In this article we will review the gross and microscopic appearance of Blunt Force Traumatic Injuries and the mechanisms which causes them. The Blunt Force Traumatic Injuries covered are abrasions, contusions, lacerations, fractures, compression and hemorrhage.

**General Concepts**

The appearance of blunt force traumatic injuries is determined by the physical characteristics of a moving object, which has an impact on the victim or the nature of the surface, which the moving victim strikes. Whether an impact results in injury is depended upon its components, one of which is the kinetic energy displaced by the object in motion. This kinetic energy is expressed by the formula \( \frac{MV^2}{2g} \), in which \( M \) is the weight of the object in pounds, \( V \) is the velocity in feet per second and \( g \) is the acceleration of gravity which is 32 ft. per second. Thus, the ability of an object to induce injury is dependent on its weight and velocity.

When the victim is struck by a moving object, the resulting injury is in part due to the ability of the moving object to displace the tissues in the same direction the object is moving in. Likewise, when a victim is placed in motion and subsequently strikes a stationary object, the resulting injury to the victim is dependent on the degree which the stationary object can stop the forward motion of the victim. In essence, the rate at which tissue is accelerated when a moving object strikes the victim or decelerated when the victim strikes a stationary object is the underlying factor in the production of injury to tissue. As an example, a boxer will turn his body in the direction of the blow, thus, dissipating some of the kinetic energy of the blow, rather than remaining stationary and, thus, receiving the full kinetic energy of the blow. A parachuter on reaching the ground will flex his head, spine and extremities rolling with the fall, thus prolonging deceleration and minimizing or completely negating injury. This is in contradistinction to the
parachuter striking the ground holding their legs rigid, thus absorbing the full kinetic energy of the impact and sustaining injuries to his feet, legs, thighs, pelvis, vertebral column and possibly the base of his skull. Another factor which has important implications in survivability in those who have fallen from a height, they may collide with objects on their descent, which will ultimately decrease the kinetic energy on colliding with the ground allowing them to survive.

Although, well coordinated muscular action, as in the parachuter who collides with the ground, can alleviate serious injury, improperly coordinated muscular action can cause serious injury. What is often forgotten is that complete muscular relaxation of a flaccid body can result in minimal injury. Thus, an infant, intoxicated person or a person who can completely relax before a collision, may sustain little or no damage to skeletal structures.

Another factor to keep in mind is the structure of the object, which the victim is struck by or the victim collides with. If the object or the surface area is very plastic, the period of impact, and thus the period-of-time over which the kinetic energy is released will be lengthened. This period-of-time is due to the colliding surfaces changing in shape or conformation. For example, the kinetic energy released by a blow to a fixed skull will be much quicker, and thus induce far more energy, as compared to the same blow delivered to the buttocks. Likewise, a strike with a metal bar will induce far more kinetic energy than a flexible hose.

Although the total amount of energy discharged and the rapidity of its discharge play an important role in the production of traumatic injury, they are not the only factors. The size of the area over which the kinetic energy is released is very important. As has been previously discussed, the amount of kinetic energy released is related to its weight and velocity, expressed in-terms-of foot-pounds. One foot-pound is the amount of work required to raise 1 pound 1 foot against the force of gravity. The rapidity with which the kinetic energy is released is expressed in foot-pounds per second, however, the full expression of the potential destructive force of any given collision is expressed in-terms of foot-pounds per second per square inch of application. What this means pragmatically is the smaller the area over which a fixed amount of energy is discharged, the greater will be the achieved disruption of the colliding tissues. Thus, more kinetic
energy will be dissipated on being struck by the flat surface of plywood than by a crowbar. The kinetic energy induced by being struck by a crowbar is released over a much smaller surface area as compared to the flat surface of plywood. Likewise, more kinetic energy is dissipated by a fall on the buttocks than by a fall on which one lands on their heels.

Another important factor to keep in mind when evaluating impact-injury is whether the kinetic energy was released slowly or over a larger surface area. In such instances, no external surface blunt force traumatic injury is produced, but the resulting motion induced in the underlying tissues or organs may cause extensive damage. Where these injuries occur within the body is dependent upon how the kinetic energy is released, and propagated throughout the body, and the differences in the plasticity of the various structures lying in the path of the dissipated waves of kinetic energy. Thus, a blow delivered to the abdominal wall may not show any evidence of injury to the soft tissues of the anterior abdominal wall due to its plasticity, but show extensive injury to the diaphragmatic surface of the left and right lobes of the liver. There may also be occasions in which there is not only no evidence of injury to the soft tissues of the abdominal wall or diaphragmatic surfaces of the liver, but extensive disruption of the parenchyma of the liver with consequent hemorrhage. This is due to the lack of cohesive strength of the liver parenchyma, which manifest itself through the act of squeezing your thumb and forefinger together through a slice of liver 1 to 2 cm thick. Another example is a person falling, striking the back of their head producing no injury to the scalp, but a ring fracture to the base of the skull in the region of the foramen magnum.

