POSTMORTEM CHANGES IN DOMESTIC ANIMALS: FORENSIC IMPLICATIONS AND HUMAN CASE COMPARISONS

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ABSTRACT

Thanatology, the study of death, examines physiological and biochemical postmortem changes, aiding forensic and veterolegal investigations. Death progresses through somatic and molecular stages, followed by immediate, early, and late postmortem changes. Immediate changes involve the cessation of vital functions, while early changes include pallor mortis, algor mortis, livor mortis, and rigor mortis. Late-stage changes involve decomposition, including autolysis and putrefaction, which lead to tissue breakdown. These changes help estimate the postmortem interval (PMI) and determine the cause of death. Algor mortis follows a sigmoid cooling pattern, with temperature dropping by 0.5°C per hour in summer and 0.7°C in winter, influenced by environmental and body factors. Postmortem caloricity, an exception, occurs in conditions like heatstroke. Livor mortis, the gravitational pooling of blood, fixes within 8–12 hours and provides clues to body positioning and toxicological causes. Rigor mortis follows Nysten's Rule, peaking at 12 hours and resolving by 36 hours, while cadaveric spasm occurs instantly, preserving the final position of muscles. Decomposition advances through autolysis and putrefaction, with green discoloration appearing within 12–18 hours. Gas formation leads to bloating and marbling, accelerating tissue breakdown. Casper's Dictum states that bodies decompose fastest in air, slower in water, and slowest underground. Adipocere, formed in moist conditions, preserves tissue for months, while mummification occurs in dry climates. Embalming extends preservation artificially. These postmortem changes collectively provide crucial forensic insights into death circumstances and Postmortem Interval (PMI) or Time Since Death (TSD) estimation.

Keywords : Embalming, Pallor Mortis, Suspended Animation, **S**upravital Period, and Thanatology.

I. INTRODUCTION

Thanatology, the study of death, encompasses the various changes that occur in the body following death. Death is legally defined under Section 46 of the Indian Penal Code (IPC). The process begins with **somatic death**, marked by the cessation of vital functions such as respiration, circulation, and brain activity. After a few hours, **molecular death** sets in, where individual cells begin to die, initiating tissue degradation. The interval between somatic and molecular death is known as the **supravital**

period, a critical window during which organs can be harvested for transplantation.

Suspended animation is a state of apparent death in which vital functions are either temporarily halted or reduced to their minimal levels. This condition can result from various factors, including concussion, shock, hypothermia, and electrocution. In humans, it can sometimes be voluntarily induced through practices like yoga. During this state, the body's organs consume oxygen at extremely low levels to sustain survival. Resuscitation is often successful if intervention occurs within a few seconds to a few minutes. Suspended animation is commonly observed in newborns, drowning cases, and individuals experiencing severe electrical shock.

