Chapter 1 Crime Scene Investigation

Introduction

Crime scene investigation is the first and most critical step in any investigation. There are three important steps at a crime scene: recognition of evidence, proper collection of evidence, and adequate preservation of evidence. In homicides, the medical examiner's involvement begins at the scene, and so should it be for animal cruelty cases. Involving the veterinarian at the very beginning of the investigation helps ensure that all the evidence is properly identified and analyzed. The goal of any criminal investigation is to solve the forensic triad: Link the victim to a suspect and connect them to a crime scene.

There are actually two crime scenes present: the macro crime scene, which is the location and surrounding area of the crime, the victim, and the suspect's body; and the micro crime scene, which is the body itself. Forensic science begins at the crime scene. There must be a thorough forensic exam of the crime scene and those findings must be shared with the veterinarian. The veterinarian must rely on evidence (or lack thereof) gathered at the scene to help determine proximate cause of death/injury, mechanism of death, and manner of death/injury. Physical evidence may be classified as chemical, biological, or pattern evidence. In may be further classified as transient, conditional, pattern, transfer, or associative. *Transient* refers to evidence that is easily lost or changed over time, such as body temperature and decomposition. *Conditional* refers to evidence resulting from an action or event that can be transient as well, such as rigor, weather conditions,

entomology, and stomach contents. *Pattern* refer to evidence with imprints, markings, or other patterns such as bite marks, blood spatter, wound patterns, and weapon patterns. *Transfer evidence* is the physical exchange of material between objects after contact, such as fibers, hair, or plant matter. *Associative evidence* is anything that can link a suspect or victim to the scene or each other. All evidence must be preserved for later testing and use at trial. In addition to testing performed for the prosecution side, the defense has the right to perform independent tests and analysis. Every reasonable effort should be made to preserve evidence and make it available for the defense team, even though this may not take place for a lengthy time period.

The Veterinarian's Role

The veterinarian's role at a crime scene is to assist investigators and examine any animals at the scene. The veterinarian should assist in the collection of evidence, examine the evidence, and assess the evidence and the crime scene. It is important to meet with the lead investigator, discuss the situation, and develop a plan for investigating the scene. Several things usually need to be addressed simultaneously, and the veterinarian can be of invaluable assistance. The situation often requires working under time pressures resulting from weather or other environmental conditions that could alter or destroy potential physical evidence.

- There may be sick or critical animals that must be triaged for transport to veterinary hospitals.
- It is important to record all initial observations of the scene and the animals.

- The status of each animal at the scene must be recorded because it may change for better or worse after arrival at a veterinary facility.
- There may be decomposing animals that must be examined to preserve any evidence.
- There are certain on-scene tasks that must be performed in order to determine the time of death.
- An investigator should interview the owner and any witnesses to determine when the animal was last seen alive.
- A rectal temperature should be taken of all deceased animals. A minimum of two readings should be taken over a 1-hour period to determine the rate of cooling.
- Any entomological evidence related to the body should be gathered at the scene
- It should be noted if the animal was in direct sunlight, shade, under any cover, or exposed.
- A determination of the state of rigor and lividity should be made and recorded.
- Paper bags should be placed on the feet of all deceased animals at the scene. Plastic bags should not be used, especially if the body is placed in a cooler prior to examination because of water condensation, which can destroy potential evidence.
- The body should be wrapped in a clean white sheet and then placed in a clean body bag or plastic bag prior to transport.

Weather Data

- The environmental temperature, either with indoor or outdoor settings, must be recorded, noting the time it was taken. A temperature should be taken of the general area, where any deceased animals were found, and at the level of the body. This information is critical to time-of-death estimates. It also can be a factor in neglect charges.
- Often, the first responders open doors or windows, which changes the enclosed structure's temperature; however, temperature information can be determined.
- Was the air conditioning or furnace on; if so, at what setting? Confirmation should be made that the power is on and that the heating and air conditioning works.
- If it has been turned off, or the power has been disconnected, it is important to get the outside temperature. The times of any temperature readings must be noted.

Photography and Videography

- Photography is the most important function at a crime scene. Photographic preservation of evidence helps minimize the possibility of overlooking something important.
- In some cases, a crime scene unit may be called in to take general photographs and video. The veterinarian may need to take additional
- Photographs, especially close-ups, of evidence as well as the individual animals and any injuries.

- It is important to take pictures of the general area, the animals, the housing, all areas the animal could have access to, any insects on the animals, any fluids, weapons, and anything else pertinent to the case.
- The type of camera used is important. Digital cameras are ideal because the quality of the photograph is immediately evident. There are digital SLR cameras with interchangeable lenses that are great for clear close-up shots.
- For 35-mm cameras, the negatives must be kept to authenticate the photographs in court. For digital cameras, the pictures may be copied and preserved on a CD for authentication and the digital card may be re-used.
- Photographs should be first of the general area and then more detailed. It is standard to start with photographs of the home or facility, including a picture of the address. A photo log should be kept for each picture taken. When taking pictures of the animals, there should be a card next to the animal identifying the case number, animal ID number, and date.
- Ideally, a video should be taken of the scene and the animals. It is important to show the condition of the animal, such as weakness, limping, injuries, or vocalizing. When taking video, it is important to inform the others at the scene so they can minimize other noise.

Evidence Recognition and Collection

- Crime scenes are complex and chaotic, requiring systematic evaluation. The recognition of evidence involves the ability to identify probative evidence that is among irrelevant or unrelated evidence at the scene.

- It is especially important to recognize evidence that warrants further testing. At the beginning of the case it is difficult to know what evidence may become important to the case.
- Often the seemingly most insignificant piece of evidence breaks the case wide open.
- When evaluating the scene for evidence, the investigator is looking for evidence of what happened, what can support or refute the suspect's statement, and evidence of how the animal was or was not cared for.
- A diagram of the scene should be done describing and assigning a name to the different areas.
- Any evidence collected must be labeled with the location it was found. All evidence collection should be done with the proper equipment and follow the proper chain of custody procedures.
- Evidence should be gathered that points to the length of time for the conditions of the animals and their injuries. Any evidence that shows ownership or how the suspect obtained the animals should be collected.
- The refrigerator and freezer should be searched for medications. The housing of the animal, the availability and condition of the food and water, and the appropriateness of the food should be recorded.
- The level of the water should be measured from the surface to the top of the container to determine if the animal could reach it. The area should be searched for any extra food the owner had to feed the animals and the expiration date on the package.

- It is important to ask the owner what food they normally fed the animal for comparison of vomit or stomach contents, especially in suspected poisoning cases.
- If the animal was tied up, the length of the tie should be measured and collected with the knot intact. If a chain was used, it should be collected and weighed. Any dog house should be evaluated for size and condition. The bedding and underneath the dog house should be searched for hidden evidence.
- The scene should be examined for bodily fluids, such as vomit, urine, or feces. The lack of feces can be indicative of starvation and the lack of urine can be indicative of dehydration. The condition of the feces should be noted, such as diarrhea or formed, and fresh or moldy.
- Animals often lose bladder and bowel control under extreme fear or distress. Samples of any urine, vomit, or feces should be collected.
- The feces should be inspected at the scene and for the following 24 hours for evidence of any foreign material. When animals are starving they may exhibit pica and ingest inanimate objects
- The investigator should look for blood and note the location and quantity by taking measurements of the blood stain (see Blood Evidence). Samples should be taken of any blood found. If blood has soaked into an absorbent surface, the item may be weighed and compared with a clean similar item as a control. The difference in weight provides the estimated blood volume loss (1 kg _ 1 L).

Blood Evidence

Overview

Although in-depth interpretation of blood spatter and blood stains require extensive training, an investigator should be able to look at blood patterns and make some basic deductions. Veterinarians have the best understanding of animal behavior and therefore should be most qualified to re-create the events of the crime. It is imperative that veterinarians be able to understand basic blood spatter analysis to analyze crime scenes, crime scene photos, and assist law enforcement. Interpretation of blood spatter can reveal the position of the victim, attacker(s),

presence of a witness, type of weapon used, number of blows, movement of the victim and/or the attacker, height of the attacker, and sequence of events. This evidence can determine the events, what did not occur, and the presence of other individuals at the time of the event. When analyzing a crime scene, the absence of blood spatter is just as important as the presence of blood spatter. It should be recorded where blood is present and where it is not, such as where something blocked the spatter, creating a void. One needs to look for voids where the victim, attacker, or an object blocked the blood from striking a surface. This also can help determine if a crime scene was staged. It is important to gather all the evidence at the scene for a blood spatter expert to analyze. A diagram should be made of all blood spatter and photographs taken using a photographic scale. Measurements should be taken from the floor, walls, and/or ceiling. Additional measurements should be taken from the nearest object to the blood drop. The width and length of the blood drop should be measured. The shape and characteristics of the drops should be documented

Blood Stain Analysis

An investigator can interpret certain basic patterns at the scene, such as drag marks, smears, or blood trails. There are some generalities of blood stain interpretation. When a drop of blood falls to a smooth floor, it remains basically spherical depending on the velocity. If it lands on a rough surface, it may appear star shaped.

Drops that fall from great heights also are star shaped. If the blood strikes the wall at a right angle, it is round. Blood drops that land at other angles are elongated, with the narrower part indicating the direction of travel. The blood drop may have a wave cast-off as it lands, giving the appearance of an exclamation point.

The direction of the smaller drop of blood indicates the direction of travel.

Passive bloodstain patterns are drops formed by the force of gravity alone.

Transfer or contact stains are formed when something comes into contact with the blood and transfers it onto another surface. Projected blood stains are created when an exposed blood source is subjected to a force greater than the force of gravity. These may be externally or internally produced, such as with an arterial spurt. A flow pattern may be seen whenever there is a change in the shape and direction of a blood stain caused by the influence of gravity or movement of the object. A drip pattern is the result of blood dripping into blood. A perimeter stain consists of only its outer periphery, the central area having been removed by wiping or flaking off after the blood has partially or completely dried. A swipe pattern is caused by the transfer of blood from a moving source onto an unstained surface. The direction of travel may be determined by the feathered edge. A wipe pattern is created when an object moves through an existing stain, removing

and/or altering the bloodstain's appearance. It is important to keep in mind that an injured animal may be mobile and may shake his head or body, causing spatter. Sneezed blood may be diluted or have air vacuoles, creating ghost drops or bubble drops. Ghost drops, or ghost-centered drops, are hollow-centered blood drops. They are formed when blood mixes with air, creating an air bubble; this eventually pops, leaving a hollow center. Ghost drops are indicative of coughing or vomiting blood and can be a mixture of sizes. Insects can cause blood artifacts moving through the blood and creating a false blood trail.

Blood Spatter Analysis

Blood spatter can be categorized based on the size of the blood drops, which is directly related to the force that caused the spatter. The velocities of blood spatter refer to the force that caused the blood to move and is measured in feet per second (fps). It should be noted that there is a gap of 25–100 fps in the following categories. The analysis of these blood drops requires interpretation of events such as weapon acceleration.

High-Velocity Blood Spatter

High-velocity blood spatter (HVBS) are drops of blood propelled by an explosive force greater than 100 feet per second. The blood spatter droplets are less than 1 mm in size, and the spatter pattern is usually called a mist. HVBS does not travel

far because of the small mass and resistance of air. Tissue fragments may be propelled further because of their larger mass. These are seen usually with gunshots, explosives, machinery, expired air, coughing, and sneezing.

Medium-Velocity Blood Spatter

Medium-velocity blood spatter (MVBS) are drops of blood propelled by an external force of greater than 5 feet per second and less than 25 feet per second. The drops are usually 1–3 mm in size. Usually they are caused by blunt- or sharp-force trauma, such as stabbing, weapons, punches, arterial spurts, and some cast-off from weapons. Arterial spurts create a large amount of blood that can be confusing to interpret. The victim may still be under attack while bleeding, creating overlying patterns. There may be swipes caused by transfer from the attacker or the victim falling against the arterial spurt pattern. Or there may be swipes where blood is transferred and smeared from the attacker or victim's body or clothes. The arterial spurt pattern is created by the contraction of the left ventricle of the heart. It creates an arcing pattern because it begins with low pressure that then increases followed by low pressure again.

Weapon cast-off blood spatter, also known as cast-off, is caused by blood flung off a weapon when the weapon is swung upward or backward. These blood stains may be elliptical or oval. They are more spherical when the blood hits at a 90-degree angle to the attacker. When the body is struck with a weapon, there is no cast-off blood spatter from the weapon with the first strike. The first strike initiates the bleeding. The second and any subsequent strikes to the now-bloody area produce cast-off blood spatter. This cast-off can be very small and easily

missed at a crime scene. Cast-off also can be classified as low velocity blood spatter if the drops measure 8 mm or larger.

Low-Velocity Blood Spatter

Low-velocity blood spatter (LVBS) is caused by a force less than 5 fps, equivalent to the force of normal gravity. These drops usually measure 3 mm or higher. They are most often caused by blood dripping from someone who is still, walking, or

Crime Scene Investigation 25 running. Blood dripping from a body or object usually falls at a 90-degree angle, forming a sphere when it hits a flat perpendicular surface. The sphere is usually smooth if the surface is smooth. Spikes or crenations may be caused if the surface has texture, or several drops repeatedly land in the same spot or fall from a distance. When blood falls from someone who is walking or running, the blood stain is more elliptical or angular with a point at one end. These stains also may have a wave cast-off, a smaller drop. The point of the angular drop and the wave cast-off indicate the direction of travel. Larger pools of blood may be seen if the animal or person who is bleeding paused in one area.

Grave Detection and Excavation

The veterinarian may be called to assist the detection and excavation of a grave suspected to hold the body of an animal. A forensic anthropologist or archeologist

should be called to the scene to assist in these cases.

Stratigraphy

Stratigraphy refers to the study of the soil layers in a grave. These are different than the surrounding natural and undisturbed area. One cannot dig a grave and then fill it back in without causing disruption to the normal soil layers. The top layer may

be mixed with deeper layers. The fill may contain artificial layers such as lime.

These layers can help determine how the evidence became interred. When excavating a grave, care should be taken to document these layers through photography and sampling.

Tool Mark Evidence

Tool mark evidence may be present at a grave either subterranean or on the surface

ground. These marks are important forensically in that they indicate the tool or

tools used to dig the grave. They may provide insight to the planning if the tool used was one of opportunity and readily available at the grave site. If more than one tool was used it may indicate there was an accomplice. Subterranean tool marks have been found many years later and under different environmental conditions. The type of soil affects the retention of tool marks; gravels or dry sandy soils are the least conducive. Handle tool marks may be at or near the

surface edges of the grave. As the grave is dug deeper with a long tool, such as a

shovel, the edges of the grave may be used as a fulcrum for the handle. This can

indicate the position of the person digging the grave. The back fill dirt may hold

tool marks such as in clumps of sod or clods of soil. The surface of the grave often contains hand, shoe, or knee impressions. These impressions are unavoidable in digging a grave regardless of the instrument used. When using small digging tools, the digger must kneel or sit around the edges. Fabric impressions on the soil may be found where the person was kneeling. When using a shovel, the digger must put his foot on the top of the blade, often leaving heel impressions. These may be misinterpreted as two different instruments used instead of the heel of the foot in conjunction with a shovel blade tool mark. Often the digger stomps or tamps the surface to pack the fill, leaving shoe or foot impressions.

Bioturbation

Bioturbation refers to the environmental factors that turbate or naturally churn, displace, or modify the position and nature of the remains (Hochrein 2002). These can include plant (floral turbation) and animal (fauna turbation). The animal factors include rodent burrows, which are often at the upper edge of the grave pit. The species of animal and the behavior of burrowing related to season may be used to determine timelines related to the burial. These burrow routes also may be examined for scavenged evidence. The roots inside a grave also may provide evidence of the instrument used to dig the grave. Large, broad-bladed tools, such as shovels, tend to tear and slice the roots at the edges of the walls. Thinner-profile tools, such as pitchforks and crowbars, tend to preserve root networks within the grave usually located beneath the body.

Burial Features

Burial features are affected by water as well. The pooling, evaporation, drying, and fine sediments in an open and filled pit create patterns of cracking. Surface cracks may occur at the edges of the grave, helping to identify the boundaries for excavation. The cracks may disappear with rainfall but consistently reappear along the same margins. In contrast, sedimentation over shallow or flat surfaces causes patterns of cracking in irregular, spider web–like fashion. The width of the cracks should be measured. In dry conditions, the crack widens at a rate of pproximately 1 mm per day. Heavy rain falls cause rapid closure of these cracks. By using the soil type and historical weather information, it may be possible to determine the post-deposition time interval to the last rainfall. Compression and depression of the burial site change the surface contours in and around the pit.

These changes may be seen through aerial or pedestrian observations. Primary depressions occur as the fresh fill dirt settles. Secondary depressions are caused by the body bloating because of putrefaction and then collapsing of the abdomen as the gases and fluid are released. The body must be placed in such a way that the bloating and subsequent collapse causes the change within the burial pit and enough time has elapsed for the putrefaction process to have occurred. If a body has been placed in a container, the secondary depressions also may be created as the container deteriorates. Holes dug by scavengers may be misinterpreted as secondary depressions. As time goes on, the evidence of both primary and secondary surface depressions becomes more subtle and harder to detect. Eventually, all evidence may be obliterated by bioturbation, sedimentation, and other changes to the landscape.

Excavation of the Grave

Excavation techniques of a grave must take into account the forensic nature of the case. It must be systematic, consider the environment and context of the burial, and take precautions to look for all evidence and document accordingly. The grave should be examined initially for defects and trace evidence. A grid should be established using a three-dimensional system around the immediate grave site boundaries to use as recording points for evidence collection. This should take into account the surrounding topography and elevation, which should be documented to address groundwater flow, surface erosions, and biota .The surface should be examined for evidence of surface sedimentation and cracking, bioturbation, compression and depression, and tool marks. Botanical and entomological samples should be taken. Progressive defoliation of the surface of the grave site should be done taking plant samples. Plants surrounding the area beyond the grave site boundaries should be taken for comparison. The removal of vegetation reveals the surface of the grave, allowing for closer examination.

Photographs of all samples should be taken prior to their collection and their location should be documented in relation to the grid system. A small rectangular window should be excavated at the center of the site down to the level of the remains. This allows a quick assessment of the depth and stratification of the soil. All excavated fill must be sifted through screens for evidence related to weapons, projectiles, ligatures, textile materials or other trace related to the suspect, the cause of death, and what was used to transport the body. The excavation should proceed from the center outward, keeping one to two sides of the box intact to maintain a profile section above and across the remains. Any evidence or stratigraphic change should be photographed and mapped. To map the area, a

transparent plastic sheet may be placed across the section and the evidence and strata traced onto the sheet. Heavy equipment should be avoided if at all possible. The tools used to excavate should not be something the suspect may have used, to avoid contamination of the scene with false or confusing tool marks. A wet dry vacuum should be used to evacuate the excavated fill from the burial site and the contents screened for evidence. Care should be taken to avoid ground water or purge contamination.

Examination of the Body

An important consideration is whether or not the victim was alive when buried. Depending on the amount of decomposition and if the body was wrapped, it may be impossible to determine if the soil was inhaled. Evidence of internal compaction has been seen in cases of humans buried alive. The soil was compacted where the victim pressed on the sides with bound legs; their shoulders, knees, or any other area of the body could have moved side to side or bottom to top. Scratching and biting of any external wrapping and the compacted areas may be expected findings with animals. The animals may have frayed or broken nails on their feet. Any roots in the grave and around the body should be noted. The position of the roots and the penetration in and around bones should be documented. These roots should be collected for analysis of the rate of growth. This can provide a time line for the date of interment of the body.

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Chapter 2 Injuries

Overview It is a breach of continuity in any body tissue, whether it is external as skin or mucous membrane or internal as muscles, bones or viscera following application of violence to the body. According to legal point of view wounds may be classified into, simple wound, a small superficial one, which heals rapidly in less than 20 days without leaving any permanent disfigurement or infirmity; dangerous wound, is extensive and takes more than 20 days to heal or heals in less than that period but leaves a permanent infirmity or lies in a dangerous place of the body, thus being difficult to treat and fatal wound which leads to death immediately or within a short period. According to their extent, mode of infliction and medico legal importance may be classified into, abrasions, contusions, incised, stab (punctured - penetrated), and lacerated wounds. in case of wound examination points must be clarified are type of wound and causal instruments; whether ante-mortem or postmortem; time passed between infliction of wound and death and cause of death and its relation to the wound. A careful and complete description of every wound separately is necessary and as regards the nature of the weapon one should state that a similar weapon could cause a certain wound. Sometimes, however, one may be allowed to be more dogmatic in ones statements if apiece of the weapon is broken in the wound and is found to fit accurately the seized one.

In examining a case for wounds one has always to be very careful and exact in his description. This should comprise the number of wounds, their position,

nature, direction, depth, character of edges and base, presence of foreign bodies, amount of hemorrhage and the appearance of inflammatory reactions or color changes must be recorded. These data should be written immediately with appropriate sketches. If possible photographs of the place and each wound to be taken separately. Actual measurements of each wound are to be always undertaken as regards the width, length and depth. A complete postmortem examination should be done in case of dead bodies, taking care to avoid interrupting or changing the present injuries whether these be superficial or deep. The incisions of dissection are to be adapted for each particular case, to clear the cause of death which may be disease, injury or poison. It should never be tried to compare a seized weapon with any wound by introducing the weapon in the latter. This may change the shape and size of that wound. The instrument may be thus stained with blood. The cause of death must be clearly ascertained and the relation between the injury and death must be clarified.The presence of any disease that might cause death or predispose to death from minor injuries should be mentioned. If the body shows more than one wound, the fatal one should be stated if possible.

A- Non-penetrating Injuries

1- Abrasions (Scratches)

Abrasions involve injury to the epidermis resulting from compression and destruction of the epidermal layers or friction against a rough surface. Antemortem abrasions are red to reddish-brown in animals. In humans, postmortem abrasions are yellow, translucent, and have a parchment-like appearance. Abrasions may be caused by blunt force trauma from a weapon,

dragging, or crushing and tearing of the skin. Abrasions may have gross or microscopic debris embedded in the wound related to the object that caused the injury or the area in which the injury occurred. The three categories of abrasions are scrape or brush abrasions, impact, and patterned.

a) Scrape/Brush Abrasions

Scrape or brush abrasions are created when a blunt object scrapes off the superficial layers of the skin, sometimes exposing the deeper dermal layers and causing fluid leakage from the vessels. This causes a serosanguineous fluid covering on the abrasion. When the area is incised, there usually is no hemorrhage in the underlying soft tissue, indicating the injury is confined to the epidermis. Grating or sliding abrasions are caused when the body slides across a surface such as a pavement. Ligatures, nooses, and dragging a body across a rough surface cause scrape abrasions. It is theoretically possible to find a bunching of epidermis at one end of the abrasion indicating direction of movement, but this is not commonly seen in humans.

b) Impact Abrasions

Impact abrasions are crushing injuries caused when the blunt force impacts perpendicular to the skin. This may occur from a weapon or a fall. Impact abrasions usually affect the skin over bony prominences in which there is less underlying protective tissue.

c) Patterned Abrasions

A patterned abrasion shows the imprint of the weapon or surface that caused the abrasion. This may be seen with ligature injuries that leave a patterned imprint

revealing the type of ligature used. In humans, intermediary material on the body such as clothing may leave an imprint from the crushing force of the weapon used.

d) Artifacts

Postmortem artifacts may be misinterpreted as abrasions. Insect feeding postmortem may be mistaken for abrasions. The drying of areas of skin may resemble abrasions. Careful examination should allow the differentiation between true abrasions and artifacts.

Dating Abrasions

- In humans, the dating of abrasions is possible with histological examination by documenting the stages of healing.
- The first stage is scab formation. The deposit of serum, red cells, and fibrin indicate survival after the injury. Infiltration of neutrophils in a perivascular formation may start in 2 hours but is clearly visible in 4–6 hours, indicating the injury is 4–6 hours old. Under the area of epithelial injury, a zone of infiltrating neutrophils in the bed of the scab is present by 8 hours. A surface zone of fibrin and red cells, followed by a zone of infiltrating neutrophils, and then a layer of damaged abnormally staining collagen appear by 12 hours. In impact abrasions, the surface zone is comprised of crushed epithelium.
- Epithelial regeneration marks the second stage of healing. The regeneration comes from the margins of the abrasion and the surviving hair follicles. In scrape abrasions, this growth of epithelium may appear in as little as 30 hours. In most other abrasions, it is visible by 72 hours.

- The third stage of healing is subepidermal granulation, which occurs only after epithelial covering of the abrasion is complete. Perivascular infiltration and chronic inflammatory cells are present. It becomes prominent during days 5 to 8. During days 9 to 12, changes in the overlying epithelium are most prominent as it forms keratin and becomes progressively hyperplastic. Collagen fibers may begin to appear. The fourth and final stage of healing is regression, which starts around the 12th day. The epithelium is remodeled, becoming thinner and atrophic. The collagen fibers become more prominent. The vascularity of the dermis decreases, and a definitive basement membrane develops.

Medico-legal importance

1- Suggest the cause of injury or crime, e.g. nail abrasions around the throat suggest throttling, around the mouth and nose suggests smothering and about the thighs and genitalia suggest rape or sodomy.

2- Dating Abrasions

3- The pattern of abrasions may gives a fair indication of the causal agent, fingernails cause small semi lunar abrasions, if the body is scratched with the nail, long linear ones are produced.

4- The presence of abrasions around the edges of wounds of the scalp and other wounds is a clear differentiating evidence between contused and cut wounds

5- Abrasions inflicted after death leave a whitish surface, which soon dries, and become brown and parchment like, thus simulating an ante-mortem

abrasion, but without any hyperemia or extravasations of blood around. Postmortem abrasions may results from ants and cockroaches, etc., eating the cuticle of dead bodies especially around the mouth and nose. Spontaneously around the scrotum or anus from the irritation caused by urine or feces, which may, expelled by rigor mortis.

2- Bruises (Echymoses or contusions)

A contusion or bruise is an area of hemorrhage caused by blunt force trauma that ruptures the blood vessels. The size and severity of the contusion depends on the blunt force applied and the vascularity of the tissue. Because animals have a reduced blood supply to their skin compared with humans, external bruising is not as commonly seen on the surface of the skin. When it is seen in animals, it is usually caused by severe force that may not only cause bleeding in the skin but also in the underlying tissue structures. Other causes for apparent bruising include a variety of clotting disorders. In addition, the apparent surface bruise may actually result from bleeding that has followed the path of least resistance through tissue from a deeper or adjacent area of injury such as a fracture. Contusions also may be present in the internal organs and internal body walls. The appearance of bruising may be delayed for several hours, especially if it is located in an anatomical site in which there is reduced blood supply. Therefore, continued monitoring of the live animal is essential to detect bruising. The absence of a bruise does not indicate the absence of blunt force trauma. A live animal may have suffered severe blunt force trauma but not have any external signs except for tenderness in the injured areas, which may be difficult to discern. In these cases as

with all suspected cases of abuse, radiographs and blood work may reveal evidence of acute and chronic injuries.

Postmortem Findings

Postmortem exam can reveal the true extent of the bruising, which is usually quite a bit larger than what was apparent on the surface. The fur may need to be shaved to look for bruising. It is possible to see bruising even in dark-pigmented skin. Bruising may take minutes to several hours to form in animals and may fade quickly depending on the extent of damage. Photographs must be taken of the bruising pattern hourly as they form to capture the full representation. It is possible that early on the bruise may be more reflective of what caused the bruise and as bleeding continues, especially from the deeper tissue, the pattern may become obscured. The skin should be reflected over the entire body to look for subcutaneous or deeper tissue bruising. At first appearance, contusions may be grossly difficult to differentiate from postmortem lividity. A contusion involves hemorrhage into the soft tissue and the blood cannot be wiped or squeezed out when incised. This is not the case in areas of lividity. Over time, decomposition can make it extremely difficult to differentiate antemortem bruising and lividity. Hemolysis of the red blood cells creates diffuse discoloration of the soft tissue. The blood within the vessels and the erythrocyte leakage caused by the breakdown of the blood vessels from decomposition hemolyze. The erythrocytes in the soft tissue from antemortem bruising also hemolyze, making it impossible to distinguish from an area of livor mortis.