The dissipation of kinetic energy can cause the displacement of gas or liquid within the victim with the resulting injury being due to pneumo- or hydrostatic forces. As an example, a severe blow to the chest while the glottis is closed, will prevent air from escaping from the lungs, which will lead to the displacement of air to other parts of the lungs causing widespread lacerations of the pulmonary parenchyma. In addition, a severe blow to the chest, most especially over the right atrium and ventricle can cause sudden displacement of blood in to the superior vena cava producing widely disseminated lacerations of capillaries and venules throughout the head and neck. A
forceful blow to the abdomen may cause rupture of the stomach, small and large intestines due to the explosive effects of suddenly displaced fluid or gas. In children the most commonly injured anatomical structures in the midline of the upper abdomen (epigastric space) are the duodenum and jejunum. The duodenum and jejunum in young children are especially vulnerable to traumatic injury due to the unique characteristics of their abdomen, wide and comparatively flared costal margins and a short anterio-posterior distance. Injury to these structures manifest themselves as duodenal hematomas, serosal avulsions, and full-thickness perforations leading to peritonitis. What is important to remember, hollow viscera, because of its visco-elastic nature, responds as a rigid, non-deforming structure, if the force is rapidly applied, such as in a punch or kick. In contradistinction to this, if the force is slowly applied, these structures display their elastic quality and are far more pliable or deformable.

Secondary Effects of Traumatic Injury

One of the most important secondary effects of traumatic injury is the escape of blood from lacerated vessels. The most substantive consequence of such loss of blood is hypovolemic shock with its attendant consequences. Another consequence of traumatic ruptured vessels is bleeding into interstitial or serous spaces of the body followed by compression of the contents of that space leading to functional injury. In addition, such blood may serve as a locus for growth of bacteria. This is especially true in the elderly due to their declining immune system. Individuals who are malnourished or who do not eat properly and thus lack vitamins, nutrients or have protein deficiency are at risk too. Those who are chronically ill, especially those who have disease processes, which cause severe compromise of their immune systems, bedridden or non-ambulatory also have high risk factors, as well as people who have undergone prolonged corticosteroid use or have been administered an immunosuppressive drug. Radiation therapy or chemotherapy patients, as well as diabetics, the obese, and those that have had a stroke or some sort of peripheral vascular disease are also most likely to develop some sort of wound infection. Bleeding into a closed space may give-rise to little loss of blood, however the resulting compression of the contents of that space may give-rise to catastrophic consequence,
such as bleeding within the cranial vault an its resultant compression and displacement of the brain followed by brain swelling and herniation. Another secondary effect of traumatic injury to blood vessels is the interference of blood flow followed by necrosis or the development of thrombosis due to stasis. Such thrombi can then cause emboli with its attendant consequences. Focal post-traumatic neurogenic disturbances of blood vessels may result in chronic circulatory disorders long after the wound has healed.

**General Adaptation Syndrome**

A process which results in stress, such as that caused by the destruction of tissue, whether that be caused by blunt force trauma or some other mechanism, may give-rise to a sequence of systemic changes, which come under the heading of General Adaptation Syndrome.

Selye published an article, “General Adaptation Syndrome and Diseases of Adaptation,” the Journal of Clinical Endocrinology, 1949. He indicated there were two mechanisms by which peripheral trauma may result in increased pituitary adrenocorticotrophic activity. One is humoral and self regulatory and the other mechanism involves the hypothalamus. Today the basic neurochemistry of the stress response is well understood. In response to stress, such as that brought on by blunt force trauma, neurons within the paraventricular nuclei of the hypothalamus secrete corticotropin-releasing hormone and arginine-vasopressin into the hypophyseal portal system. The locus ceruleus and other noradrenergic cell groups of the adrenal medulla and pons referred to as the LC/NE system, also become active and use brain epinephrine to execute autonomic and neuroendocrine response, serving as a global alarm system. The autonomic nervous system gives-rise to the immediate response to stress referred to as the fight-or-flight response. In this response the sympathetic nervous system is stimulated and the parasympathetic nervous system is depressed, the net effect of which gives-rise to cardiovascular, respiratory, gastrointestinal, renal and endocrine changes. The hypothalamic-pituitary-adrenal axis (HPA), a major part of the neuroendocrine system involving the interactions of the hyhypothalamus, the pituitary gland, and the adrenal glands, is also made active by release of corticotropin releasing hormone and arginine-vasopressin. This results in the release of adrenocorticotropic
hormone (ACTH) from the pituitary into the vascular system, which results in secretion of cortisol and other glucocorticoids from the adrenal cortex. Cortisol functions as an anti-inflammatory agent and suppresses the immune system. These corticoids involve the whole body in the organism's response to stress and ultimately contribute to the termination of the response via inhibitory feedback.

The General Adaptation Syndrome consist of three components or stages: Alarm, Resistance and Exhaustion.

Alarm is the first stage. When a stressor is realized the body reacts by manifesting a state of alarm. It is during this stage that adrenation is produced to cause the fight-or-flight response. There is also some activation of the HPA axis producing cortisol. In 1975, Selye published an article in which he divided stress into eustress and distress. When stress enhances function, whether that be physical or mental, such as through strength training or challenging work, it is referred to as eustress. Persistent stress that is not resolved through contending with the difficulty as adaptation is referred to as distress, which may lead to anxiety or depressive behavior.

Resistance is the second stage. If the stressor persists, it becomes necessary to attempt some means of dealing with the stress. Although an effort is made to adapt, the body cannot maintain adaption indefinitely due to the fact its resources are gradually depleted.

Exhaustion is the third stage. During this stage the body’s resources are eventually depleted and is no longer able to function normally, resulting in various forms of cardiovascular, renal and immune dysfunction.

