studies of C.S.F., Vitreous Humor, Aqueous humor, etc. have been tried to reach the accuracy regarding time since death.

The other methods such as study of decomposition changes and study of entomology are in practice to estimate time lapse after death. Since among these three criterions as described above, rigor mortis gives the longest estimation interval regarding the time since death, hence we had chosen this topic for the review purpose. Rigor mortis is the state of postmortem stiffening and some shortening of the muscles of the body, both voluntary and involuntary, following the period of primary flaccidity. It is due to kchemical changes affecting the proteins of muscles fibers that is acting and myosin. This is sign of the end of cellular life of the muscles [1].

# **History Of Rigor Mortis Findings**

The first investigation of rigor mortis is attributed to Nysten in 1811 [2]. He is the first to demonstrate rigor mortis. Bendell and Smith (1946) postulated the role of ATP in the onset of rigor mortis by their experimental studies on pso as major muscles of rats. This study has given the biochemical basis for development of rigor mortis [3]. In 1950, Shapiro demonstrated the sequential progression of development of rigor from the head downward. Further in 1960, Bendell studied the biophysics of muscular contraction, which led to a better understanding of rigor mortis. He stated that voluntary muscles consist of bundles of long fibers of dimension of a human hair. Each fibre is formed of densely packed myofibrils extending through its whole length. These myofibrils are the contractile elements, and they are made up of proteins filaments, myosin filaments and actin filaments [4]. Due to this concept, study of rigor got a microscopic approach. H. A. Husband (1877), Krompecher, Bergerioux (1988), Keith Simpson (1969), I. Gordon (1988) and many others has been the pioneer in understanding the various factors affecting rigor and its relation with the cause of death [5].

Huxley (1974), the separation of actin and myosin filaments [6]. The consequent extensibility and softness of the muscle and the energy needed for the contraction are all in one way or another dependent on adenosine triphosphate (ATP) [7]. This is in high concentration in resting muscles and its level, though constant, is always a balance. After death, the failure of re-synthesis of ATP led to a fall in its concentration within the muscles and accounts for the hardness and rigidity of muscles in rigor. The development of rigor mortis is a process of change in the intensity of muscular rigidity over time. Initially the muscular intensity of rigor mortis is weak; subsequently it increases and reaches to the maximum, then decreases and practically disappears after certain period of time. Rigor mortis firsts appear in the involuntary muscles and then in voluntary muscles. Rigor mortis starts appearing within 1-2hrs after death and takes around 10-12hrs for full development and remains steady for next 12hrs and then disappears in next 12hrs. Rigor is said to develop sequentially, but this is by no means constant, symmetrical or regular.

Typically it starts first in the small muscles of eyelids by approximately 1-2hrs of death and then progresses in the muscles of the lower jaw, neck, muscles of trunk, upper extremities and lower extremities [8]. Lastly the muscles of the fingers and toes are affected. The disappearance of rigor mortis is the same as its order of appearance. The nature of its gradual development, order of progress and ultimate disappearance give the rigor some striking characteristic. Developed rigor mortis once damaged by any means will never recur again in the body. The disappearance of rigor mortis denoted by flaccidity of muscles is caused by the action of alkaline liquids produced by putrefaction [9]. Since last two centuries, Rigor mortis is one of the important factors in determining time since death. Various authors studied this phenomenon the review of which is taken as follows. Nysten (1811) has published the first scientific description of rigor mortis [10].

According to him, no measurable shortening of muscles occurs during rigor mortis unless the muscles are subjected to tension. When rigor is fully developed, 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 development of rigor. The joints of the fingers and toes are often markedly flexed due to the shortening of the muscle of the forearm and the legs. Cadaveric rigidity affects successively the masticator muscles, those of the face and the neck, those of trunk and arms and finally those of the lower limbs. This after him called as Nysten Law. It is often added that resolution occurs in the same order. Sommer (1833) has promoted the view that the development of rigor mortis could produce significant movements of the body in special circumstances such as an extreme position of the body at the moment of death [11].

This postulation became known as "sommer's movement". But further studies criticize this view and give the justification that the body is moved before the onset of rigor then the joints will become fixed in which the body is placed. For this reason, when a body is found in certain position with the 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 body, then it can be concluded that the body was moved after the rigor mortis had developed. Husband H.A. (1877) Quoted in the Student's hand book of forensic medicine, the factors influencing the rigor mortis: Effect of enfeebling disease prior to death, rapid is the invasion and passes of rapidly, Effect of robust frame at period of death, the accession may be prolonged, Effect of violent exercise prior to death, rigor supervenes and disappears rapidly, Effect of poisons: The poisons which causes violent contraction for some time prior to death e.g. strychnine, influence the rapid invasion of rigor mortis, it's short duration and the subsequent putrefaction.

Where the death in poisoning by strychnine is almost instantaneous with a short convulsive stage, the rigor mortis comes on rapidly and remains a long time. Swenson Arne, Wend Otto and Fisher, Barry A.J. (1914) Stated that the biochemical changes in the body produce stiffening known as rigor mortis, which usually appears within 2-6 hrs [12]. After death. Rigor mortis begins in the muscles of the jaws and neck and proceeds then downwards in the body and trunk and extremities and it is completed within 6-12hrs. The rigidity remains for 2-3 days and disappear in the same order in which it appeared. The very young and very old will likely developed less rigidity than adults with well developed musculature. Wordsworth S. William (1916) [13]. Stated that rigor mortis or cadaveric rigidity is the result of coagulation of at least part of the muscle substance and it appears to start with the plasma, the clots become firmer and probably involves the other part of the muscles substance. It comes on quicker in the recently fatigued muscles.