Patterned Bruising

A patterned contusion may be seen that reflects the shape and sometimes detailsof the object that was used to cause the bruise. Asymmetry to the bruise may be a clue because bleeding follows the path of least resistance. Depending onthe area of skin and gravity, it tends to flow from the center outward, forming irregular or blurred lines. If the contusion was caused by a weapon, it may be possible to find evidence of the tool in a bruising pattern or indentation of the skin, tissue, or even bone. If a flat object is used, there may be two parallel lines of bruising. If a rod was used, the contusion pattern is linear in shape. Rods and similar objects also can cause skin lacerations if used with enough force or if the skin is more susceptible to tearing, such as with young or geriatric animals. The imprint of a shoe or fingertips may be seen from grabbing or holding the animal. Bruising may extend beyond the site of impact, obscuring the pattern created by the object. It is important to find out if investigators have a suspected weapon that was used in the incident. A weapon may be identified or discovered later in the investigation, so it is important to preserve any evidence that may be linked to the offending object for later comparison or confirmation. The patterns and indentation of the skin may fade or resolve very quickly, so it is important to take photographs immediately. A photographic scale must be placed next to the contusion for a forensic specialist to use the photograph for later comparison with a suspected weapon. For any indentations, a cast should be taken. Mikrosil is a rubber casting material that is inexpensive and easily used for this purpose. It may be ordered from any forensic supply company. It comes in a variety of colors, although brown is the preferred color for tool mark examiners.

Dating Bruising

The healing of a bruise is dependent on the number of bruises in the area, vasculature of the injured area, amount of subcutaneous tissue and fat, type and severity of the force that caused the bruise, and any underlying disease that may impair local tissue reactions. Contusions undergo color changes over time because of the breakdown of hemoglobin, but the color change and time for change are variable. In animals, bruising initially may appear red, purple, or dark blue. As time progresses the bruise may fade and turn brown. At best, one can say the bruise appears recent or is older. It is possible to cause contusions postmortem if a severe enough blunt force is directed at the body within a few hours after death. This causes rupture of capillaries and forces blood into the surrounding tissue. Ante mortem contusions that occur immediately prior to death may not have had enough time to show a vital reaction detectable by histology. If the contusion occurred ante mortem with enough time for the body to mount a response, evidence of a vital reaction may be seen with microscopic examination of the injury. . Sometimes after putrefaction, hemolysed blood permeates into the tissues and stains them red, which may be mistaken for a bruise. Usually a bruise resists putrefaction for some time and in early-putrefied bodies it appears as a collection of blood and not a diffuse red staining.

3- Fractures

Direct Force

Fractures may be caused directly or indirectly by blunt force trauma. When a long bone is struck, it tends to bend, causing a fracture in the opposite side of the bone where there is greatest tension on the convex aspect of the bend. With significant force, crushing of the concave side of the bone can occur. Focal or crushing

fractures can occur because of blunt force trauma depending on the amount of force and size of the area to which it is directed.

Indirect Force

Indirect fractures are caused by the application of force distant from the fracture site. Bone is weaker to tension forces than compression forces. The classifications of indirect fractures are traction; angulation; rotational; vertical compression; angulations and compression; and angulations, rotation, and compression

4- Evidence of Long-term Abuse

The presence of multiple fractures in different stages of healing is highly indicative of repetitive abuse. There may or may not be bruising on the skin. If the fracture is acute, there will be hemorrhage from the marrow and adjacent tissue injury in the immediate vicinity of the fracture site and into the surrounding tissue. The rate of healing callus formation depends on several factors: the age of the animal, amount of displacement of the fracture site, and stability or amount of movement at the fracture site. Production of a callus begins with the external callus produced by the periosteum and the internal callus produced by the endosteum. Periosteal proliferation may be underway within 24 hours of injury. Mineralization can begin in 3 days, but this early fibrocartilaginous callus may not be visible in radiographs for 2 weeks. It may be palpated as a hard thickening but it must be differentiated from a resolving hematoma. On postmortem exam a gross and microscopic exam of the fracture area should be conducted to determine the stage of callus formation. At best, a time estimate for the fracture may be given as acute, recent, or older.

5- Hit-by-Car/Motor Vehicle Accident Injuries

HBC victims have characteristic injuries related to the incident. The impact of the vehicle to the body can cause several injuries depending on where the animal was hit, if the animal rolled under the car, how far it was flung, and where it landed. In addition to the blunt force injuries, the animal will have frayed nails, dirt or debris embedded in the fur or mouth, and abrasions. These are usually drag or sliding abrasions located on more than one area of the body. They are almost always found lateral on one side of the body and medial on the other side. Primary impact injuries are caused by the first impact of the vehicle to the victim. After this, the victim may be thrown under, flung to the side or up onto the car, causing secondary impact injuries. The animal may have further injury from the undercarriage or wheels of the vehicle, and blunt force trauma from landing on the ground or on top of the vehicle. This may include head trauma, additional fractures, abrasions, debris, or glass from the site of injury embedded into the fur or wounds. If the animal had a secondary impact with the vehicle, then the final contact with the ground causes tertiary injuries. If the animal is lying on the ground and run over by a vehicle, then these types of impact injuries are not present. Instead, the expected injuries are crushing and/or flaying of the body where the vehicle contacted it.

6- Fall Injuries

When evaluating victims of falls, it is important to consider how the animal fell, the distance, the mass of the animal, and the animal species. In addition, it is important to know how the animal landed and if the animal could have landed on

any objects. All the information regarding the fall is crucial to properly analyze and interpret the cause of the injuries. With injuries caused by falls, it is important to consider that it was not accidental but possibly the animal was thrown or dropped by a human. If the animal accidentally fell, one would expect the animal to have frayed nails from frantically clawing at the edges before or during the fall, depending on the circumstances and surrounding surfaces. This may or may not be present in accidental falls. However, it is important to consider that the fall may have been non-accidental if the nails appear normal. The animal should be examined for other injuries that may be inconsistent with the fall and are more indicative that the injuries occurred prior to the fall. To know what injuries to expect, it is important to know how and on what the animal landed. A fractured lower spine is not expected in a cat that accidentally fell off a one-story balcony to the asphalt. In suspected animal cruelty cases the animal always should be examined for evidence of repetitive abuse. High rise syndrome is a term given to a complex of injuries associated with cats that have fallen from a significant height, usually 2–32 floors. The triad of injuries associated with this syndrome is epistaxis, fracture of the hard palate, and pneumothorax. Findings also can include pulmonary contusions, limb fractures, and possible bladder rupture resulting from the acute increase of intra-abdominal pressure. The hard palate fracture or split is caused by the force of the lower canines being thrust upward between the upper dental arcade.

B- Penetrating Injuries

Forensic classification of penetrating injuries, excluding gunshot wounds and other projectiles, are lacerations (split wounds), incised wounds (cuts), and stab wounds.It is important to know if law enforcement has the instrument or weapon suspected to have caused the injury or death of the animal. When evaluating penetrating injuries, the veterinarian must consider that the investigators will eventually have the weapon and take steps to ensure that evidence is collected from the animal for comparison.

1- Lacerated and contused wounds

Lacerated wounds are open injuries as a result of a tearing or splitting of the tissues. They include deep damage to the skin and may involve the subcutaneous and muscular layer. They are often associated with injuries to internal organs. They occur most commonly over bony prominence where the skin is crushed and split against the bone. Blows cause them from blunt instruments, such as sticks or stones or by falls on the ground. The edges are irregular, the angles are not sharp, the base is uneven and usually there are few bruises and abrasions in the edges and surrounding parts. At the base tags of tissue, especially vessels and nerves, may be still seen crossing from one edge to the other. Slight external bleeding but it is very liable to sepsis. The hairs are seen compressed to the edges of the wound and may be crushed but never sharply cut. Healing takes place by second intention. Lacerated wounds, especially over the animal head may simulate cut wounds but by hand lens, the irregularity, bruising of the margins and crushed hair can be seen in the lacerated wounds.

Crushed wounds, is a special type of contused wounds, caused by the passage of a vehicle or heavy object over the body. A contused wound in tense skin, as in the scalp may like a cut wound, but careful examination reveals the irregular bruised edges.

2- Incised wound

These are wounds produced by sharp instruments (razor or knife). Drawing the edge of the sharp cutting instrument along the body surface makes it. The sharper the instrument, the cleaner will be the injury and the more typical will it be. They having sharply cut edges and cleanly cut appearance of the deeper tissues at the base. They are more long than deep, and there is no relation between the length of the wound and the instrument that caused it, nor is there any relation between the shape of the wound and the blade of the instrument, whether sharp on one or both sides. The extent of gaping of the edges depends on the direction of the cut and the elasticity of the skin. If the cut is in the direction of the underlying muscle fibers, the wound gapes less than if the cut is perpendicular to the direction of these fibers. Incised wounds bleed freely.

They are regular in shape and direction unless they are made in places covered with loose skin, where the wound may appear irregular (e.g. in the neck). Wounds from broken glass may simulate incised wounds, but usually the edges are irregular and show minute bruises, which could be seen by a hand lens. The examination of the hairs around the wound may help, as these will be sharply cut in case of incised wounds and crushed in case of contused wounds. Incised wounds mad by heavy instruments like and axe, grabber (Fass), sword and butchers knife are clean cut but deep and may cut bones underneath. It is usually heals by first intention with minimal scar formation. In about 12 hrs,

red swollen edges. In 24 hrs, proliferation of the vascular endothelium. In 36 hrs, the wound is covered with lymph. In 3 days their edges are strongly adhered. In about 7 days healing takes place leaving a red linear scar. If gross sepsis occurs, the wound may remain open almost indefinitely, and scars are larger and deeper.

The danger of incised wounds depends on the position of the wounds, so that wounds of the neck are more dangerous than similar incised wounds in the limbs, owing to the superficial position of the big vessels in the neck thus causing more hemorrhage, the presence of vital structures e.g. big nerves, liver spleen etc…and the infection by pyogenic organisms leading to either local or general septic complications.

3- Stab and punctured wounds

Stab wounds are more deep than wide and have sharp edges (both angles may be sharp if the instrument used was a double sharp edged one). They frequently take the shape of the instrument, especially if they affect moderately solid structures as cartilage. Unless the wound is enlarged during the withdrawal of the instrument, its length is usually smaller than the breadth of the blade and its width more than the thickness of the instrument. Stab wounds take the shape of the instrument that caused them, so that the wound will be triangular with one sharp and one broad lacerated end, if caused by a single sharp edged knife and in case it is caused by a double sharp edged one the two ends of the wound will be sharp. Stab wounds occurring in loose skinned areas as the neck and axilla may be angular or irregular in shape. The wounds may also be angular or irregular if the victim moves, while the instrument is still in the body, or if the

hand of the assailant moves before the instrument is withdrawn. More than one wound of the deeper tissues may occur with only one skin wound, if the victim moves before the instrument is withdrawn, or if the assailant only partially withdraws the instrument and thrusts it back.

Stab and punctured wounds, is a stab wounds may caused by pointed and sharp edged instruments as knives and swords, but pointed blunt edged instruments as nails, files and pokers cause punctured wounds. Punctured wound is differ from stab wounds in shape as they are not linear and sharp as stabs, but take the shape of the instrument that cause them so they are rounded if caused by a nail, rectangular or star shaped if caused by a rectangular nail or poker and rhombic if caused by scissors. *Transfixing and penetrating wounds* is a stab or punctured wound transfixes the whole body or limb from one side to the other, it is then called transfixing wound. If the stab reaches a body cavity as the abdominal or thoracic cavities it is called penetrating wound.

Documentation

When examining a victim of stab wounds it is necessary to assign a number to each wound and record the location of the wounds. This should be done in two ways. One way is to use a diagram of a dog or cat showing the shape and location of the wounds, and assigning a number to each wound. The second way is to record the location by measurements to the nearest landmark on the body such as the midline, the spine, or a specific nipple. The wounds should be identified with the corresponding number on the drawing and the appearance fully described. When examining stab wounds it is important to take measurements of the width

and depth of the wound. Photographs should be taken of each wound with a photographic scale next to the wound.

The danger of stab wounds:

1- Hemorrhage is more difficult to stop because it usually originates from deep vessels (ligature may need major operation) and hemorrhage may occur inside body cavities may be missed in its early condition or it may accumulate and press on vital organs as the heart and brain.

2- Sepsis because their depth and difficulty of cleaning.

3- Injuring of vital organs is more liable.

Medico-legal importance:

1- The presence of foreign particles or broken pieces of the instrument, when it is thrust against bone may help in identifying the instrument used.

2- Age of wound may be identified, after about 8 to 12 hours, the edges become red and swollen. In 2 or 3 days a sticky serous or purulent material covers the wound. In about 5 days, definite granulation tissue appears. Complete healing occurs in about 10 to 14 days, unless sepsis sets in when healing will be delayed.

3-They may help in identification of the instrument used as they usually take the shape of the instrument so that the wound will be triangular with one sharp and one broad lacerated end, if caused by a single sharp edged knife and in case it is caused by a double sharp edged one the two ends of the wound will be sharp.

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Chapter 3 Fire arm wounds

Injuries from firearms are common in Egypt. The veterinarian should be familiar with the different types of fire arms especially those types commonly used in crimes. Most animal gunshot victims have a common history of being outside and unattended. A gunshot study of animals revealed that most gunshot wounds involved animals that were allowed to wander outdoors unsupervised. In urban areas, dogs were more likely to be shot in the evening and early morning hours. Handguns were the most common firearm documented in urban areas. High-velocity rifles and shotguns were most common in rural areas. Gunshot injuries may easily be mistaken for puncture wounds, bite wounds, or lacerations. Any animal with unexplained wounds should have full body radiographs taken. Additional clues of a gunshot injury include pneumothorax, pneumomediastinum, cardiac tapenades, dyspnea, lethargy, limping, fractures, peritonitis, emoabdomen, and hemothorax. The animal also may present with symptoms of lead poisoning from a retained projectile in the body. This is commonly seen when a projectile is inside the joint, where there is slow dissolving of the bullet and slow absorption. The questions that may arise in an investigation of a fire arm injury are: 1) What kind of weapon was used? 2) From what distance the missile was shot? 3) From what direction the firing was made and what were the relative positions of the victim and the assailant? 4) Is the case accident or homicide? 5) If a weapon is seized; is it the weapon used in accident or not?.In order to understand and answer these questions one should have a thorough knowledge of the weapons commonly seen and the missiles one is likely to meet with every type of weapon.

Fire arm weapons and cartridges:

Fire arm weapons:

Firearm weapons are classified according to the length of the barrel into, long weapons, medium weapons and short weapons and according to the method of loading into, muzzle loaders and breech loader:

1- Muzzle loaders, are guns are filled or loaded through the muzzle. Through the use of capsules on the upper part of the barrel, at its breech end or to one side. The flint lock type in which there is a special apparatus carrying a piece of stone which is hammered upon by a pereussor and the result of such hammering is the evolution of a spark which goes inside the barrel to the loading resulting in firing of the ammunition. Such an apparatus is usually applied to one of the breech end, opposite to it directly there is an opening through which the flash passes. These are the old carbine guns which have either one or double-barreled. (Double barreling in the carbines is however rare). Most of these weapons are usually cut into two halves of the original length of the barrel so as to be easily carried. They are obsolete weapons no longer in use nowadays and their interest lies in the historical evolution of the manufacture of weapons and also in the museum. Their loading is usually of the home made variety and consists of different amounts of gun powder of the black type guarded with some wads which are usually pieces of rags of paper and then some homemade shots (cylinders or balls of lead) then the whole with another set of rag wads.

2- Breech loaders, the gun is usually loaded from its breech end. The gun is opened through some device either through a spring mechanism to one side or the use of a handle underneath. They are loaded with cartridges made of

brass or cartoon of different colors. The cartridges are either machine or home loaded. A cartridge containing the powder, wads, shots and cap into the breach end of the barrel. To fire such a weapon, one simply pulls the trigger (after raising the hammer if there is an external one), when the hammer will strike a needle at the breech block, which in turn strikes the percussion cap of the cartridge, producing the spark and igniting the powder. This group comprises rifled and non-rifled weapons

A- Rifled weapons (guns firing single missile)

They fire bullets (solitary shots). The rifling is the longitudinal ridges alternating with grooves that are running spirally inside the barrel purposely during its manufacture, either in clockwise or anti-clockwise direction. They produce a spinning missile or projectile, to direct the missile fired and gives it more power of penetration and a longer range of firing. Rifling marks or grooves, which are marks present on the surface of the fired bullets, which help in identification of the causal weapon as they are as individual as fingerprints. These characteristics are number, diameter and width of lands and grooves, depth of grooves and direction and degree of rifling twist. Comparison of the bullet recovered from the body with bullets fired from various weapons will enable the forensic scientist to identify the gun that was used. Similarly a cartridge case bears marks produced by the firing mechanism from which it is possible to identify the gun which it was fired. The presence of magazine markings, the types of breechblock mark, and the size, shape and location of ejector and extractor marks are important in making such identification. The size, shape and location of the firing pin on fired rim fire cartridge bases can

also be used to determine the mark of weapon. Rifled guns are classified according to the length into long, which is Service rifled weapons (automatic-non-automatic), and short, automatic pistol, which is loaded by automatic manner or Revolver, which is loaded by a revolving magazine (container).

B- Non-rifled (Guns firing a mass of small missile)

The non-rifled weapons have a smooth barrel from inside. They fire a collection of shots which may be a machine made or a homemade manufacture. They are all-long, include, sporting guns (shotgun) and gaffer guns (Greener- Schneider-Remington). The non-rifled weapons are weaker than rifled.

Fire arm cartridge:

Cartridge of fire arms is formed of tube, missile, percussion cap and powder. Missile is made of lead, percussion cap, is a smaller tube fixed in the center of the base of the cartridge (at the base of the tube) and it is filled with a paste formed of powdered glass to produce heat on friction, mercury fulminates as a highly inflammable substances and K-chlorate as a source of O2. There are two types of gun powders a) Black powder, is formed of 15% carbon, 10% sulfur and 75% potassium nitrate and when it is ignited it produces 300 volume gases causing high pressure, and leaving an alkaline residue in the form of carbonates, bicarbonates, sulfides and sulfates. In practical examination it is present in the form of black irregular small granules in a small test tube, which are staining the cover of the tube black. It is only present in Ghaffir's gun cartridges. b) Smokeless powder is formed of nitrocellulose or nitro glycerin. When it is ignited it produces 900 volume gases leaving a neutral residue in the form of nitrites and

nitrates. The most common type smokeless powder is called Carotid which is in the form of small brown cords and formed of nitrocellulose or nitroglycerine impregnated in gun cotton and jelly. It may be also in the form of small rods, scales or plates taking any color.

Table 1 Difference between black and smokeless powder

Black powder	Smokeless powder
Composed of 15% carbon+10% sulphur+75% nitrates	Composed of Nitroglycerine (liquid)+nitrocellulose (solid)
Presence of Old revolvers, ghaffir's guns (Schneider and Remington), and shot guns	Presence of Service rifles, automatic pistols, recent revolvers, shot guns and ghaffire's guns (Greener)
On ignition One volume give 300 volume of gases leaving alkaline residues	On ignition One volume gives 900 volume of gases, leaving neutral residues.
Date of firing can be estimated	Date of firing can't be estimated
Powder	Cordite, brown sticks composed of nitroglycerine, gun cotton and felly, Scales or Amorphous.

Non-rifled Weapon's cartridges:

The cartridge case is made of cardboard with a brass base and central percussion cap fixed to the base. The cap is a small cylindrical copper

container lined from the inside by a paste containing fulminate or cyanate of mercury, potassium chlorate and powdered glass. Such cap is struck by a needle, the mercury fulminate takes fire and a spark is produced. Immediately above the cap there is the powder (black or smokeless). The inner wad which is made of felt or thick cardboard. The missile is composed of different number of variable sized shots, depending on the type of intended shooting. The total weight of shots in each cartridge is about 30 g. External wad is mass of cardboard.

Results of explosion

When the trigger of a firearm is pulled the hammer or needle strikes the percussion cap. Spark is produced which ignites the powder which results in the production of a great volume of gases (200-300 volumes, per volume of black powder and about 800-900 volumes, per volume of smokeless powder). The gas try to escape through the only opening in the barrel (muzzle) pushing everything, shots, wads and unburned black powder in front of them. The powder does not always get completely ignited especially in case of black powder when particles of unburned powder are always present.

The explosion products

1- The gases come out in the form of blast and when entering the body, they expand in all directions causes tearing of the edges of the wound at the entrance. These gases travel for short distance (15 cm in long weapons and much less than this in short weapons.

2- Flame and smoke, produce burning and blackening of the body at the entrance. if the weapon has been fired within 1.25 meters in long weapons and

less than this in short ones have. These are much less marked in case of smokeless than in case of black powder. The burning is totally absent beyond few centimeters and the smoke does not blacken the target, as in black powder but only gives it a grayish metallic color.

3- The unburned particles of the powder (tattooing), strike the body irregularly resulting in the so-called tattooing. These particles are black in case of black powder and gray minute in smokeless powder. The tattooing reaches about 3 meters in long weapons.

4- The first wad (internal wad), It can penetrate the body at a distance of about 3 meters, reaches a distance of about 8 to 10 meters.

5- The second wad (external wad), penetrates the body for a distance of about one meter in long weapons. Is lighter and thus travels for a shorter distance, so that it travel for a distance of about 3 meters.

6-The shots or bullets, cause wounding of the body. The shape of the wound varies with the type of weapon, distance and direction of firing. These distances are only approximate; They differ very much according to the quality of the weapon, length of barrel, type of powder and relative proportion of powder and shots.

Rifled Weapon's cartridges:

It is formed of long (Service rifle) or short (pistol & revolver) brass tube and a long or short bullet made of lead, which is covered, with copper or nickel and it has a pointed end. The cartridge may have a rim around the base in case of

non-automatic weapons or a groove around the base in case of automatic weapons. All rifled weapon's cartridge is filled with smokeless powder. *Dum Dum bullet* is a bullet having defects in its cover, or it may contain explosive substances, which leads to another explosion on striking hard objects like bone.

Bore or Caliber (diameter of the barrel)

In rifled weapons, it is measured directly in mms or fractions of an inch and it equals to the diameter of the base of the bullet. In non-rifled weapons, it is measured in relation to balls made of lead and weighs a certain fraction of pound. E.g. Weapon No. 12 it means its diameter equals the diameter of a lead ball weighing 1/12 of a pound.

Determination the time of discharge of the weapon

It is possible only in case of black powder. A characteristic smell of burnt powder can be detected in the barrel up to 10-12 days after firing depending on the conditions to which the weapons has been exposed. A rough estimation is by the chemical examination of the residue in the barrel in case of black powder has been used. The muzzle of the weapon would smell strongly of sulphurated hydrogen (chemically detected within 20-30 minutes, by putting a moistened lead acetate paper over the muzzle of the barrel, if H_2S is present the paper gets blackened).

Examination of Gunshot Victims

It is important to have all the crime scene investigation information prior to examining a gunshot victim. This includes photographs, witness/suspect statements, and any recovered ammunition, casings, or weapon. All the wounds should be photographed before and after treatment. When examining a gunshot victim, it is important to consider that the animal may have suffered additional injuries, such as blunt force trauma. The skin of deceased animals should be reflected to look for subcutaneous bruising. All gunshot victims should have full-body radiographs to locate the projectile and injuries. The sequence of gunshot wounds often is hard to determine. The wound findings should be compared to witness and suspect statements. To properly interpret gunshot injuries, the veterinarian needs to understand wound ballistics. Gunshot wounds can be very complex and a large amount of information must be analyzed and documented.

The veterinarian must be able to classify the gunshot wounds as entrance or exit, recognize what can affect their appearance, and determine the gunshot range that caused them. The veterinarian also should recognize wound patterns associated with specific weapons and ammunition. It is important to properly retrieve gunshot residue and the projectile from the body, and cartridge casings at the scene without damaging the evidence. The trajectory of the projectile through the body must be determined and compared with investigation findings. Finally, the veterinarian must properly record the injuries and prepare the examination report. Most of the following information on gunshot wounds comes from human gunshot victims. The same principles and guidelines should apply to animals.

Wound Ballistics

The amount of damage caused by the gunshot is directly related to the amount of kinetic energy from the projectile that is absorbed by the tissues. The greater the energy transferred to the tissues, the greater the tissue damage. Kinetic energy refers to the energy possessed by a moving object. In gunshots, the projectile causes tissue to balloon outward, stretching and tearing in a process known as cavitation. Cavitation also creates a vacuum affect as it passes through the target, pulling debris and hair deeper into the wound track. This shearing, compression, and contraction continue as it travels through the body, causing injury to the surrounding tissue, sometimes distant from the bullet's path. Depending on the kinetic energy of the projectile, this may cause rib fractures without a direct impact from the bullet. In addition to the kinetic energy of the projectile, the degree of injury depends on the characteristics of the tissue through which it travels. If a high-velocity bullet passes through a leg without impact to the bone, the amount of damage depends on the kinetic energy transferred to the surrounding tissue. The thicker and denser the tissue, the more energy is absorbed. The lung and muscle are more resilient and elastic, which results in less damage because of the ability to absorb a portion of the cavitation process. A bullet can penetrate or perforate bone depending on the velocity, construction, angle of impact, type of bone, its thickness, and its surface configuration. Fracture patterns depend on the type of bone and angle of impact. Cancellous bone is softer and tends to absorb more of the energy, causing less fragmentation. Cortical bone tends to fracture and shatter. The angle of the bullet impact (such as 90 degrees versus tangential) and the weight and position of long bones also can affect the fracture pattern.

Fire arm wounds

Firearm wounds are characterized by a) loss of tissues especially in the entrance depends on the size of the missile and whether it is a bullet or shots. b) Presence of powder marks in form of burning blackening or tattooing around the entrance wound in the skin. c) Presence of two wounds, entrance and exit, unless the missile enters and remains in the body. In such case, the missile can be easily seen under the screen (x-ray) in the living or by dissection in the dead. d) if a flat bone is perforated e.g. skull, the rounded perforation with beveling of the bone is characteristic. The shape of the wound always has the previous characters, varies according to type of weapons (firing bullets or shots), distance of fire (effect of gases, powder or dispersion of shots, position of the wound (a bullet wound in an area with loose corrugated skin as the scrotum may appear irregularly lacerated, whereas in other positions as the limbs or trunk, the wound appears as a nice circular opening or slit) and direction of the fire, which if perpendicular to the surface of the body, causes a rounded wound, and if slanting, causes an oval wound or a longitudinal track with loss of substance (this latter type of wound may be mistaken for contused or incised wounds).

Wounds of rifled weapons

A single missile traveling at high velocity produces these wounds.

Determining Gunshot Range.

Gunshot range is defined as the range from the muzzle to the target. There are four categories of gunshot range: contact, near-contact, intermediate, and distant.

Characteristic findings of gunshot wounds help define the distance from which the animal was shot

1- Contact or close range wound

When the muzzle of the weapon is held against the surface of the animal body at the time of discharge. Skin at the point of entrance is torn often in a cross shape due to the expanding gases. Soot, powder and vaporized metals from the bullet, powder, and discharge gases are deposited in and along the wound tract, the last being found up 1-1.3 meter.

2- Near-contact or Close range firing wound

Explosive effects of soft and the high initial velocity of the bullets will produce hard tissues at entrance. If there is no resistance to their passage, the size and shape of the entrance and exit wounds may be very similar. The products of explosion (flame-soot-unburned powder) escaping from the muzzle with the missile will mark the skin around the entrance wound with a ring of blackening by soot and tattooing by fragments of unburned powder driven into the skin. Hair may be signed.

3- Intermediate range wounds

Clean punctured wound. The entrance wound is approximately the size of the causal bullet.

4- Distant range firing wounds

There are no any marks. The characteristics of the entrance wound are entirely due to the bullet. As the later penetrates the skin it inverts and abrades the margins. The inlet wound is irregular and lacerated owing to the loss of velocity. The wound will appear as a round hole with a band of abrasion

around the margin of the hole. In rifled wounds, it is important to distinguish between inlet and exit wounds in order to decide the direction of fire. *It is based on the following:*

Table 2. Differences between inlet and exit wounds

Inlet wound	Exit wound
More loss of substances with small splits or tears (microtears), radiating outward from the edges of the perforation	Less, and it is apparently tears in the skin produced by eversion of the skin
Inverted edges with a punched - out clean appearance	Everted edges with a cone like manner if bones or solid organs have been perforated
It may show evidence of powder marks as burning, blackening or tattooing.	No
Hemorrhage and laceration, are usually less	More
More regular	Less
The dermis collagen fibers adjacent to the wound stain from deep red to gray-blue and appear swollen and homogenous	Not seen
Embedded grains of powder may be seen in the skin adjacent to the perforation	No
Have a gray rim around entrance called bullet wipe or soiling that is the contamination of wound edges by the greases covering the bullets or oil carried on the bullet from the barrel of the weapon	No
The wound of entrance is small, surrounded by a reddish zone of abraded skin (the abrasion ring). It may be eccentric or concentric depending upon the angle between the bullet and the skin.	Large

The edges of the entrance may be everted in the following exceptions. By firing the weapon (point blank) or at few centimeters, the tissues are blown from inside out by the blast of air and gases, thus rendering the edges everted. If the entrance wound lies in a fatty area, the skin will contract by its elasticity, while the underlying fat will not and thus the wound appears everted. Putrefaction everts the edges by the liberated gases. Sometimes, the inlet may be larger than the exit as in very near injuries with tearing of the edges, in an oblique injury (the missile is small and passes only through soft tissues, and the exit having the appearance of a small slit in case of shot gun injuries, where the shots enter in one mass and most of them remain in the body, while only few of them come out. In case the missile strikes a flat bone, this is perforated, the hole being beveled internally at the entrance and externally with some radiating fissures at the exit. The size of the exit wound may be extensive with marked laceration. If the missile strikes a bone before it comes out (the bone will be comminuted and pieces there are pushed through the skin in front of the missile, causing this excessive laceration.