**Morphologic Manifestations of Blunt Force Trauma**

The initial reaction to blunt force traumatic injury is dilatation of capillaries, which is immediately followed by a reduction in the rate of the flow of blood. In addition the capillaries develop an increase in permeability, which results in the passing of plasma and cells into the traumatized tissue. The edema that develops is in part due to plasma passing through the capillaries, but also due to fibrin occluding the lumen of lymphatics. Some believe it is the extent of lymphatic obstruction, which determines the severity of the edema, the greater the obstruction, the more severe the edema.
Marchand in 1901 was the first to demonstrate margination and emigration of leukocytes immediately after traumatic injury. Margination of leukocytes is seen within a few minutes of traumatic injury. The leukocytes are seen to adhere to the endothelial lining of the dilated capillaries. The actual migration of leukocytes between the endothelial cells into the traumatized tissue is seen within 30 minutes to one hour of the injury.

There is an important point to remember and that is neither margination, nor the beginning emigration of leukocytes, is conclusive evidence that the traumatic injury occurred before death. This is due to the fact the death of the victim does not coincide with the death of the individual cells within the tissues. The reality is that the cells composing the tissues are capable of vital reaction after the person has died, albeit for a limited period-of-time. As an example, Carscadden published a paper in 1927, in which he observed margination of leukocytes in the sinusoids of postmortem injuries to the liver up-to 30 minutes following cessation of cardiac activity.

Another important point to remember is that bleeding from injuries may occur for several hours following death. For example, penetrating wounds of the chest or abdomen in which large vessels have been incised or lacerated, may be followed by escape of blood into the pleural or abdominal cavities for several hours after death. A postmortem penetrating wound of the trunk may give-rise to several 100 cc of blood in the pleural or abdominal cavities. This phenomenon also applies to traumatic injuries to dependent parts of the body. However, postmortem bleeding from a superficial wound is usually minimal or absent and rarely gives-rise to more than a few cc of blood.

You also need to be cognizant of the fact that traumatic injuries with minimal or no gross evidence of bleeding does not necessarily mean the injury occurred in the postmortem period. The lack of bleeding from a traumatic injury may mean it occurred at the same time the victim suffered an acute cardiac arrhythmia, such as while operating a motor vehicle. Another example would be a penetrating missile or stab wound of the chest or a deep laceration of the scalp, even though inflicted in the antemortem period, may lead to little bleeding, if it has been preceded by another injury, which has resulted in circulatory collapse, such as severing or contusing the spinal cord above T6, thus causing an autonomic dysarrhythmia.
On occasion it is difficult to differentiate between antemortem and postmortem contusions even following microscopic examination. When there is traumatic injury in the antemortem period (the person is alive), red blood cells readily leave the disrupted blood vessels and infiltrate the adjacent tissue. However, should a deceased person receive a traumatic injury, similar, but less extensive hemorrhage can occur in the tissues, especially if the body part is in a dependent position. Generally, the quantitative difference between the antimortem and postmortem hemorrhages are great enough so that in many instances they can be resolved.

**Emigrated and Extravasated White Blood Cells**

When evaluating the number of white blood cells in an area of hemorrhage into tissue you need to be cognizant of the fact that white blood cells will escape from lacerated capillaries as well as red blood cells. The number of white blood cells in the extravasated blood is likely to be proportional to their number in the circulating blood at the time the hemorrhage occurred. Whether neutrophils (polymorphonuclear leukocytes) in the area of hemorrhage have emigrated or extravasated they will begin to disintegrate within three to five hours and are completely fragmented within twenty-one hours, assuming there is no additional bleeding. Their primary purpose is to release proteolytic enzymes that remove necrotic tissue and bacteria. Within approximately thirty hours the basophilic nuclear fragments have undergone autolysis or have been ingested by phagocytic monocytes. The neutrophils are first seen in the peripheral region of the traumatized tissue, albeit few in numbers, appearing within approximately thirty minutes. They are typically readily identified within two hours. The neutrophils migrate toward the central zone of the traumatized tissue using the meshwork of fibrin. What should be borne-in-mind is the number of neutrophils seen is predicated on the severity of the injury. For example, if the trauma is such that it only causes a few capillaries to be damaged with resulting bleeding occurring into the interstitial tissue, but no appreciable damage to adjacent tissue such as muscle, there may be no substantive neutrophilic reaction. The mere presence of bed blood cells in interstitial tissue does not invariably lead to a neutrophilic response. However, if the traumatic injury damages muscle there will be a substantive neutrophilic response. In those cases of traumatized tissue in which there is no superimposed infection, the neutrophilic reaction reaches its
peak in forty-eight hours. If however, the traumatized victim becomes moribund after the injuries, the cellular reaction to the traumatized tissue may be substantively depressed.

Neutrophils are largely replaced by phagocytic monocytes (macrophages) by 48 to 96 hours. Unlike the neutrophils, the monocytes and lymphocytes within the area of hemorrhage do not ordinarily disintegrate, but aid in subsequent repair. Lymphomononuclear cells, which have emigrated, do not typically appear in the traumatized tissue until between twelve and twenty-four hours after the injury. These macrophages, as well as the subsequently appearing fibroblasts, have their origin primarily from cell-precursors, which migrate to the area from the bone marrow. The quantity of the lymphomononuclear cells is proportional to the severity or extent of the traumatized tissue. Lymphomononuclear cells, which were part of the initial bleeding process from the lacerated capillaries, show evidence of metamorphosis into fibroblasts within six hours. Such mononuclear cells, as well as the vascular endothelial cells, will show evidence of swelling within one hour of the injury.