It comes earlier in the smaller and more active muscles that have a smaller amount of plasma. Rigor depends on such physical conditions or influences as heat, electricity or the amount of fluids, it seems to depend far more on the chemical conditions specially fatigue stuffs and poisons. The period of rigidity varies to a considerable degree with that of relaxation usually lasting about 2 days, but the onset and fading being usually gradual, it is difficult to determine accurately. The passing of the rigidity usually begins in the smaller mobile muscle and follows the general order of onset both varying largely under local conditions. Aitcheson, Robertson Stated that rigor mortis consist in hardening and stiffening of the muscles of the whole body, which sets in some hours after death. Rigor mortis appears in a definite order affecting the various groups of muscles. The stiffening affects the muscles in the following order: the eye lids, the muscles of neck, lower jaw, the muscles of face, Chest, Upper extremities, Trunk, Lower extremities.

Rigor mortis passes away in the same order as it comes on. Time of onset varies greatly in different cases. But on an average it may be said to commence 2-4 hrs after death and to persist until decomposition comes on in from 1-3 days. He further state some conditions in which the rigor mortis to delay or appears early [14]. The death followed by convulsions, muscular exertion, racing, the rigor mortis will appear earlier. In wasting diseases like cancer, phthisis, rigor mortis will appear early. In newborn, the postmortem rigidity comes on very rapidly and passes off equally soon. In warm climate, the onset is rapid and so is the disappearance. The delayed onset of rigor mortis is seen in conditions like Sudden death while in previous good health (apoplexy, pneumonia etc), Asphyxia, Narcotic poisoning, it is even said to be absent in such cases. In paralyzed muscles it is long delayed. Curran William, Mcgnrry, and petty Charles 6 stated that the body normally begins to stiffen 2-3hrs after death [15].

It becomes apparent in the small muscle groups first and later progresses to larger muscles group in order of increasing mass. Complete stiffness of body usually occurs in 6-8hrs before receding in approximately the same sequence in which the stiffness occurred. They also stated that external factors produce marked changes in the onset and duration of rigor mortis like cold will delay the rigor while heat will hasten the rigor. Internal factor like in cases of infants rigor have rapid onset, emaciated built will delay the rigor and in extremely obese persons the rigor is totally absent. Owens T. F Reported that rigor appears first in the jaw and face, it rapidly spreads on the muscles of the neck and back then to the upper extremities and finally to the lower extremities. The time of onset varies somewhat but the rigor is generally present in the jaw by the ends of 2 hours after death, the upper extremities are probably rigid in 3 hours, and rigidity is complete in another hour. The order of disappearance as same as that of the onset and all trace of rigidity in the majority in the cases, disappeared in the20 to 24 hours after death (Table 1).

 Table 1: Postmortem At Which Rigor Was Complete (Hrs) Verses Cases.

Number of cases	postmortem at which rigor was complete (hrs)
2	2
14	3
31	4
14	5
20	6
11	7
7	8
4	9
7	10
1	11
1	12
2	13

Nidrekon cited by Owens TF Found rigor mortis fully developed before the end of the seventh hour after death in 92 out of 113 cases [16]. Mallach has given the chronology of development of rigor mortis based on the survey of literature from 1811-1960 i.e. 150 years as per following table where he compared and evaluated the observations statistically. The observations are highly subjective. He stated that the reasons for the great difference in the development of rigor mortis between one corpse and another is biological or more precisely biochemical and also it can be affected by number of intrinsic and extrinsic factors. He studied 108 publications on rigor mortis and evaluated them statically. He had shown that before rigor mortis there is a delay period for about 3 hrs and the complete rigidity is seen within 8 hrs and its persistence is around 57 hrs and resolution of rigor mortis is seen around the 76 hrs. Simpson Keith (1969) cited that rigor mortis results from stiffening of the fibers of cell muscle, voluntary and involuntary. The fibers shorten and stiffen, and all group of muscles become prominent and rigid fixing the limbs.

Rigor can be detected in the face in a 5- 7hrs and spreads during the next 2-3hrs to the shoulders and arms, reacting down to the trunk to involve the legs and becoming fully established by about 12hrs. It last for some 12hrs and takes further 12 or more hours to pass away, leaving the body in same order as it came. Further he stated that rigor might come early when metabolism is pronounced at death, as in fever and exercise or in summer, or it may fail to develop properly at any stage. It develops earlier in babies and disappears more quickly. John Cyril, Polson and Gee DJ stated that the rigor mortis is likely to be apparent at about 4hrs and complete about 6hrs. It may be complete at the end of 4 or even only 2hrs. and its completion may be delayed until the end of 10 or even 13hrs. It is reported first to appear in the eyelids, face, lower jaw and neck, subsequently it involves the trunk and limbs. It passes off in the same order and the body again becomes flaccid.

It is usually established in 6hrs and last about 36hrs but it may persist for much longer period. They also reported that the time of onset of rigor mortis is varied by several factors e.g. Atmospheric temperature, humidity and movements of air around the body but in general it is likely apparent of about 4hrs and complete in about 6hrs after death. It may be complete at the end of 4 or even only 2hrs. Its completion may be delayed until the end of 10 or even 13hrs. Rigor mortis is early and it's passing rapid in deaths from septicemia in from wasting diseases. Delay in the appearances of rigor mortis occurs when the body is exposed to cold and in any other circumstances, which delay purification e.g. poisoning by arsenic or mercuric chloride. Rigor mortis is delayed also in asphyxia death notably by hanging or carbon dioxide poisoning and when the death has been immediately preceded by severe hemorrhage. The violent exertion shortly before death, deaths caused by convulsing poisons, electrocutions, hastens the onset and passing off rigor mortis.