Determination the distance of firing in weapons firing bullets

It would be very difficult to estimate the distance beyond the range of the powder marks. Therefore one has to rely in such rough estimation on the degree of penetration of the bullet into the tissues. Estimation of the distance of firing is quite approximate so it is always advisable to do experiments with the seized weapon after it is carefully examined chemically. Results of these experiments are then compared with the findings in the body, clothes and other targets.

Wounds of smooth weapons

At close range the shotgun is the most formidable and destructive of all small arms. Unlike bullets, shotgun pellets rarely exit the body. As the distance of firing increases, there is dispersion of the shot with resultant decrease in the number of pellets that strikes the target. If the muzzle of a shotgun is held in loose contact or near contact with the animal body, there will be a circular area of soot deposited on the skin surrounding the entrance hole. As the range increases the diameter of the soot deposit increase but the density decreases. Deposition of soot continues out to a range of approximately 30-cm. As the range increases beyond 1 to 2 cm from muzzle to target, powder tattooing will occur. As the muzzle of the shotgun is moved farther from the animal body, the diameter of the circular wound of entrance increases in size until a point is reached where individual pellets begin to separate from the main mass.

In deaths from shotgun wounds, the size of the shot pattern on the body should be measured so that the range can be determined accurately by conducting a series of test shots as to reproduce on paper the pattern of the wound on the body. The diameter of the shot pattern in centimeters is some 2.5-3 times the muzzle distance from the wound in meter's (or the spread in inches is equivalent to the distance in yards); estimates derived in this way must be checked by test firing whenever possible. At close range, when there is only a single large wound of entrance, the wad from a shotgun shell will be found inside the body. As the range increases, the wads gradually fall and separate from the main shot mass. As still relatively close range the wad may impact the side of the wound of entrance before sliding into the body. Thus one will have a circular entrance surrounded by a symmetric abrasion ring with a large, irregular area of abraded margin on one side where the wad impacted.

As the range increases (3 m), however the wads will drift laterally until they impact on the skin adjacent to the entrance site and don't enter. At this time, the wad will leave a circular or oval imprint on the skin. As the range increases, the wads will miss the body or strike with so little energy that will not leave a mark on the skin. The direction of fire may be deduced from a careful examination of the passage of individual shots through the tissues.

Distance of firing determination in smooth weapons

It depends on the nature of the weapon and the type of powder. The gases reach about 15 cm and would cause tearing of the inlet. Flame and smoke reach about 1 - 1.5 meters causing burning and blackening respectively (faint and grayish reaches about one meter in smokeless powder. Unburned particle travel for about 3 meters causing tattooing. In case of smokeless powder, where combustion is more complete, the powder marks would be less marked and reach shorter distance thus burning may not be present at all or only at a distance of few centimeters and gases reach 15 cm. Blackening is rather faint and grayish, reaches about one meter and the range of tattooing which is lighter in color than that of black powder, is about 2 meters. In case of short weapons, whether rifled or not, the powder marks only reach a distance of not more than 50 cm.

Wadding

The wads can also give an indication about the distance, whether they have entered the body or simply struck it, and to what depth one or both of them penetrated into the tissues. External wad, (made of thin cardboard), may penetrate the body at one meter and reach a distance of 3 meters in the air. Internal wad (made of thick felt or compressed cardboard), may travel about

10-12 meters in the air and penetrates the body at a distance up to 3 meters. Between distances of 3-10 meters, it may strike the body, causing a circular bruise just at the edge of the dispersion area of shots or outside this area, generally on a lower level. Approximate formulae are given as a way of estimating the distance from the powder marks, which can be used in all types of weapons. Flame and smoke reach a distance equal to about 1-1.5 times the length of the barrel. Unburned particles reach a distance equal to about 2 or 3 times the length of the barrel. At longer distance, the estimation in case of shotguns is based on the extent of dispersion of the shots. The longer the distance the bigger the space between neighboring shots and the less the power of penetration that at 50 meters or even less in old weapons, the shots may simply strike the body and fall on the ground causing only bruises.

Table 3. Distance of firing determination in smooth weapons

Shape of the wound	Distance of firing
The whole number of shots enter in one mass	Up to one meter
A central hole and few surrounding separate shot holes	At two meters.
The number of dispersed shots increased and the central hole gets smaller in size	At 3 meters
Complete dispersion of the shots occupying a circular area of about 16 cm in diameter	At 4 meters
A circular area of about 32 cm in diameter	At 6 meters
A circular area of about 50 cm in diameter	At 8 meters
A circular area of about 60 cm in diameter	At 10 meters

Direction of firing

The direction of firing can be easily made out by joining the entrance and wound with an imaginary line. In case the missile is still inside the body by joining the entrance wound with the site of the missile in the body, as detected by x-rays in the living and dissection in the dead. Such imaginary line will indicate the direction of firing. If the shots are dispersed, the site of the biggest collection is taken as that of the missile. It should be noted that the missile might be deviated from its original direction especially with more or less spent bullets being fired either from a long distance or a weak weapon. In such case, when the bullet hits a bony surface, it gets deflected instead of perforating the bone.

Table 4. Differentiate between ante-mortem and postmortem wound

Ante-mortem wounds	Postmortem wounds
Presence of gaping and eversion of edges	No gaping or eversion
Presences of blood oozing from the wound	No blood oozes
Presence of blood clots infiltrating the tissues.	No blood clot
Edges usually swollen	No swelling of edges.
Usually accompanied by Hemorrhage either external or Internal with blanching of viscera if extensive.	No accompanying hemorrhage.
Vital reaction in the form of healing or sepsis	No
Microscopic examination shows leukocytes and fibrin Threads infiltration and may be granulation tissue	No

Time between infliction of wound and death

Time that has passed between wound and the occurrence of death could be judged from the different changes that occur in the wound color, healing or sepsis.

Causes of death in wounds

In all cases of wounds, complete and thorough external and internal examinations of the body are essential. There are direct and indirect causes of death in wounds

Direct causes, include hemorrhage, shock and injury to vital organs.

1- Hemorrhages, the amount of hemorrhage that causes death depends on:

a) Site of hemorrhage, external hemorrhage causes death when it is accompanied by loss of at least one third of the total volume of blood. With internal hemorrhage, the amount of blood varies with the locality, so that death may result after an effusion of one or more litters of blood in the pleural or peritoneal cavities, whereas effusion of only one fourth of a liter or even less of blood in the pericardial cavity is quite sufficient to cause death. Only few cubic centimeters of blood effused in the brain may be fatal. b) Rate of hemorrhage as rapidly effused blood is much more dangerous than slow hemorrhage. c) Age, sex and state of health, as Small and large animals are more susceptible. Female could stand hemorrhage somewhat better than male. The state of health has also an important effect on its resistance to hemorrhage.

2- Shock, death from shock has got no characteristic postmortem signs and it could be only suggested from the history of the case and the absence of other causes of death. It may follow minute injuries, which may leave no mark, e.g. slight burns, slight violence to the generative organs or even severe fright. It may follow a number of minor injuries, as occurs in beating by sticks, where no one injury could alone cause death.

3- Injury to vital organs, injury to brain or liver is an evident cause of death and could be easily detected in the postmortem examination. In such cases, death may occur immediately follow the injury or even few days or weeks after the injury.

Indirect or secondary causes include Secondary hemorrhage, tetanus, septic inflammations, septicemia and pyaemia, hypostasis, aspiration pneumonia or any other complications that may follow operations may cause death of the victim. Embolism, bone necrosis, tissues gangrene or asphyxia during animal slaughtering may also of the secondary causes.

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Chapter 4 Burns

Physical agents like; temperature, atmospheric pressure and radiation may produce serious injuries to animals or even deaths. Burn generally, is the destruction of tissues through the action of flame, hot solids, hot liquids, corrosives, electricity or lightening but a burn is generally restricted for the effect of flame, hot solid, molten metal, friction with a moving object, sun rays, electric currents, X-rays and radium. While the effect of hot liquids or steam is generally termed scald. The effect of corrosive fluids as acids and alkalis is termed corrosion.

Interpreting Burn Patterns

The suspicion of deliberate infliction of burns on an animal is raised when the history offered by the owner does not match the presentation of the burn, the environment, or the behavior of the animal. Some owners may blame the animal laying too close to a hot object as the cause of the burn. Although it is possible for an animal to sustain a thermal burn injury before registering the pain it is causing, the burn pattern may not match a contact burn. In animals with unexplained eschar the possibility of some kind of burn should be considered and investigated. The appearance of the burn provides several clues as to its cause. Burns are usually a patterned injury that reflects the cause of the injury. Proper interpretation of the burn patterns can reveal the exact nature of events that may support or refute the history and help direct the investigation. A determination of where the burn started on the body may be made when there are more severe burns confluent with

more superficial burns. Splash or spill burns have trickle-like burns where the offending liquid ran down the body. These trickle-like areas are usually more superficial than the place where the liquid first contacted the body. A burn pattern that is evenly distributed with the same degree of injury is indicative of an even rate of burn. This can result from a flash fire or chemical agent. The location of the burn can indicate whether the burn could have been accidental versus intentional. When an animal is set on fire and the fur burns for a short period of time there may not be large areas of burns to the underlying skin. Instead, there may be a wide distribution of isolated circular burns on the skin where the fur acted as a wick for the fire to reach the skin. There may be a larger burn area on the skin where the fire was initially ignited on the body.

Collection of Evidence

Evidence pertaining to a fire includes any substances and or devices that were used to cause the fire or produce the burn and related injuries. The accelerant, chemical, or liquid that caused the burn may be on the animal or nearby objects. There may be an odor associated with the injured area that may indicate the cause of the burn. It is imperative to take samples in all burn injuries. The burn and adjacent tissue should be swabbed for accelerants or chemicals. Samples of the fur and skin should be taken for residue testing. The soil beneath the animal and adjacent to it should be collected along with any bedding. The device used to ignite the fire may be a cigarette lighter, matches, or blow torch and may contain residue of the accelerant on the surface. When collecting evidence related to a fire it is important to use proper containers. Accelerants are volatile substances and

may escape through plastic. The best way to preserve this evidence is by placing it in a clean, uncoated paint can or a special container for arson evidence.

Classification of burns:

Burns are classified clinically into six degrees depending on the intensity of the heat and the time of exposure. It depends mainly on the time of heat application than the amount of heat.

1- First degree burn (superficial redness), simple redness of the skin due to dilatation of the superficial capillaries (hyperemia) which get less apparent after death. It heals without leaving scar.

2- Second degree burn (vesication), the formation of vesicles due to the exudation of fluids between the cuticle and the cutisvera. The vesicles will be tense and filled with an albuminous fluid. The edges and base of the vesicles show either diffuse redness or multiple hyperemic points. These burns also heal without leaving any scar.

3- Third degree burn (destruction of the epidermis), burn cause destruction of the superficial layers of the skin down to the sensory nerve in the dermis. They heal by formation of scar, quickly covered by epithelium from the base of the raw area intact hair follicles and sweat and sebaceous glands. Scar formed is thin and elastic usually does not interfere with the integrity of the part and causes little or no contraction or disfigurement.

4- Fourth degree burn (destruction of the whole skin), causes destruction of the whole skin. The scar resulting from healing of such burn is fibrous, corrugated and causing disfigurement or disability.

5- Fifth degree burn (destruction of the muscles), causes destruction of the skin, underlying muscles and the deep fascia and other soft tissues. It results in great scaring and deformity. It is not very painful.

6- Sixth degree burn (complete charring), causes complete charring of the tissues is preventing the reform. Ends in inflammation of the subjacent tissues and organs if death is not the immediate. When a blister is formed as a result of burn fluids collects between the dermis (true skin) and the epidermis (the horny protective layer). The nerve endings (tactile corpuscles) are situated in the true skin close to the epidermis where a network of blood vessels and nerve is found; the sweat glands also lie nearby. The skin cells are called epithelial cells (epithelium) which can only grow from epithelium and accordingly after a burn, in which the epidermis is totally destroyed, there is no epithelium left at the bottom of the wound where the epithelium has been left intact.

Factors affecting gravity of burns:

It is much more important than the degree of burn. The extent of the burn as burn covering more than one third of the body surface is usually fatal even if superficial. Degree of burn: burns of the third degree are most serious owing to the severe shock from the exposure of the sensory nerve ending and excessive evaporation of body fluids. Position of the burn: burns of the neck or trunk are more serious than those of the limbs. Age as burns in small and old age are more serious. The general health of the victim is important in burn resistance.

Causes of death in burns according to their rapidity:

1- Immediate or quick death, may be due to, nervous shock (severe pain), syncope from fright, falling objects injuring vital organs or from asphyxia through the inhalation of CO or CO2.

2- Death within 6-48 hours, may be due to any of the following

a) Toxemic shock: absorption of histamine and histamine like substances produced in the burnt area due to the process of tissues and cells destruction may lead to paralysis of the vasomotor center, producing extreme vasodilatation with stagnation of the blood in the capillaries.

b) Blood over concentration: 80% of deaths in burns may be due to evaporation of the fluids from the burnt surface.

c) Fat embolism: the heat liquefies the fat and destroys blood vessels so those droplets of fats are absorbed into the circulation causing embolism in the pulmonary arteries especially in fatty animals.

d) Acute edema of the glottis may supervene especially in burns around the neck and face.

3- Delayed death (beyond two days up to a number of days or even weeks). It may be due to any of the following:

a) Hemorrhage in the suprarenal glands occurs about 4th or 5 th day and lead to death from suprarenal insufficiency.

b) Internal inflammatory complications occur within one week of the burn as pericarditis, peritonitis and pneumonia etc...

c) Rupture of the duodenal ulcers, considered a common complication of burn owing to the excretion of the burn toxin by liver in the bile, which irritates and ulcerates the duodenum. Rupture of such ulcer, however rarely occurs and may kill the patient about the tenth or twelfth day.

d) Sepsis of the burn may take place in the burnt area causing death due to septic absorption, toxemia or septicemia, which occur at any time after the first week.

Post-mortem findings:

You must examine for the appearance of the burn whether it is vital or not (anti-mortem or post-mortem) and for wounds or signs of violence. The stomach contents should be examined; the nature of any food or foreign substances present should be noted and retained for analysis. Samples of blood from each side of the heart to compare the percent and quantity of CO (it depends on length of time the victim breathed after the fire started).

A) External signs

1- The animal is contracted, the limbs are raised in position of defense by heat stiffening and certain parts may be found ruptured**.**

2- General redness due to CO.

3- Blisters and a ring of redness around the blisters. Blisters having the preceding characters cannot be caused after death and the presence of such blister is indicates that life was not extinct when the heat was applied.

4- Deeper burns which show an intense zone of redness at the periphery or there may be complete incineration in which case it is impossible to state whether the burn is anti or postmortem.

B) Internal signs

1- Carbon particles in the mucous membranes of larynx, trachea, bronchi and lungs (vital).

2- Congested lungs, signs of bronchitis, bronchopneumonia or pneumonia.

3- Congested liver and kidneys.

4- Effusion in the pleural and pericardial cavities.

5- Reddened stomach and duodenal ulcers.

6- The heart is usually filled with cherry red clotted blood.

Table 1. Differences between ante-mortem and post-mortem

Ante-mortem burn	Post-mortem burn
Redness of the skin (Hyperemia)	No
Vesicles are filled with fluids Rich in albumin and chlorine	Absence of vesicles, if present, they will contain gases and very scanty fluids
Presence of soot	No
Red blood may contain CO	No
may show signs of healing or sepsis	No
No possible causes of death	May be present

Burn dating

A recent burn is free from pus or such swelling and edema is probably less than 36 hours. There is pus on the surface or under the unseparated sloughs and red inflammatory zone surrounding the burn has disappeared; the age is from 36 hours to few days. The superficial sloughs of third degree burns will usually be thrown off in about a week and deep sloughs may take a fortnight or are attended with more suppuration. A red granulating surface free from sloughs when the whole thickness is destroyed indicates a burn two weeks old. Older burns are estimated by the amount of granulation tissues, its depth and by the extent of epidermis growth from the circumference. Scars of burns should be dealt with as an ordinary scar in deciding its age.

Table 2. differences between burn, scald and corrosion

Burn	Scald	Corrosion
Coverings burnt	Coverings wetted and may be colored by liquid	Coverings are colored and eaten up depending upon type and concentration of corrosives
Singed and curved hair	Wet hair may be discolored	Hair may be discolored
Spread usually from below upwards in a wide irregular area	Spreads from above downward in form of streaks.	Fixed.
Any degree is possible.	First and second degree	Only first, third and fourth

	only.	degrees
Vesicles only present at the edges	Vesicles all over	Absence of vesicles
Skin with or without deep tissues may be charred	Sodden and bleached skin	Skin is destroyed, sodden or charred.
Soot may be found on the surface and in the air passages.	Absent soot.	No soot.
Blood may contain CO	No CO in the blood	No CO in the blood
Resulting scars thick and may cause much disfigurement	Scar is thinner and cause less contraction & disfigurement	Scar is thinner and cause less contraction & disfigurement

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Chapter 5 Asphyxia

Asphyxia is a form of death resulting from impairment or suppression of respiration. It takes place in case of:

1- Obstruction of the air passages from inside, either due to:

a) Diseases (inflammatory conditions of the air passages, edema of glottis, tumors or disease of lungs as tuberculosis, pneumonia etc.).

b) Accidental inhalation of dust or sand as may occur when a heap of sand falls over an animal or in heavy sand storms in the desert; inhalation of food as occurs in vomiting during unconsciousness or due to accidental aspiration of artificial teeth (human) or other foreign bodies into the air passages. Obstruction may also be due to aspiration of water or other fluid (drowning).

2- Obstruction of the respiratory openings as in smothering.

3- Obstruction of the respiratory muscles movements as in overlying or spasm of these muscles as in strychnine poisoning.

4- Obstruction of the air passages from outside, as in strangulation, hanging and throttling.

5- Paralysis of the respiratory center in the brain as occurs in opium poisoning.

6- Breathing irrespirable gases.

Signs of Asphyxia

The signs of asphyxia are usually well marked and occur in three stages; these stages are very short in duration and are usually 2-5 minutes:

1- First (Dyspnoeic) stage

This stage may be short or long, depending on the physical condition of the patient and the extent of obstruction, whether partial or complete and sudden or gradual. The patient appears cyanosed (face, lips and conjunctiva) with rapid breathing and the muscles of respiration are in full action as lack of oxygen and increase carbon dioxide level in the blood, will stimulate the respiratory center in the brain leading to increase in the number and depth of respiratory movement to get maximum amount of oxygen. Violent excitation increased blood pressure and increased pulse rate.

2- Second (convulsive) stage

In this stage the patient still breathes regularly, with very short inspirations and prolonged expirations as accumulation of carbon dioxide in amounts more than stimulant amount, increase expiration to get rid of the biggest proportions of carbon dioxide. Increased intrapulmonary pressure, which may lead to rupture of some air vesicles under the pleura, causing the so-called silvery spots and very high blood pressure, leading to rupture of the unsupported capillaries as those under the pleura, pericardium and meanings, etc. Convulsions set in, the reflexes get weaker and then abolished and the patient soon passes into coma (the first sign of the third phase).

3- Stage of irregular breathing

The patient become unconsciousness with irregular breathing the respiration becomes slow and irregular taking the form of single gasps separated by long intervals or long apnoeic phase or period between gasps. All the muscles relax, the head falls to one or other side, the nose and cheeks become blown, the mouth open and respiration completely stops. The heart usually continues to beat for some time after complete cessation of respiration and so long as there is any heart beat, their remains hope to resuscitate the patient by artificial respiration (after removing the obstruction).

Post Mortem Signs of Asphyxia

I- External signs

1- Severe congestion on the face, the lips and conjunctiva due to lack of oxygen and increase carbon dioxide level.

2- Bulging of the eye with subconjunctival hemorrhages may be found.

3- Well marked blue to blackish in color postmortem hypostasis.

4- The signs of the casual agent as a rope mark round the neck.

5- The tongue protrudes from the mouth.

6- Fine froth is found around the mouth and nose (very large in case of drowning). The color of froth may be bright whitish in case of drowning, tinged with blood in mechanical asphyxia or green, large particles with putrefactive odor in postmortem hypostasis.

II- Internal signs

1- Congested lungs with petechial hemorrhages under the serous membranes, the tissues of internal organs and sometimes in the sub mucosa. When found under the serous membranes of the lung (pleura) and heart it is called Tardieu's spots or it may be large echymoses when the blood escaped from large veins. Silvery spots are found on the surface of the lungs due to rupture of the lungs alveoli (drowning), it is characteristic when found with other signs of asphyxia, where it may be found in other cases like secondary nervous shock and in some poisonous conditions.

2- The air passages contain fine bloody froth similar to that found about the mouth and nostrils.

3- The heart is dilated and filled with dark fluid blood especially the right side. There may found petechial hemorrhages under the pericardium (it is not characteristic where it is found in all types of death).

4- All viscera are congested, but it is not characteristic for asphyxia, as it is present in some cases e.g. in secondary nervous shock and in chronic cardiac depression. The brain shows neither severe congestion as occurs in coma, nor pallor as occurs in syncope.

5- Some petechial hemorrhages under the dura-matter

6- Higher temperature than normal.

7- Inability of the blood to clot, after death. In the start of blood clotting blood vessels produce enzyme dissolve the fibrin (fibrinolysin) if the amount of the enzyme decreased the blood clotted by the action of thrombin.

8- Histologically, the lungs show congestion of the vesicles, edema of the air cells and rupture of some of the air vesicles, especially under the pleura.

Types of Asphyxia

Central asphyxia, suffocative asphyxia, static asphyxia, tissue asphyxia, paralytic asphyxia and mechanical asphyxia

1- Central asphyxia

It may be derived from external poisons e.g. opium, datura and barbiturates or internal poisons e.g. uremia.

2- Suffocative asphyxia

It is resulted from inspiration of toxic gases as carbon monoxide, methane and carbon dioxide. Deaths from suffocating gases are not due to the toxic nature of the gases, but to displacement of oxygen from the atmosphere. Reduction of atmospheric oxygen to less than 25% of normal by displacement of oxygen by inert gases such as carbon dioxide and methane produces unconsciousness in seconds and death in a matter of minutes. Determination of the cause of death in such cases is by knowledge of the circumstances surrounding the death case. There are no specific findings at autopsy. If death is prolonged, the individuals appear cyanotic with petechial hemorrhages of the epicardium and visceral pleura. Blood analysis unless in case of carbon dioxide, because it is a normal constituent of blood while methane can be detected in the blood.

3- Static asphyxia

It is resulted from depression of all respiratory functions due to blood stasis e.g. in heart failure, digitalis poisoning, very severe hemorrhage or due to haemoconcentration resulted from burning.

4- Tissue asphyxia

Deaths occur as a result of inhalation of toxic chemical substances which prevents utilization of oxygen at the cellular level. It includes four types:

- **Anoxic asphyxia**, in this case the Hb does not transport oxygen to the cells due to the overcoming of other gas as carbon monoxide.
- **Hemolytic asphyxia**, the Hb escaped into the serum and loss its affinity to transport oxygen as in venom poisoning.
- **Enzymatic-tissue asphyxia**, the oxidase enzyme loss its functions in helping oxygen transport from blood cells. e.g. arsenic and mercury poisoning.
- **Histotoxic asphyxia**, the cells itself become insensitive to oxygen due to the effect of some poisons as cyanide.

5- Paralytic asphyxia

It take place due to paralysis of the respiratory muscles (animal unable to inspirate sufficient quantity of air) e.g. strychnine poisoning and in the use of some muscle relaxant as succynil choline.

6- Mechanical asphyxia

A- Pathological mechanical asphyxia

It is resulted from obstruction of the airways especially the larynx due to pathological cause. e.g. Edema of the epiglottis, supportive inflammation of the throat, dephetric membrane expanded to the larynx, paralysis of the laryngeal muscles or diaphragm, bronchitis or pneumonia, pleural effusion or hemorrhage in the lungs or emphysema.

B- Violent mechanical asphyxia

It may be accidentally or homicidal and called according to the affected part of the respiratory system into, smothering, strangulation, overlaying, throttling or manual strangulation and hanging

Hanging

Hanging is a type of asphyxia death caused by suspension of the body from the neck, either totally or partially. Suspension is made through the application of a band or ropes either in form of a loop or running noose and the constricting force being the weight of the body then the base of the tongue occluding the air passage. This may be occurs accidentally in animals especially in horses when trying to get rid of tightly fixed stopper-ring. Also homicidal hanging may occur in dogs and may be suicidal in human.

Causes of death

1- Occlusion of the gullet by the base of the tongue, which passed upwards and backwards by the ring of the ligature which, stretched around the neck.

2- Cerebral anemia from obstruction of the lumen of the carotid arteries in the neck through their elongation, being pulled between the rope and the weight of the body (the brain cannot sustain anemia more than 5 or 6 seconds).

3- Syncope due to stretching of the vagus nerve and carotid sinus which lead to slowing the heart rate or stooping the heart (reflex cardiac inhibition).

4- Fracture dislocation of the odontoid process of the axis vertebrae and tearing of the medulla oblongata with immediate suspension of the functions of the vital centers.

5- In most cases of hanging, death may be due to two or more of these causes. Delayed death in hanging may occur if a victim is saved but death may even take place after many hours due to acute pulmonary edema or edema of the glottis.

Treatment

1- Remove at once the rope from around the neck by cutting it.

2- Artificial respiration until respiration is regained automatically.

3- Warmth and cardiac stimulants.

4- Clarify the mouth and pharynx from froth.

PMP

1- The face is pale or congested and cyanosed with bulging eyes, protruding tongue and congestion under the conjunctiva, saliva is seen dribbling from the mouth due to pressure of the rope on the sub maxillary salivary gland.

2- Postmortem hypostasis is present in the lower parts if the body is kept suspended for sometimes after death.

3- Ligature mark is shown as an oblique incomplete constriction abrasion situated high up in the neck (above the larynx) and having the characteristic pattern of the rope with hyperemic or contused edges. The ligature mark is characterized by the presence of hemorrhages and contusions especially in the internal tissues and between muscles in the site of rope.

4- Brain may be congested or anemic.

5- Transverse initial rupture in the carotid sinuses.

6- Hyoid bone may be fractured (rarely).

Throttling (manual strangulation)

This type of asphyxia is only homicidal. The assailant usually uses excessive force, thus producing well-marked signs in the neck, which together with the general signs of asphyxia, may be the only proof of throttling. It occurs by pressure of one or two hands around the neck on the larynx and trachea continuously until death. **Causes of death are mainly due to** prevention of the air entry to the trachea, cerebral anemia resulted from the pressure on the carotid arteries or due to reflex action resulted from the pressure on the carotid sinus.

Signs

Multiple small semilunar abrasions on both sides of the neck- generally four nails abrasions with contusion produced by pressure with the fingers, (which may

be only seen in the deep tissues of the neck) on the left side of the neck under the angle of the jaw one abrasion with contusion in the right side when the right hand is used and vice versa occurs when the left hand is used. The numbers of abrasions increase if the position of the hand on the neck is changed and use of both hands. The number of these abrasions may be very important to identify the assailant if he has one or more fingers missing. Abrasions may be absent when the neck is pressed between arm and radius and ulna from behind.

PMP

1- Multiple contusions in the deep tissues, mucous membranes and blood vessels of the neck.

2- Contusions in the gullet and under the tongue.

3- Fracture of the hyoid bone or laryngeal cartilage or both which is surrounding by effusion of blood. Fracture of the hyoid bone is much more frequently found in throttling than in strangulation or hanging. Thus, the hyoid bone should be carefully dissected out of the neck then tested for the presence of any fracture.