What is of interest is an observation of Menkin who noted the quantity of lymphomononuclear cells, which appear in the traumatized tissue is in part influenced by the pH of the tissue, the greater the acidity, the greater the number of lymphomononuclear cells seen.

The macrophages produce chemokines, TNF, PDGF, TGF-β and FGF, which cause the fibroblasts to migrate to the site of injury. The fibroblasts subsequent proliferation is triggered by PDGF, EGF, TGF-β, FGF, and cytokines IL-I and TNF. Although, macrophages are the main source of these factors, other inflammatory cells and platelets can also produce them.

The fibroblasts may also be derived from existing fibroblasts in or near the site of injury. According to Maximow, mitotic division is first seen in these fibroblasts in about 15 hours. Another source of fibroblasts is from a metamorphic process involving macrophages, which begins to occur within six hours of the appearance of the macrophages.
Repair
The second phase of the inflammatory reaction is constituted principally by a proliferation of fibroblast and the formation of new blood vessels. If the traumatized surfaces of the disrupted tissue are not separated by extravasated blood or necrotic tissue, and the edges of the traumatized tissue are closely approximated, the defect is bridged first by clotted blood, which contain entrapped red blood cells, fibrin, fibronectin, and complement components. In essence, the clot provides scaffolding for subsequent cells migrating into the defect, neutrophils followed by monocytes, which are attracted by growth factors, cytokines and chemokines released into the traumatized area. The release of these substances is enhanced by VEGF (vascular endothelial growth factor), which leads to increased vascular permeability and edema.

The fibroblast initially produce a network of type III collagen, which is a weaker form of the structural protein that can be produced rapidly. This is later replaced by the stronger long-stranded type I collagen, which is seen in scar tissue.

Another aspect of the repair process is the proliferation of surface epithelial cells, which fuse in the midline of the defect below the surface scab, producing a thin, continuous epithelial layer that closes the wound. While this process is occurring macrophages stimulate fibroblasts to produce FGF-7 (keratinocyte growth factor) and IL-6, which enhance keratinocyte migration and proliferation. Other mediators of re-epitheliatation are HGF and HB-EGF. Signaling through the chemokine receptor CXCR 3 also promotes skin re-epitheliation.

Cicatrization (Scar formation)
After the first few weeks the mass of the extravasated blood and the repair tissue in and about the wound begin to decrease. The leukocyte infiltrate, edema and increased vascularity for the most part disappear during the second week. The original scaffolding formed by the granulation tissue is converted to a pale, avascular scar, composed of fibroblast dense collagen, fragments of elastic tissue and other extracellular matrix components. In most instances a scar of the skin becomes static within a month. In a study of scars of the skin, vonSchroter found that the elastic fibers first appeared in about 36 days and appeared in a substantive amount between 3 and 6 months. Some scars, however, did not show elastic fibers after a year. The pigment of the skin often
returns incompletely or not at all in scars of the skin. Dermal appendages (hair, sweat glands, sebaceous glands and nails) that have been destroyed near the scar do not regrow. However, under experimental conditions rats have re-grown new hair follicles in healing wounds under Wnt signaling pathway stimulation. The Wnt signaling pathway is a network of proteins best known for their roles in embryogenesis and cancer, but also involved in normal physiologic processes in adult animals. Howes, Sooy and Harvey, in a study of the healing of wounds of skin, fascia, and stomach, found that within 14 days the tensile strength of the scars was usually as great as that of normal tissue.

Types of Wounds Produced by Mechanical Trauma

Abrasions: An abrasion is a traumatic wound produced by scraping or rubbing, resulting in the removal of either the superficial layer of the skin (epidermis) or it may
extend deeper into the dermis, or still deeper into the soft tissue beneath the skin (subcutaneous or muscle). If the abrasion involves only the epidermis, it is declared ‘superficial’ and typically there is no bleeding. However, Bernard Knight reminds us, due to the skin’s corrugated nature and hence exposure of the dermal papillae, which have fine blood vessels, a superficial abrasion can produce bleeding, albeit, minimal. If the abrasion extends into the dermis and/or the subcutaneous tissue, blood vessels will be lacerated with its attendant bleeding.

Abrasión con extensión hacia la dermis, o aún más hondo hacia la tejido subcutáneo debajo de la piel (subcutánea o muscular). Si la abrasión involucra solo la epidermis, se declara ‘superficial’ y típicamente no hay sangrado. Sin embargo, Bernard Knight nos recuerda que, debido a la naturaleza corrugada de la piel y por lo tanto, la exposición de las papilas de la dermis, que tienen vasos sanguíneos finos, una abrasión superficial puede producir sangrado, aunque sea mínimo. Si la abrasión se extiende hacia la dermis y/o el tejido subcutáneo, los vasos sanguíneos se lacerarán con sus consiguientes sangrados.
Any mechanical force that traumatizes the epidermis, removing the keratinizing layer, thus exposing the underlying cells will change color and have a moist appearance due to exuded tissue fluid. The acute color is typically red should the person remain alive. If death occurs within the same period of the generation of the abrasion, the abrasion will become stiff, leather-like, and take on a parchment-like brown color due to drying of the moist exposed surface. A classic example would be the ligature mark produced by hanging or strangulation.

An abrasion produced by a ligature used in a hanging

An abrasion produced by a ligature used in a hanging
In actuality it is impossible to tell whether a superficial abrasion occurred immediately before or after death. If however, there is evidence of a substantive inflammatory reaction, not just a few scattered leukocytes, which are normally circulating in the blood, than the injury is antemortem.