It is usually established in 6hrs and last about 36hrs but it may persist for a much longer period. Edgar Rantoul and Hamilton Smith Stated that the earlier manifestation of rigor mortis would found in the muscle of the eyelid and those of the lower jaw. Rigidity of the eyelids usually precedes that of the jaw muscles, which are affected in about 3 to 4hrs of death. The rigidity next appears progressively in the muscles of neck, the face, the thorax, the upper extremities, the trunk of the body, and the muscles of the lower extremities. Rigor mortis brings about slight shortening of the muscle fibers [17]. The general stiffening will be established after 10 to 12hrs. In majority of cases, rigor will have commenced to pass off in about 36hrs, but in cold weather and for other reasons a much longer interval may elapse. They also stated that stronger the muscularity of the person at the time of death, the later is the time of onset and longer the duration. HF Burton stated that the rigor mortis is best known of all signs of the death.

He also stated that the rigor is the most deceiving sign of the triad. Rigor seems to be a common occurrence in the bodies of the extremely obese, although it occurs occasionally in the senile, bony and emaciated. Rigor is enhanced by heat and retarded by cold. He also differentiated the rigor from the stiffness due to cold and stated that both are chemically different. Shapiro HA and Gordon I stated that order of appearances of rigor mortis in striated (voluntary) muscle as commencing in the muscles of neck and lower jaw and then spreading throughout the muscles of the body (Scheme 1). It is generally accepted that rigor mortis passes off in the same order in which it develops. When the environmental temperature is high, the onset of rigor mortis is accelerated and duration is short. A low temperature retards the onset and prolongs the duration of rigor mortis. R. Van Den Oever stated that the postmortem rigidity i.e. rigor mortis is dependent on age, sex, physical conditions, muscular built and muscular activity immediately ante mortem and directly connected with the environmental temperature.

Scheme 1: Spread of Rgor Mortis in Conventionally Represented In the Following below Diagram

Right	Face	Left
Hand <forearm<arm<neck &="" lower<br="">jawàArmàForearmàHand</forearm<arm<neck>		
Thorax		
Foot < Leg < Thigh < Abdomen Pelvis > Thigh> Leg> Foot		

He also stated that rigor mortis begins to develop on an average at 2 hours after death and reaches to its maximum extent in about 10-12 hours. The muscular mass of the corpse seems mainly to decide the moment of onset, the degree of complete rigor and the returning of the rigidity once it has been interrupted by an external force. He also stated that ATP and ADP shift plays a major role in the development of rigor mortis. Francis E. Camps stated that the rigor mortis is the result of chemical changes involving the proteins of the muscles fiber. When it fully develops in the skeletal muscles it assumes a board like rigidity and the forcible bending of the joint against the force of rigor actually tears the muscles and stiffness of the muscle never returns. Ordinarily the rigor mortis appears within 2-4 hours, but sometimes it is seen within 30 minutes of death and sometimes the onset is delayed for 6 hours or more. In human, temperature might have play a smaller role in the onset of rigor mortis because during the first 6 hours after death ordinarily

environmental temperature will have a small effect on the main muscles masses of adequately nourished and clothed bodies.

According to him the time since death can be estimated by assessing the condition of body, which is given in the following tabular form in (Table 2). Rigor mortis first appears in the muscles of eyelids and then spreads to the neck and upper limbs, trunk and lower limbs. Those muscles first to develop rigor are first to become flaccid again and rigor usually stays longest in the lower limbs. Rigor will not start to disappear for perhaps 36-48 hours after death and some rigidity will persist in the lower limbs for 3-5 days. However when the body temperature is kept raised or in warm room in a bed, or when the body is fat or well clothed, the complete displacement of rigor by putrefaction might takes place within 24 hours. Sengupta BK stated that rigor mortis affects all the muscles of the body-voluntary, involuntary, as well as cardiac muscles. It usually occurs whilst the body is cooling, in about1-2 hours after death and takes 1-2 hours more to spread all over the body, so that after about 4 hours after death, all muscles are stiff.

 Table 2: Time Since Death Can Be Estimated by Assessing the Condition of Body.

Condition of body	Time since death	
Warm but no rigor mortis	Died within 3 hours	
If rigor mortis present	Died between 3-9 hours probably	
Rigor mortis fully established	Died 9 hours previously	

It lasts for about 18-24 hours in summer and longer in winter. In the voluntary muscles it is first noted in the eyelids, next back of neck, and lower jaw, then the front of neck, face, chest upper limbs. It next spread downwards to the muscles of abdomen and lower limbs. Small muscles of hand and fingers are last to develop. Arne Svensson, Otto Wendel and Berry Fisher (1981):34 stated that after death the body becomes flaccid. Biochemical changes in the body produce stiffening, the rigor mortis that usually appears within 2 to 6 hours after death. Rigor mortis begins in the muscles of the jaw and neck and proceeds downwards in the body to the trunk and extremities and complete within 6 to 12 hours. The rigidity remains for 2 to 3 days and disappears in the same order in which it appeared. Guharaj PV stated that in rigor mortis, the muscle undergoes a decrease in both plastic and elastic deformation. Elasticity constantly decreases in rigor mortis whereas plastic deformation first increases and then decreases.

The initial increase in plastic deformation is attributed to the ATP content of the muscle. Decrease in elasticity is mainly responsible for rigor mortis. Classically rigor first appears in the small muscles of the eyelids and the jaw and then spreads to the muscles of the trunk and limbs. Joints surrounded by lesser amount of muscle mass become completely involved earlier than those surrounded by a large muscle mass. Thus the smaller joints such as the tempero-mandibular joint and those of fingers and toes are involved earlier than larger joints such as knees, elbows, shoulders, and hips. He further stated that the rigidity might commence on the eyelids 4-5 hours after death. Complete involvement of the body may require about 12 hours. It starts to pass off from the body after 36 hours (Table 3). AK Mant stated that the rigor mortis in sudden death usually commences within 2-4 hours. It reaches the peak in about 12 hours and starts to disappear within next 12 hours. The cadaver becoming limp 36 hours after death.