Strangulation

Strangulation is a form of violent asphyxia in which the air passages are blocked from outside by applying pressure to the neck by means of a ligature. It always homicidal, but may be accidentally during casting the animal down. It is generally asphyxia owing to the pressure of the ligature on the air passages and great vessels and nerves of the neck. In all forms of strangulation, the cause of

death is cerebral hypoxia, secondary to compression and thereby occlusion of the vessels supplying blood to the brain. The carotid arteries by virtue of their location are easily compressed by direct pressure to the front of the neck. In contrast the vertebral arteries are resistant to the direct pressure, but can be occluded by severe lateral flexion or rotation of the neck.

Signs

- Signs of resistance on the body, congestion and swelling of the face and neck, protrusion of the eyeballs with congestion of the conjunctiva.
- Sub conjunctival hemorrhage, wide dilatation of the pupils, protrusion, swelling and congestion of the tongue which may be found bitten.
- Fine, viscid blood- stained froth may be found around the mouth and sometimes there are few petechial hemorrhages under the skin of the face and neck.
- Dark violet and well-marked hypostasis.
- The constriction mark of the neck varies in shape and depth depending on type of ligature used. It is usually encircles the neck completely in a horizontal direction below the larynx (it differ from the mark of hanging which is incomplete, oblique at high level.
- On dissection of the neck, The hyoid bone, laryngeal cartilage and tracheal rings are rarely fractured. Bruises and lacerations under the skin and in the deeper structures, especially under the site of the knot. . The strangulation mark resist putrefaction, although the strangulation carcasses are putrefied rapidly.

Smothering

Smothering is a form of violent asphyxia arises from obstruction of respiratory orifices by the hand or other soft objects as clothes. It is a common way of killing infants, old people and other debilitated persons. The placing of a plastic bag over a child's head is the most common form of smothering. Accidental smothering can occur with defective cribs. Abrasions and bruises are seen around the face, mouth and nostrils. Bruises and contused wounds may be found on the inner aspects of the lips and cheek. Contusion present due to pressure of tissues on the bones of jaw and teeth. Abrasions may be not present if soft object is used. The signs of asphyxia but in a less marked form may be noticed internally.

Overlaying

Overlaying is a special type of violent asphyxia due to compression of the chest and abdomen, thus preventing respiratory movements. A large weight falls onto or presses down on an individual's chest or upper abdomen, making respiration impossible e.g. the mother overlaying her small animal (traumatic asphyxia). Or as a result of crowdness of small and large animals. An infant is put in bed with one or more adults. Fall of heaps of earth, collapse of buildings. when an individual are buried in loose earth or sand, they die as a result of occlusion of the nose and mouth, plus immobilization of the chest and abdomen by external pressure sufficient to prevent the respiratory movement. (it is smothering combined by overlaying asphyxia). Heavy crowds in the farm or in riots.

PMP

The external signs are very few or even absent but the internal signs of asphyxia are usually present as in smothering and throttling.

1- Contusions in the chest near the sternum and this bone may be fractured, also ribs may be fractured near its angles.

2- Contusions and abrasions on the back.

3- Congestion of the eye with subconjunctival hemorrhages.

4- The hypostasis may be black.

Choking

It is a form of asphyxia caused by the impaction of foreign bodies inside the air passages. It commonly occurs accidentally from the aspiration of foreign bodies as vomited matter or from the inhalation of sand or dust in case of falling heaps or dust on the victim. Presence of foreign body in the larynx. Mucous secretion until the bronchioles with general signs of asphyxia.

Causes of death

1- Complete obstruction of the air passage by large foreign body.

2- The foreign body is not necessarily so large to completely block the larynx but partial obstruction may be completed by spasm of the glottis and secretion of mucus, which reflexly result from the foreign body.

3- Pressure on the trachea from behind when large foreign body obstructs the esophagus.

Drowning

This type of asphyxia caused by submersion of the body in water or other fluids, which will fill the air, passages preventing the later from entry. The inhalation of fresh water may cause rapid and fatal haemodilution. It is not necessary to have the whole body immersed in such fluid but immersion of the face is sufficient for few minutes to cause drowning. Experimentally, if a dog is strangled, it can still be saved after 4 minutes from starting its strangulation. But if the head is submerged, resuscitation will be only possible within 1 to 1.5 minutes

Causes of death

1- Obstruction of the air passages where it filled by water, which prevents entrance of air. In this case the signs of death would be those of asphyxia.

2- Concussion or other head injury due to falls down to the bottom of the water channel or through striking some hard object as rocks or trees in the water.

3- Syncope produced by reflex inhibition of the heart, which resulted from fear and cold water.

PMP

External signs

1- Presence of fine whitish froth around the mouth and nostrils due to the thorough admixture of the water with air and mucus during the forcible attempts at respiration. When the current of water wash off this froth, it soon

comes again without or with pressure on the chest. This type of froth must be clearly differentiated from coarse, dark, bloody; greenish fouled smelling froth, which appears with putrefaction.

2- Presence of stones or sands or aquatic weeds in the hands of the body (cadaveric spasm) resulted from the effort of the victim in a trial to pull him up. It indicates that the person was living when immersed.

3- Coolness of the body as it loses its temperature much more rapidly than in air, paleness of the skin and congestion of the conjunctiva and peeling of the epidermis.

4- Folding of the skin (due to contraction of the erector muscles).

5- Hypostasis is marked at the head, neck and the upper part of the chest.

Internal signs

1- The air passages are filled with whitish froth similar to that found externally around the nostrils and mouth also sands and aquatic weeds may be present. The mucous membrane of the air passages is generally congested and may show petechial hemorrhages.

2- The lungs are voluminous and completely fill the chest cavity with characteristic ribs marks on its surface. They are pale due to the blood being forced out of the vessels by the filling up of the air cells with water. They are filled with water, when the lungs is cutted a large amount of water oozes out from the cut surface, intermingled with air and mucus, thus taking the form of fine froth. Silvery spots from the

rupture of the air vesicles under the pleura may also be seen as small bright spots. Tardieu's spots are usually less marked.

3- Presence of water in the stomach, which is not mixed with the food (salty or muddy water). After putrefaction, the only evidence of drowning is the presence of mud, sand, aquatic weeds or other foreign bodies characteristic of the water stream in the tracheas, bronchi as well as the stomach as well as the stomach together with the circumstances of death and the exclusion of other possible causes

4- The heart is flabby with few sub-pericardial hemorrhages. The right side of the heart is filled with dark fluid blood while the left side is more fluidy than in the right due to absorption of water from the lungs.

5- If the chlorine contents is increased in one side (25 mg) than the other side this indicate drowning, if not more than 12 hours pass to the carcass in the water. In a body drowned in fresh water, the amount of sodium chloride in the blood of the left side of the heart is less than that in the blood of the right side. The reverse occurs in bodies drowned in salty water, due to the dilution of the blood of the left side of the heart with water in the first case, and absorption of sodium chloride and other salts in the second case.

6- Spleen is usually pale and contracted.

7- Congestion of other organs includes the brain.

8- The demonstration of diatoms or plankton's in any of the body organs such as the brain, bone marrow, kidneys or the liver is strong if not conclusive evidence of drowning, being distributed throughout the body by the circulation during drowning. The organs should be digested with

concentrated mineral acids leaving only the acid-resistant silica shells of the diatoms before subjected to microscopic examination.

Is death due to drowning?

The answer to this question may be easy in a fresh body from the presence of the already described signs of drowning, together with the absence of signs of any other cause of death in form of wounds, injuries, strangulation, etc. In putrefied bodies, the presence of characteristic water weeds, sand or mud in the air passages or stomach, together with the presence of blood stained fluid in the pleural and peritoneal cavities and absence of other cause of death. If the characteristic foreign bodies are not present but the pleural and peritoneal cavities contain some blood watery fluid with no other causes of death- drowning is then a possible cause of death.

Diatoms

Overview

Diatoms are microscopic unicellular algae with a uniquely extracellular coat composed of silica. There are over 10,000 morphologically distinct varieties of diatoms that range in size from 5 to greater than 500 _m. They are present in every naturally occurring body of water, from a puddle to the ocean. They may be found also in moist soil and the atmosphere. The type of diatoms found in a certain location is unique and specific to that area. The season also affects what type is found. The diatom populations have monthly fluctuations in their concentrations in a particular body of water. In one body of water, several types of

diatoms may be found, but all are located in a separate and specific area. These characteristics can help identify the location and even season of death. Because of the presence of diatoms in all types of water, the analysis for diatoms has been developed as a conclusive test for drowning. Diatoms can enter the body in three different ways: through inhalation of airborne diatoms, ingestion of material containing diatoms, and aspiration of water containing diatoms. This last route is the foundation for forensic diatom testing, which in conjunction with other findings provides a diagnosis of drowning. When water enters the lungs, either through aspiration if the victim was alive or by postmortem submersion, diatoms may enter the lung tissue passively. They stay in the lungs and do not disseminate unless the heart is beating. When the diatoms perforate the alveolar-capillary barrier they enter the bloodstream and are disseminated to various organs, including the femoral bone marrow. The detection of diatoms in the bone marrow is then compared with the water that was aspirated into the airways or stomach, or from the site at which the body was recovered. A positive match indicates that drowning was the cause of death and the victim was breathing upon entry into the water. A negative test may be seen with dry lung drowning, i.e., when there is no aspiration of water. However, a negative test does not rule out drowning.

The testing of soil for diatoms has been used also to determine cause of death or site of death in severely decomposed or skeletonized bodies found on land. If a body was submerged, presumably drowned, and then pulled from the water and then dumped on land or buried, the diatoms from the outside of the body and within the lungs are deposited in the soil underneath the body. Because diatoms may be found in moist soil, samples from underneath and adjacent to the body are tested and compared to soil samples further away but near the body. These samples also may be compared to water diatoms from nearby bodies of

water to determine the site of submersion. A higher concentration of diatoms in the soil associated with the body than the surrounding soil is indicative that the victim died by drowning.

Diatom Testing

The acceptance of diatom testing has been questioned because of the ubiquitous nature of diatoms in the environment. The validity of this test is supported by the criterion of concordance, which demands that the diatoms recovered from tissue be comparable to the diatoms in the putative drowning medium. The concentration of diatoms in bone marrow and other tissues is directly proportional to the concentration found in the drowning medium. Aquatic diatoms are diagnostically different that those living in other environments. Any contamination is detected by the investigator when comparison is made with the putative drowning medium. This removes the ambiguity of the origin of the diatoms and proves the diatoms were introduced during the drowning process. New testing modalities may further increase the sensitivity and reliability of the diatom test. A recent study using a quantitative diatom-based reconstruction technique was able to confirm drowning as the cause of death and the site of drowning . The diatom test may be conducted on bone marrow or from other tissue in the body. It has been found that the sternum may be the best source for diatoms because the depositional interval for diatoms is shorter than for the femur. The testing of other tissue may provide additional confirmation to bone marrow findings; however, caution must be used when testing other tissues to prevent contamination. The body must not be decomposed and the chest cavity must not have been damaged while submerged.

When collecting and preparing samples for diatom testing, certain precautions should be taken to prevent contamination from other water supplies.

This includes changing gloves when touching or handling a single area and limiting contact of samples to only triple-distilled water. A sample of the drowning medium should be collected from the scene at the site where the body was recovered for comparison testing. Approximately 500–1000 ml should be collected in a clean container. Additional samples should be taken of the water in the stomach, sinus, or airways. All water samples should be kept separate from the body and stored in separate containers to prevent contamination. Water samples should be refrigerated to prevent microbe growth. The tissue test for diatoms is conducted on the femoral bone marrow. Tests may be performed also on other tissue from closed organ systems such as an encapsulated kidney from a non-decomposed body. The body and leg should be cleaned prior to the removal of the femur to prevent contamination with exogenous diatoms. Before removing the bone from the body, it is important to change gloves to prevent contamination of the surface of the bone. The femur should be washed in distilled water. The femur should be placed in a sealed plastic bag and frozen prior to submission to the laboratory for testing

Gettler method

It is a chemical method or test is of a more diagnostic value in cases of drowning in seawater, which can be safely presumed if an appreciable increase of sodium chloride is found in the blood of the left ventricle. On the other hand, a reduction in the chloride content of the left ventricle cannot be taken as evidence of drowning in fresh water, as the reduction in the blood chloride is a common post-mortem phenomenon, which need not progress at the same rate in the two sides of the heart.

Two samples of blood are separately taken from the two ventricles and sent for analysis in clean dry tubes. It is advisable to send, at the same time, a sample of the water from which the dead body is recovered. Yet, if a body is examined within twelve hours of being recovered from fresh water, drowning can be presumed if a disproportionate depression of the chlorides in the left ventricle of 60 mg chloride per 100 cc or more is found.

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Chapter 6 Asphyxiating Gases

1- Chemical warfare agents

The Geneva Protocol of 1925 was drawn up and signed at the Conference for the Supervision of the International Trade in Arms and Ammunition, which was held in Geneva under the auspices of the League of Nations from 4 May to 17 June 1925. The conference adopted a Convention for the Supervision of the International Trade in Arms, Munitions and Implements of War, which has not entered into force and, as a separate document, a Protocol on the use of gases. The earlier treaties prohibiting the use of gases to which the Protocol refers are in particular the Hague Declaration concerning asphyxiating gases of 1899 and the Treaty of Versailles of 1919. Poisoning due to chemical warfare agents in veterinary practice may take place due to their use in war. Accidents arising from their normal commercial use or accidents from military exercises e.g. malfunctioning of an aircraft storage tank filled with the nerve gas (VX) led to the contamination of a wide stretch of the Skull valley Utah in 1968 and the consequent poisoning of over 6000 sheep, three quarters of which died and contamination persisted for over three months. It is classified according to: Chemical nature, into esters, ketones and nitrocompounds. Use, into lethal and harassing. Volatility, into persistent and non-persistent. Physiological action into: Systemic poisons, Lungs irritants, Lacrimators, Sternutators, Nerve gases and Vesicants,

1. **Systemic poisons** , lethal compounds intended to kill e.g. hydrogen cyanide and cyanogen bromide.

2. **Lungs irritants** , lethal compounds act on the respiratory system e.g. chlorine and phosgene, both of them are widely used in the chemical industry.

3. Lacrimators , non lethal compounds affect the eyes and are used for riot control e.g. CN, CAP and CS.

4. **Sternutators**, they are arsenical smoke produces sneezing, malaise and vomiting e.g. Adamsite (DM).

5**. Nerve gases**, cholinestrase inhibitor (Tabun and Savin) similar to but much more toxic than the OPI.

6. **Vesicants**, chemical compounds cause blistering of the skin, blindness (may be permanent) and edema of the lungs if inhaled. Visicants include a) Mustards (dichloroethyl sulfide). b) Arsenical mustards (Lewisite) may cause systemic arsenical poisoning. c) Nitrogen mustards (Mustine). d) Nettle gases (dichloroforoxime) causes an externally painful irritation of the skin and are used as harassing agent.

Treatment:

Chemical warfare agents affecting general animals less severely than human, particularly by lachrymators'. In case of whole sale atmospheric pollution by toxic industrial chemicals or war gases, the initiation of specific treatment necessitates identification of the gas involved so non-specific treatment is recommended. Remove the animal into cleans air and in case of lung irritants, exercise should be avoided. In case of Vesicants, a serious problem arises from the contamination of pasture, which may remain dangerous for considerable time,animals should be driven from such areas, and kept under cover if possible.

Any liquid adhering to their coats should be hosed off with much water.

2- Carbonmomoxide (CO)

A colorless and odorless gas liberated from incomplete combustion of organic matter, during the war, from coalmines, defective stoves, cocking machines and brick or limekilns. Toxic industrial asphyxiate gas generated from incomplete combustion of carbonaceous substances (tobacco, fuel, wood and coal burnt in furnaces). It is present in high concentrations in coal gas, exhaust gases from spark-ignition engines, in locomotive smoke. CO may be produced during coke and coal processing and crushing in coal stores. It is used as a reductant for metal ores, for chemical synthesis and as a duel. CO poisoning in large domestic animals is rare. The risks to cats and dogs are greater. In small stables with bad ventilation, the hazard of poisoning may arise from leakage of the gas from coke burnt in furnaces used in heating.

Toxic action:

- Anoxemia, CO is readily absorbed across the respiratory tract and binds avidly to hemoglobin (Hb). The affinity of Hb to react with CO is about 200-300 time more than oxygen, so the gas is rapidly absorbed into the blood stream, is combined with blood Hb forming carboxy hemoglobin (COHb).COHB is difficult to be dissociated and incapable of combining with oxygen causes, anoxaemia. The most affected organs due to anoxaemia are heart and brain leading to death. The gas have cumulative effect so repeated short exposure may have the same results as one prolonged exposure.

- Direct effect *on the lungs* tissues, it affects the pulmonary surfactant, which affects the tissue permeability leading to lungs edema.
- Muscles, it combines with myoglobin causing primary muscle toxicity.
- It suggested that the formation of carboxymyoglobin in the subendocardium could explain the electrocardiographic changes and decrements in work capacity. CO combines with respiratory enzymes containing bivalent iron, thus reducing cell respiration, and leading to damage to the nervous *system.*

Clinical signs:

The clinical symptoms of CO poisoning depend on CO concentration in inhaled air and on the length of exposure to a contaminated atmosphere. The course of poisoning is usually very rapid. The first symptoms are staggering, general veariness, followed by immobility and by apparent signs of paralysis, particularly in the hind legs. Spasms and suffocation occur later**.**

In man:

It varies according to the percent of blood hemoglobin combines with CO. When the gas combines with 20% of the blood Hb, only headache and lassitude. When the gas combines with 30% of the blood Hb, headache, giddiness, nausea, vomiting, muscular relaxation, rapid pulse, dyspnoea and mental confusion. When the gas combine with more than 30% of the blood Hb, dyspnoea, drowsiness, confusion, impairment of hearing and vision and the animal is unable to move. When the gas combines with more than 50% of the blood Hb, animal falls in deep sleep (coma), shallow irregular respiration, rapid pulse, fall down of blood pressure and subnormal temp, red skin and m.m.(homogenous or patches), asphyxia developed slowly and imperceptible, so the dead body appears as a

living sleeping person. There were no convulsions; no involuntary movements (if it is occur it is a rare case).

Chronic poisoning in man:

It takes place due to prolonged exposure to traces of the gas for long period as in case of leakage in gas pipes, in chemical laboratories and in certain occupations. Signs appear in the form of Pale yellow face, giddiness, looking upward, lassitude and headache, muscular weakness, exhaustion, loss of power and irritability and general weakness. These symptoms especially general weakness is mainly due to central thalamic affection, real peripheral neuritis, and degeneration of the nerves. A person exposed to the action of CO and recovered generally shows weakness, headache, nausea and giddiness.

PMP :

Body show crimson red color especially in blood vessels, lips, conjunctiva, tissues and viscera. Lungs edema, congestion and fine froth in the air passage. Brain, shows small numerous hemorrhages with patches of softening all over the brain.

Diagnosis :

- History.

- Determination of atmospheric CO (The maximum admissible CO concentration in the air is 0.036 mg/l) and blood CO-Hb content.

-PMP (Confused with death due to cold, cyanide and nitrites but detection of gas in the blood and presence of COHB in blood indicate CO poisoning).

Laboratory diagnosis:

- Detection of the gas must be rapidly because if animal exposed to fresh air for long time the detection of CO gas fails. Detection of CO using spectrophotometer or gas chromatography. Blood from poisoned animals and air samples from suspected poisoning area.
- Increase the number of WBC, high PCV, high SGOT and SGPT.
- Chemical detection (Kunkel's test): ½ ml of blood diluted 3-4 times by water + equal volume 3% tannic acid solution---- crimson red ppt. (positive) or dirty brown ppt. (negative).

Treatment:

- Move the animal to fresh air and artificial respiration 100% oxygen under pressure.
- Circulatory stimulants (Coramine).
- Katalysin (provides heart and brain with their oxygen requirements).

3- Hydrocyanic acid (PRUSSIC ACID - HCN)

Hydrocyanic acid is one of the most toxic and rapidly acting of the common poisons. Pure acid: Colorless and volatile liquid with a characteristic odor similar to that of bitter almond. Na and K salts of the acid only slightly less toxic. Complex cyanides (ferrocyanides & thiocyanates) practically harmless and dissociate if exposed to light. The acid is present in certain medicinal drugs as , aqua laurocerasi contain 0.5-1% HCN, dilute HCN 2% used in treatment of gastric

affection, Scheels acid contain 4% HCN and Crude oil of bitter almonds contain 2-10% HCN. Industrial effluents from gold mines causes deaths in cattle drinking water contaminated by HCN. Industrial effluents from other sources like Chrome and Nickel plating works, have resulting in poisoning. Electroplating, tanning, case hardening steel, Iron, photographic solutions and cleaning and coating silver, and Acrybritrile. Agriculture toxicity: poisoning of calcium Cyanamid (fertilizer), cyanides found in certain rat and pest poisons,or cyanide used for destruction of plant vermin. Feeding on green plants as barseem grown under the fumigated trees. Suicidally in human being. Homicidally (criminal): addition of the poison to drinking water or stuffed into the maize corn for animals.

Cyanogenetic plants

Plant materials (cyanogenitic plants) are the most important source of HCN poisoning in animals. Many species of plants contain HCN either free or more usually in the form of cyanogenetic glycoside which is an organic compound containing a sugar , capable of yielding cyanide on hydrolysis. The glycoside is itself is non toxic, but if brought into contact with the appropriate enzymes it is decomposed and HCN is liberated (various Prunus species contain the glycoside amygdalin, which is hydrolyzed by the enzyme emulsin to glucose, benzaldehyde and HCN). In the intact plant no such action takes place, it is not until the plant tissues are damaged or starts to decay that liberation of HCN begin. Stunted or wilted plants or these which have been damaged by frost,hail or trampling are considerably more dangerous than intact specimens. Eg. F.Gramineae (sorghum sudanese - sorghum vulgaris or millet). Plant treatment with nitrogenous fertilizers or with herbicides such as 2,4-D also increases the cyanide contents.

Cyanide is rapidly liberated under the action of ruminal organisms, provided conditions obtain in the rumen. Boiling although it inactivates the enzyme, will not necessarily detoxicate a cyanogenetic glycoside.

Examples of cyanogenetic plants:

Bitter almond, seeds of peaches, cherry, apples sorghum vulgaris, unripe fruits of gawafa, and young millet or maize contain amygdalin.Linseed contains linamarin.White clover contain 20% linamarin and lotaustralin.-Parts of wild cherry contain prunasine.-Lotus arabicus contain lotusine.Millet contains durine. Beans contain phaseolunatine.

Toxic dose:

Minimum lethal dose in HCN in all animal species is ranging from 2 to 2.3 mg/kg. The cyanide contents of the feed (high in young actively growing plants). The rate of food intake or the total cyanide intake because rhodenase enzyme is widely distributed in the animal body and it is capable of converting cyanide to the much less toxic cyanate, if an animal eat the plant containing cyanide so very quickly that the rhodenase cannot detoxify all the ingested amount, poisoning occur. The speed of liberation and metabolism of cyanide in the digestive tract and it depends on: PH of the stomach, the concentration of cyanide liberating enzymes in the plant and the previous diet nature. The relative velocities of absorption and of detoxification of plants by the tissues.

Animal susceptibility:

1-Ruminants are more susceptible to poisoning by cyanogenetic plants than are horses and pigs since the enzymes concerned in the release of HCN are destroyed by the gastric hydrochloric acid.

2-Sheep are less susceptible than cattle.

3-In man the absorption of cyanides is helped by the stomach HCL,in its absence, however absorption is delayed as in case of RASPUTIN who was given cyanides enough to kill a horse but it was effect less because he had a chlorhydria due to excessive drinking.

Absorption and fate:

Absorption, from GIT and lungs. -*Detoxification,* through cystine binding forming 2 amino - 4 thiazolidin carboxylic excreted through the kidneys, reacting with sulfur compounds and excreted through the thiosulphate pathway or through binding with hydroxycobalamine to form cyanocobalamine. Owing to this rapid detoxification, it is possible for animals to ingest amount of cyanide only slightly less than the lethal dose over extended periods without harm. All of these routes can't prevent rapid death. *Excretion,* from lungs (give bitter almond smell of the exhaled air). The greater part is quite rapidly detoxified by conversion into thiocyanate and excreted in urine over a period of several days also in feces.

Toxic action:

1-Red asphyxia or histotoxic anoxia: Cyanide cause inactivation of the cytochrome oxidase enzyme system necessary for tissue respiration. CN free

cyanide ion react with ferric ions of mitochondrial cytochrome system (cell respiratory enzyme system) forming cytochrome-CN-complex. Cytochrome-CN-complex inactivate or paralysis the enzyme leading to histotoxic anoxia or tissue anoxia(oxygen reach the cells but the cells are unable to utilize the oxygen carried for them, thus the blood oxyhaemoglobin remains as such un reduced).

2-Direct effect on the CNS, the cerebral tissues is the most affected tissue due to histotoxic anoxia.

3-Direct effect on heart.

4-Direct protoplasmic poisons on the body cells.

5-It affect on more than 40 of the body enzymes.

6-Large amount of sulphocyanide affects the formation of Thyroid hormone (hypothyroidism).

Signs of toxicity:

I- Peracute toxicity,

It is caused due to cyanide gas or pure HCN. Death occurs within few seconds. Signs appear in the form of, convulsions, paralysis, stupor, froth around the mouth, opened glistening eyes, subnormal temperature, bitter almond odor of in breath, cessation of respiration before that of heart beats and laryngeal paralysis (cyanide cry).

II- Subacute toxicity:

It is caused due to eating of cyanogenetic plants. Death without symptoms may occur. Symptoms appear immediately or delayed depending on the amount ingested and the rate of cyanide liberation. It appears in the form of: excitement, contraction of throat, burning sensation in mouth, profuse salivation, retching, and foaming at the mouth, convulsions of varying degrees and duration.*(opisthotonus position),* Jerky movements of eyeballs, lustrous cornea, glistening, fixed eyes and dilated insensitive pupils, cold clammy skin, involuntary micturation and defecation, face at first is pale, but soon becomes deeply flushed and just before death become cyanosed, deep labored respiration soon becomes slow and gasping then getting irregular before complete apnea supervenes and death occur after 15-60 minutes after onset of symptoms.

Treatment:

Treatment depends on Nitrite-Thiosulphate therapy (in cyanide toxicity CN radical inactivate cytochrome forming CN-cytochrome complex) . Amyl followed by sodium Nitrites convert some oxyhaemoglobin into methaemoglobin, cyanide combine with methaemoglobin forming non-toxin cyanomethaemoglobin and free cytochrome. Sodium thiosulphate acts as a sulfur donor for the conversion of cyano-met Hb to thiocyanate under the action of Rhodenase enzyme. Vitamin C or MB converts methaemoglobin into oxyhaemoglobin.

1- Sodium nitrite or Methylene blue (I/V) injection and Sodium thiosulphate (I/V) injection.
2- Stomach washes using potassium permenganate or sodium thiosulphate 3-5% + animal charcoal.
3- Specific antidotes:

- Injection of vitamin B12a (hydroxycobalamine) not Vitamin B12 only (cyanocobalamin) producing cyanocobalamin (Vit.B12) without producing Met-Hb.
- Monosodium di-cobalt edetate.
- Cobalt sulfate increased the therapeutic effectiveness of the thiosulphate nitrite mixture.
- Sodium cobalt nitrite.
- Chloropromazine will antagonize cyanide intoxication in mice, rat and pigeons.

4- If poisoning by inhalation: amylnitrite inhalation and Oxygen + 5-8% CO_2 therapy is must and Hydroxycobalamine or cobalt edetate.

Treatment of dog

Sodium nitrite ($NaNo_2$) 1% solution 25 mg/kg I/V. followed by Sodium thiosulphate ($Na_2S_2O_3$) 25% solution 1.25 g/kg. Treatment should be repeated as need using half the initial dose.

Treatment of cattle

Sodium nitrite 3 g and Sodium thiosulphate 15 g in 20 ml water is given S/C. In sheep reducing the amount into 1g +2g in 15 ml water. sing of Amyl nitrite inhalation cause rapid formation of Met-Hb in blood so it is used in toxicity through lungs.

PMP:

1- Dilated fixed pupil and luster of cornea.

2- Reddish pink hypostasis and mucous membranes.

3- Reddish blood in the right side of the heart doesn't coagulate.

4- All organs appear reddish pink.

5- Rigormort comes rapidly. Reddish pink froth from nostrils along trachea excreted from the nostrils.

6-Fine petichae on surface of lung, heart and sometimes other organs.

Differential diagnosis

It must differentiated from CO poisoning, death from frostbite and potassium chlorate poisoning. Characteristic smell of bitter almond. Alkalinity to litmus paper (HCN and cyanide). Spectroscopically: two absorption bands between D & E (OxyHb) give reduced Hb on addition of reduced agent indicate Frost bite and two bands between D &E but not affected by reducing agents (carboxy-Hb) indicate CO poisoning. Cyano-Met-Hb indicates cyanide. HCN is rapidly vaporized and this is hastened with putrefaction so the test should be rapidly done and no alcohol is added.