The above is an illustration noting the various layers of the skin

**Types of Abrasions**

1. **Tangential:** The pattern of the abrasion may indicate the shape or something of the nature of the abrading object. As an example, a brush abrasion is produced by some portion of the body passing across a rough surface producing linear furrows across the skin. On occasion, such abrasions will show the epidermis to be rolled-up at one margin, indicating the direction of movement of the body across the abrading surface. Likewise, if a person is struck by an object in a glancing blow fashion, the epidermis may be rolled-up at one end of the abrasion indicating the direction of the blow.
This is an example of a tangential abrasion

2. **Crushing**: This is an abrasion that is produced by an object, which strikes the surface perpendicular to the skin surface. This causes the surface of the skin to be

Blunt rubber-bullet crushing injuries of the back, some are circular crushing abrasions, others are penetrating.
crushed by the surface of the object and on occasion with an imprint of it’s surface appearing on the skin. An example would be the imprint of the sole of a shoe which was used to stomp the victim. If the impact is substantive than the crushing abrasion will be associated with an underlying contusion. Also, if the impact is particularly forcible, as shown in the above picture, and the area of contact small a puncture wound can be produced.

3. **Fingernail abrasions:** A common form of an abrasion is that produced by fingernails, in which instance the number and distribution of the marks may be highly characteristic. Fingernail abrasions are seen most commonly on the neck, face, arms and forearms. Such wounds on the neck may constitute important evidence in cases of manual strangulation. Fingernail induced abrasions may be linear if the nails are dragged down the skin, however, they may be short or curved if they are applied to the skin in a static fashion. The pattern of fingernail abrasions on the neck may be linearly irregular when the victim of either a manual or ligature strangulation attempts to remove the assailant’s hands or the material used to strangle the victim. Typically under these circumstances the fingernail marks produced by the victim are linear, whereas those produced by the assailant are more irregular and random.

![Image of traumatic injuries produced by manual strangulation (throttling)](image_url)
Traumatic injuries produced by strangulation (ligature)
Static fingernail abrasions are often seen on the arms when the victim is being restrained and thus gripped. This is especially true in child abuse. Not uncommonly such abrasions are associated with contusions. Be very careful on giving a definitive opinion on how the assailant’s fingers were applied based on the curvature of the fingernail abrasions. Bernard Knight clearly points out that although it is natural to assume that the concavity of the fingernail abrasion indicates the orientation of the fingertip, Shapiro et al (1962) has shown this is not often the case. They showed that when the skin is put under lateral tension by fingernails, they may distort the skin, so that when the tension is released the elasticity of the skin causes it to return to its original position, carrying the fingernail abrasion with it. The produced curve may then reverse to form either a straight line or a convexity.

Of collateral importance is the indication of fragments of skin and blood beneath the fingernails of the victim, indicating abrasion will be found on the assailant. Before DNA analysis, such a determination would constitute circumstantial evidence as to the identity of the assailant. However, with the applications of DNA technology such a determination would constitute definitive identification of the assailant.

4. Patterned abrasions: These are classically represented by abrasions produced by the grill of a car or projections of motor vehicles or tire tread patterns. They can be of great value in identifying the vehicle or object that produced the abrasion. Objects with a patterned surface used to strike a person, especially if the object strikes the person perpendicular to the surface of the skin will induce abrasions of the surface of the epidermis that follow the ridges of the pattern on the surface of the striking object.
Patterned abrasions produced by a biting

5. **Postmortem abrasions:** These can be produced by a myriad of causes including dragging the deceased, as part of postmortem dismemberment, insect bites, deceased floating in water rubbing against rocks, limbs of trees, fish bites, etc. Postmortem abrasions can also be the result of handling of the body by medical and mortuary personnel, law enforcement, etc. These abrasions often present with a stiff leather-like appearance and a parchment-like orange to brown color.

Abrasions produced after death
Postmortem abrasions produced by roaches

Dating of abrasions at best can only give you a rough estimate of the age and that is after a careful microscopic examination. The age, however, cannot be determined with precision due to interpersonal variability in human physiology, underlying pathology, and the mechanism of trauma. Under the best of circumstances, an abrasion may be categorized as of very recent origin (within a few hours), recent (a few hours to a day), a few days old, or older than several days.

Contusions (Bruises): A contusion or bruise represents the effect on tissues of an impact or pressure and is ordinarily recognized by the presence of extravasated blood without gross disruption of tissue continuity. An extravasation of blood larger than a few millimeters in diameter is usually referred to a a ‘contusion’ or a ‘bruise.’ This size overlaps with the older seldom used term ‘ecchymosis’, which in reality is a bruise. Another area where you will see the term ‘ecchymosis’ used is in the clinical physical examination of an elderly patient, who due to the fragility of the superficial blood vessels, lose of subcutaneous tissue and poor skin turgor, will show diffuse hemorrhages of varying ages often on the upper and lower extremities due to minor unintentional trauma. This is an attempt to distinguish such hemorrhage beneath the skin from the hemorrhages induced by intentional blunt force trauma.
Ecchymoses commonly seen on the upper and lower extremities of the elderly
Another term you will often see is ‘petechial hemorrhage,’ which is the size of a pinhead or less (1 to 3 mm). These hemorrhages appear as red or purple spots on the body, typically caused by the rupture of capillaries. The most common cause of petechiae is through a vigorous bout of coughing, vomiting or crying, which can result in facial petechiae, especially around the eyes. Such petechiae disappear within a few days. They may also occur when excessive pressure is applied to tissue (e.g., when a tourniquet is applied to an extremity, the person is struck across the face with an open palm of the hand (slap). If the petechiae are of a more diffuse nature they may be a sign of thrombocytopenia (low platelet count) or inhibition of platelet function. Thrombocytopenia is technically any value below a platelet count of 150,000. However, a common definition of thrombocytopenia is a platelet count below 50,000 per microliter
of blood. Generally, the normal platelet count ranges from 150,000 to 450,000 platelets per microliter. Typically, if a person’s platelet count is between 30,000 to 50,000/mm³, bruising with minor trauma can occur; if it is between 15,000 to 30,000/mm³, spontaneous bruising will be seen, mostly on the arms and legs. Inhibition of platelet function can be the result of medications, infections, genetic disorders such as clotting factor deficiencies.