Postmortem changes	Time since death
Rigor on the jaw	More than 1 hour
Patchy mottling	More than 2 hours
Rigor not seen	Less than 3 hours
Postmortem changes	Time since death
Pupillary response to chemical stimuli present	Less than 4 hours
Hypostasis confluent, rigor fully established	More than 4 hours
Electrical excitability of muscles present	Less than 5 hours
Hypostasis fixed	More than 7 hours
Rigor progressing	Less than 9 hours
Body completely cooled, rigor disappearing	More than 16 hours

 Table 3: Sequential Changes after Death.

He also stated the conditions influencing the onset and disappearance of rigor mortis. Rigor mortis comes on slowly and uniformly in healthier subjects and the onset is rapid in case of deaths in exercises prior to death, convulsions, and sudden hemorrhage. He also stated that the rigor mortis is influenced by some external conditions like environments. In cold, the appearance and disappearance will be slower while in heat accelerate the rigor mortis in cases of deaths from lightning and electrocution the rigor mortis develops early and passes away early due to violent convulsions of muscles. The rigor mortis first appears in the muscles of eye, face, neck Trunk and then spreads to the muscles of upper extremities and lastly in the legs. Basu SC stated that rigor mortis first appears in the involuntary muscles of the hand and then in the voluntary group. Orbicularis oculi is the first voluntary muscle involved. The heart is affected an hour after death. Earlier it comes on, more quickly it disappears. Longer it takes to appear, longer it persists. Rigor mortis follows a definite order as it appears in the voluntary muscles (Table 4). Order of rigor mortis appearance

Condition of body	Time since death	
If body feels warm and flaccid	Dead less than 3 hours	
If body feels warm and is stiff	Dead from 3-8 hours	
If body feels cold and is stiff	Dead from 8-36 hours	
If body feels cold and flaccid	Dead more than 36 hours	

- a. Eyelids, lower jaw, face.
- b. Muscles of the back of neck and trunk
- c. Muscles of the abdomen and lower limbs

It disappears also in the same order with the commencement of third stage of cadaveric changes in muscles. He further stated the conditions which affect the onset of rigor mortis like in children and old people the onset is hastened or accelerated in feeble, fatigued and exhausted muscles and death from lightning, strokes, cholera, tetanus, opium and strychnine poisoning. While the onset delays in healthy adults and strong muscles also in apoplexy and asphyxia death. He also differentiated the rigor mortis from the heat stiffening. Heat stiffening is seen due to the coagulation of muscle albumin by heat. This is seen in death due to burns or when a body is suddenly dipped into the boiling fluid. The temperature must exceed 75 degree centigrade. Parekh CK quoted that every muscle in the body, voluntary and involuntary, takes part in the process of rigor mortis. Rigor first appears in the involuntary muscles and then in the voluntary muscles. It first appears in the muscles of eye then in the muscles of face, neck, trunk followed by muscles of upper extremities and then the legs and lastly muscles of fingers and toes.

It passes off in the same order in which it appeared. In India, it commences in 1-2 hours and takes 12 hours to develop and persist for another 12 hours and takes 12 hours to pass off. It is not depended on the nerve supply as it develops in the paralyzed limb also. It is tested by gently bending the various joints of the body. Rigor can be broken by mechanical force, if once broken the limb become flaccid and will remain so thereafter. He further quoted that the factors affecting the process of rigor mortis are:

a. Age and condition of the body:- In foetus rigor is rapid in onset and passes off quickly, rigor occurs for a short period in the still born bodies and occasionally occurs in a dead foetus before birth. In the early youth and old age the onset is earlier than in adult life. The onset is late and duration is longer in strong muscular persons. The more feeble or exhausted muscular conditions the onset is rapid and duration is shorter.

b. Mode of death: Rigor may appear early and pass of quickly due to depletion of glycogen stores in long continued febrile and chronic diseases and some convulsive disorders. In death from drowning rigor may appear early due to muscular exhaustion but last longer due to coldness of water.

c. Surroundings: rigor is delayed by cold and accelerated by heat. Spitz U. Werner (1993) reported that in temperate climate, under average condition, 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.

Rigor mortis appearance and disappearance is accelerated by prior exercise, convulsions, electrocution, and hyperpyrexia or hot environmental temperature. Hypothermia and cold environment delay the rigor process. Rigor mortis development is affected by total body muscle mass and develops poorly in young children, the elderly and debilitated. He also suggested that when the appearance of rigid limbs is inconsistent with gravitational forces, rigor is reliable indicator of postmortem shift in the position of the body. They further stated that rigor develops and disappears at a similar rate in all muscles. However because of lesser volume, small muscles (e.g. Masseter, hands) become totally involved by rigor before the large, volume muscles (e.g. thigh muscles), a phenomenon which, formerly led to misleading belief that rigor progresses from the head downwards. In temperate climate, under average condition, 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.

The appearance and disappearance is accelerated by prior exercises, convulsions, electrocution, hyperpyrexia and hot environmental temperature. Mason JK stated that the rigor mortis presents in the face at 3 hours, in the arms at 6 hours and is complete in the legs at 12 hours is little more than a useful rule of thumb which is open to considerable variation and the sequence is by no means constant. He further stated that the rigor will remains well marked for 12 hours and then disappears over the next 12 hours in the order in which it appeared. Infants and elderly often do not demonstrate rigor. The onset of rigor will be accelerated in conditions involving a high ante-mortem muscle lactic acid e.g. after a struggle or other exercise. Mukherjee JB stated that in Eastern India, rigor mortis commences within one to two hours after death. It takes another one to two hours to get fully developed in the whole body. In temperate climate, ordinarily it commences within 3 to 4 hours of death, takes further 3 to 4 hours to get fully developed.