4-Chemical tests:

Kunkel's test: 0.5 ml of blood is diluted three or four times with water is added to equal volume of 3% tannic acid ----------► (+ve) Crimson red ppt. It is (CO).

b-Prussian blue reaction:

Reagents: (ferrous sulfate 1.5% - ferric chloride 10% - HCL 10% - NaoH 10%)

Procedure: 10 ml of the suspected fluid + 2 drops NaoH + 2ml ferrous sulfate + 1ml ferric chloride, then warm and add HCL drop by drop.

Result: Blue ppt. of prussian blue (sodium ferrocyanide compound)

4-Ozone (O3)

Ozone (O3), a reactive species of oxygen, is an important natural constituent of the atmosphere. Background levels of ozone in the lower atmosphere may reach 0.1 ppm and are modified by geographic elevation, solar radiation and climatic conditions. Since some effects of ozone are radiomimetic, its action may be enhanced in the presence of ionizing radiation from background and / or man – made sources. While stratospheric ozone spares the earth from excess solar ultraviolet radiation; high levels of ozone in the environment are toxic and result in health hazards. Ozone is one of the most powerful oxidizing substances. It is formed in the troposphere by the action of sunlight on nitrogen dioxide . direct emission of ozone into the atmosphere as a result of industrial activity is only very limited . Ozone is an important component of photochemical smog and its formation in the atmosphere depends to a large extent on the absolute and relative concentrations of volatile organic substances on the one hand and nitrogen oxides on the other. The maximum natural background concentration, expressed as the average over a period of 24 hours, is 120 ugm-3 (0.06 ppm), the 50% values lying between 40 and 60 ugm-3. On a global level, the main concern with ozone is the

reduction in its concentration in the upper atmosphere. The well-publicized ozone hole" which occurs over the Antarctic (but now detected at high latitudes in the Northern Hemisphere), is caused by the degradative effects of Chlorofluorocarbons (CFCs) on ozone molecules. CFCs are released from aerosol containers, from the coolants in domestic refrigerators when they are broken up or leaked , and from foam packaging . The ozone layer absorbs ultraviolet light so that one hazard associated with its thinning this ozone layer, is an increase in the rates of skin cancer . Environmentally, it has been suggested that the increased radiation could decrease photosynthesis of phytoplankton in the Antarctic.

Mechanisms of ozone toxicity:

The toxicity of ozone depends upon its oxidative properties. (1) Ozone acts by initiating peroxidation of polyunsaturated fatty acids (PUFAs) present in the cell membrane. The peroxides and secondary reactive oxygen species which ensue produce their toxicity by damaging the integrity of the cell membrane and other cellular molecules. (2) Ozone exerts its toxicity by oxidation of compounds of low- molecular weight like those ontaining thiol, amine , aldehyde and slcohol functional groups and by oxidation of proteins. Both soluble peptides, such as glutathione and protein in lipid bilayers provide potential targets for ozone action. Protein modification takes place via oxidation of amino acid side roups So, two mechanisms of ozne toxicity may be interrelated. Peroxidation of PUFAs gives rise to water-soluble products such as aldehydes, peroxides and hydroxyl radicals which diffuse into the cytosol and initiate oxidation of amino acids and proteins.

Direct oxidation of amino acids and proteins by high ozone levels or oxidation by secondary reaction products of PUFAs peroxidation can inhibit a variety of

cellular protective systems. These include glutathione, a scavenging thiol, glutathione peroxidase, superoxide dismutase and catalase, which detoxify peroxides, enzymes which supply reducing cofactors such as glucose-6-phosphate dehydrogenase and ant proteases, which play a role in the inhibition of ozone-mediated leakage and edema. Both thiols and enzymes may be restored metabolically to control levels or rebound to higher protective levels following intermittent or continuous ozone exposure.

The degree to which ozone reacts with proteins is determined by the presence of ozone-susceptible amino acids at their active sites and the location of the amino acids in the tertiary structure of the protein. For example, cystein, methionine and tryptophane are very susceptible to ozone and the oxidation of tryptophane produces peroxides which are toxic and give rise to other reactive toxic oxygen species.

The hypothesis that lipid peroxidation is the primary factor in ozone mediated toxicity has its strongest support in the findings that vitamin E, a dietary antioxidant, is a powerful protective agent in ozone toxicity. Its effectiveness is further enhanced by other antioxidants such as ascorbic acid and butylated hydroxytoluene.

Peroxidation of PUFAs by ozone results in the generation of fatty acid hydroperoxides. These are destroyed by glutathione peroxidase-consuming glutathione. Oxidized glutathione is reduced y glutathione-reductase consuming NADPH. Thus, the loss of glutathione following ozone exposure promotes lipid peroxidation indirectly through the inhibition of glutathione peroxidase or by direct oxidation and depletion of glutathione. Peroxides formed via lipid

peroxidation induce glutathione peroxides and this in turn induces enhanced levels of the enzymes required to supply reducing factors such as NADPH to glutathione peroxidase. Vitamin E suppresses spontaneous formation of lipid peroxides. The supplementation of vitamin E in the diet would therefore decrease the utilization of glutathione peroxidase and maintain a high level of protection in the cell . there is a cross function between various antioxidants. Selenium, an essential factor in selenium-dependent glutathione peroxidase and an inducer of the enzyme, prevents vitamin E deficiency and increases the transport and utilization of the vitamin

Following exposure to high levels of ozone, the relative importance of PUFAs peroxidation and the oxidation of proteins and comounds of low molecular weight depends on many factors. These include (a) membrane composition of PUFAs and proteins, which determine ozone accessibility and degree of interaction and damage,(b) enzymatic pathyays to decompose peroxides, (c) pathways to generate thiols and (d) the presence fo antioxidants (vitamin E, vitamin C and selenium) to prevent peroxide formation and to partake in scavenging free radicals arising from secondary reactions.

Toxic effects of ozone:

Ozone is an extremely reactive oxidant molecule. Its toxicity is complex because of the large number of biological systems that can be affected and the variety of effects that can result from ozone interactions with cellular components. The toxic effects of ozone are manifested upon its inhalation and absorption in the lungs. The pulmonary system is therefore the primary target for ozone toxicity through extrapulmonary effects.

The levels at which ozone toxicity becomes evident are influenced by a variety of parameters. These include genetic factors (species, airway anatomy, stage of development) as well as host factors such as preexisting disease state, age, dietary and hormonal status and cellular protective systems. The lattar, which directly or indirectly suppress the oxidative damage induced by oone, serve as important determinants in establishing the consequences of ozone health effects.

A- Pulmonary effects of ozone:

Ozone is an air pollutants and a major component of photochemical smog, It is a respiratory irritant producing irritation of the upper airways and high concentration may even produce fatal pulmonary edema in both humans and experimental animals.

The sensitivity of lung tissue to ozone-induced damage varies with cell type and location. The ciliated cells in the airway passages and the squamous alveolar epithelial cells (type 1 cells) are the most sensitive to ozone. Type III alveolar epithelial cells are more resistant to ozone and in fact serve as progenitor cells which differentiate into type I cells during the process of repair of ozone lesions. Morphological changes following ozone exposure also differ with the state of the animal and are modified by an altered nutritional or immunological status. For example, rats maintained on vitamin E deficient diets tend to develop more morphological lesions as compared to those maintained on vitamin E-supplemented diets.

Studies of airway reactivity following ozone exposure indicate a hyperactive state resulting from a disruption of the respiratory epithelium and a sensitization of the underlying nerves to chemical and mechanical stimuli. This sensitization

may be an underlying factor in the reflex broncho-constriction and the repid shallow breathing observed following ozone exposure; conditions which are enhanced if exposure takes place during exercises. Epithelial damage induced by ozone has also been implicated in the enhanced allergic reactions to inhaled foreign proteins. Chronic exposure to ozone causes pulmonary arterial lesions that result in thickening of the arterial walls. Ultra structural changes in the alveolar capillaries have also been found.

A large number of enzymes are inhibited following ozone exposure. The cytochrome P-450 enzyme system, important in carcinogen and drug metabolism, is inhibited in hamsters and rbbits following short exposure to ozone (0.75 – 1.0 ppm). The inhibition of cytochrome-P4-450 activity increases the hazard of ozone exposure due to the decrease in detoxification of inhaled chemicals including pneumotoxicants and carcinogens. The inhibition of prostaglandin synthetase, a membrane bound enzyme, has also been reported. The decreased lung cholinesterase activity which has been observed following ozone exposure could result in elevated acetylcholine concentration in the bronchial muscle that end by enhancement of bronchial concentrations following a given stimulus.

The action of ozone on pulmonary protein synthesis falls into two general categories: (a) an effect on the synthesis of collagen and structurally related proteins, which is directly related to ozone-associated lung fibrosis;(b) an action on glycoprotein synthesis and mucous secretion, which affects their role in protecting underlying cells from ozone toxicity and in removing adventitiously inhaled particles.

Continuous exposure to ozone for 7 days (0.5 – 0.8 ppm) results in a significant enhancement of collagen synthesis and precursor protein. The inhibitory effect of

ozone on the synthesis and secretion of mucus glycoprotein synthesis in tracheal explants varied with the species.

B- Extrpulmonary effects of ozone:

1- Central nervous system and behavioral effects:

Rats and mic exposed to low levels of ozone (0.5ppm) resulted in a depression of motor activity. Limited data on the effects of ozone on the enzymes in the central nervous system indicate a variability depending on the studied enzyme. For example, catechol-O-methy1 transferase is decreased while the levels of monoamine oxidase are increased. Despite reports dizziness, throat and nose irritation, and visual impairment in humans exposed to ozone; there is limited information on ozone-induced changes in animal behavior.

2- Effects of ozone on the immune system:

Marked impairment of the pulmonary host defense mechanisms. Ozone increase the incidence of pulmonary infections induced by a number of other pathogenic organisms, including streptococcus sp., Pasteurella haemolytica and Mycobacterium tuberculosis. The increased susceptibility may relate to ozone induced impairment in alveolar macrophage function.

- Ozone-induced systemic immune dysfunction has also been demonstrated and cannot be ruled out as an addition ional factor, which impair host defense. Alterations in rabbit alveolar macrophage production of arachidonic acid metabolites (increased prostaglandin E2, PGE2) following in vitro and vivo ozone exposure have been also reported. Non immunological mechanisms may

contribute to decreased host resistance. Ozone-induced impairment in the mucuciliary clearance and increased mucous secretion could result in an accumulation of pathogenic organisms. Lung inflammation occurs in humans exposed to extremely low level of ozone.

3- Hematological effects:

The effects of ozone exposure on hematological endpoints have been investigated in many in vivo and in vitro studies. Exposure of red blood cells (RBCs) to ozone resulted in some changes including increased fragility, potentiation of complement-dependent membrane damage, formation of Heinz bodies, inhibition of Na+/K+-ATPase and spherocytosis RBCs from humans exposed to 0.5ppm ozone for 2 hr showed enhanced glutathione and glucose-6-phosphate dehydrogenase activity, while that from rats and monkeys at the same dose and conditions failed to show such changes. Oxidation of intracellular glutathione has been observed in human RBCs exposed to 0.84 ppm ozone for 2 hr supporting earlier studies which showed a decrease in RBCs glutathione following long-term exposure of rats to 0.5 ppm ozone for 23 days. Other changes have been detected in serum of animals. These include decreased albumin and enhanced levels of globulins in rabbits and increased lysozyme activity and prostaglandin levels in rats.

4- Endocrine effects:

The endocrine system is adversely affected by ozone inhalation. Morphological changes are observed in the parathyroid after a single exposure to ozone. - Reduced levels of thyrotropin and thyroid hormones were shown in rats exposed to 1 ppm ozone for 24 hr, suggesting changes in hypothalamic function.

Hormones have been recognized as playing a role in modulation ozone toxicity. A protective effect has been observed by removing the thyroid, hypophyseal or adrenal glands, suggesting that hormones such as thyroxin may potentiate the toxic action of ozone.

5- Reproductive effects:

The reproductive and teratogenic effects of ozone have been investigated. Mice parentally exposed to 0.2 ppm ozone showed reduced infant survival. Progeny of dams exposed to 1 ppm ozone during late gestation showed slower growth rates and retarded early reflex development.

6- Genotoxic effect:

The genotoxic effects of ozone have been predicted from its radiomimetic character. Free radicals produced upon decomposition of ozone in water, such as OH-radical, are similar to those produced by ionizing radiation and thought to play a role in some of its genotoxic actions. An induction of chromosomal aberrations in human fibroblasts due to exposure to 1960 ug ozone m-3 was observed. Different results were obtained when human lymphocytes were exposed to ozone both in vivo and in vitro. No effect was shown in hamster and mouse peripheral lymphocytes exposed in vitro to 1 ppm ozone or less 5 hr. in contrast, a significant chromosomal aberrations were found in hamster peripheral lymphocytes when exposed to ozone under similar previous conditions.

7- Carcinogenic effects of ozone:

- The role of ozone in carcinogenesis is unclear. Some of its actions as a powerful oxidant and producer of free radiation, a complete carcinogen, Produces chromosomal aberrations in synergistic fashion with ozone, These may play a role in the neoplastic process. Free radicals produced gy oxidants damage to DNA and modify cellular genetic integrity, could lead to carcinogenic events. The role of free raicals in carcinogenesis is seen by the fact that scavengers of free radicals, such as catalase, inhibit oncogenic transformation in vitro by radiation and chemicals. In addition, dietary factors such as selenium enhance the breakdown of peroxides in the cells exposed to radiation and chemicals and prevent oncogenic transformation.

5- Nitrogen oxides

They are mixture of oxides of nitrogen and including: 1- Nitrous oxide (Laughing gas), Nitric oxide (No) and Nitrogen dioxide (NO2). They are formed by the thermal decomposition of atmospheric nitrogen and thus can be formed by almost any combustion process.

1-Nitrous oxide:

Nitrous oxide (dinitrogen oxide , N2O) once commonly known as "Laughing gas" is used as an oxidant gas and in dental surgery as a general anesthetic. It is the only inorganic gas used for clinical anesthesia. It has both anesthetic and analgesic and analgesic actions, and equilibrates with blood much more rapidly than halothane or ether. It is an asphxiant, colorless gas, with slightly sweetish odor and taste.

Toxic effects of N2O:

It is an asphyxiant gas, it is a central nervous system depressant, severe bone-marrow depression, decreases the activity of methionine synthetase due to displacement of cobalamin from the enzyme, in chronic inhalation, polyneuropathy was reported. N2O affects on the reproductive system for both sex male and female (A-When pregnant rats exposed to N2O 70-75% on the 9^{th} day of gestation they suffered from fetal resorption. Skeletal anomalies and, other teratogenic effects, and B-In an another study, pregnant rats exposed 0.5% N2O in air they had a significant reduction in litter size, No evidence of fetal resorption or skeletal malformation, and C-A reduction in mature spermatozoa in rats exposed to 20% nitrous oxide for 35 days was found).

2-Nitric oxide:

It is a colorless gas used in the manufacture of nitric acid. It is produced in combustion processes from organically bound nitrogen endogenous to fossil fuels (particularly coal, heavy fuel oil, and shale oil) and from atmospheric nitrogen under the conditions that exist in an internal combustion.

Toxic effects fo No:

On inhalation, NO causes cyanosis, ataxia, tachycardia and other respiratory system symptoms that may lead to respiratory arrest.

3-Nitrogen dioxide:

Nitrogen dioxide (synonyms: nitrogen tetroxide, NTO, dinitrogen tetroxide, nitrogen peroxide) is a dark-brown fuming liquid or gas with a tetroxide, nitrogen

peroxide) is a dark-brown fuming liquid or gas with a pungent, acrid odor. It is highly toxic and is the main precursor of ozone. NO2 is formed from nitric oxide in presence of ozone or oxygen as following:

(organics, photochemical processes)

2NO + O2 ----------► 2NO2

(stagnant air, low humidity, intense sunlight)

It is generated in large quantities as a result of burning fossil fuels for heating, power generation and as petrol and diesel oil in motor Vehicles.

Toxicokinetics of NO2:

NO2 is poorly absorbed by the upper respiratory tract and peak tissue doses occur at the Junction of the conducting air-ways and the gas doses occur at the junction of the conducting air-ways and the gas exchange zone of the lung. As is the case for most inhaled compounds, exercise leading to increased ventilation and an increased oral inspiratory flow, raises the proportion of the inspired gas which reaches the lower air ways.

Effects due to NO2 poisoning:

1- Inhalation of NO2 causes severe irritation of the upper airways and lung tissues resulting in pulmonary edema, fatal bronchiolitis fibrosa obliterans, damage to cilia, stripping of bronchiolar epithelium, emphysema, interstitial fibrosis, impairment of macrophage function. (Inhalation of air containing 200-600 ppm of NO2 for even very brief periods of times can be fatal).

2- Disruption of some enzyme system, lactate dehydrogenases.

3- Acts as an oxidizing agent result in formation of free radicals and undergo chain reactions in the presence of O2 resulting in their oxidative destruction).

4- NO2 combines with water to form nitric acid resulting in damaging of cell membranes.

5- It increases the susceptibility of lower respiratory tract to infection.

Treatment: Corticosteroids.

Ammonia

Ammonia is a naturally occurring substance that plays a vital role in protein metabolism. It is a colorless gas at ambient temperature and pressures with a strong, irritating odor, and is classified as a flammable gas by the National Fire Protection Association. Most of the ammonia produced worldwide is used for fertilization . It is used extensively as a refrigerator in commercial installations . It is also used in the manufacture of chemicals such as plastics, explosives, nitric acid , urea, hydrazine, pesticides and detergents. In addition to being handled as a compressed gas, NH3 is commonly encountered as aqueous solution of 28% (aquammonia), called ammonium hydroxide and 10% called household ammonia.

Mechanism of action and toxicity:

The toxic action may be attributed to the inhibition of Kreb's cycle of citric acid in the brain cells. In ruminants, the toxicity of ammonia depends on the pH of the rumen contents. At a low pH the toxicity is low while at high pH the toxicity is high.

Clinical symptoms due to ammonia poisoning:

The clinical signs are increased respiratory rate tremor, fibrillar muscular twitching, salivation, bloating, and tonic- clonic spasms.

Diagnosis:

The diagnosis of ammonia poisoning depend on the case history clinical signs, evaluation of feed samples, digesta, rumenal contents and blood. The contents of the rumen are examined laboratory for pH value and for concentration of ammonia.

Therapy:

Ammonia intoxication should be treated by causal and symptomatic therapy

1- Modern causal therapy of ammonia poisoning is aimed at :

A) Reducing the generation of ammonia in the animal or slowing down its absorption. It is necessary to avoid administration of andy feeds which would serve as a source for the production of ammonia by the microflora of the digestive tract . Acids should be given in order to substantially decrease the pH of the rumen content, thus reducing the activity of urease and other ammonia generating enzymes. The recommended dose for cattle are 3-5L of 2% acetic acid , about 3-5L of 2% vinegar (the percentage indicate the content of acetic acid) and about 1L of 0.2% HCL. The doses for sheep should be about three to seven times lower.

B) Promoting reactions in which ammonia is converted into non- toxic substances. E.g. arginine i. p. Protamine reduces the levels of blood ammonia. Glutamic acid therapy gave good results in cattle which were treated with 50-2000 g of the acid administered orally as a suspension in lukewarm water (the protective action of glutamic acid in the complex metabolic processes of

ruminants can be attributed to the effect of a decrease in the rumen liquor pH value).

2- Symptomatic therapy depends on the symptoms in the clinical picture. For circulatory system caffeine is effective . Also i.v. glucose is effective. In severe symptoms of tetany, calcium infusion is indicated.

Sulfur dioxide (SO2)

It is an intermediate in the production of sulfuric acid. It is a common air pollutant produced by the combustion of pyrite (FeS2) in coal and organically bound sulfur in coal and fuel oil. These sources add millions of tons of sulfur dioxide to the global atmosphere annually and largely responsible for acid rain. SO2 is an irritant to the eyes, skin, mucous membranes and respiratory system. As a water- soluble gas, it is largely removed in the upper respiratory tract. When SO2 dissolved in water it produces sulfurous acid , (H2SO2), hydrogen sulfite ion (HSO3) and sulfite ion (So32-) Sodium sulfite (Na2SO3)has been used as a chemical food preservative. It is used in the manufacture of sodium sulfite, sulfuric acid, disinfectants , fumigants, glass, wine, ice and industrial and edible protein.

Toxicokinetic of SO2:

SO2 is soluble in water and considerable absorption occurs in the upper airways including the nose and mouth. Absorption is concentration dependent with more than 90% of inspired SO2 being absorbed at high concentrations.

Effects of SO2:

It produces bronchoconstriction when inhaled in high concentrations, asthma, pulmonary edema, respiratory paralysis. SO2 can cause rhinitis.

Conjunctivitis, corneal burns and opacity, rales, pneumonia, altered sense of smell, dyspnea, a thickening of the respiratory mucous layer and inhibition of ciliary movement. Acute poisoning can lead to death by asphyxiation. SO2 has also been shown to enhance the carcinogenicity of polycyclic aromatic hybrocarbons.

6- Hydrogen sulfide (H2S)

It is a colorless gas with a foul rotten- egg odor. It is produced in large quantities as a byproduct of coal coking and petroleum refining and massive quantities are removed in the cleansing of sour natural gas. It is a major source of elemental sulfur by a process that involves oxidation of part of the H2S to SO2 followed by the Claus reaction. H2S is a very toxic substance, which in some cases can cause a fatal response more rapidly even then hydrogen cyanide. It affects the CNS. Causing symptoms that include headache, dizziness and excitement. Rapid death occurs at exposures to air containing more than about 1000 ppm H2S, and somewhat lower exposures for about 30 minutes can be lethal. Death results from asphyxiation as a consequence of respiratory system paralysis.

Uses H2S:

Hydrogen sulfide is used in the synthesis of organic and inorganic sulfides and is generated in the decomposition of sulfur- containing organic matter and in industrial processes involving sulfure compounds.

Mode of action of H2S:

The most common route of entry into the body is by inhalation. H2S is an acutely toxic gas that may bring about immediate coma. It is an irritant to the eyes and respiratory system. The systemic action of H2S is due to inhibition of cytochrome oxidase and death, when it results , is due to respiratory failure. Although H2S is an extremely toxic gas, fatalities are less common than might be expected due to its foul odor , to which the human olfactory sense is particularly sensitive, acting as an early warning system.

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Chapter 7 Neglect

Introduction

Neglect is the most common form of animal abuse encountered. The term neglect is commonly used to refer to the failure to provide adequate food, water, and shelter. Neglect can be more generally defined as the failure to provide for an animal's needs. This includes proper medical care, adequate space, appropriate food, maintaining the animal's hair coat and nails, and providing sanitary conditions. Neglect can also apply to any situation that has a negative impact on the animal, such as embedded collars, short tie-outs, and heavy chains. Some state laws and local ordinances have vague definitions that are open for interpretation, whereas others have clear, detailed definitions of what constitutes neglect. Neglect can be an act of omission or commission. In cases of neglect, it is important to note what would have been obvious to the owner, such as a foul odor from an embedded collar. In criminal cases, the issue of intent is a vital criterion in deciding on appropriate charges and sentences. Neglect is often a continuum of action or lack of action by the owner over a prolonged period of time. The issue becomes at what point in this continuum the owner had to have knowledge of the problem and still failed to take appropriate action. This can be defined as impliedmalice. In addition, it can help define the animal's suffering. The veterinarian's assessment of the animal's condition and environmental findings is the foundation for these legal issues.

Environment Examination

The animal's environment usually holds the most critical information in neglect

cases. The veterinarian needs all this information, especially photographs, to analyze the physical findings in context with the environment and determine what tests to perform. Often multiple problems are found at the scene. Common findings include crowding, short tie-outs, lack of shelter, and exposure to the elements. Usually there is a lack of potable water, containers that the animal cannot access (i.e. 5-gal buckets with food or water only at the bottom), inappropriate food (dog food for puppies, large kibble for small-breed puppies), and food unfit for consumption. Neglect cases often have environments that are malodorous, filthy, and cluttered with hazardous material, with urine and feces covering most surfaces. The level of ammonia in the environment can contribute to eye and respiratory problems. It is important to look for evidence of infectious diseases at the scene, such as vomiting and bloody diarrhea. The presence of infectious disease may be a primary or secondary problem. Severe environmental infestations of fleas can cause anemia in the live animals. Any deceased animals should have a necropsy performed to determine cause of death and rule out any infectious disease. A time estimate should be given for the conditions to have been present. This may be evident by old, dried, moldy feces; debris covering the fronts of pen/cage doors; moldy food; and algae in the water container. There may be botanical evidence, such as vines growing over pens and the lack of grass around the place dog was tied-out. In cases in which there are deceased animals, entomological evidence may provide an estimate for the time of death.

Death due to starvation

It is usually occur due to neglecting, losing way in vast desert, criminal withdrawal of food and due to entombment of pits in fallen houses. During the first few days, glycogen stores of the liver and muscles are called upon but these are of limited value because the main source of energy is the reserve of fat. The breakdown of tissue proteins also takes place to supply glucose needed for the combustion of fat. The fat used in starvation is that stored in the subcutaneous tissues and muscles and that fat which form part of the cell structure is spread till the end. When the fat stores are exhausted protein alone is available and death rapidly occurs so females stand starvation better than males (fatty).

Signs

General weakness and acute pain in the stomach, which is relived by, pressure. Nervous signs, due to acidosis result from incomplete combustion of fat. Consequently and after 7 days, sunken face, wild eyes, dilated pupil and hot breath. Dry rough skin and wrinkled emitting a peculiar disagreeable odor. Dry mouth and thick saliva, constipation, oliguria and intolerable thirst. The body is dried up and appears like mummies with marked prostration and prominent bony projections, abdomen is sunken and the extremities become thin and flaccid with loss of muscular power. Before death diarrhea or dysentery, subnormal temperature and death may be preceded by convulsions. Death occurs when weight reach 2/5 or 40% of the weight.

Treatment

1- A very gradual increase in the amount of warm liquids. If the stomach is suddenly filled, vasomotor disturbance may occur and may even led to death

2- Stimulants.

3- Milk in small amounts.

Post-mortem appearance

1- Shrunken, dries skin and free from fat.

2- Soft muscles and internal organs deprived of fat and much reduced in size. The heart and kidneys are free from any surrounding fat.

3- Collapsed and empty stomach and intestine and the mucous membranes thinned out and may be transparent.

4-The liver is small but the gall bladder is distended with bile and the omentum is free of fat.

5- You must bear in mind that there are certain pathological conditions which to progressive wasting and emaciation of the body. It is very necessary to examine all the internal organs to search for the existence of any of these diseases. N.B. In some criminal cases food may be given before death to hide the crime, this food will however be found undigested in the stomach.

Death due to thirst

It is mainly due to water deprivation. The desire for water quickly becomes overpowering, disappearance of the saliva, the throat and tongue become dry and swallowing of other food become impossible. Dry skin, which is hot and later, becomes shriveled. Muscular power diminishes rapidly, vertigo, dimness of vision set in together with diminution of urine and restlessness. Wasted body fats, dries

and shriveled skin, sunken eyes and thick blood. Small amounts of warm liquids. The symptoms of thirst are of central origin and will not disappear unless the entire system has received enough fluid. It is well known that left for them, these subjects are likely to die from overloading their stomach with water ending in acute heart failure.

Death due to cold

Hypothermia is usually caused by exposure to low environmental temperatures. It occurs when the body heat loss exceeds heat production. Immersion in cold water can cause more rapid loss of body heat than exposure to cold air temperatures. Animals are more sensitive to the cold if they are not properly acclimated. Cats are much more sensitive to sudden changes in temperatures than dogs. Hypothermia is much less commonly reported in cats most likely caused by lack of detection. Small, aged, bad nourished, diseased, fatigue, and exhausted animals are more susceptible also humidity is from the predisposing factors. Tissue anoxaemia due to the delayed action of the respiratory enzymes so that the blood and tissues exchange oxygen less readily, the metabolism is much lowered and the body temperature reaches so low a level that the vital processes cease (inhibition of respiratory enzymes). Shivering, sense of fatigue and sleepiness. Coma and death (sometimes convulsions occur before death).

PM: (not distinctive)

1- Cold pale body and red congested conjunctiva.

2- Red congested viscera (owing to combination of oxygen with HB), multiple thrombosis and gastric erosions.