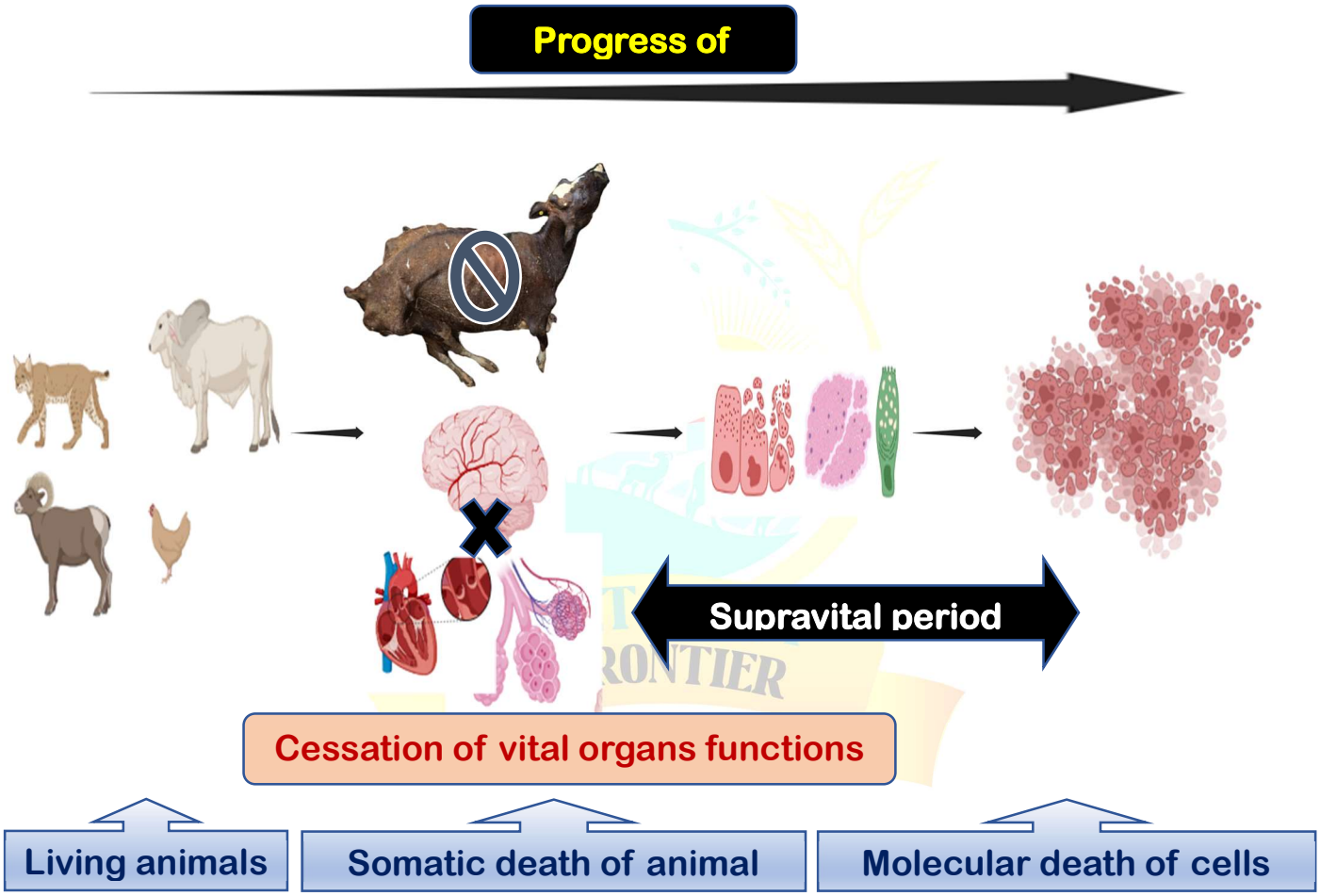


Figure 1: Schematic illustration of the progress of death

Postmortem changes are categorized into immediate, early, and late stages. **Immediate changes** include the loss of voluntary movements (earliest sign of death), cessation of respiration, and the halting of circulation. **Early changes** involve observable phenomena such as pallor mortis (loss of elasticity and paleness of skin), eye alterations, primary flaccidity of muscle, **algor mortis** (postmortem cooling), **livor mortis** (postmortem staining due to blood pooling), and **rigor mortis** (postmortem stiffening of muscles). Finally, **late changes** are characterized by decomposition, which occurs through **autolysis** (self-digestion by cellular enzymes), **putrefaction** (breakdown of tissues

by microbial action) adipocere formation and mummification. Together, these sequential events provide critical insights into the timeline and circumstances surrounding death, aiding forensic, medicolegal and veterolegal investigations in humans and animals respectively (Poloz and O'Day 2009; Sutherland *et al*., 2013).