In Eastern India rigor mortis usually lasts in summer for 18 to 24 hours and in winter may lasts for 24 to 36 hours. It starts in the muscles of eyelids by 2 hours approximately of death, then progresses in the order i.e. in the muscles of the back of the neck and lower jaw, in the muscles of front of neck, face, in the muscles of chest, in the upper extremities, lastly in the muscles of abdomen and the inferior extremities. The small muscles of toes and fingers are last to be affected by rigor mortis. The course of disappearance of rigor mortis is the same as its order of appearance. The rigor mortis is early to appear in early youth and old age and its duration is shorter. The onset of rigor mortis is rapid and duration is short in cases of bodies exposed to warm and moist climatic conditions. Mackenzie SC cited by Mukherjee JB that the period of onset of rigor mortis being 40 minutes, latest 7 hours, average is 1 hour 56 minutes in Eastern India. Narayan Reddy stated that rigor mortis is a state of stiffening of muscles, sometimes with slight shortening of the fibers. Individual cell death takes place in this stage.

He further stated that the rigor mortis is seen in both voluntary and involuntary muscles of the body [2]. It begins in the eyelids, neck and lower jaw and passes upwards to the muscles of the face and downwards to the muscles of the chest, upper limbs, abdomen and lower limbs. Such a sequence is not constant, symmetrical or regular. It disappears in the same order in which it has appeared. Rigor mortis always sets in, increases and decreases gradually. He further stated that in India, it begins to appear within 1 to2 hours after death and takes further 1 to 2 hours to develop. In temperate countries, it begins in 3to 6hours and takes further 2 to 3 hours to develop. In India, usually it lasts 24 to 48 hours in winter and 18 to 36 hours in summer. It lasts for 2-3 hours in temperate region, when rigor sets in early; it passes off quickly and vice versa. In death from diseases causing great exhaustion and wasting e.g. cholera, typhoid, tuberculosis and cancer and in violent deaths as by cut throats, firearms or by electrocution, the onset of rigor is early and duration is short.

In deaths from asphyxia, severe hemorrhage, apoplexy, pneumonia, nervous diseases causing paralysis of muscles, the onset is delayed. In strychnine and other spinal poisons, the onset is rapid and duration is longer. He also differentiated the rigor mortis from the heat stiffening. When a body is exposed to the temperature above 65 degree centigrade, rigidity is produced which is much more marked than rigor mortis. It is seen in cases of burns, high voltage electric shock and from falling into hot fluid. Heat stiffening occurs due to the denaturation [17]. Dalbir Singh and Indrajeet studied the onset, duration, and sequence of rigor mortis, on 376 subjects (303 Males and 73 females) who had died in Nehru Hospital of PGI, Chandigarh during the last 4 years. They carried the study on only those cases, which had been kept under room temperature and in which the exact time of death was known. Rigor Mortis was observed by lifting the eyelids with the help of pulp of index finger, depressing the jaw by exerting the slight pressure over the chin, gently trying to bend the neck forward and backward and by trying to elicit passive movements at various joints of the limbs.

The sequence and time taken by various muscle groups to become stiff was recorded. To observe the effect of atmospheric and seasonal conditions on the rigor mortis, 202 cases were studied in summer [April to September] and 174 cases in the winter [October to March]. They grouped all the subjects under three age groups i.e. 0-17 yrs., 18-50 years and above 51 years. They found that in summer, the average time of onset of rigor mortis varied from 1:47 hours in the eyelids [40 minutes to 2:15 hours] to 8 :32 hours in the toes [6 :10 hours to 11: 45 hours]. For its disappearance in summer, the average time was between 12:32 hours in the eyelid [9:15 hours to 16:50 hours] to 25:39 hours in toes [21:50 hours to 32:10 hours]. In winter, the average time of onset was between 2:26 hours in the eyelids [1:15 hours to 3:40 hours] to 10:40 hours to 10:23 hours in the toes [9 hours to 12:50 hours] and for its disappearance average time was 23:50 hours in the eyelids [18:20 hours to 29:30 hours] to 38:54 hours in the toes [32 hours 15 minutes to 46 hours 50 minutes].

They observed that the average duration of rigor mortis in summer varied from 10:45 hours in the eyelids [8:35 hours to 14:35 hours] to 17:7 hours in the toes [15:40 hours to 20:25]

hours] while in winter its average duration was between 21: 24 hours in the eyelids [range 17: 5 hours to 25:50 hours] to 28:31 hours in the toes [23:15 hours] to 34:45 hours. They observed that in 339 (90.1%) subjects in order of frequency, rigor mortis (commenced first in the eyelids followed by lower jaw, neck, upper limbs and lower limbs with fingers and toes in the last. It disappeared in the same order as it appeared. In remaining 37 cases (9.8%) they found that the duration remained the same, appearing disappearance of rigor mortis in various muscles was erratic and had not followed the pattern as described above, making the sequence of rigor mortis unreliable. They observed that rigor mortis appeared and disappeared earlier in the younger (10-17 yrs) and older (51 years and above) subjects in comparison to the adults in the age groups of 18-50 years.

Bernard Knight described the method of testing the rigor mortis by attempting to flex or extend the joints though the whole muscle mass itself becomes hard, and finger pressure on quadriceps or pectoralis can also detect the changes. The stiffness may develop within half an hour of death or may be postponed indefinitely [18]. Usually infants, the cachectic and the aged may never develop recognizable rigor mortis mainly because of their feeble musculature (Table 4). A rigor mortis spread to involve the whole muscle mass and reaches to maximum within 6-12 hours and remains in full around 18-36 hours. He also stated that biochemically the rigor is initiated when the ATP concentration of muscle falls to 85% of the normal and the rigidity of the muscle is at maximum when the level declines to 15% [19]. He also stated that the factors modifying the speed of onset of rigor mortis were physical activity shortly before death. The reason may be the availability of glycogen and ATP in the muscles is a crucial element in rigor formation.