3- Stiff and frozen body (stiffness should be differentiated from rigor mortis, as the cold stiffness will gradually disappear if the body is removed to a warm place. Rigor mortis will set in later (slows to appear and lasts longer) Putrefaction in such bodies is suspended. If we find frozen body showing any evidence of putrefaction this would mean that putrefaction was there before freezing and death could then be ascribed to causes other than cold.

Death due to heat stroke

It is condition caused due to the exposure of the body to high atmospheric temperatures, especially in the presence of excess of humidity resulting in either damage to the cells of the CNS and so failure of heat regulatory mechanisms (Heat hyperpyrexia) or damage to the cardiac muscles and heart failure (Heat exhaustion). Exposure to the direct sun rays is not necessary to cause heat stroke, but the condition may occur in the shade, or inside houses, or even at night in humid hot places, so it may occur in animals while playing in a closed, hot and bad ventilated room. Non acclimatization to high atmospheric temperature, thus it is more common in European breeds live in tropics, fattened animals and heavy hair coat, fatigue, overcrowding and inadequate ventilation, previous affection with heat stroke and pyrexia diseases like fever may be Predisposing factors for heat stroke:

Heat hyperpyrexia (heat or sunstroke)

It is due to exposure to both high temperature and high humidity. Signs set in sudden in the form of rise of temperature, coma, and convulsions or set in gradually in the form of headache, rise of temperature, restlessness, nausea and vomiting with hot and dry skin. Gradual rise of temperature to 41-42 c°, the face

is flushed and cyanosed. Delirium and convulsions. Coma with stertorous breathing and contracted pupils and muscular paralysis which end in death.

Heat exhaustion

It is may be due to exposure to high temperature and low humidity. The onset is sudden with weakness, fainting, inability to walk, mild rise in temperature, sweating, oliguria, rapid and weak pulse and dilatation of the heart. The skin is cold, pale face and dilated pupils.

Heat cramps

They are very severe and painful cramps affecting the muscles of the limbs and abdomen in the working animals caused by loss of sodium chloride in the blood due to excessive sweating.

Post-mortem appearance:

1- Early rigor mortis.

2- Continues rise of temperature after death.

3- Putrefaction may set in within few hours of death.

4- Generalized congestion. Edema of the brain and meninges with marked degenerative changes in the cortex and basal ganglia cells.

5- In severe cases, petechial hemorrhages under the skin and m.m.

6- The right side of the heart is dilated and filled with dark and venous congestion is marked in all organs.

Treatment:

1- Rapid thorough cooling of the body and cold compresses to the skin. Ice bags to the head and ice cold rectal enema.

2- 10 g sodium chloride in heat cramps.

3- Symptomatic treatment

Death from lightning

Lightning always travels in the air in places of least resistance. The commonest sites of lightning are places near good conductors, as high buildings, railways and similar objects. Lightning is quite rare in Egypt. If the animal stroked by lightning and does not instantly die, it may get noises in the ears, temporary loss of vision and congestion of the face, rapid irregular and weak pulse, stertorus breathing and dilatation of the pupils, tremors, convulsions and psychic shock or nervous irritability which may be permanent and abortion of pregnant females.

The most important causes of death in lightning are the passage of the current through the nerves to the brain, causing a severe nervous shock and suspension of its important functions. Sometimes, the patient recovers with a permanent tingling and numbness or even paralysis of limbs and face and psychic disturbances.

Post-mortem appearance

External injuries

1- Burns at the site of current entrance and exit vary from simple hyperemia to complete charring. They occur at the entrance site, causing thick raised edges and coagulative necrosis. Lightning may either cause it or from the clothes taking fire or opposite metallic objects as watch, piece of coin.

2- Singed hair if burn occur in hairy area.

3- Arborization or crystallization in the direction of current due to, dilatation of the superficial blood vessels in the skin due to hyperemia or even clotting of the blood in these vessels.

4- Wounds in the form of punctured or contused wounds bruises or even fractures of bones. Extensive wounded limb or the neck may be severed, and they look clearly cut as if done by a sharp instrument, however no hemorrhage or extravagation of blood can be found, probably due to the accompanying coagulative necrosis.

Internal injuries (no characteristic post-mortem picture)

1- Sometimes pale brains and congested in other cases.

2-Skull fractures, the brain may be lacerated with effusion of blood around.

3- Other internal organs lacerations.

Death due to electric current

Electric currents of high potentials as those are used in factories, picture hoses and tramways are quite dangerous. Any contact with them leads usually to deaths. Currents of low potentials, as those are used in household lighting (200 volts and below) under ordinary circumstances, don't usually cause death but a few temporary symptoms in form of spasm of glottis, burning pain or dizziness causing the victim to fall to the ground. The effect of electric current on animal body depends on voltage, amperage and duration of current. Death from electric current depends on the parts of the body through which the current passes. Paralysis of the medullary centers if a current of high voltage passes through the head. Ventricular fibrillation of the heart muscles may occur if the current passes through the trunk. Spasmodic contraction of the respiratory muscles, with resultant asphyxia occurs if the current passes as well through the trunk.

If the victim does not die immediately signs appear in the form of loss of consciousness, stertorous breathing with slows pulse and subnormal temperature, symptoms of kidney dysfunction due to the excretion of the changed proteins liberated at the site of passage of the current or signs of cerebral irritation and Other delayed symptoms may appear even months after the accident in the form of, skin or bones necrosis, chronic kidney troubles, disturbances of the central nervous system, optic atrophy and other eye changes due to the occurrence of cell degeneration.

Post-mortem appearance:

External signs

1- External local burns may be found in the sites of entrance or exit of the current in the hands and feet, these are small, dry, parchment-like areas

with pale indurate edges especially at the entrance and surrounded by a line of hyperemia.

2- In currents of high voltage, splitting of the skin, especially at the exit may occur together with ordinary burns opposite metallic objects,

3- In some cases, we may found arborization or hyperemia of the superficial vessels of the skin in the direction of the current passage.

4- Metallization or spreading of the melted wire into the skin in the neighborhood may also occur in cases of high voltage currents.

5-The body shows generalized congestion, congested eyes, well-marked hypostasis and early appearance of rigor mortis.

Internal signs

Appear in the form of generalized congestion of the viscera with dark fluidy blood in the vessels (picture of asphyxia) and a number of petechial hemorrhages along the path of the current.

Treatment:

1- Cut the current off.

2- Artificial respiration evens if it appears dead.

3- Respiratory and cardiac stimulants.

4- Warmth to keep the body temperature.

5-Lumber punctures only in the presence of nervous irritability. Ttaking care not to remove much fluid or to have a rapid flow.

Untreated Injuries

Some cases of neglect involve untreated injuries. The veterinarian must document the original injury and the results caused by lack of treatment. The degree of pain and the affect on the animal's mobility, appetite, and ability to perform normal functions should be documented. It is important to document the prognosis of regaining full function and the likely outcome of treatment now versus if it had been initiated when the animal was first injured. It may be helpful to give an estimate of the cost of current treatment and follow-up and compare it to what the cost would have been if treatment had been initiated after the injury.

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Chapter 8 Sexual Offences

Overview

Sexual assault of animals is a sensitive and uncomfortable issue for those involved and is often met with silence and inaction. It is a crime of abuse and cruelty shrouded in sexual violence. One cannot ignore the fact that this form of abuse inflicts serious harm and sometimes death on the victim. Sexual abuse of animals encompasses a wide arrange of behaviors, including vaginal or anal penetration; genital fondling; penetration with an object; or the injury or killing of an animal for sexual gratification. It is the eroticization of violence, power, and control. To recognize this type of animal abuse it is important to understand the types of sexual abuse the animal may be subjected to and its significance to society. Sexual abuse of animals has also been recognized as one of the early warning signs of psychological dysfunction, including conduct disorder in children and adolescents, and antisocial personality disorder in adults. Sexual abuse of animals has similar characteristics and distinctions as child abuse. A correlation has been found with childhood sexual abuse in some of the cases of animal sexual abuse. This type of animal abuse also has been linked with violent sex offenders. A connection has been seen between sex with animals and those engaged in sadomasochistic practices. The type of sexual interaction between a human and the animal includes masturbating the animal, receiving oral sex, performing oral sex, performing vaginal intercourse, performing anal intercourse, receiving anal intercourse, sodomy with objects, and the animal as a surrogate for a behavioral fetish such as sadomasochistic practices or sexual murder. Necrophilic tendencies have been

seen with the animal first killed during sexual gratification; then the dead body may be used for masturbation, or may be dissected and mutilated. The species of animals that offender's abuse include chickens, dogs, cats, horses, cows, sheep, and goats with either a male or female animal involved. Invariably, cases of sexual abuse are seen in the practice of veterinary medicine. A study in Germany revealed that 36 percent of surveyed veterinarians had seen animals involved in bestiality. The owner may or may not be aware that the animal has been assaulted; only bringing in the animal if it is injured or has developed an infection. The range of injuries and physical findings of the study by Munro and Thrusfield parallels those found in human sexual assault victims. The findings in dogs and cats from the study include: vaginal trauma, vaginal hemorrhage, recurrent or refractory vaginitis, knife wounds in the vagina, uterine tears near the cervix, cervical scarring, uterine or peritoneal hemorrhage, necrotic anal mucosa, anal dilation, anal tears, ligature around the genitalia (penis or scrotum), necrosis of the scrotum or testicles with a ligature no longer present, castration, and penetrating wounds around the anus, vulva, or perineal area. In addition, intrauterine, intracervical, or vaginal foreign bodies were reported, including a candle, knitting needle, sticks, a broom handle, and a possible tampon.

Crime Scene Investigation

As in all cases of suspected abuse, the circumstances surrounding the injury must be considered and it is important to rule out other causes of the injury. Equally important is to show due diligence by performing diagnostics to either support or exclude sexual assault. At the crime scene, all bedding around the animal and anything he or she could have sat or laid on should be collected for analysis.

Semen can leak from an orifice onto other surfaces. If present on the fur, it could have been transferred to the surrounding surface items either by contact or the animal scooting after the event. The scene needs to be examined for evidence the animal may have defecated or urinated after the assault, and the feces or urine must be collected.

General Findings

The injuries from animal sexual abuse parallels that found in child abuse and human forensic pathology. The injuries range from severe and even lethal to the complete lack of any abnormal finding. The injuries may involve the anus, perineal area, rectum, colon, vulva, vagina, uterus, scrotum, or penis. Any abuse that involves injuries to the anorectal region or genitalia by definition qualifies as sexual abuse. The injuries found in sexual assault victims depend on what was used to commit the assault, the type of assault, the size of the object, and the size of the animal. With penetration injuries, the size of the animal may be a factor in the degree of damage sustained. There may be evidence of acute or chronic abuse. These injuries may be related directly or indirectly to sexual assault or result from a history of repetitive abuse. The animal may have been stunned or physically beaten to gain control over it. There may be evidence of head trauma and other blunt force injuries to the body. It is possible for animal sexual assault victims to have peritonitis from rectal or uterine tears. Examination of the eyes and ears may reveal evidence of trauma, including retinal hemorrhage, retinal detachment, ear canal hemorrhage, ear canal petechiae, or injuries to the pinnae. The ears may show acute or chronic injury if the perpetrator restrained the animal by grabbing the ears. Other injuries may be present on the rest of the body from restraints used on the animal or additional inflicted trauma. In human cases of sexual assault the

most common mechanism of death is asphyxia. This may be the case with animal victims, especially if they were surrogates for sexual murder. With any deceased animal that is thought to be the victim of a sexual assault, the neck should be carefully examined for evidence of strangulation or attempted strangulation. The animal's neck may be fractured during the assault or the throat cut. It is possible to see injuries to the tail, which may or may not produce bruising in the perineal region. The perpetrator may pull the tail and force it up or to the side, causing separation or fractures to the coccygeal vertebrae. This usually occurs in the proximal tail region, close to the pelvis. This is not an area of the tail that lends itself to getting caught in something, which is the most common cause of accidental tail fractures and vertebral separation. This injury causes bleeding in the surrounding tissues, which often dissects ventrally around the anus. This looks like bruising to the perianal region. Ligatures around the genitalia should not be labeled as just a childish prank. This type of abuse is a common finding in abused male children. In addition to the sadistic injury to the animal, there should be an investigation into why the offender had the idea to inflict the abuse in that manner. This issue should be addressed whenever any sexual assault is perpetrated by a juvenile on an animal.

Examination of the Victim

The initial exam should start with photographic documentation of injuries. In addition, a diagram of all injuries should be made. The external body should be examined carefully with a UV light source to find any trace evidence or bodily fluids, such as semen, saliva, urine, blood, fibers, or pubic hair. With deceased victims, the feet should be placed in paper bags and the body wrapped in a clean white sheet or body bag. These items should be examined for obvious evidence

after removal from the body and then preserved for further analysis at a laboratory. Fiber and hair found on the body should be collected and swabs taken of any fluids. These may be related to the assailant or the victim. In human sexual assault crimes it is common for saliva to be transferred to the victim during the assault. Consider the circumstances surrounding the crime in relation to the injuries to determine where the assailant would have likely deposited saliva on the animal. There may be ejaculate found on or near the animal if the attacker did not wear a condom. To collect samples of dried fluid, first moisten a sterile cottontip applicator with sterile water, and then swab the area, which will rehydrate the cells. Next, roll a dry sterile swab on the moistened area. Both swabs should be saved for testing. After the coat has been inspected with a UV light and evidence collected, the animal should be placed on white roll paper and the fur combed thoroughly to look for embedded trace evidence, such as pubic hair or fibers. During the assault, the animal may have transferred his or her own DNA or fur to the attacker. Samples for DNA and a sample of fur, including one of each color on the coat, should be collected and held for comparison testing. It is important to consider that the animal may have been drugged by the assailant to facilitate the act. Blood and urine should be collected immediately for toxicology testing. The drugs used on the animal may include illegal substances, human tranquilizers, or similar narcotics. The assailant may have given veterinary tranquilizers such as acepromazine, a commonly prescribed drug for animals that have anxiety related to travel, thunderstorms, or fireworks. It is possible the victim fought and scratched the attacker during the assault. The feet and legs need to be inspected for evidence of injury, ligature marks, nail injuries, and embedded trace evidence using a magnifying glass and a UV light source. After the collection of evidence, the nails should be scraped and then clipped, saving all scrapings and clippings for

analysis. In deceased animals, the nails should be removed using clean instruments to prevent contamination and saved for trace and DNA testing at a crime lab. Full-body radiographs, including the tail, need to be done on the live and deceased victim, to look for evidence of additional injuries. The external muzzle and oral cavity should be examined for evidence of trauma, foreign material, or trace evidence. It is important to consider what the animal may have done to defend itself during the assault or in response to pain. The mouth of an animal contains certain bacteria that may be linked to any bite wound infection inflicted by the victim to the attacker. The teeth need to be inspected for any tissue or trace evidence caught on the teeth. The oral cavity needs to be swabbed for possible DNA or other potential evidence related to the assailant or the crime. Swabs should be taken from the outside and inside of the lips, under the tongue, inside the buccal mucosa, and along the gums and teeth. The assailant's DNA may be present in the vagina or anorectal region. A human rape kit may be used to collect samples, which contain sterile swabs, microscope slides, slide holders, labels, gloves, and envelopes for hair evidence. Separate swabs should be taken all around the perineal and genital areas even if there is a lack of visible fluids with the UV light source. Swabs need to be taken of the vagina and rectum. Several rectal swabs should be taken prior to taking the animal's temperature or treatment of a rectal prolapse (see below) to prevent any cross-contamination. Slides should be made and air dried. A wet slide should be made and immediately examined for sperm, making note of any motility. Evaluation of sperm and semen is covered later in this chapter. Separate swabs should be saved for DNA, cultures, trace evidence, and other tests. Swabs should be placed in a cardboard box, allowing the swab to air dry. The animal's first bowel movement should be collected and preserved for DNA testing (the assailant's DNA). If it is known that the animal

had a bowel movement prior to examination, for example, during transport, that fecal sample also should be collected. A urine sample should be obtained in female victims. Because the opening of urethra is inside the vagina in dogs and cats, the assailant's sperm may travel from the vaginal area to the bladder. When examining the genitalia it is important to know what is normal to be able to recognize what is abnormal. An otoscope may be used for vaginal and anal exams, which are done preferably while the animal is under anesthesia. There is a vaginal speculum that may be used with the Welch-Allyn otoscope base. Colonoscopy may be indicated, especially if blood is found on the fecal swabs or the animal exhibits painful defecation. If there is vaginal or rectal penetration by the perpetrator, there may be severe trauma to these areas, causing deeper lacerations and subsequent peritonitis. If there is evidence of penetration with an object, trace evidence may be present from the object. Evidence from the animal also may be transferred onto the offending object. In addition to the injuries found in the study by Munro and Thrusfield, findings may include vulva and vaginal edema, abrasions in the perianal or perivulvar region, or along the vaginal mucosa or rectal mucosa. Bruising may be visible in these areas or on the surface of the cervix. Depending on the animal, the vulva folds may be forced inward by the penetrating object, protecting the internal vaginal mucosa. In this case, bruising, abrasion, or laceration injuries may be seen to the outside of the vulva folds and the cervix, if penetration reached that area.

Suspicious Exam Findings

There are certain physical findings that should raise the index of suspicion sexual abuse. These findings may be related to acute or chronic abuse. It is important in every situation to rule out other causes and take appropriate samples for testing. Sexual assault victims often go undetected by the veterinary community. Vaginal Penetrating vaginal assault can cause hemorrhage from the vulva caused by vaginal lacerations. Vaginal strictures may be result from previous sexual abuse. Recurrent vaginitis may be indicative of sexual abuse. Vaginitis can occur in sexually intact or neutered bitches, but is rare in queens. It may be caused by bacterial or viral infections, immaturity of the reproductive tract, androgenic stimulation, chemical irritation as with urine, or mechanical irritation. This mechanical irritation may result from neoplasia, anatomical abnormalities of the vagina or vestibule, foreign bodies, or human sexual penetration. The vaginal discharge associated with vaginitis may be mucoid, mucopurulent, or purulent, and rarely contains blood. Cytological findings may be non-septic or septic inflammation without hemorrhage. The vaginitis causes mucosal inflammation, hyperemia, and edema. A concurrent urinary tract infection commonly is found but it is not the cause of the vaginitis. In addition to vaginitis, other causes of vaginal discharge include vulvitis, pyometra, metritis, abortion, uterine stump granuloma or abscess, or retained foreign body. Vaginoscopy is needed to rule out anatomical abnormalities and other mechanical causes. Vaginal prolapse rarely occurs in the bitch or the queen. When seen, it is normally due to tenesmus, dystocia, or forced extraction of the male during the genital tie. Vaginal prolapse may be seen in dogs with estrogen stimulation, as seen in proestrus or estrus. It also may recur after parturition or even at the end of diestrus. Vaginal prolapse is primarily seen in young, intact, large breed dogs. The tissue becomes swollen because marked edema. The presence of a vaginal prolapse in a spayed female or

without other predisposing causes is highly suspect and the possibility of sexual assault should be thoroughly investigated.

Anus, Rectum, and Colon

There may be injury to the anus, rectum, or colon in sexual assault victims. Anal tears can be caused by anal penetration. Dilation of the anus can be indicative of spinal cord disease, spinal injury, or anal penetration and trauma. However, after death the anus relaxes and can be mistaken as traumatically stretched. Proctitis is the inflammation of the rectum. This may be seen because of trauma from rectal foreign bodies and sexual assault. It may cause recurrent rectal prolapses. Clinical signs of hematochezia, dyschezia, tenesmus, and pain may be seen with proctitis. Abdominal radiographs, proctoscopy, and colonoscopy should be performed on any animal showing these signs. Rectal foreign bodies can cause rectal fistulas, perirectal abscesses, and peritonitis. Rectal strictures may be caused by neoplasia or previous sexual assault. Sexual abuse must be considered as a possible cause for rectal prolapse. Appropriate swabs should be taken prior to treatment until the history is evaluated to determine the cause for the prolapse. Rectal prolapse is usually secondary to straining caused by rectal irritation. This irritation can be secondary to several predisposing factors, including enteritis, diarrhea, colitis, rectal foreign bodies, or sexual assault. Also it may be seen from a blow to the abdomen in which there is a sudden increase in abdominal pressure. Rectal prolapse is generally uncommon in animals with longstanding tenesmus and dyschezia. Rectal prolapse may be partial, involving the protrusion of the rectal mucosa through the anal orifice, or complete, with the protrusion of all layers of the rectum. It may or may not involve layers of the anal canal. The rectal mucosa exposed increases straining, which aids and promotes further prolapse.

Zoonotic Disease

A consideration for any sexual assault case is the possibility of zoonotic disease transmission during the attack. This pertains to any disease that is unique to the animal that could have been transferred to the perpetrator, such as a bacterial or parasite infection of the genital, intestinal, or urinary tract. Any intestinal parasite that has zoonotic potential, such as roundworms, may be transmitted to the assailant through the fecal–oral route. Because there may be residual fecal material around the genitalia, this transmission may occur through any sexual contact with the animal. To confirm the source of any human infection, perform DNA typing of the parasite found in the human if possible and compare the results with the profile of the parasite carried by the animal. Several bacteria that the animal may normally carry may cause infection in the assailant. The aerobic bacteria normally found in the vagina have been isolated from 59 healthy, breeding bitches. The bacteria, listed in descending order by percentage of isolates, are as follows: Pasteurella multocida (98 percent of dogs), _-hemolytic streptococcus, E. coli, unclassified gram-positive rods, unclassified gram-negative rods, Mycoplasma, a-hemolytic and non-hemolytic streptococcus, Pasteurella, enterococci, Proteus mirabilis, Staphylococcus intermedius, corniforms, coagulase-negative staphylococcus, and Pseudomonas st. (10 percent of dogs). In addition, there are bacteria with zoonotic potential that the animal may carry that can cause infection in the animal, assailant, or both. Coxiella burnetii is found in cats. The symptoms of cats may be subclinical, can cause abortions, or stillbirth.Humans can become ill from direct contact through aerosol exposure to birthing fluids passed by parturient or aborting cats. Humans develop symptoms 4 to 30 days post-exposure. These symptoms include fever, malaise, headache, pneumonitis, myalgia, and arthralgia. Approximately 1 percent of infected people

later develop hepatic inflammation or valvular endocarditis. Leptospira spp. can infect dogs and rarely cats. It can be transmitted to humans via urine from animals, usually by human contact with abraded skin or mucous membranes. The symptoms in Sexual Assault 231dogs can be fever, malaise, inflammatory urinary tract disease, inflammatory hepatic disease, uveitis, and central nervous system disease. Humans have similar symptoms, depending on the serovar of the Leptospira. Brucella canis is an infection of dogs that causes orchitis, pididymitis, infertility, abortion, stillbirth, vaginal discharge, uveitis, diskospondylitis, fever, and malaise. It is transmitted between dogs primarily by venereal transmission. Humans can be infected by direct contact with vaginal or preputial discharges from the dog. Symptoms in humans are usually fever, depression, and malaise.

Evaluation of Assailant's Sperm and Semen

Another special consideration in sexual assault cases is the condition of any recovered sperm. In humans, the motility and condition of the sperm can help establish timelines that enable an estimate of the time of the assault. In human rape cases, the sperm remains motile in live victims up to 6 hours and less often up to 12–24 hours. Sperm can survive longer in the cervical mucus than in the vaginal area. Non-motile sperm with tails can be seen up to 26 hours in living rape victims. Sperm heads without tails from the vaginal area may be seen up to 120 hours later. Sperm has been found 7 days later in a cervical swab. In anal and rectal swabs, sperm with tails are not commonly found, particularly if more than 6 hours have elapsed. Sperm heads were found in anal swabs 45 hours later; in rectal swabs 65 hours later. The sperm survival in deceased victims is shorter because the sperm are destroyed by decomposition. In humans sperm has been found in the vagina up to 2 weeks after death. Any sperm on a piece of material

and air dried may be recovered years later. Sperm may not be present in sexual assault cases. This may be because of the lack of ejaculation, use of a condom, aspermia resulting from a vasectomy or disease, or drainage of the semen out of the vaginal area. Human semen contains high quantities of acid phosphatase for which the swab can be analyzed. The presence of acid phosphatase is usually 8–24 hours after the act, then begins to disappear and is completely gone by 48–72 hours. Because non-motile sperm can be identified 2–3 days later, this test can indicate the act was more recent. P30 is a semen-specific glycoprotein of prostatic origin. It is present in semen only, regardless of the presence of sperm. This P30 can be detected 13–47 hours later. It has been found that there are cases in which the acid phosphatase was negative and the P30 was positive, confirming the sexual act. In cases in which there are no visible sperm it still may be possible to get male DNA from epithelial cells in the ejaculate or premature lysis of sperm. In human cases, the female DNA can overwhelm the quantity of male DNA, causing problems with interpretation of the mixed profile results. In these cases, better results may be obtained by performing DNA tests using Y-chromosome short tandem repeats (Y-STRs). This test may be helpful in compromised sexual evidence of any sexual assault case.

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Chapter 9 Time of Death

Overview

Death is defined as a complete and permanent cessation of all vital functions of the body. The main direct causes of death are heart failure, respiratory failure (asphyxia), or cerebral dysfunction (coma). There are two types of death: ***Somatic death***, (death of individual animal) means permanent stoppage of the main three systems in the body (circulation, respiration and central nervous system). The causes of somatic death are usually injuries, poisoning, acute illness and occupational nature. These causes may be classified to direct or indirect causes concerning these effects on the main three systems in the body heart failure, which is caused as a result of diseases, injuries or poisoning of the heart and blood vessels. Asphyxia, many causes can induce asphyxia as toxicants, mechanical and pulmonary diseases. Coma, is the cessation of cerebral functions induced by some diseases as encephalitis, meningitis, brain abscess, head injuries, brain tumors, and toxicants (morphine and hypnotic). ***Molecular death***, (cellular or tissue death) means death of the cell occurs sometimes few minutes to few hours after somatic death. Different tissues and organs die at widely different rates, because of their sensitivity to hypoxia. Bone, skin, muscles and many of the structural connective tissue can survive hypoxia a long time. E.g., the skeletal muscles responses to an electrical stimulus some hours after death. Neurons of the cerebral cortex die after 3-7 minutes of complete oxygen deprivation. This lead to the new concept of whole brain death, which is important both legally and ethically in relation to organ transplantation. ***The molecular life***, is the time between somatic and molecular death, during which organs transplantation can be done, the muscles

response to the electric stimuli, some enzymatic changes still occur and sperms retain their motility.

Assurance of death occurrence and its causes are a very important job of the veterinary practitioner especially in crimes. The living body depends on the integrity of three principal interdependent systems, circulation, respiration and CNS. Failure of one of them will lead to failure of the others and death. In some cases the respiration stop alone while the circulation continue for a few minutes as in cases of anesthesia and hanging, during these minutes the victim resuscitated by artificial respiration. The circulation may also stop while the respiration continues, as in cases of heart injuries. Permanent and complete stoppage of respiration and circulation for five consecutive minutes suggests occurrence of death, which is detected clinically by cessation of circulation and respiration, brain death, primary flaccidity, contact flattening and ocular changes.

Signs of circulation cessation

Auscultation of the chest and heart over a period of five minutes. The cardiac sounds may be heard so feeble because of a thick chest wall or emphysematous lungs or they can't readily be heard. Electrocardiogram (ECG) may be used for assurance of death. If these detectors are not available we can do some simple tests: a) Absence of pulsation in the principal arteries. b) No redness or congestion on applying a hot material to the skin. c) No distribution of color in distant areas after injection of the green inert fluorescing dyes. d) No bleeding on cutting a small peripheral arteries during surgical operations. e) The webs between fingers are opaque on trans illumination due to loss of normal translucency of the tissues (human). f) If a ligature is made around a finger, the tip will not become swollen or blue (Human).

Signs of respiration cessation

Absence of breath sounds on auscultation and respiratory movements are signs of. Place a feather on the lips and notice its movement, hold a clean hand- mirror in front of the animal mouth and nostrils and observe its dimnessm and putting a small basin of water over the abdomen and chest of animal, observe the movement are subsidiary tests.

Signs of brain death

Loss of reflexes & complete muscular flaccidity (primary flaccidity), no response due to electrical shock, complete flaccidity and flat Electro-encephalogram (EEG).

General muscular flaccidity (primary flaccidity)

It is general relaxation or flaccidity of the body muscles (voluntary and involuntary) with absence of reflexes, ends after two hours in winter. These conditions had been termed primary flaccidity to distinguish it from the condition of secondary flaccidity, which occurs after the departure of rigor mortis.Dilatation of pupilsm, drooping of mandible and disappearance of the face wrinkles and peaceful expression due to loss of muscle contraction (human). Appearance of some muscular response due to electrical stimuli because the molecular life remains for a variable time after somatic death. Dilatation or contraction of the pupil will occur by atropine or physostigmine to the eye. It is flattening of the convex parts of the body in contact with the ground due to loss of muscle elasticity and compression of these parts (Contact flattening).