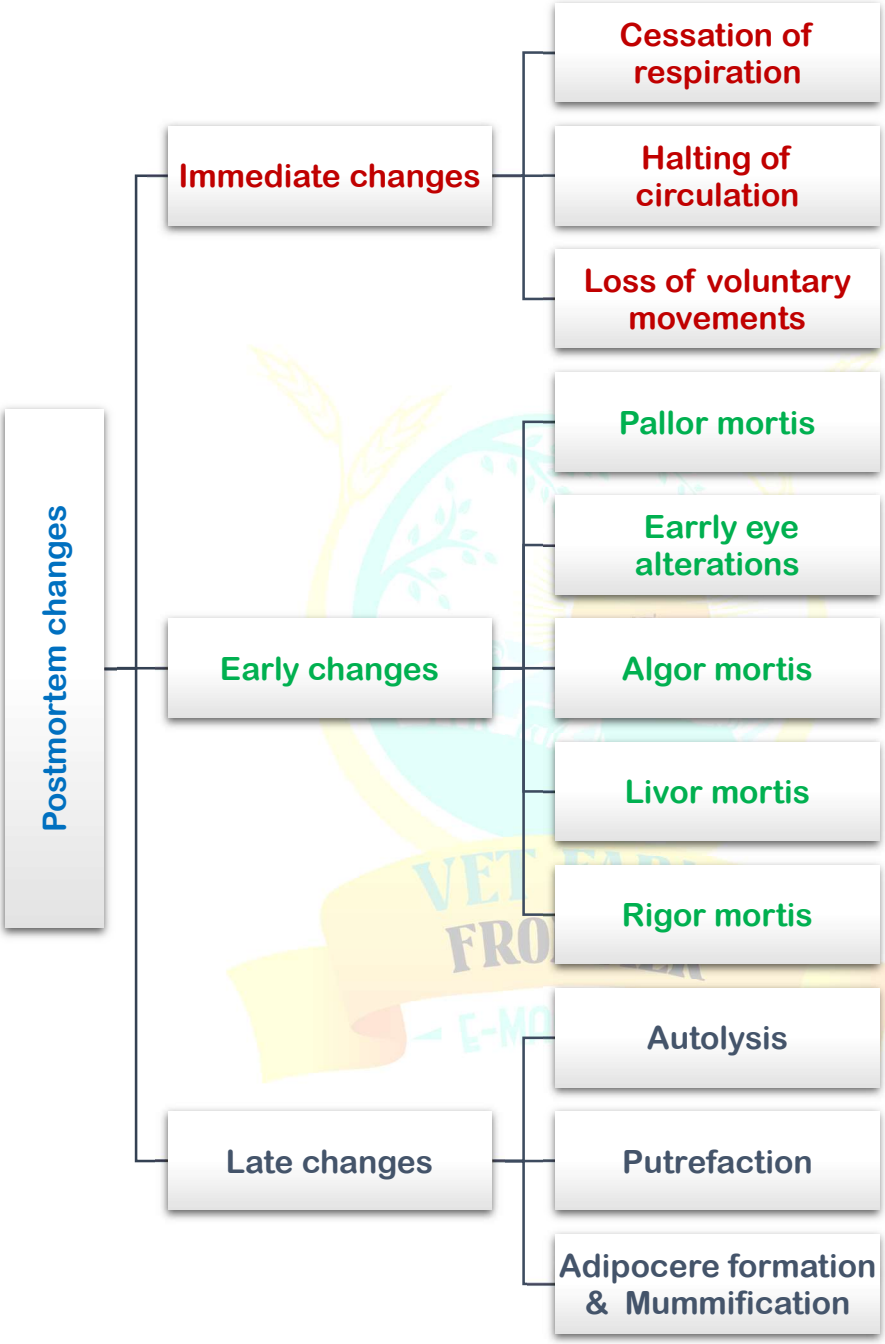


Figure 2: An overview illustration of postmorterm changes

II. EARLY OCULAR CHANGES

Eye changes are among the earliest postmortem alterations and play a vital role in determining the time of death. One of the earliest observable signs of death is the loss of the corneal reflex, followed by corneal opacity due to dehydration, which typically occurs around six hours after death in humans but varies in animals. Pupillary diameter, retinal vessel striation, retinal color changes, and a reduction in intraocular pressure are other notable postmortem changes.In dogs, corneal clouding usually begins no earlier than 8 hours postmortem. If the eyelids remain closed, corneal dehydration is delayed, making this observation a more reliable indicator for estimating the postmortem interval (Jaafar and Nokes 1994; Balci *et al.,* 2010).

Postmortem drying of mucous membranes and delicate skin can cause changes in color and texture. This desiccation begins immediately after death and progresses rapidly in moist areas, particularly the eyes in humans. It forms a yellow or dark brown horizontal band with triangular discolorations on either side of the cornea, with the base facing the cornea and the tip pointing toward the eye's angle, a condition called Tache Noire Sclerotica. Tache Noire develops when the eyelids remain open, causing the sclera to dry and accumulate mucus, dust, and cellular debris. However, this is less prominent in animals with larger corneas. Skin desiccation occurs more slowly, primarily affecting thin areas like the lips and genitalia, which darken to red or black with an irregular surface. A well-formed Tache Noire typically indicates that death occurred approximately 3–6 hours earlier in humans (Brooks 2016).