The muscular exertion affects the interaction of these substances and hastens the onset of rigor mortis. Nandy A stated that rigor mortis occurs in both voluntary and involuntary muscles of body. In summer, it takes 1hour to appear and another 3-4 hours for all round distribution. During this season the average period of stay of rigor mortis is between 12 to 18 hours. In winter, it takes about 2-3 hours to appear and another 4-6 hours for all round distribution, with an average period of stay for about 24-48 hours. In foetus of less than 7 months, the rigor mortis does not develop. In thin built subjects with weak musculature, it comes and passes off early while in well built with strong musculature; it comes late and stays longer. In high atmospheric temperature, it comes early and passes off early while in cold atmospheric temperature; it comes late and stays longer. Deaths in exhausted diseases or when the convulsions proceeds death, and also in wasting diseases, the rigor mortis comes early and passes off early.

In deaths due to strychnine and HCN poisoning, it comes early and passes off early. Subramanyam BV Stated that rigor mortis first appears in the involuntary muscles and then in the voluntary muscles. In the heart, it appears within an hour after death. In the voluntary muscles, rigor mortis follows a definite course. It first appears in muscles of eyelids, next in the muscles of the back of neck and lower jaw, then in the of front of neck, face, chest and upper extremities and lastly extend downwards to the muscles of toes and fingers are lastly affected. It passes of in the same sequence. The average period of onset of rigor mortis may be regarded as 3 to 6 hours after death in temperate climate, and it may take 2 to 3 hours to develop. In India the rigor usually commences in 1 to 2 hours after death. In temperate region, rigor mortis usually lasts for 2 to 3 days. In Northern India, the usual duration of rigor mortis is 24 to 48 hours in winter and 18 to 36 hours in summer. In adolescent and healthy adults bodies, the occurrence is slow but well marked, while it is feeble and rapid in children and old people. Mackenzie cited by Subramanian B. U. investigated in Calcutta and found average duration of rigor mortis the 19 hours and 12 minutes, and shortest duration being 3 hours and longest 40 hours [20].

# Basic back ground of rigor mortis

# Influencing Conditions: On onset and duration of rigor mortis

a. Condition of Body: Rigor mortis sets in quickly in thin built emaciated subject and its duration is also shorter in such cases. The more muscular and healthier the subject, rigor mortis will be more late to set in and long to continue.

b. Age of Deceased: In case of foetus, rigor mortis is both earlier to appear and also to disappear. Its duration is short in case of the stillborn. It is said that rigor mortis does not occur in foetus less than 7 months. It is early to set in the bodies of newly born infants and may last for some hours. Rigor is early to appear in early youth and old age and its duration is also shorter.

c. Cause Of Death: Rigor will be earlier to set in but shorter in duration in case of death due to tetanus, strychnine poisoning, in death preceded by violent spasms and convulsions as in status epilepticus, electrocution, lightening or in case of death of soldiers after many hours of violent exertion in the battle fields, and in cut throat and firearm injures. It is early to set in death due to cholera , typhoid , T.B ,uremia , plague, cancer and nephritis .It is late in onset of death from pneumonia ,apoplexy, asphyxia especially hanging , carbon monoxide and dioxide poisoning ,arsenic or mercuric chloride poisoning ,nervous disease producing the paralysis of muscles.

d. Environmental Factor: The onset of rigor will be rapid and duration will be short in case bodies exposed to warm and moist climatic conditions. The factors such as movements of cold air, presence of wearing apparels will indirectly influence the onset and progress of rigor by their effects on the body temperature. In cold, rigor will be delayed in onset and its duration will be prolonged. Temperature below 5°C will retard the onset of rigor indefinitely, but when temperature is raised, rigor sets in the usual manner.

Body if exposed to temperature of 75 °C or little above, the rigidity becomes more pronounced due to coagulation of all muscles proteins and the body will show the rigidity in the form of heat stiffening [21,22].

# Forensic significance of rigor mortis

To study

- a. Time since death.
- b. In relation to age.
- c. In relation to sex.
- d. In relation to weight.

e. The effect of environmental temperature on rigor mortis.

#### Patho-physiology of rigor mortis

Rigor mortis is certainly the most fascinating cadaveric sign because it gives a putrified appearance to the deceased person; rigor mortis is noticed by everybody who encounters a corpse. Rigor mortis is a result of postmortem muscle contraction, therefore to understand the development of rigidity, we must first study the mechanism of muscle contraction and hence the structure of muscle. The muscles are of three types: (1) Skeletal, (2) Cardiac and (3) Smooth muscle. Szent Gyorgy discovered two muscle proteins which he named Actin and Myosin. These two proteins form interdigitating thick (Myosin) and thin (Actin) filaments, which build the sarcomere, the contractile unit of muscle. The sarcomeres are organized head to tail in series i.e. 4000/cm and that forms the fibrils.

The muscle cell is a fiber composed of 1000 to 2000 fibrils. What so ever the histological type may be, but every muscle is a molecular motor which converts chemical energy into mechanical work (kinetic energy). The body musculature is organized in accordance to need and be major anatomical movements are named as flexion, extension, abduction, adduction, supination, probation, medial and lateral rotation and some other movements like blinking, swallowing etc. Thus the muscles are architect zed on the skeleton in such a way that they will give appropriate movements. The program of contraction and relaxation of skeletal muscles materializes these appropriate movements. The skeletal muscles are having following properties i.e. excitability, conductivity, contractility, relaxibility, fatigue, elasticity, all or none law and tetanus etc [19].