Ocular changes

Loss of corneal and pupillary reflexes, loss of luster of the eyes which assume a glaze appearance and the pupil is dilated and may react to atropine and physostigmine for few hours after death. The eyeball becomes flaccid and clouded of the cornea, on the account of the formation of a thin layer of mucous over it but the formation of a thin layer of mucous over the cornea may be delayed if the eyelids are closed just after death or in some cases of death like CO or cyanide poisoning. Dimness of the cornea may be formed during life as a result of some pathological causes. Softening of the anterior chamber of the eye due to stoppage of aqueous secretions. Ophthalmoscopic examination shows pale papilla, segmentation of retinal veins and empty retinal arteries

Time of Death

Determining the time of death is very important in animal cruelty cases. The time of death is determined by the postmortem interval. The time of death may be usedto determine the charges in the case. For example, some state felony statutes requirethat the defendant "knowingly and maliciously" committed animal cruelty. In ahoarding case, time of death was used to meet the felony statute. Using forensic entomology the time of death was determined on the numerous dead animals in the home. In addition, it was discovered that the defendant then obtained more animals after the death of the others. It was viewed as a "knowledgeable and malicious" act to bring more animals in to a home in which dozens were already dead, qualifying as a felony animal cruelty charge.

Determining the postmortem interval may be used to support or refute the defendants or witness statements. It also may be used to invalidate a suspect's

alibi. The author had one case in which a dog was shot and killed. The main suspect was arrested and put in jail the same day that gunshots were heard. The time of death was determined by the use of forensic entomology and it was proved that the dog died prior to the suspect's arrest. Another consideration is the possible time lapse from the fatal injury to the time of death. In these cases, time of death does not equal the time of the fatal injury. This time lapse may be a short period of time or an extended period if death was slow to come, and is called the survival period. An animal may have suffered injuries and subsequent maggot infestation of the wounds. By using forensic entomology, the time of injury may be determined.The environmental observations must be recorded to estimate the postmortem interval. When at the scene it is important to know if the air conditioning or furnaceis on and at what setting. Confirmation must be made that the power is on and that the heating and air conditioning are working. Often the windows or doors have been opened at the scene to allow dissipation of the foul odor, thereby changing the temperature inside. These windows and doors must be closed, the temperature allowed to stabilize, and then a temperature reading recorded. If heating or air conditioning unit has been turned off or the power has been disconnected, the inside temperature must be taken in addition to the outside temperatures. The times of any temperature readings must be noted.

Examination of the body for Post-Mortem Changes

After an animal dies, the body undergoes a variety of changes. These postmortem changes include livor mortis, rigor mortis, algor mortis, and decomposition. Certain things that do not change may assist with time of death determination, such as gastric emptying time. An assessment of the various changes in the body

after death enables us to arrive at an approximate idea as to the length of time that has elapsed since death. It is impossible to fix the exact moment of death.

Pallor of the skin

It is mainly due to drainage of blood from the capillaries and venules of the skin after stoppage of circulation. It is hardly reliable sign otherwise because this might be noticeable only when the body is under observation at the time of death. Certain coloration is present during life such as jaundice. It may help to know the cause of death, pinkish color of the skin or certain parts of the body (death due to Co inhalation).Reddish irregular areas over the exposed parts of the body (death of cold exposure). Pink patchy coloration may be seen upon the body surface (death of HCN and cyanide poisoning.

Algor Mortis (Post-mortem cooling)

It means cooling of the body in cold weather. It takes place due to heat loss without heat production (stoppage of metabolism) until the temperature of the body and that of the environment becomes the same. The most reliable method of determining the body temperature after death is to record the visceral temperature by making a small midline incision into the peritoneal cavity and placing the bulb of the thermometer in contact with the inferior surface of the liver and the reading must be taken in situ also the rectal temperature and the ear temperature are very important. In estimation of the postmortem interval, the body temperature reaches the medium temperature in 8-12 hours for large animals and 5-7 hours for small animals. Human loss 1- 1.5 degree/hour in ordinary cases. The rate of heat loss is not constant in relation to the beginning and the end of conduction.

Factors influence the rate of cooling

1- The medium temperature (atmospheric temperature): cold medium opened places and absence of bedding affects the rate of cooling. Bodies immersed in warm stagnant waters; water containing sewage effluent or water containing putrefying materials cool more slowly than bodies immersed in cold running water. The surface of the trunk cools less readily than the extremities.

2- Cause of death: in deaths due to asphyxia or strychnine poisoning, the body may retain heat for long period. Bacterial infection (septicemia) the body temperature may even rise after deaths for a short time while in deaths due to hemorrhage cool rapid.

3- Age of the animal: old and small animals cool rapid.

4-Sex: females cool later due to its fat.

5-The body mass, obesity, which acts as heat insulator and the initial temperature of the animal.

Livor mortis (post-mortem lividity or staining- Hypostasis)

It is bloody (light violet) coloration of the most dependent parts skin, viscera, and organs of the body after dressing. It takes place due to gravitation of fluid blood inside the paralyzed vessels towards these parts after stoppage of circulation. It appears as mottled patches, which gradually extend and coalesce but in certain cases, isolated patches of lividity and such patches can remain separate from the large areas of lividity resembles bruises and contusion. You must differentiate a zone of inflammatory reaction in a viscous from an area of hypostatic engorgement by naked eye due to changes such as the presence of an

exudate or any sign of inflammation. In case of difficulty, portions of tissues should be examined histologically. It must be differentiated from bruises according to the following principles:

Table 1. Differences between bruise and PM lividity

Bruise	**PM lividity**
During life	After death
On any place	In the dependent portions only
Accompanied by swelling	No
Show multiple colors	No
If cut over, diffuse infiltration of blood in the tissues & it is firmly clotted and can't be washed	Drop of blood appear oozing from blood vessels and easily washed away by water stream
Accompanied by abrasions or signs of sepsis	No
Edges are well marked	**No**

Medico-legal importance

1- It is a sure sign of death.

2- It commences 2-3 hours after death, the staining is usually fixed as the result of blood coagulation in 6-8 hours and it is fully pronounced on body 8-12 hours (due to liberation of thrombokinase from the dying endothelial cells lining the blood vessels) so it help to estimate time of death.

3- Detection of death cause, which is mainly, depends on, the extent times color and site of hypostasis:

a) The extent and time of hypostasis, appearance of hypostasis is mainly, depend on the volume of blood in circulation at the time of death and the length of time that the blood remains fluid after death. Decreased total blood volume as in deaths from acute massive hemorrhage, the lividity is usually limited in its extent. Increased total blood volume as in congestive heart failure or asphyxia, the extent of lividity is usually marked and the time of its appearance is accelerated. When the concentration of active fibrinolysin is high, the rate of intravascular coagulation is slow, the blood remain fluid and gravitates rapidly into the dependent capillaries and veins over extensive area.

b) Hypostasis color, in relation to Hb state before death the color of hypostasis is different, in natural deaths it is deep purplish blue (light violet) in color and the intensity of color depends upon the amount of reduced HB before death. In cases of cyanide or co poisoning the areas of lividity show a cherry red color due to the presence of carboxy haemoglobin in the blood. In case of potassium chlorate, hypnotics, nitrates, nitrites or sulfonamides poisoning, a chocolate-brown appearance is observed where met-Hb is formed in the blood during life. Areas of bright pink mottling of the skin are seen in deaths from exposure to cold. It is very dark blue in asphyxia.

c) Hypostasis site, it appears in the lower limbs in case of hanging, in the back or on any side in natural death and in the head in cases of drowning.

4- Detection the body position, changes of the body position after death will lead to changes in the site of lividity so long as the blood is fluid because

blood in capillaries coagulates after prolonged period of death. When the body position is changed after the blood in the dependent vessels has undergone complete coagulation (8 Hours after death), the distribution of lividity can't be altered. When the position of the body is changed before the blood has coagulated; fresh areas of lividity persist in the old site of distribution. Therefore, if a body is found in a certain posture with PM lividity distributed over a region of the body, which is not the most dependent part in that posture it can be assumed that the body was moved after death. Hypostasis is absent in areas subjected to pressure (areas of contact flattening). N.B. It is of great importance to differentiate between ante-and post- mortem clot. The clotted blood in the heart and great blood vessels, induces by hypostasis, shows a comparable condition. The lower part is composed of dark clot and the upper of stream, while between a light red as buff colored layer.

Rigor mortis (PM rigidity)

It is a state of progressive muscular rigidity affecting all muscles (voluntary - involuntary) of the body, replaces the primary flaccidity conditions and disappears gradually due to the autolysis of the muscle proteins (putrefaction). It begins first in small muscles of the eyelids followed by the jaw muscles, which are affected in about 3-4 hrs after death. Next it appears in the muscles of the neck, face, thorax, anterior extremities, trunk, muscles of the posterior extremities and tail. It appears 2-3 hours after death in the face and completes established in 10-12 hrs or shorter in the whole body. Commenced to pass off (disappear) again in about 36 hrs in winter and 24 hrs in summer. The normal soft consistency of the muscles is mainly due to degree of hydration, which caused by ATP adsorbed on

the muscle myosin. After death, gradual chemical breakdown of ATP, leading to loss of water from the muscles (dehydration results in rigidity) or it may take place. Normally during the life, the ATP is converted into ADP and energy necessary to muscles contraction. After death, due to the absence of ATP and oxygen, the muscles use the anaerobic cycle for yielding energy which leads to accumulation of the lactic acid which leads to the actin and myosin fuse irreversibly into un dissolved actomyosin.

Factors influence the onset and duration of PM

1- Environmental temperature: high temperature accelerates onset of RM and shortened its duration as the presence of coverings or bedding.

2- The degree of muscular activity before death: the stronger muscularly animal at time of death, the later is the time of onset and the longer is the duration, while the exhausted the muscular conditions, the more rapid is the time of onset and the shorter is the duration. Fatigue reduces the muscle glycogen content and may ultimately bring about its complete disappearance with an accumulation of acid substances in the muscle plasma.

3- Causes of death: long standing diseases causes its early appearance while sudden death may cause its retardation.

4- Age: in fetuses and in old age the prices are rapid in both onset and passing off and it occur in a fetus in uterus and even during the life of the mother.

Medico-legal importance

1- It is a sure sign of death

2- Estimation of approximate time of death.

3- Suggestion or may point to the cause of death, as it is rapid in onset in cases of convulsions, as tetanus and strychnine poisoning.

4- It gives an idea about the position of the body during or a short time after death as the muscles is stiffened in this position. Preservation of the position of the body as agonists and antagonists undergo rigidity in the same degree without shortening of the muscle fibers. N.B. false signs due to rigor mortis like, the state of pupils, which has no indication to their ante-mortem appearances they may appear, constricted and the state of the heart, which may look like hypertrophy (contraction of the heart muscles).

Conditions simulate rigor mortis are cadaver spasm, heat stiffness and cold stiffness. **Heat stiffness (stiffening),** is due to burn, which causes coagulation of the muscles proteins leading to rigidity with shortening in the affected parts. **Cold stiffness (stiffening),** stiffness occurs in bodies exposed to very low temperature. It take place due to consolidation of the body fluids especially the synovial fluids thus the joints become fixed crepitate on passive movements. If such a body is removed to a hot atmosphere, this stiffness disappear, the body relaxes then rigor mortis sets in usually in less than one hour

Cadaver spasm (instantaneous rigor-mortis)

A sudden contraction of certain groups of voluntary muscles, which were already in a state of strong contraction due to extreme nervous tension without any preliminary flaccidity. It occurs in certain conditions only in conditions of

extreme nervous tension like, suicidal cases where the suicide is usually found firmly catching the weapon in his hand, homicidal cases where the victim is found firmly catching something belonging to the assailant or in drowning where the victim is found firmly grasping seaweed or gravel. CS occurs in certain group of voluntary muscles while other muscles in the same dead body may be in a state of primary flaccidity. occurs just before or at the moment of death or just immediately after death. This condition is very rare in animals.

Table 2. Differences between rigor mortis and cadaver spasm

Rigor mortis	Cadaver spasm
Occur in all conditions.	Occurs in certain conditions.
In the whole body.	In some parts of the body.
Develops gradually after death.	Suddenly with death.
Affect all parts of the body.	Certain groups of muscles.

Decomposition (Putrefaction)

It is the last or final change takes place in the dead body. It is called autolysis, tissue digestion or body resolution. It means autolysis of the body by the action of microorganisms resulting in breakdown of the complex proteins into simpler constituents gas, fluids and salts or body resolution from organic to inorganic state associated with foul smelling gases and certain color change. It begins 24-36 hours after death in winter and 24 hours after death in summer. The normal peristaltic movements, which drive away the decomposed materials from the body during life are stopped. The putrefactive bacteria in the decomposed food

attack the stagnant blood in the vessels, so they spread widely to continue their process in the following order, larynx, trachea, stomach, intestine, spleen, liver, brain, heart, lungs, kidneys, pancreas, pharynx, blood vessels, bladder, uterus, skin, hair and teeth. When the body in air, organs putrefy as follows, abdomen, chest, face and neck, hind limbs, shoulders, and anus and forelimb. When the body in water, organs putrefy as follows, head and neck, shoulder and the front part, fore limbs chest, abdomen and hind limbs. E.coli, staphylococcus, non-hemolytic streptococcus viridance, clostridium welchi, diphtheroid and proteus types are the most frequently isolated strains of bacteria are known to be normal inhabitants of either the respiratory or the intestinal tracts. Other isolated bacteria are usually associated with disease process within the body or due to wound infection. (mainly anaerobic gas forms mostly clostridium welchi).

Signs:

The putrefactive changes occur in larynx, trachea, stomach, intestine, spleen, liver, lungs, brain, kidneys, heart, bladder and uterus respectively. greenish coloration (sulpho-met-Hb) on the surface of the anterior abdominal wall opposite the caecum which extended over the abdomen and other parts of the body. The body distends with putrefactive gases (sulphurated hydrogen, carburated hydrogen, ammonia, carbon dioxide, and phosphorylated hydrogen) especially abdomen, chest, scrotum, face and limbs). Small blisters filled with foul-smelling fluid under the cuticle and these gradually enlarged, coalesce and rupture causing large areas to be denuded of cuticle. The subcutaneous tissues become emphysematous, eyes bulge, tongue forced out between the swollen lips.The hair and hoof or claws are loosen and are easily detached. Froth bubble gases come out from the mouth and nostrils. The presence of gases may cause

food to be forced from the stomach into the larynx, fetus may be discharged (postmortem delivery of a fetus) and the anus or the uterus may be prolapsed. Putrefactive venous (*marbling phenomenon*) tree due to the presence of frothy blood in the venous tree (DD from new and old wounds bleeding). In drowned bodies, the head tends to sink to a lower level than the rest of the body, blood and body fluids gravitate there so putrefaction is firstly appeared in head, neck, fore limbs, chest, abdomen and hind limbs respectively. Putrefaction is hindered by cutting the body into pieces and so limbs cut before putrefaction are putrefied later on (This is why the butchers always do this with carcasses of animals after slaughtering).Liver becomes spongy; later it is transferred into semi-pultacous material inside the capsule. Due to gases and raises tension inside the abdomen, bile passes from the gall bladder to stain the nearby organs and abdominal wall. Kidneys may undergo putrefaction earlier than the liver. Heart is filled with frothy putrefied blood, its wall become friable and gas can be felt and seen along the coronaries. The endocardium, aorta, and coronaries resist putrefaction. Brain is transformed into a semi fluid materials, varies in color from green to somewhat purple.

Factors affect the rate of putrefaction:

1- Temperature between 25 - 40 ºC favored putrefaction while below 15 ºC and above 40 ºC retard putrefaction.

2- Increase humidity enhance putrefaction so hair and eye putrefy soon and so do dropsically asphyxiated animals when carcass brought out of water into air.

3- The presence of air may favor putrefaction as it carries putrefying Organisms.

4- Cause of death may affect putrefactions, e.g. previous injuries as open wound or bruises enhance putrefaction; sudden death from acute infection enhances putrefaction, animal dying from hemorrhages gastrointestinal irritation or burning retard putrefaction. A newly born or stillborn fetus putrefies slowly.

5- Certain chemicals: certain cases of poisoning especially chronic toxicity of arsenic may retard putrefaction. Strychnine, phosphorus, antimony and mercury retard putrefaction. N.B. In drowned bodies, the head tends to sink to a lower level than the rest of the body, blood and body fluids gravitate there. Putrefaction is firstly appeared in head, followed by the neck, fore limbs, chest, abdomen and hind limbs respectively.

Other Death Phenomena

Adipocere (saponification)

It takes place when the carcass is kept in moist conditions either in water or in damp earth. It begins in about three weeks and is not completed before six months. It is mainly due to the gradual hydrogenation of pre-existing fats (non-saturated fatty acids) such as oleic into higher fatty acids (saturated fatty acids) which keep the natural form of the body, so it may be identified after years. It has a rancid or sweetish smell, greasy and friable feelings. It floats in water and dissolves in ether and alcohol. Eventually the whole of the fat is converted into palmitic, stearic and hydroxy stearic and a mixture of these substances

constituents adipocere. Adipocere is formed first in the subcutaneous tissues later in the adipocere tissues elsewhere in the body. This changes were previously referred to as saponification owing to a belief that the fats were broken down to glycerin and fatty acids and that the later was gradually converted into insoluble calcium and magnesium soaps but adipocere is formed whether there are salts in the water or not. It does not occur naturally in the viscera or in the fatty tissues.

The medico legal importance:

1- The easily identification of body features for many years.

2- Some causes are identified e.g. firearm wounds.

3- Determination the time elapsed after death, it starts within few weeks, as little as 3 weeks and takes few months to develop all over the body about 6 months.

Mummification

This occurs in bodies exposed to air in deserts or in sandy areas especially in tropics. Dryness is absolute on dry weather, putrefaction stops, process of dehydration of the body by the effect of air currents and heat occurs through evaporation of the body fluids, the skin becomes parchment like, the tissues dry on the bones and at the end outward appearance of the body is remarkably preserved. After complete mummification no further changes may occur. The post-mortem changes is usually appeared 3 weeks after death and completed all over the body within 3-6 months.

The Post-Mortem Intervals:

Determination of the post-mortem interval is important in criminal but it is difficult to determine the moment of death. The previously mentioned post-mortem changes are the most important factors in estimating the time of death. Also there are some chemical changes as blood, muscles and CSF pH and potassium and ascorbic acid levels in aqueous and vitreous humor are helpful factors in estimating time of death.

Table 3. Postmortem intervals

Item	Time
PM cooling	The body loses its temperature in 8-12 hours for large animals and 5-7 hours for small animals
PM lividity	Start 2-3 hours after death and fully pronounced 8-12 hours.
PM rigidity	Appear 2 hours after death and complete 10-12 hours and start to pass off again in 36 hours in winter and 24 hours in summer.
Putrefaction	Starting after 24 hours in summer and 48-96 hours in winter.

Flow Cytometry

Flow cytometry has been investigated as a possible instrument to determine time of death in the early postmortem period in humans. The test involves looking at the degradation of nuclear DNA using flow cytometry and comparing it to the

degradation in known controls. The percentage of degradation is then correlated to the postmortem interval in hours. The spleen, peripheral blood, and liver have been looked at as possible sources for testing. Research has shown that hepatocyte degradation has a linear correlation with the time elapsed since death. The presence of hepatic neoplasia does not alter the findings. In addition, hepatic tissue is ideal because of the ease of obtaining samples through a biopsy needle

Vitreous Humor

Overview

The potassium concentration in the vitreous humor has been used in human forensics to aide in the determination of the postmortem interval. After death, autolysis starts when cell metabolism stops and subsequently the integrity of all tissues throughout the body are lost. Selective cell membrane permeability and the active cell membrane transport ceases. In turn, this causes ions to diffuse across the membranes, depending on the gradients. The vitreous humor is more isolated than other structures in the body and more resistant to bacterial degradation resulting from decomposition. It is relatively more stable postmortem compared with blood or cerebrospinal fluid. The potassium gradient reverses postmortem and diffuses from the lens and retinal blood vessels into the vitreous humor. There may be different levels in the anterior, central, and posterior layers of the vitreous until equilibrium has been reached. As much vitreous as possible should be removed to eliminate the problem of concentration variation in the layers (Henssge et al. 1995). In large animals, the aqueous is sampled for electrolyte testing. The laboratory should be contacted regarding sample collection and submission.

Sampling and Testing

Sampling technique of the vitreous is important. The sample should be aspirated carefully with a syringe and small needle, applying gentle pressure to minimize contamination. Only a clear, colorless sample should be used. The sample is centrifuged and then the supernatant is tested. The sample may be frozen and held prior to testing. A sample from each eye should be taken. It is normal to have up to a 10 percent difference between the right and left eye in humans. Although there are differences between the individual eye measurements, the mean value does not change and the regression lines used for analysis are the same.

The relationship of potassium concentration and postmortem interval (PMI) is linear up to 120 hours. The equation used by Henssge is:

PMI = 5.26 X K+ Concentration - 30.9

The 95 percent confidence level of the formula is 20-100 hours postmortem. The estimate for PMI may be undervalued by 0.3 hour, with a standard deviation of 19 hours (Henssge et al. 1995). A study by James et al. on postmortem vitreous potassium levels was conducted on 100 human bodies that came to the forensic center in which the PMI was known. The study showed similar results to the formula from Henssge.

The vitreous potassium is of more value after the first 24 hours after death because other measurements are accurate in that postmortem interval. The vitreous potassium increases as postmortem time increases, but there is great variability, which increases the longer the postmortem interval. Potassium levels

are controlled by the rate of decomposition, so anything that affects this rate also affects the rise in potassium levels.

Gastric Emptying Time

Gastric emptying time and the gastric contents are helpful in human cases to helpnarrow down the postmortem interval. In an animal, when it is known what and when it last ate, it may be possible to use that information. Gastric emptying time is affected by many factors, including solid or liquid food, the fat and caloric content of the food, water intake, volume of food ingested, and whether the animal was fed meals or free-choice. It can be affected by the age and size of the animal, although in cats increasing age does not slow down the gastric emptying time as it does in humans.

Forensic Entomology

Overview

Forensic entomology involves the analysis of insects for legal cases, primarily to help determine the postmortem interval. With appropriate collection and documentation, forensic entomology can provide the most accurate time of death. The foundation for the use of forensic entomology is that the time of colonization, rate of growth, and stages of insect succession can be determined by analyzing climate data. Because maggots, with rare exception, only colonize a body after death, this time determination represents the time of death, or the minimum postmortem interval. The veterinarian, as part of the death investigation team, must become knowledgeable in forensic entomology to properly recognize

evidence, and collect, preserve and ship the entomological samples. The deceased body of an animal serves as a food source for insects. The decomposition of an animal attracts insects of certain species at different times, depending on season and weather conditions. The changing composition of the body biologically, physically, and chemically alters which insects are attracted over time; this continues until no more food source remains or the environment conditions change to prevent further feeding. Insects have additional forensic value because they may be analyzed for drugs that were present in the body at the time of death. Certain drugs can affect the rate of larval development, so the presence of any known drugs should be documented for the entomologist's consideration. It is possible to assay the gut contents of maggots for DNA from the body on which they were feeding. This has been used in cases in which a body was moved and maggots that were not even observed on the body were linked by DNA testing. Maggots can help determine the presence of wounds and whether or not the body has been moved or disturbed postmortem. The analysis of insects should be conducted by a forensic entomologist. All samples collected should be shipped immediately to the entomologist. It is recommended to always contact the entomologist prior to shipment to discuss the case and verify that someone will receive the samples and handle them accordingly.Myiasis refers to the colonization by maggots of a live body. This is usually found on injured or debilitated animals in which blood or excrement is present, which attracts flies. Forensic analysis of the maggots can help determine the time of injury. Myiasis may create confusion forensically on a deceased body when

determining the postmortem interval. It always should be noted when examining the body if there are conditions that may have caused myiasis prior to death, such

as injuries that may not have resulted in immediate death; then the time frame must be taken into consideration. In severe neglect cases in which the animal may have had excrement on the body for a period of time prior to death, it is possible for myiasis to have been present. Because blow flies are attracted to decomposition, the wounds of an animal may not be colonized until infection and dead organic matter are present. Numerous insects are of forensic importance, including blow flies, flesh flies, muscoid flies, skipper flies, dung flies, black scavenger flies, small dung flies, minute scavenger flies, soldier flies, humpbacked flies (scuttle flies), month flies, sand flies,owl midges, carrion beetles, skin beetles, leather beetles, hid beetles, carpet beetles, larder beetles, rove beetles, clown beetles, checkered beetles, hide beetles (family Trogidae), scarab beetles, and sap beetles. In addition, venomous arthropods are forensically important in that they can be the underlying cause of death. Scavenging insects are important in that they can feed off the animal and cause postmortem damage

to the tissue, which may be misinterpreted as other types of injury. These insects include paper wasps, yellowjacket wasps, ants, cockroaches, pillbugs, and sowbugs. Pillbugs and sowbugs are usually found in the protected area underneath the remains next to the soil. Fire ants can cause tissue damage that resembles burns antemortem. Acrobat ants feed on fly eggs and maggots affecting the initial colonization, sometimes causing a time delay for 2–3 days.

Climate and Weather Documentation

After the initial observations, climatological data should be recorded. While these data are being collected, insect sampling and collection can begin. Climatological data are critical to estimate the postmortem interval using forensic entomology

and for analysis of other postmortem findings. The time of colonization and life cycle development are largely dependent on temperatures and may be influenced by other factors such as rainfall, sun exposure, and snow. All temperature readings should be done with shading the sensing element of the thermometer to protect it from the influence of direct sun rays. Ideally, several temperature readings should be taken at the scene in close proximity to the body: ambient air temperature at 1- and 4-foot heights; ground surface temperature on top of surface ground cover; body surface temperature on the upper surface of the body; under-body interface temperature, sliding the thermometer between the ground surface and the body; maggot mass temperatures at the center of the mass; soil temperatures beneath the body immediately after the body is removed; and additional soil temperatures 3–6 feet from the body starting at the ground cover, 4 inches deep, and 8 inches deep. If the body was buried, soil temperatures should be recorded at the depth of burial.

An estimate of the body's exposure to direct sunlight, broken sunlight, or shade during the daylight hours should be made by looking at the surrounding vegetation and structures. Blowflies do not like to lay their eggs in direct sunlight. In addition to data collected at the scene, data must be obtained from the nearest weather station to the scene and for the previous 1–2 weeks or longer in severely decomposed remains. This information is used to evaluate temperatures prior to the discovery of the body. This information is usually available from nearest the National Weather Service station, through a local university, or from a qualified climatologist or meteorologist. In some cases, only the highs and lows and any precipitation accumulation for the time period are available. In some cases hourly readings are needed. Often, this information is only available through a climatologist or meteorologist. The forensic entomologist compares this

information to readings taken at the scene. All effort should be made to collect entomological evidence at the scene. If this cannot happen, then temperature data must be documented every time the animal's body changes environment and/or location. The time and temperatures at the scene are still taken as described. The temperature of the transport vehicle's holding area for the body must be taken and the time recorded for transport in that environment. If the body is placed in a cooler, the time and temperature readings must be documented. This documentation protocol is repeated until the entomology samples are collected. Failure to keep this documentation can cause problems with the subsequent forensic analysis. Collection of Entomological Evidence at the Scene. All preparation for entomological collection should be done distant from the remains. When collecting insect samples there should be minimal disturbance to the body and any unavoidable disturbance should be documented and photographed. Every effort should be made to get a sample of live flies at the scene where maggots or maggot eggs are present for species identification. Collection of any flying or fast-crawling adult insects is best done with an aerial insect net. One technique is to perform several sweeps over the body, reversing the opening 180 degrees after each pass, ending with a reversal to seal the flies inside the net. This should be repeated three to four times to ensure that a representative sample of flies has been collected. The surrounding vegetation should be swept because the flies may be resting in plants or grasses 10 to 20 feet away. Another technique is to hold the bottom of the net vertically with the mouth of the net over the body using a downward swatting motion. The flies will naturally fly up and into the net. After the capture of the flies, the end of the net with the insects may be placed in a kill jar for 2 to 5 minutes. Kill jars, made of ethyl acetate and gypsum cement, are commercially available and inexpensive. After the insects are immobilized, they

can be transferred to vials of 75 percent ethyl alcohol using a small funnel. An alternative is carefully transferring the netted insects into 75 percent ethyl alcohol by holding the end of the net upward and reaching in and up with the jar of alcohol toward the flies, which have a natural tendency to walk upward, tapping them from the net into the vial. Any ground-crawling adult insects may be collected with forceps or fingers. They should be preserved in the same way as flying insects. Blow fly egg masses should first be photographed and their location documented. Using forceps, break a small piece of egg mass off approximately the size of a dime, taking care to collect from the center as the eggs at the edge may be desiccated and no longer viable. Each egg mass collected from each location on the body should be kept separate. The mass collected should be broken in half and one-half placed in 75 percent ethyl alcohol. The other half should be placed in a larval-rearing pouch. These pouches are made taking a piece of aluminum foil and folding it to create a three-dimensional rectangular pouch, crimping the corners together. A small piece of beef or pork liver should be placed inside as a feeding substrate should the larvae hatch. The top should be crimped together sealing the sample. This pouch then should be placed inside a plastic container for shipment with approximately 1 inch of soil or vermiculite in the bottom and small air holes punched into the plastic top. This substrate absorbs any fluids that leak from the pouch and, for late-stage larval samples, provides a burrowing substrate. Two labels should be created for the larval feeding pouch with the date and time, case number, location of the sample collected, and sample number. These should be filled out in pencil to avoid any destruction of the writing. There should always be

a double labeling system used in which one label is placed inside the plastic container and the other affixed to the outside of the container. For all samples, note the time they were placed in the container and when they were shipped.