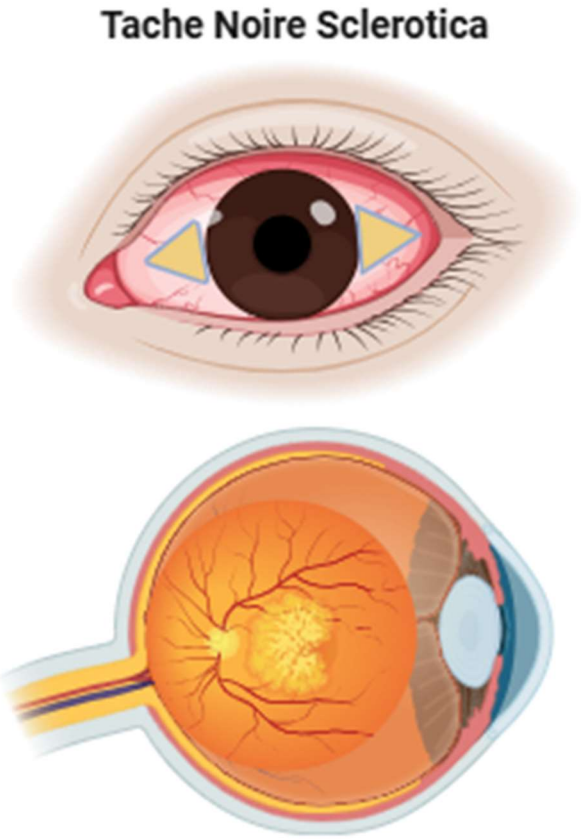
The **flaccidity of the eyeball** is another postmortem change due to a drop in **intraocular pressure (IOP)**. During life, the IOP ranges from **14–25 mmHg**, but immediately after death, it drops below **12 mmHg** and further decreases to less than **3 mmHg within half an hour**. The pupils become **dilated and fixed**, showing no reaction to light. However, in humans, the pupils may respond to atropine for up to an hour postmortem but not to strong light. Once rigor mortis sets in, the pupils may constrict, and while their shape remains unalterable during life, it can be modified after death. One of the earliest and most distinct ocular changes is the Kevorkian Sign (Trucking Sign), characterized by the fragmentation or segmentation of retinal vessels, resembling "cattle tracks." This phenomenon occurs within minutes (as quickly as 10 seconds) after death and is observed using an ophthalmoscope. Additionally, changes in the vitreous humor provide crucial biochemical markers for estimating the postmortem interval. In humans, the potassium (K^+) level in the vitreous humor rises predictably at a rate of 0.2–0.8 mmol/hour over the first 100 hours postmortem. Formulas such as Starner's Formula and MADEA's Formula are commonly used in forensic investigations for precise time-of-death estimation (Jaafar and Nokes 1994; Balci *et al.,* 2010; Brooks 2016). **(Figure 3: Schematic illustration of early ocular changes and Tache Noire sclérotique).**

III. ALGOR MORTIS AND POSTMORTEM CALORICITY

Algor Mortis or postmortem cooling, refers to the gradual decline in body temperature following death. It begins approximately 15–30 minutes after the cessation of life and is driven by the cessation of metabolic processes, which halts the production of heat energy and with no energy being produced, the body begins to lose heat, transferring it from the core to the surface and eventually to the surrounding environment. The cooling process follows a sigmoid curve, indicating an initial slow decline, a linear rapid cooling phase, and a final plateau as the body temperature approaches ambient temperature

(*Marshall and Hoare,*1962; *Henssge and Madea,* 2004; *Kaliszan* et al., 2005).

Generally, an animal's body will cool to ambient temperature within 24 to 48 hours postmortem. However, if the carcass is open, rectal temperature is not a reliable method for measurement; instead, deep intranasal or thigh temperature should be taken for a more accurate reading. The rectum is the most frequently used site for measurement due to its accessibility and reliability, where the thermometer is inserted 6–10 cm deep and kept in place for 2 minutes. In situations where rectal measurements are not viable, such as in cases of rectal trauma or obstruction, alternative sites include the subhepatic space (inferior surface of the liver), which is considered an ideal location for precise measurements, or the tympanic membrane via the ear canal.The most common method for measuring core body temperature during algor mortis involves the use of a chemical thermometer or a thanatometer (a specialized device that is 25 cm long and calibrated between 0°C and 50°C) (*Henssge and Madea,* 2004; *Kaliszan* et al., 2005).