### **Physiology Of Contraction And Relaxation Of Muscles**

The contraction of muscle can be explained by the ATP theory of Erdos and sliding filament model of contraction proposed by Hanson and Huxley. According to this model, contraction or tension in the muscle is achieved by the contrary motion of the interdigitating filaments. The myosin filament carries myosin heads on both ends. This heads are attached to the actin filament and act as cross bridges to form the actinmyosin (Ac-My) complex. During contraction the heads swivel and thin filament is pulled past the thick one. As the heads on each end of the myosin filament swivel in the opposite direction, the Z-lines approach each other and the sarcomere shortens. The limit of contraction is reached when the thick myosin filaments but against the Z-lines. Both filaments slide without changing their length, motion is achieved by cyclic formation and breaking of the cross-bridges. The fibrils may shorten by 30-50%, thus requiring that the swivel cycle be repeated many times. The driving force for the sliding motion comes from the myosin heads.

The kinetic energy is derived from chemical processes. The myosin heads bind ATP and form myosin ATP which in turn has a very high affinity for acting, resulting in the Ac-My complex. When the Ac-My complex is formed, Atlases activity displaced by free myosin heads is increased, and ATP is hydrolyzed. The energy released through the ATP hydrolysis is used for the dissociation of the Ac-My complex. The ATP used is immediately regenerated; this may be achieved through three different processes (Energy kinetics of muscle fiber) which are as follows.

- a. Phosphorylation of adp by creatinine phosphate.
- b. Oxidative Phosphorylation of adp in the mitochondria.
- c. Substrate level Phosphorylation of adp by the glycolytic pathway.

Phosphorylation of ADP by the cells creatine phosphate (CP) provides a very rapid means of forming ATP at the onset of contractile activity. When the chemical bonds between keratinize (C) and the phosphate is broken, the amount of energy released is about the same as that released when the terminal phosphate bond in ATP is broken. This energy, along with the phosphate group, can be transferred to ADP to form ATP in a reversible reaction catalyzed by creatine kinase.

#### CP + ADP -----C + ATP

Creatine kinaseIn resting muscles fiber, the concentration of ATP is more than that of ADP, leading by mass action to the formation of creatine phosphate. During period of rest, muscles fiber build up a concentration of creatin phosphate to a level of approximately five times that of ATP.At the beginning of contraction, the concentration of ATP begins to fall and that of ADP rises owing to the increased rate of ATP from creatine phosphate.

This transfer of energy from CP to ATP is so rapid that the concentration of ATP in a muscle fiber changes very little at the start of contraction, wherever the concentration of CP falls rapidly. Since the formation of ATP from CP is very rapid and requiring a single enzymatic activity the amount of ATP that can be formed is limited by little concentration of CP in cell. Therefore for continuation of contractile activity of muscle fiber, the muscle must be able to form ATP from other sources, which are listed above. The use of CP at the start of contractile activity provides few seconds necessary for the slower, multi enzyme

pathways of oxidative Phosphorylation and glycolysis to increase their rates of ATP formation to the levels that match the rates of ATP breakdown. For the moderate level muscle activity the most of ATP used for muscle contraction is formed by oxidative Phosphorylation. During first 5-10min. of exercise, muscle glycogen is major fuel contributing to oxidative Phosphorylation. For next 30 mins or so, blood born fuels like blood glucose, fatty acids contribute equally oxidative Phosphorylation of the muscles.

Beyond this period, role of fatty acid becomes more important and that of glucose decrease. Here the glucose for glycol sis is obtained from the two sources-blood or stores of glycogen within the muscle fiber. Since ATP act as the main fuel of contraction and relaxation of muscles fiber for swiveling of myosin heads to acting to form Ac-My complex. This ATP is generated in muscles through three different processes-

- a. Hydrolysis of creatinine phosphate.
- b. Glycol sis.

c. Oxidative Phosphorylation (combustion of glucose in presence of oxygen).

Since after death all these processes are stopped, ATP is not generated. The head of myosin gets fixed to the actin head and the normal swiveling and de-swiveling of the head of myosin from the actin does not occur. Therefore the Ac –My complex is not split and become stable. This stable Ac-My complex is the basis for the development of the rigor mortis in the postmortem states i.e. rigor is a normal muscle contraction occurring after death and fixed by lack of ATP.

It is last evident vital event in the muscles. With cessation of myocardial and respiratory functions, the muscle tissue becomes anoxic and all oxygen dependant processes cease to function. Within a certain period after death, the muscle fiber contract and retain this shortened state for a variable period of time before passively relaxing. This state of muscle contraction depending entirely on physiochemical change, and devoid of electrical excitation is known as rigor mortis. The persistence of this rigid inextensible state depends upon both external and internal factors. The external factors are those such as environmental heat or strenuous physical exertion before death, which hastens the depletion of ATP and glycogen, producing early onset of rigor state. The absence of exertion before death and cooling of body after death delay the onset and prolong the period of rigor. As ATP inhibits the activation of linkages between actin and myosin the internal changes responsible for development of rigor relates to the muscle ability to maintain the adequate level of ATP.

The major area for resynthesis of ATP depends on the supply of phosphocreatine in the muscle and also on anaerobic glycol sis. Anaerobic glycol sis continues until most of the glycogen is depleted and results in increasing levels of pyruvic or lactic acid. Production of lactic acid and breakdown of glycogen remains linear until the PH reaches 5.8 after which the process of glycol sis slows down at which time very little glycogen remains. As the level of ATP decreases beyond a critical level, the process of rigor proceeds rapidly. Rigor mortis under these circumstances, associated with a low PH is known as Acid rigor [1]. On the other hand where the individual was exhausted or starved before death, the glycogen stores are minimal and rapid or precipitin rigor may occur. Pyruvic and lactic acid are not formed and the muscle remains alkaline and is commonly known as Alkaline rigor by Bernard. Mitochondrial activity ceases abruptly at death while myofibril ATP ase activity is active at normal PH and become very active at high PH level. With a fall in PH.