When collecting maggots for analysis, it is important to look for the oldest

(largest) larvae because they are the ones that first hatched and in turn were the first eggs laid. At first, the body and surrounding area should be examined for pre-pupal maggots (post-feeding). These will be found most likely off the body but may be found in the fur, carpet, the first 3–5 cm of soil, or up to 50 meters from the body. If none are found, then samples of the largest instar larvae should be collected, noting their location on the body. Temperature recordings and time of collection should be documented as described. A sample of the collected maggots should be preserved at the scene. Place a sample of the largest maggots and some of the next size down into hot or boiling water for 5 minutes to kill and blanch them, documenting the time of blanching. They should then be transferred to a vial of 70–85 percent isopropyl alcohol. They may be placed in 70–85 percent isopropyl alcohol at the scene if hot water is not available for blanching. The vial should be double-labeled as described with egg masses, with one label in the liquid and another affixed to the outside. Another live sample of the maggots should be preserved for examination using the larval-rearing pouches. Do not put too many maggots in the pouch because they need air and too many could cause the majority or all of them to die. Fold over the foil, leaving air above and seal the edges well. The migratory larvae and puparia may be found usually within 20–30 feet of the body, depending on the species. They may be found under surface debris, in the top few inches of soft soil, vegetation, under rocks, or on tree trunks. The soil and surface debris should be sifted to find migratory larvae or puparia. These samples are post-feeding and do not require a food substrate. They should

be placed in the plastic container with vermiculite or sand on the bottom and a damp paper towel to prevent desiccation. The presence of the empty pupa cases indicates that a complete blow fly life cycle has taken place on the body and indicates a minimum elapsed time since death. These casings are often mistaken for rat droppings. They may be found in the same areas as the pre-pupal maggots and pupae. It is important to look for the 256 Veterinary Forensics ecdysial caps to assist with species identification. These caps are tiny, delicate, and easily missed. They may be found separate from the pupa cases or still attached. The pupa cases and caps should be placed in a dry vial with tissue paper for cushion. A soil sample from underneath the body, adjacent, and up to 3 feet away, should be collected and placed in a separate container with a solid lid, filling it half full to allow for air if any insects hatch from the soil. Litter samples (leaves, bark, and grass) and any other debris on the ground surface close to the remains should be collected and placed in plastic containers for later examination for insect evidence. Newly emerged adult flies should be collected in dry vials and a description of their appearance noted, as it will change by the time it reaches the forensic entomologist. As time goes on, there is sequential colonization of the remains by other insects. The succession of arthropods can be used to determine the minimum and maximum postmortem interval (Wells and Lamotte 2001). Analyses of these later-appearing insects can help with the estimate of the postmortem interval. The successive colonization is dependent on season, weather, and other environmental conditions. The presence of any insect evidence may be forensically important because some insects parasitize others. These insects may be on, below, or flying above the body.

All insect evidence should be collected and preserved separate from the maggot

samples. Live adult insects should be placed in 70–85 percent isopropyl alcohol and larvae treated the same as blow fly larvae. A soil sample should be collected as with blow fly collection.

Collection from the Body

Collection of entomological samples from the body during examination follows the same basic rules of collection. The time, temperature, and location should be documented for each sample, keeping each location separate. The hand bags, body bags, or any cloth wrapping should be inspected for insect evidence, noting the adjacent body location. If the body was in a cooler and any maggot mass is present, the temperature of the center mass should be taken to see if there was any temperature decrease and if so, by how much. Any botanical evidence may contain hidden insect evidence and should be carefully inspected . The areas of insect activity on the body start preferentially around the head, followed by wounds and the urogenital-anal area. The presence of any unhatched eggs may be significant and should be documented. In dried or mummified remains, there may be the accumulation of insect feces (frass). This mass of material can appear like sawdust or pencil shavings. With submerged bodies, there is the possibility of aquatic insects colonizing the remains. This may occur in conjunction with terrestrial insects if part of the body was floating for a sufficient period of time. These aquatic insects can help determine the postmortem submersion interval. Usually only the immature stages of the aquatic insect are found. Care should be taken when removing the body from the water to use a sheet or fine-weave mesh under and around the body to prevent the loss of these insects. They can be very

small and fragile, with some having been mistaken for fiber trace evidence. The collected specimens should be preserved in 70–80 percent ethyl alcohol for examination. For live rearing, a forensic entomologist should be contacted for current recommendations. Entomological Evidence in Enclosed Structures Enclosed structures present several problems. The flies may have limited access into the structure and may be found outside the structure or very few inside. The internal temperature may be very different than the outside temperature. An adjustment can be made if temperature readings are taken from inside the structure over 3–5 days during the peak times and compared with data from the National Weather Service. Often, the first responder opens the windows or doors because of the odor, changing the original inside temperature. First, it must be determined if the air conditioning or heat was on; that temperature setting may be used by the entomologist. To verify this, the windows and doors should be closed and the inside temperature allowed to stabilize. If it is a programmable thermostat, the previous week's program may be retrieved for temperature readings.

The migratory larvae and puparia may be found under carpeting, rugs, or other covering. Dark fly specks, which are fecal spots, may be found on the floors, walls, or ceilings from the flies. Food regurgitation spots that are lighter in color may be found in these same areas. The density of this spotting can indicate the relative size of the fly population attracted to the remains

Determining the PMI: Examination of the Crime Scene

Several types of evidence can be found at the crime scene or on the animal's body

that can help determine the time of death. All findings, including examination findings, must be analyzed together to reach the most accurate time of death. Blood Stains. It is possible to evaluate blood stain findings at the scene to assist in the estimation of time of death. The blood may be fresh or clotted, and the serum separated or dried. It may be in a large pool, or there may be blood spatter. The amount of blood loss should be calculated to help determine if death was caused by exsanguinations. Blood can continue to seep from the body after death because of gravity. By recording the blood loss, condition of the blood stains, and environmental factors, scientists can help determine the postmortem interval. Using these findings, they can perform experiments to determine the length of time for that quantity of blood to clot, the serum to separate, or the blood to dry on similar surfaces under similar environmental conditions.

Scene Markers

Other findings at the scene may assist in estimating the time of death. Investigators need to question neighbors to find out when the last person saw the animal alive and the condition of the animal. If the animal was abandoned in a home, the investigator needs to look for signs of when the owner was last there, such as newspapers and mail. The animal's environment and housing should be inspected for clues as to time. It is important to look for what is present as well as what is not present that one would normally expect to find. These may include cobwebs on the body or shelter, fresh urine or feces, lack of fresh urine or feces, botanical evidence, debris build-up around the entrance to a run or crate if the animal was confined, and the presence of mold on the body, feces, or food.

Forensic Botany

Forensic botanists can analyze plant evidence found at the scene and on the body to help determine the postmortem interval, whether the body was moved, and the original location of the body. The time frames for the postmortem interval are often months, years, or seasons. The plant specimens may include leaves, twigs, roots, pollen, fungi, and algae that can be analyzed. These plant specimens may be found on the body or in the gastric contents.

Final Analysis: The Report of Exam Findings

The reports generated for an animal cruelty case are very important. These reports are legal documents and are examined throughout the legal process of the case. They are submitted to the investigating and prosecuting agency. As the case progresses, the report is examined by the defense counsel, the defendant, their expert witness, and the judge. It is from these reports that decisions are made by both sides, including whether or not to prosecute and how to charge the defendant. The report may be used to determine plea bargains or sentencing. It is used to map out defense and prosecution strategies. The contents of the report should be laid out in a logical, factual manner. Often, a report is needed prior to the receipt of all test results and final determination of conclusions. In these cases, a verbal report may be given to the investigating officer or a preliminary report issued. Any preliminary reports should be written with great caution and should not contain anything other than known facts, confirmed findings, and pending tests. If any changes are made in the final report, a valid explanation must be given in the final report. Both the preliminary and final reports should be kept for comparison later.

They are both considered evidence and part of discovery to be turned over to the defense attorney. In addition, all notes taken at the scene or during the examination, laboratory results, photographs, radiographs, and any treatment or medical records must be preserved as evidence for the case. It is a medical report, which is written by a jurist after examination of crimes concerned with animals.

The report

It consists of three parts

1- Introduction

The part contains Job, name and address of the practitioner who writes the report, authority ordering the examination, number types, date and time of the sign and summary of the case must be mentioned.

2- Subject

The date, time and place of examination and the presence of the official witness must be recorded. In case of alive animal, a complete description of the internal and external examination of the case. Clinical sign samples for analysis, described treatment, time needed for treatment and percentage of losses expected should be recorded. In case of dead animals, identification, carcass position, external examination, postmortem changes, samples and internal examination should be recorded.

3- Conclusion

It contain in alive animal, the most characteristic signs, treatment, expected time for treatment and percentage of losses in alive animals. In dead animals, the postmortem findings, the cause of death and time elapsed after death followed by the signature and qualifications of examiner.

Precautions

Cancellation of any fault is forbidden. Put a mark on the faulty word and write the corrected one on the border side with your signature. Don't use different scientific terms in report language.

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Chapter 10 Animal doping

Introduction

Doping is comes from Dutch word (doop), meaning a thick liquid or sauces a reminder that it originally referred to a South Africa drink. In days gone by, (dope) was something you drank to help you work hard, if only for a short space of time. The use of substances and any other available methods of artificially enhancing performance in a sporting event, or when preparing for it. It is the administration to animal of any substances, other than a normal nutrient for the purpose of affecting its speed, stamina, courage or conduct in race. In other words, animal doping means the use of drugs or similar substances or preparations to artificially lift lower or maintain an animal's performance.

During prehistory, the plants being the only natural preparations available were overrunning the sports. From the end of the XIX century, the constant wish to enhance human possibilities is perpetuated, whatever the price to pay, so the more we wear on the more the formulas are sophisticated. It is the era of magie beverages and speed up potions, directly imported front racecourse including higgledy-piggledy. Atropine, caffeine, strychnine, cocaine and arsenic…. Second World War will deliver to the sport planet, via the sky (sportsmen), the amphetamines, synthetic substance acting on the central nervous system. The big era of the alert amines will last in the beginning of the 70. Their decline will take place with the first doping tests. During the last thirty years, the story of doping substances will go forward the rhythm of the performances of toxicologists, being

in the cheater side or in the antidoping side. Every fight will bear the naine of one or two molecules: corticosteroids, anabolic steroids (1970), testosterone, growth hormone (1980), and erythropoietin (1990)

Uses:

1- Giving a stimulant to make the horse run faster, common at youth livestock competitions and for animal fattening.

2- Sedative to slow the animal down in case of nervous animals.

3- A local anesthetics to mask lameness or tranquilizer to quite obstreperous horses.

4- Male sex hormones to mars in estrous.

5- Analgesic drugs to treat arthritic conditions.

6- Some of show participants (30% of all) use illegal doping to improve their entries. They inject drugs or inject oil under the animal's skin to improve their appearance.

7- Artificially improving animals productivity with drugs like steroids or similar substances or preparations is likewise prohibited by the Act, unless it can be proved that these products do not harm animal's health or welfare. Special substances can't be used to increase animals' productivity, unless it has been proven in scientific studies or by extensive established experience that these substances are harmless to animals. An animal may neither be fattened nor may force-feeding increase its productivity.

Agents used:

1- Stimulants

Caffeine, which is cheap, easy to obtain, and quite effective. Morphine and its derivatives which enables a horse to run well above its normal form. Amphetamine and methyl amphetamine and similar drugs which delay the onset of fatigue. Stimulants must be given within 3 hours of the beginning of the race. For this reason it is usually recommended that during this period the horse should be in some controlled area of the racecourse where unauthorized persons can't approach it.

2- Depressants

It includes chloral, chlorbutol and the barbiturates. It must however be realized that many compounds will not fit exactly into these classifications, as their action depends on the dose given. A small dose of chlorpromazine will quite an excitable horse and enable it to run a better race, while a large dose will act as a depressant and slow it down. Similarly, while local anesthetics are generally used topically to mask lameness, they are said (possibly wrongly) also to act as CNS stimulators.

3- Equine growth hormone (EGH)

The drug increases the muscles mass giving an unfair edge to both athletes and horse, makes a horse look better and may make the horse run faster. High doses of growth hormone (EGH abuse) can damage growing bones, and could eventually cripple a young horse (against the welfare of the horse). EGH is worrying both horse racing & athletics authorities, as its detection is essentially impossible as it disappears from the body within hours.

4- Anabolic steroids

Including testosterone to make the animals grow faster than nature. This practice can be implemented in three ways, by adding beta-agonist to animal feed or implants under the animals' skin or by Intravenous injection.

5- Diuretics

There are more than 17 diuretic like amiloride, triamterene, spironolactone, canrenone, hydrochlorthiazide (Esidrex), torasemide, clopamide, indapamide, Furosemide (Lasix), Chlortaidone (Hygroton), Atenolol (Tenormin) and acetazolamide. Horse racing authorities prohibits them because they can mask the administration of doping agents by diluting their concentration. These practices are illegal in the European union but are much used in the USA. B-adrenergic agonist Clenbuterol, which used therapeutically in the treatment of airway diseases via aerosol based medicate delivery systems is prohibited by horse racing authorities.

Doping analysis

Method of drug detection must be found before approval of the drug. Samples are taken from, saliva, urine, sweat, and blood and recently from hair. Instruments used are HPLC/ UV, High resolution masses spectrometry, Gas chromatography or ELISA. The Food and Drug Administration and state officials are growing more concerned about the use of steroids and other growth drugs in animals exhibited and sold at youth livestock shows. Many steroids have been used as many as a dozen prize-winning animals across the country. Officials are not only worried about cheating at the shows. The doping tests are to eliminate the cheats from international sport. Exceeding the recommended serving may cause serious

health effects. Meat from the treated animals will harm humans. The use of clenbuterol, one of the most common animals doping drugs has been blamed for deaths in Europe, and there is a current Federal investigation of contaminated veal in the U.S. If an athlete eats an average quantity of pork, from an animal, which has a high concentration of nortesterone, the athlete can fail a doping test for nadrolone because he or she will have an accumulated level of metabolites. The same applies to beef which has been treated with anabolic steroids. The doping of racing animals is a thoroughly undesirable practice. Apart from its inherent dishonesty, it involves considerable danger to the jockey (the horse may falling or becoming unmanageable). It is liable to cause permanent impairment of the reproductive system of the female. It interferes with the selection of animals for breeding by conferring a false value upon inferior animals that have own under the influence of stimulants and not their intrinsic merits.

Gene therapy & Doping

Following the Genome Project buzz, the next question is what can we do with all that information? …..One answer is gene therapy. Scientists around the world are searching for ways to alter damaged or diseased genes and revolutionize how we treat illness. Many predict that in the near future, hemophiliacs for example will be able to have a blood-clotting factor inserted into their genes. Many people in the sports world are concerned that athletes will try to use certain therapies to secure a competitive edge. **Gene therapy** is a newly emerged field aim at finding a cure at the gene level for genetic diseases. Genetic diseases are defined as these, which result from defective gene (dysfunctional protein specific function). Examples of genetic predisposed diseases are hemophilia in man and animals,

muscular dystrophy, Alzeihmer's disease, Autosomal abnormalities (mongolism or Down syndrome), sickle shape anemia and X-fragile syndrome.

Gene therapy techniques

Gene addition type is the main. This involves attempt to provide a corrected DNA fragment of a gene to a defective one. It takes place by a variety of means, not only in-vitro but also in clinical trial inpatients. The ultimate goal is to want the therapeutic DNA to become a permanent part of the host's chromosomes. This would ensure its stability and would be replicated along with the host's chromosomes during each cell division. A number of vehicles have been developed to deliver corrective genes into cells. In this way (gene therapy), many predict that in the near future, hemophiliacs for example will be able to have a blood-clotting factor inserted into their genes.

Type I Epo (erythropoietin)

This hormone instructs the body to manufacture new red blood cells. Inserting a gene into a person's bloodstream to boost production of the hormone erythropoietin (epo). Patients who suffer from severe anemia, such as people with AIDS or kidney failure stand one-day benefit tremendously from this form of gene therapy because their bodies produce inadequate amounts of red blood cells. For athletes and racing horses, increased epo production would enhance oxygenation of tissues, in turn increasing aerobic stamina. This is different from synthetic epo, which can be temporarily injected, then flushed out of the system. Some endurance athletes to increase oxygen transport and aerobic power in an attempt to improve endurance capacity and recovery during competition use recombinant human erythropoietin (Rhu Epo). The close to perfect horology between

endogenous and recombinant Epos, its short plasma half life and the late clinical manifestation of its effect mean that no reliable analytical technique is yet available to detect its use by athletes.

Type II Muscle-boosting hormones

Inserting muscle-beefing genes into muscle cells (like epo gene therapy). Techniques to strengthen muscles are being developed to help people with illness, in this case people with degenerative muscle conditions such as muscular dystrophy. Whereas the epo therapy would be pervasive through the body, this approach would target specific muscles. Extending this treatment to athletes could one-day mean strengthening a tennis player's shoulder muscles, a sprinter's calves, a boxer's biceps. Studies with a protein muscle growth factor called (IGF-1) have demonstrated compelling results.

TYPE III Blood vessel generation

Inserting genes to help grow new blood vessels. This therapy is being developed to help elderly people with peripheral arterial disease, the death of tissues in the body's extremities due to inadequate oxygen supply. The gene would turn on (or turn up) production of new vessels. If athletes used treatments for blostering vessel production, the result could be a hypersupply of oxygen and other nutrients to the tissues with better supply lines, muscles, lung, the heart and other parts of the body would not tire as easily. The technology that would enable gene doping is currently under development, and the stream of reports about successful animal tests continues. The cover story of the issue of scientific American assents that by 2012 gene therapy will

probably be a well-established and widely used medical technique with animal testing in full swing coupled with the push by pharmaceutical companies to pioneer this huge new market.

Gene Doping & Health Risks

Boosting muscles mass and increased oxygenation of tissues already developing gene therapy to increase red blood cell production, which raises the amount of oxygen delivered to cells. The treatment is meant to help people with severe anemia, but also bloster the aerobic capacity of healthy people. Another biotech firm is working on a gene therapy for patients with degenerative muscle disease such as muscular dystrophy. Researchers have successfully beefed up the muscle with genes demonstrating that this technology could one day be used to boost muscles of athletes as well. Healthy people who choose to unnaturally boost their epo levels will increase their chances of stroke and heart attack because adding red blood cells makes the blood thicker, as it gets thicker, it become more difficult for the body to pump blood successfully to all tissues of the body, causing clots wherever vessels can't compensate for this increased thickness. Athletes using synthetic epo today face similar risks but after a few weeks the drug is flushed through the system and red blood cells production returns to normal levels. But with permanently altered genes, abnormally high red blood cells production would continue indefinitely and the blood could get thicker and thicker until the stress damaged the circulatory system. With muscular alterations, muscles would likely become disproportionately strong pulling on surrounding tendons and bones causing tears or fractures. Scientists emphasize that the unknowns of this treatment may in fact be the most dangerous factors of all. No one knows for sure

if these gene alterations would be detectable. Experts in the U.S. Anti-Doping agency said, there would be no way to test for this type of doping with current technologies (detection would be nearly impossible).

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Chapter 11 Animal Euthanasia

Introduction

When efforts to prolong the life of a beloved pet serve only to prolong death, euthanasia (a Greek for good death) should be considered. Veterinarians have the means, and training to put animals, which are beyond treatment and recovery to sleep humanely. It meaning production of quiet, painless death in an animal for humane reasons. It is performed for, the hopeless case (treatment is ineffective and animal suffering pain), animal having behavior problems, animals their owners no longer wish to own the said animal. The veterinarians should present the alternative and try to give the client the facts upon which to make a decision and they should not advocate or recommended euthanasia in any specific case.Personnel administering a euthanatizing agent must know:

Agent:

Whether the agent is appropriate for the species. What route, dose or concentration the agent should be administered. If the agent may result in fear behavior, struggling or vocalizations. If the agent causes tissue changes.

Method:

The method should be reliable, safe for the personal involved, have a restricted environmental impact, minimizing undesirable psychological stress,

economically feasible, compatible with its purpose, minimize the emotional effect upon observers and operators and should produce death without pain, the time to produce unconsciousness and death should be short. The technical skills for appropriate administration.

Methods of Euthanasia

The selection of the method of euthanasia is dependent upon the species, available means of control, number of animals, economic considerations and the wishes of the owner. The ideal euthanasia method involves sedation prior to death. The various agents that sedate or anesthetize animals prior to euthanizing include tranquilizers, barbiturates, carbon dioxide and inhalant anesthetics. Once the animal is sedated / anaesthetized, any number of methods can be used to euthanize, i.e. cervical dislocation, decapitation, various chemicals and physical agents. To assure euthanasia sufficient attention must be given to assure the cessation of heart beats or the lungs is collapsed. Inhalant agents, as Enflurane, isoflurane, ether, cyclopane, nitrous oxide, carbon monoxide, carbon dioxide, halothane, methoxyflurane, hydrogen cyanide, nitrogen and anesthetics.Non-inhalant agents includes pharmacological agents as barbiturates, chloral hydrate, strychnine, hydrocyanic acid, magnesium sulfate and curariform drugs. The preferred route of administration of the noninhalants is by intravenous injection. Physical methods, include Electrocution, shooting (gunshot), captive bolt pistol, cervical dislocation, decapitation, microwave irradiation and rapid freezing.

1-Inhalant agents

Include inhalant anesthetics, Carbon Monoxide, Carbon dioxide and Hydrogen Cyanide.

Inhalant anesthetics

As chloroform ether, halothane, and methoxyflurane. Are used for pups and kitten. The animals are confined in a small, closed chamber. Place cotton or gauze saturated with the anesthetic liquid. The animal inhales the vapors until death ensures. *it is* valuable in young animals where vein puncture is difficult. *the disadvantages* struggling and excitement, stimulation of the CNS during the induction stage, ether is flammable, chronic exposure to chloroform, halothane or methoxyflurane is injurious to personnel , halothane or methoxyflurane are expensive. Ether and chloroform are not acceptable methods for sedation, anesthesia or euthanizing, unless that method is part of the research objective.

Carbon Monoxide

Carbon monoxide can be effectively used for the euthanasia of small animals including dogs and cats provided that proper equipment is available and adequate safety precautions are observed. Inhalation of pure CO causes rapid and painless death. It converts the hemoglobin to carboxyhaemoglobin and causes an anemic anoxia. Chemical interaction of crystals of sodium format and sulfuric acid, exhaust fumes from engine combusting petroleum or the use of cylinder gas. The animals are placed in a closed chamber 6% carbon monoxide concentration gives the fastest results. Personnel using the gas must be thoroughly instructed in its use and understand the hazards and limitations. The lethal chamber must be equipped with internal lighting and view ports. The gas generation process should be adequate to achieve the desired CO concentration throughout the lethal chamber.

Carbon dioxide

Carbon dioxide (CO2) is heavier than air and nearly odorless. In low concentration (7.5%) it is an analgesic (pain reliever). At medium concentration (30-40%) it can be used as an anesthetic, causing rapid loss of consciousness without struggling, distress or excitation. At high concentration, more than 80% causes quick death. High concentrations, however, painfully irritate eyes and the respiratory tract, so it is important to first induce an analgesic effect, then bring about deep anesthesia (within 1-2 minutes) before exposing the animal to high concentrations. Carbon dioxide has been effectively used to euthanize most small laboratory animals such as mice, rats, guinea pig, reptiles, amphibians and rabbits. The combination of 40 % carbon dioxide and 3 % CO is relatively cheap, noninflammable, non explosive, and odorless and presents no hazard to the operator.

Hydrogen Cyanide (It should not be used for euthanasia).

2- Non-inhalant agents

Include Barbiturates, Chloral hydrate, Strychnine, Hydrocyanic acid and Magnesium sulfate

Barbiturates

Any of the barbituric acid derivatives can be used in excessive dosage to produce euthanasia of individual dogs, cats and other small animals. The barbiturates depress the respiratory center. The barbiturates are generally given intravenously. Intra thoracic, intra cardiac or intra peritoneal injection may be

employed. Pentobarbital sodium is often used for euthanasia in small animals in dose 27 mg per pound for I/V and 40 mg per pound for intra pleural or intra peritoneal injection or it can be given orally in bait to vicious animals. It produces death in quietly and animal appears to go to sleep naturally.

Chloral hydrate

It is used for euthanasia in large animals. It fatally depresses the respiratory centers. It is not recommended for use in small animals because of its slow action and death is preceded by unpleasant manifestations such as crying, muscular spasms and gasping. The fatal dose is 1 mg per five pounds in horses and is given I/V. It is relatively inexpensive.

Strychnine

Strychnine in any form should not be used for euthanasia for any animal because it increases the excitability and produces violent muscular contractions, which produce excruciating pain.

Hydrocyanic acid

HCN acid causes histotoxic anoxia and paralysis of the tissue enzyme system. It is not recommended for euthanasia because it causes death appears to be painful and it produces muscular tetany and the animal cries loudly before becoming unconscious.

Magnesium sulfate

The magnesium ion depresses all parts of the central nervous system and unconsciousness occurs prior to fatal respiratory paralysis. It is used for small and large animals. Saturated solutions (1:1) of magnesium sulfate should be injected

intravenous or intracardiac in very small animals. The temperature of the solution should be around 40 to 50 C at the time of injection. Magnesium sulfate solution should be injected rapidly to produce a lethal concentration of magnesium sulfate ion in blood. Dosage for euthanasia depends upon the concentration of the solution, rates of injection and the condition of the animal. Magnesium sulfate should be administered until euthanasia is produced. Doses 10 ml for small animals, 30 ml for large dogs and 0.5 g per pound body weight. Magnesium sulfate is inexpensive and does not produce excitement.

3- Physical methods

Electrocution

Electrocution as a form of euthanasia has been used for various species of animals. The usual procedure for electrical destruction of cats is to pass on alternating current from the forefeet to hind feet for about one minute, with an open-circuit voltage of 500 or 1000 volts. In this method the current passes through the thorax but not through the head and brain. Small currents cause death by asphyxiation, while larger ones produce ventricular fibrillation. Experiments in dog and man have shown the necessity of directing the electrical current through the brain in order to produce unconsciousness. The method of electrocution, which does not stun may include a period of at least 12 seconds of intense pain; this pain may cause the animal to faint or electrical curarisation in which the muscles are paralyzed. It is possible to electrocute individual animals relatively humanely. Electrocution cannot be used for mass euthanasia because of the time required to deal with each animal. Electrocution is aesthetically objectionable.

Shooting

Accurate and careful shooting is the most humane and the most rapid method for euthanasia. Almost any gun that fires a projectile can be used. A shotgun is preferable to a rifle. The target point for the most species can be located by drawing imaginary lines from each ear to the opposite eye. Where the lines intersect in the middle of forehead is the proper target point. The gunman should stand at a distance of five to eight feet. Instantaneous unconsciousness is produced if the projectile is accurately placed to enter the brain. For dogs and cats the captive-bolt pistol is employed with the barrel and held against the target spot. Shooting causes excessive hemorrhage and may be objectionable to the owner of the animal. Adequate precautions must be taken the assure the safety of operating personnel.

Cervical dislocation & decapitation

It is used if the animal cannot be sedated prior to euthanasia. Methods to use only on poultry, other small birds, mice, rats weighing less than 200 g., rabbits weighing less than 1 kg, other small amphibians, fish and reptiles. Personnel giving the responsibility for performing the technique are properly trained and consistently apply them humanely and effectively. Decapitation machine must be kept clean and sharp.

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