The rate of cooling varies based on environmental factors including environmental temperature, humidity, body size, and insulation from fur or external coverings and body characteristics. On average, body temperature drops at a rate of 0.5°C per hour in summer and 0.7°C per hour in winter. However, this rate is influenced by external conditions and other variables. During the first hour postmortem, the cooling is typically negligible as the body retains residual heat. From the second hour onward, a linear pattern of cooling ensues at a rate of 0.4–0.6°C per hour, lasting approximately 12–16 hours. The rate of temperature decline is most rapid during this phase. As the difference

between the body temperature and the ambient environment reduces to around 4°C, the cooling rate slows significantly, and the body eventually reaches thermal equilibrium with its surroundings in approximately 20 hours. A larger temperature gradient between the body and the environment accelerates the cooling process. Higher ambient temperatures slowing cooling and lower temperatures accelerating it. The medium surrounding the body also has a significant effect: a body immersed in stagnant water cools twice as quickly as in air, and in running water, the cooling rate triples. Additionally, the physical characteristics of the deceased influence cooling; for example, an obese individual's insulating fat layer slows the rate of cooling, whereas lean individuals cool more rapidly. Cooling may begin even before death pre-mortem conditions such as chronic illnesses, infections, or long-standing debilitating diseases can further alter the cooling dynamics. The "rule of thumb" suggests that the body cools at a rate of 1°C (1.8°F) per hour after death, with an additional 3-hour adjustment for the temperature plateau effect (TPE). Other sources propose a cooling rate of 1.5°F to 2.0°F (0.83°C–1.11°C) per hour for the first 12 hours, followed by 1°F (0.55°C) per hour thereafter. This leads to the formula for estimating the postmortem interval (PMI): {**PMI (hours) = [98.6°F – rectal temperature (°F)] / 1.5} and { PMI (hours) = [37°C – rectal temperature (°C)] / 0.83}.** This method provides an approximation, but multiple factors, such as ambient temperature and body conditions, can influence cooling rates. The cooling process is hastened in cases involving hemorrhage, chronic illness, or wasting diseases, while it is significantly retarded in instances of sudden death due to accidents, acute diseases, apoplexy, asphyxia, lightning strikes, carbon dioxide poisoning, and suffocation. To estimate the time since death using algor mortis, ambient temperature, and body weight, Henssge and Madea's refined 2-exponential model is available as a user-friendly nomogram, widely accessible online and in textbooks (*Henssge and Madea,* 2004).

Postmortem Caloricity, on the other hand, is a phenomenon where body temperature either remains elevated or increases for about 2 hours after death. This occurs due to the paralysis of the body's heat regulation mechanisms. Common causes include conditions like heatstroke, pontine hemorrhage, and convulsive deaths (e.g., tetanus, strychnine poisoning, or electrocution). Sepsis or high fever prior to death can also contribute to this phenomenon. It is important to note that burns do not cause postmortem caloricity. Both algor mortis and postmortem caloricity provide valuable insights into the postmortem interval and the physiological processes occurring after death, aiding forensic investigations.

IV. Livor Mortis

Livor Mortis, also known as postmortem staining, postmortem hypostasis, lividity, suggilations, hypostatic congestion, darkening of death or vibices, refers to the purplish-blue or purplish-red discoloration that develops in the superficial layers of the skin due to the settling of blood in the dependent parts of the body due to gravity after death. This occurs due to capillovenous distension as blood settles under the influence of gravity following the cessation of circulation, leading to blood stagnation. The process begins within **30 minutes to 2 hours** after death, becomes well-established by **4 to 6 hours**, and gets fixed between **6 to 12 hours** (typically **8–12 hours**). If compression of the area causes blanching, the lividity is not yet fixed, whereas if the area remains discolored without blanching, it is considered fixed. Contact pallor occurs at pressure points where blood flow is restricted, such as the back of the head or foot in humans. Lividity distribution depends on body position: in a supine position, staining appears on the back except at pressure points; in a prone position, it occurs on the face

and front; in a side position, it develops on the resting side. In hanging cases, lividity is circumferentially distributed over the lower limbs and dependent areas of the upper limbs. In animals, lividity depends on body positioning, and during internal examination, the lungs and kidneys prominently show hypostatic congestion. Special forms include glove and stocking hypostasis in submerged bodies, while drowning cases show lividity on the face, chest, abdomen, and lower limbs. Notably, postmortem staining does not form in areas exposed to continuously flowing water (DiMaio and DiMaio 2001; Erlandsson and Munro 2007; Lew and Matshes 2005).

The color of lividity can provide forensic clues, especially in cases of poisoning. Under normal conditions, it appears **blue or purple** due to deoxygenated hemoglobin. **Cherry-red lividity** is associated with **carbon monoxide poisoning**, while **bright red** suggests **cyanide poisoning** due to oxygen retention.. **Chocolate brown** lividity indicates poisoning substances like **phosphorus**, **potassium chlorate**, **aniline**, or **nitrates**, the staining may take on a **brown** color due to methemoglobin formation, whereas **black discoloration** is linked to **hydrogen sulfide poisoning,** while **reddish-brown** may indicate **opium poisoning**. **Greenish-brown discoloration** can be seen in **lead poisoning**, and **deep blue lividity** is characteristic of **strychnine poisoning** (DiMaio and DiMaio 2001; Erlandsson and Munro 2007; Lew and Matshes 2005).