The sarcoplasmic ATP ase become hyperactive and ultimately ATP are degraded. With progressive decomposition and disappearance of ATP from muscle due to dephosphorylation and deamination down to critical level, overlapping arrays of actin and myosin filaments combining as rigid links actinomyosin, forms viscous, inextensible dehydrated stiff gel like state which accounts for the rigidity and stiffness of muscle in rigor mortis, when the muscle do not respond to electrical or any other stimulus. Thus this irresponsive stiff rigid contracted state of body musculature constitutes the rigor mortis. With this explanation, it is easy to understand the mechanism of development of cadaveric rigidity, which unfolds in four different phases [23].

a. 1<sup>st</sup> phase: Delay period: After clinical death, the muscles survives in a normal state for a short time and relaxed as long as the ATP content remains sufficient enough to permit the splitting of actin-myosin cross-bridges. Erdos (1943) who compared the hardness of the muscles with its ATP concentration proved this fact. Here decrease in ATP levels was matched by an increase in the hardness. The rate of ATP depletion depends on its contents at time of death, on the possibility of postmortem ATP production and the rate of ATP hydrolysis.

b. 2<sup>nd</sup> phase: Onset period: The ATP content of the muscles falls below a critical threshold. The cross bridges remain intact and rigidity appears. However, this state is still reversible. The addition of ATP or oxygen results in a relaxation, indicating that the muscle is still able to function.

c. 3<sup>rd</sup> phase: Rigor (Irreversible phase): Rigidity is fully developed and become irreversible. Postmortem modifications of muscle fiber destroy their ability to relax.

d. 4<sup>th</sup> phase: Resolution: Rigidity disappears and the muscles become limp. The cause of resolution according to Szent-Gyorgyi and Erdoz is a denaturation processes [23].

# Conclusion

In this review of literature, time since death from rigor mortis has been compiled on the basis of forensic prospective. In future, rigor mortis is a paramount and dominant parameter for estimating the time since death and others.

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#### References

- 1. Henssge C, Madea B (2007) Estimation of the time since death. Forensic Sci Int 165(2-3): 182-184.
- Sukhadeve RB, Khartade HK, Tasgaonkar GV, Meshram VP, Parchake MB, et al. (2016) Estimation of time since death from rigor mortis in central India. Medico-Legal Updat 16(2): 197.
- Bate Smith EC, Bendall JR (19467) Rigor Mortis and Adenosinetriphosphate. J Physiol 6(1939): 177-185.
- 4. Rall JA (2014) Mechanism of Muscular Contraction.
- Khartade HK, Tasgaonkar G V., Sukhadeve RB, Parchake MB, Meshram VP, Hosmani AH (2017) Study of rigor mortis and factors affecting its development for determination of postmortem interval. Indian J Forensic Med Toxicol 11(2): 70-74.
- Liu X, Pollack GH (2004) Stepwise Sliding of Single Actin and Myosin Filaments. Biophys J 86(1):353-358.
- Postgraduate diploma courses forensic medicine, medical jurisprudence and toxicology. (2003) Int J Med Toxicol Leg Med 5(2): 55-73.
- Holstege CP, Rusyniak DE (2005) Medical toxicology. Med Clin North Am 89(6).
- 9. Dettmeyer RB, Verhoff MA, Schütz HF (2014) Forensic Medicine: Fundamentals and Perspectives.
- Vain A, Kauppila R, Vuori E (1996) Estimation of the breaking of rigor mortis by myotonometry. Forensic Sci Int 79(2): 155-161.



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- 11. Ratzliff AH, Santhakumar V, Howard A, Soltesz I (2002) Mossy cells in epilepsy: Rigor mortis or vigor mortis? Trends Neurosci 25(3): 140-144.
- Chakravarthy M (2010) Rigor mortis in a live patient. Am J Forensic Med Pathol 31(1): 87-88.
- 13. Honikel KO (2014) Rigor Mortis, Cold, and Rigor Shortening. In: Encyclopedia of Meat Sciences 358-365.
- 14. Montali RJ (2008) Postmortem Diagnostics. In: Biology, Medicine, and Surgery of Elephants : 199-209.
- 15. Hossein SZ (2002) Modern forensic medicine and the medico-legal system in Iran. J Clin Forensic Med 9(1): 12-14.
- Leibovitz A, Blumenfeld O, Baumoehl Y, Segal R, Habot B (2001) Postmortem examinations in patients of a geriatric hospital. Aging (Milano) 13(5): 406-409.
- 17. Newman J, McLemore J (1999) Forensic medicine: matters of life and death. Radiol Technol 71(2): 169-185.
- Poposka V, Gutevska A, Stankov A, Pavlovski G, Jakovski Z, Janeska B (2003) Estimation of Time Since Death by using Algorithm in Early Postmortem Period. Glob J Med Res Interdiscip 13(3): 17-26.
- 19. Krompecher T (1981) Experimental evaluation of rigor mortis V. Effect of various temperatures on the evolution of rigor mortis. Forensic Sci Int 17(1): 19-26.
- 20. Atkinson SB (1906) Post-mortem examinations. Notes Queries 10(107): 29.
- Kobayashi M, Takatori T, Nakajima M, Sakurada K, Hatanaka K, et al. (2000) Onset of rigor mortis is earlier in red muscle than in white muscle. Int J Legal Med 113(4): 240-243.
- 22. Steward O, Balice-Gordon R (2014) Rigor or mortis: Best practices for preclinical research in neuroscience. Neuron 84(3): 572-581.
- Bedford PJ, Tsokos M (2013) The occurrence of cadaveric spasm is a myth. Forensic Sci Med Pathol 9(2): 244-248.

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