The size of a petechiae in relationship to a dime

Petechiae on the leg
Whereas petechiae are caused by the rupture of the capillaries, contusions are caused by damage to veins, venules and small arteries. The onset of bleeding is immediate and it may continue for minutes or even hours after the injury, the duration depending upon the violence of the impact, the type of tissue injured and the bleeding time (assesses platelet function) and clotting time (assesses the conversion of fibrinogen, a soluble protein and to fibrin, an insoluble protein). People vary greatly in their susceptibility to bruising. Those who are obese or suffering from chronic dietary deficiencies, such as chronic alcoholics, show extensive subcutaneous and soft tissue hemorrhage from relatively minor trauma. Extensive bruising can occur in those with clotting and bleeding disorders. Extensive bruising may be completely masked by pigment in the case of blacks and dark-complexioned individuals.

There is a term, which you will occasionally see used, ‘intradermal bruise.’ This is a bruise which occurs in the layer of skin between the epidermis and subcutaneous tissue. Such bruising is often seen with patterned objects with alternating ridges and grooves where the skin is forced into the grooves and thus distorted. Intradermal bleeding will occur here, while the areas in contact with the raised ridges may remain pale. The ribbed rubber soles of ‘trainer’ shoes can produce such injuries.

**Superficial Bruises:** These are bruises involving not only the dermis, but the subcutaneous tissue with possible extension into the superficial layer of muscle. These bruises are by far the most common. Their appearance is in part determined by the amount of bleeding into the subcutaneous tissue, the laxity of the subcutaneous tissue next to the lacerated blood vessels, the depth at which the bleeding originates and the time of examination following the trauma. The amount of bleeding is in part determined by the severity of the force, the density of the under lying vascular network, the fragility of the blood vessels, the coagulability of the blood, and the volume of the subcutaneous tissue.

Whether a bruise will appear, or if it does, it’s size is influenced by the anatomic area subjected to the mechanical force. Those areas of the body in which subcutaneous tissue and muscle directly overlie bone, such as the head, chest and anterior surface of the knee, legs, feet and the posterior surface of the hand readily show bruises due to the fact the ‘UN-giving’ bone allows the overlying soft tissue to be easily compressed.
with minimal trauma, thus producing contusions. In contradistinction to this, areas such as the abdominal wall, buttocks and posterior aspects of the thighs are less apt to show bruises with the same level of force, which readily produced a bruise over the shins.

A superficial bruise overlying the cheek
Bruises are far more apt to occur in the lax tissue of the orbit and infraorbital region due to the lack of density of the soft tissue. In contradistinction to this, it is unusual to see bruises on the palm of the hand or sole of the foot due to the density of the fibrous tissue beneath the epidermis as well as the restrictive facial planes. Also, subcutaneous hemorrhage into the eyelids and/or infraorbital region is not necessarily evidence of blunt force affect to the area. Extravasation of blood into the lids and infraorbital soft tissue may be due to blunt force trauma to the forehead or the anterior fossa of the base of the skull with subsequent migration of the blood to the lax soft tissue of the orbital and infraorbital region. If the bruise involves the deep tissue, the blood may migrate along fascial planes to appear as a bruise some distance from the traumatized area, such as involving the arm and thigh surfaces as a bruise around the elbow or knee.
Bruises involving the upper and lower eyelids and the infraorbital region due blunt force
Due to the greater volume of adipose tissue in the subcutaneous tissue in those who
are obese, they bruise much more easily as compared to those who are thin, of the
same age, considering vessel fragility and senile changes.
Children tend to bruise far more easily than adults due to the less volume of muscle and
subcutaneous tissue. In the case of deeper bruises it may take several hours, up to
more than 24 hours, before the extravasated blood makes its way to the surface, thus
becoming visible as a bruise. This accounts for why bruises become more evident with
the passage of hours or days. This is believed to be the result of extension of free
blood from its origin in deeper tissues upward to the epidermis. Some have also
suggested this phenomenon is the result of hemolysis of red blood cells, thus,
producing free hemoglobin, which in turn stains the tissue in a more diffuse way. They
go on to point out that this is the reason for not only the postmortem phenomenon of
bruises becoming more prominent after death, but new bruises appearing later where
none were visible at the time of autopsy, or bruises identified at the time of external
examination, yet none were visible at the scene. Although, there are certain aspects of
this explanation, which may be true, however, the rate of disintegration in situ of red
blood cells varies greatly. In a mild contusion it is unusual to see any intact free red
blood cells after 5 to 7 days. If the bleeding has been copious, forming a hematoma, intact red blood cells may be seen for weeks, thus escaping phagocytosis and disintegration. It is true that the initial product released from disintegrating red blood cells is hemoglobin. However, within a few hours hemoglobin is phagocytized producing hemosiderin, which is represented by yellow-brown amorphous granules. The orange, iron-free, rhombic to needle-like crystals of hemotoidin appear in about one week. The presence of iron-containing pigment, either at the site of injury or in the lymph nodes draining that region, indicates that at least 12, to probably 24 hours, has elapsed since the blunt force trauma injury was sustained.