Table 1: Postmortem lividity progression over time

Stage	Approximate Time
Variable patches appear	~4 hours
Patches become fixed	~6 hours
Begins to develop fully	~12 hours
Completely fixed	~24 hours

Table 2: Differences between postmorterm lividity and antemortem injury

Feature	Postmortem Lividity	Bruise / Contusion
Location	Dependent parts of the body	Can occur anywhere
Blood distribution	Intravascular (within vessels)	Extravasated (outside vessels)
Effect of incision	Blood remains within vessels	Blood spreads in surrounding tissues
Underlying tissue discoloration	Absent	Present

Postmortem lividity provides crucial forensic insights into the position of the body at the time of death and potential causes of death. It helps determine whether a body was moved after death and assists in toxicological investigations.

V. RIGOR MORTIS

Rigor Mortis, or cadaveric rigidity, refers to the postmortem stiffening of muscles due to biochemical changes after death. It follows a sequence of **primary relaxation**, then **rigor mortis**, and finally **secondary relaxation** as decomposition sets in. Rigor mortis begins when **ATP levels drop**, initiating stiffness at **85% of normal ATP levels** and reaching its peak at **15% of ATP levels**. The onset of rigor mortis typically occurs **60 minutes after death**, peaks by **12 hours**, remains for another **12 hours**, and disappears over the next **12 hours**, following the **Rule of 12 (**DiMaio and DiMaio 2001; Erlandsson and Munro 2007).

Rigor mortis is a reliable indicator of time of death in its initial stages, though it is influenced by various factors. A fresh body with no postmortem changes but with clotted blood of recent origin suggests a time of death between 2 to 4 hours. The progression of rigor mortis varies with environmental conditions, beginning in the head within 2 to 8 hours in winter and ½ to 3 hours in summer. It extends to the head, neck, and forelimbs by 12 hours, spreads to the whole body by 15 hours, and remains only in the hind limbs by 20 hours. Rigor mortis disappears around 24 hours, and decomposition begins, leading to body looseness, a putrid smell, and bloating after 30 hours. External temperature plays a significant role, with warm and moist climates accelerating the onset and shortening its duration, while cold and dry conditions delay its development and prolong its presence. Violent exercise, such as racing, fighting, or struggling, hastens rigor mortis, violent muscle contractions due to conditions like tetanus or strychnine poisoning can accelerate the onset of rigor mortis whereas low temperatures retard it. In well-nourished, healthy carcasses, rigor mortis is well-defined, lasts longer, and appears late, while in weak or emaciated bodies, it is weaker, shorter, and delayed. In bodies immersed in water, rigor mortis sets in quickly but disappears later than in those exposed to air (Martins *et al.* 2015; Saukko and Knight 2004).

The progression of rigor mortis adheres to **Nysten's Rule**, starting proximally and descending distally. The first site to stiffen internally is the **myocardium**, while externally, it begins with the **eyelids** and progresses to the **jaw, neck, thorax, upper limbs, abdomen, lower limbs**, and finally, the **fingers and toes**. The stiffening order reflects a proximal-to-distal pattern for both onset and disappearance. A unique variant of rigor mortis is **cadaveric spasm**, also known as instantaneous rigor. It occurs immediately at death, skipping the relaxation phase, and involves a group of voluntary muscles that were actively in use at the time of death. Unlike regular rigor mortis, cadaveric spasm cannot be artificially induced and serves as an important **antemortem sign** for forensic determination of the manner of death. These characteristics make rigor mortis a critical tool in postmortem investigations (DiMaio and DiMaio 2001; Erlandsson and Munro 2007; Martins *et al.,* 2015; Saukko and Knight 2004)**.**

Table 3: PMI detection through rigor mortis

Body Condition	PMI (Post-Mortem Interval)
Warm and flaccid	Less than 3 hours (<3h)
Warm and rigid	3 to 8 hours (3-8h)

Cold and rigid	8 to 36 hours (8-36h)
Cold and flaccid	More than 36 hours (>36h)

V.1. Rigor Mortis Onset and Duration:

Rigor mortis typically begins early in certain conditions, including **starvation**, **bacillary haemoglobinuria**, **anthrax**, **tetanus**, and in **exhausted animals** (though it passes off quickly). It also occurs quickly in animals that died due to **lightning** or **drowning**. Additionally, animals treated with **sodium salicylate** show an accelerated onset of rigor mortis, with **alcohol** and **ether** also encouraging its early development.