**Aging of Bruises (Contusions):** Both the color change and the fading of the bruises are time related. However, these color changes are not constant. The usual sequence is from dark red, through blue, dark blue-purple, brown, yellow and yellowish-green. The entire gamut of color change may run its full course within a week or the absorption may occur so rapidly that all visible color has disappeared within a few days. Clearly a yellow-green contusion is older than a blue-purple one; but how much older? A yellow or yellow-green discoloration of a contusion usually means that at least several days have elapsed since injury, but how much longer may be impossible to say. Bernard Knight handles this issue in a very pragmatic fashion. If a bruise appears fresh over all its area, with no observable color change, it is unlikely to have been inflicted more that 2 days before death, except in the elderly. The elderly may not heal their bruises at all and carry them for the rest of their lives. If the bruise has any green discoloration, it was inflicted not later than 18 hours before death. If several bruises (of roughly comparable size and site) are present and are of markedly different colors, then they could not have been inflicted at the same time. This particular statement has significant pragmatic importance in suspected child abuse, where intermittent episodes of injury have important diagnostic significance.

More recent observation have suggested the most significant change is the appearance of a yellow color (in persons less than 65 years of age), which indicates the bruise could not be less than 18 hours old. Blue, purple and red do not assist in dating; bruises of brown color are believed to be a mixture of colors and thus not considered useful.
Bernard Knight believes it is impossible to comment on the age of a bruise less than 24 hours since the induction of blunt force trauma, other than to say it is fresh.

Bruise approximately 4 days old showing purple to brown to yellow
There are other aspects to the macroscopic appearance of bruises, which must be considered. First, the size of the area of the bleeding site can play a significant role in the appearance of a bruise. A large bruise may contain the entire color sequence of the macroscopic appearance of a bruise, from purple in the center to yellow at the periphery. Second factor which affects the gross appearance of a bruise is the age and physiologic condition of the person. As previously mentioned, the elderly may not heal their bruises at all, carrying them for the rest of their lives. Lastly, is the presents of coagulation or bleeding defects.

Microscopic evaluation can provide some information as to age, however, like the macroscopic examination, the information it provides is not precise. As I have previously discussed in this article, Bernard Knight has stated from his experience stainable iron, in the form of hemosiderin, does not usually appear within the first 2 to 3 days, which is in contradistinction to others, such as Simpson, who have observed stainable iron in phagocytes giving rise to a positive Prussian blue reaction within 24 hours, with iron-free rhombic or needle-like crystals of hematoidin not appearing for 7 days.
Prussian Blue positive macrophages revealing stainable iron of hemosiderin

Another issue that you need to keep in mind is if the bleeding is rapid or copious the blood is likely to displace tissue rather than infiltrate it, and such a collection of blood in a newly formed space constitutes a hematoma. If large, a hematoma is likely to become secondarily infected since extravasated blood provides a favorable site for bacterial growth. A large hematoma may remain fluid at its center for weeks. It can become encapsulated and persist indefinitely as a cyst. Calcium may be deposited in the hematoma and the entire mass may eventually undergo ossification.

A contusion may be overlooked on external examination if it is located on a dependent surface of the body and livor mortis is already established. To distinguish between lividity and an acute contusion an incision into the suspected contusion will reveal hemorrhage into the soft tissue manifested by diffuse reddish discoloration; lividity will show no such discoloration in the soft tissue. In addition, microscopic examination will show hemorrhage into the soft tissue, whereas postmortem lividity will show no interstitial extravasation of red blood cells.

**Appearance of a Bruise suggesting Causation:** The shape of a contusion may suggest the striking object. For example, the beating of a child with a coat hanger will
cause a distinctive narrow patterned contusions. Likewise, a closely grouped circular contusions separated by a narrow distance located on the arms and anterior-lateral and posterior-lateral chest wall of a child suggest bruises formed by fingertips during the process of gripping the child. Such bruises, along with fine curvilinear abrasions on the neck can be seen in manual strangulation.

A ‘tram-line’ bruising pattern is often seen when the victim has been struck by a rectangular or cylindrical object. This pattern is marked by two parallel lines of bruising with an undamaged area in the center.
The above is an example of a Tram-line bruise

Pattern bruises produced by the tire of a motor vehicle

Often when the victim is stomped on an intradermal bruise will be produced in a pattern reflecting the same pattern on the sole of the shoe. Such a patterned injury can also be produced by the tire pattern when the victim is run-over by a car as shown above.
Postmortem bruising

**Postmortem Bruising:** Bruises not only occur before death but after. Contusions can be produced within a few hours of death. These typically occur when the deceased is subjected to substantive blunt force trauma. The underlying causation is the same as antimortem contusions in that small vessels, venules, small arteries and capillaries are ruptured leading to leakage of blood into the interstitial tissue. They typically occur in the skin and underlying soft tissue overlying bone, such as the head, nose, chin, elbows, knees, shins and dorsal surface of the feet. Microscopic examination often is of little help in discerning between an antimortem bruise and a postmortem bruise. When the body begins to decompose it can make determination of an antemortem bruise externally difficult. This is especially true in the scalp. With decomposition there is hemolysis of red blood cells, which causes a diffuse coloration of the soft tissue. This process can make it very difficult to distinguish between an antemortem contusion and an area of livor mortis. What may be of some help is in your microscopic examination to look for leucocytes, which if abundant may suggest an antemortem injury. However, if the decomposition process has reached the point that leucocytes have disintegrated
than it may be impossible to make the distinction between an antimortem contusion and lividity.