V.2. Exceptions and Incomplete Rigor Mortis:

However, rigor mortis is either absent or only scarcely visible in **killed animals**, **slaughtered animals**, those that died in a **febrile condition**, and **fatigued animals**. In these cases, rigor mortis does not develop fully or at all.

The **pugilistic attitude**, also known as the **boxer's attitude**, occurs when the body is exposed to temperatures of **65°C or higher**, leading to coagulation of heart and muscle proteins. This posture is nonspecific and can be observed in both **antemortem and postmortem burns**.

VI. DECOMPOSITION

Decomposition begins shortly after death and is driven by two primary processes: **autolysis** and **putrefaction**. **Autolysis** is caused by the action of lysosomal enzymes, starting internally at glands and the brain, with **corneal clouding** being the first visible external sign. In humans, the first observable external color change is a greenish discoloration in the right iliac fossa (caecum), which appears 12–18 hours after death in summer or 1–2 days in winter. Internally, a **reddish-brown discoloration** forms in the aortic lumen (intima). **Putrefaction**, caused by bacteria from the gastrointestinal tract, leads to progressive decomposition changes, including **color change, gas formation, and tissue liquefaction**. One notable bacterium involved is **Clostridium welchii**, which secretes **lecithinase**, facilitating tissue degradation. **Marbling**, a linear branching pattern on the skin caused by the deposition of sulfhemoglobin in vessel walls, typically appears **36–72 hours postmortem** and is an important marker for estimating the **time since death (TSD) (**Reed 1958; Wilson *et al.,* 2007; Zhou and Byard 2011; Vass 2011) .

Putrefaction is one of the stages of decomposition in a dead animal's body, primarily driven by the action of anaerobic bacteria. As decomposition progresses, the putrefied body becomes a host for different life stages of flies, which can provide valuable clues for estimating the time of death. The presence of fly eggs, which hatch into maggots within 1 to 2 days, indicates an early stage of decomposition. Maggots then develop into pupae within 2 to 3 days, and pupae transform into adult flies within 4 to 5 days. By analyzing these insect life cycles, forensic experts can make more precise estimations of the postmortem interval. Forensic entomology has seen limited development in veterinary science.

Table 4: PMI detection through forensic entomology based on fly life cycle stages

Life Stage of Fly	Approximate Time of Death
Eggs to maggots	1 – 2 days
Maggots to pupae	2 – 3 days
Pupae to adults	4 – 5 days

Gas formation during putrefaction, primarily involving **hydrogen sulfide (H_2S)**, produces foul odors and results in **gas stiffening** and **postmortem skin blisters**. The sequence of organ putrefaction begins with the **larynx and trachea**, followed by the **stomach, intestines, spleen, liver, lungs, brain, heart**, and lastly, the **prostate or uterus**, skin, tendons, and bones. The rate of putrefaction also varies by medium, as described by **Casper's Dictum**, with decomposition occurring fastest in air (1 week), slower in water (2 weeks), and slowest in buried bodies (8 weeks). These processes are crucial for forensic analysis in determining the **postmortem interval** and circumstances of death (Wilson et al. 2007; Zhou and Byard 2011; Vass 2011).

Investigators have found the traditional four-stage decomposition model (1.Fresh, 2. Bloated, 3.Decay, and 4.Dry) problematic due to the lack of clearly defined starting and ending points. To address this issue, Vass summarized existing research and presented a table based on decomposition degree. Before Vass's summary, other researchers had modified Reed's four-stage scale, first expanding it to five stages and later to six. Wilson *et al.,* developed a six-stage model that includes fresh, primary bloat, secondary bloat, active decay, advanced decay, and skeletonization. Both the five- and six-stage models provide detailed descriptions for each stage. The six-stage model is often more suitable for buried remains or cases with shorter post-mortem intervals (PMIs), while the five-stage model is better suited for exposed remains or cases with longer PMIs. The five-stage decomposition model is summarized as follows (Table -5) (Wilson *et al.,* 2007; Zhou and Byard 2011; Vass 2011; Reed 1958; Megyesi *et al.,* 2005; Lew *et al.,* 2005; Galloway *et al.,* 1989)

Table 5: Summary of decomposition stages

Decomposition Stage	Duration	Description
Fresh	0–5 days postmortem	No discoloration or insect activity.
Early decomposition	1–21 days postmortem	Gray to green discoloration, bloating, post-bloating rupture, skin slippage, and hair loss.
Advanced decomposition	3 days to 18 months postmortem	Moist tissue breakdown, sagging flesh, abdominal collapse, extensive insect activity, less than half of the skeleton exposed, and mummification.
Skeletonization	2 months to 9 months postmortem	Presence of bones with body fluids or tissue covering less than half of the skeleton, as well as dry bones.
Extreme decomposition	6 months to >3 years postmortem	Skeletonization with bleaching, exfoliation, metaphyseal loss, or cancellous bone exposure.

Adipocere, or saponification, is a postmortem change that occurs when a body is exposed to a **warm, moist climate**. It involves the conversion of body fat into a waxy, soap-like substance through the action of **lipase enzymes** and bacteria like **Clostridium welchii**. Early adipocere appears as a waxy, clay-like material with an **ammoniacal smell**, while in its later stages, the tissue becomes **hard and brittle**. This process typically takes **3 weeks to 6 months**, though in India, it can occur within **4–5 days** under favorable

conditions. Adipocere forms more quickly in water (**2 weeks**) compared to soil (**8 weeks**), where it progresses slowly. **Mummification**, in contrast, occurs in **dry, hot climates** through the process of **drying and dehydration**, leaving the body **odorless** and preserved for **6–12 months**. It is also seen in cases of **intrauterine death (IUD)** of a fetus. Both adipocere and mummification hold significant **medicolegal importance**, aiding in **identification**, determining the **cause and time since death**, and establishing the **place of disposal** of the body(Reed 1958; Vass 2011; Megyesi *et al.,* 2005).

Embalming, an artificial preservation technique, involves injecting **antiseptics and preservatives** into the body. The most effective method is **discontinuous injection and drainage**, although **ethanol** is not used in this process. These preservation methods are crucial for forensic investigations and medico-legal documentation.

VII. CONCLUSION

Postmortem changes occur in a predictable sequence, allowing for an estimation of the postmortem interval (PMI). In the early postmortem period (<24–36 hours), core temperature decreases at a steady rate until equilibrium is reached. Rigor mortis follows a typical progression, becoming fully established within 6–36 hours and resolving thereafter. Initial decomposition is minimal, with no significant discoloration or insect activity in the first few days. Entomological evidence, particularly blowfly development, begins within minutes of death, with eggs hatching into first instar larvae within 8–14 hours. As decomposition progresses into the late postmortem period (>24–36 hours), visible changes such as discoloration, bloating, and skin/hair loss become evident within days to weeks. Insect activity intensifies, accelerating soft tissue breakdown. Over months to years, the body undergoes mummification, extensive bone exposure, and eventual skeletonization. The predictable stages of decomposition, rigor mortis, and insect succession provide valuable forensic markers for PMI estimation.

Figure 4: Various stages of animal decomposition based on five-stage classification *Adapted from Forensic Field*)

Table 6: Summary of postmortem changes over time

Postmortem Period	Changes	Time Frame
Early Postmortem Period (<24–36 h PM)	**Core Temperature Decrease**	~0.5°C–1°C per hour, equilibrates to environment in ~24–48 h PM
	Rigor Mortis	Onset: 2–6 h PM, Full: 6–36 h PM, Resolving: >36 h PM
	Decomposition	No discoloration or insect activity (0–5 d PM), Gray-green discoloration, bloating, skin/hair loss (1–21 d PM)
	Entomological Evidence (Blowfly Life Stages)	Eggs: within minutes, First instar: 8–14 h, Second instar: 16–28 h
Late Postmortem Period (>24–36 h PM)	**Rigor Mortis**	Full: 6–36 h PM, Resolving: >36 h PM
	Decomposition	No discoloration or insect activity (0–5 d PM), Gray-green discoloration, bloating, skin/hair loss (1–21 d PM), Moist decomposition, insect activity, minimal bone exposure, mummification (3 d–18 mo PM), Extensive bone exposure, dry bones (2 mo–9 mo PM), Complete skeletonization with bone degradation (6 mo–>3 y PM)
	Entomological Evidence (Blowfly Life Stages)	Eggs: within minutes, First instar: 8–14 h, Second instar: 16–28 h, Third instar: 3–4 d, Pupa: 9–10 d, Adult: 18–24 d

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