**Deep Tissue and Organ Contusions:** Contusions not only appear on the skin and underlying subcutaneous tissue, but they can also occur deep within muscle as well as the organs. Such contusions suggest substantive blunt force trauma. It is important to look for these contusions. Bleeding into the thigh muscles or the liver may be of sufficient quantity to cause death. This also applies to contusions of the lungs, which in of themselves can make a substantive contribution to death. Contusions of the heart can create an acute cardiac arrhythmia and death. Contusions of the brain can cause substantive brain swelling causing herniation. Contusions of the brain stem are a cause of sudden death due to cessation of cardiac and/or respiratory activity.

**Lacerations:** A laceration is usually the result of a crushing or stretching force. Lacerations may occur externally or internally. If internally they may not communicate with the surface, as for example a laceration of the diaphragmatic surface of the liver or the visceral pleural surface of the lungs. The typical laceration is one which involves the skin and is usually due to the skin and underlying connective tissue being stretched over a superficial bone. This is often accomplished by a not necessarily sharp object crushing the skin and underlying soft tissue with force causing a disruption of the continuity of the skin and subcutaneous tissue. The resulting defect is often at right angles to the direction of the applied force and often fellows approximately definite anatomical lines of cleavage. Lacerations produced by crushing force usually have ragged margins, and frequently the adjacent skin shows abrasions caused by the flatter portions of the striking object rubbing against the skin. Since bleeding is an invariable effect of a laceration, one often sees a contusion accompanying the laceration. The margins of the laceration may suggest the direction in which the force was applied. The more undermined edge of the laceration is the side toward which the force of the striking object was directed; the sloped side of the laceration is that side from which the blow was directed. Likewise, the side of the laceration with the adjacent contusion is often the side from which the force of the blow was directed.
Markedly irregular laceration of the back of the left hand with patch contusions at the margins
Sutured laceration with evidence of an abrasion below and to the right and somewhat to the left with an infraorbital contusion.

A laceration of the skin, which is not accompanied by any significant amount of crushing may have such sharp margins as to resemble an incision. It may be necessary to examine the margins, especially the ends of the wound with a magnifying glass, to recognize that the tissue has been torn and not cut.

The shape of the laceration may indicate something of the nature of the object that caused it. However, because of the resiliency of the target tissues, much stretching may result before the tearing of tissues results. Thus, blunt force traumatic injuries inflicted by a hammer may not necessarily cause lacerations in the shape of a hammer face or for that matter semicircular lacerations. What is often seen at the end of such lacerations are tears at angles diverging from the main laceration itself, which are called ‘swallow tails.’
Should the victim survive blunt force inflicted lacerations, the lacerations will undergo a series of changes, somewhat similar to those noted in abrasions and contusions. The first change is the formation of a blood clot, which tends to overfill the depths of the laceration and to spread out into the surrounding skin or mucous membranes. The clot, together with aggregated tissue fluids and cellular debris combine to form an eschar, or scab. Scar tissue growth begins in the depths of the laceration manifested by an intense inflammatory reaction with the formation of abundant granulation tissue through a vigorous proliferation of fibroblasts and vascular endothelial cells forming new small blood vessels. These new vessels are porous allowing for the passage of plasma proteins and fluid into the extracellular space. By 5 to 7 days, granulation tissue fills the wound area. With the neutrophils being largely replaced by macrophages by 48 to 96 hours, followed by the deposition of collagen and spurs of epithelial cells extending from the edges of the wound, the new scar tissue and thus, repair are complete. The scar tissue does not contain skin appendages, such as sweat glands and hair. Estimation of the time of initiation of the blunt force traumatically induced laceration is no more precise then that possible with abrasions or contusions. The best that can be
hoped for in most instances is an estimate of very recent, a few days, and older than several days. Lacerations after death can be distinguished by the absence of bleeding. As has been intimated above, sometimes it can be difficult to differentiate a laceration from an incised wound. The following points will help you in making that differentiation:

1. Examination of the margins, using a magnifying glass if necessary, will show irregularity in a laceration.

2. Not uncommonly a laceration has an associated abrasion and/or a contusion.

3. Presents of ‘swallow tails’ at the ends of a lesion, diverging at an angle from the primary lesion indicates a laceration.

4. Bridges of tissue are seen extending from one side of the laceration to the opposite. In an incised wound no such bridges are formed.

5. When a laceration appears over bone, there is no evidence of linear injuries in the underlying bone.

6. Lacerations of the scalp often will show intact hairs extending across the defect. An incised wound will show cutting of the hairs.

Lacerations of deeper soft tissue, i.e., muscles of the thigh, buttocks, and organs can also be the result of blunt force trauma. This is especially true of those organs which contain fluid, i.e., blood, gastrointestinal contents, etc. Such organs can burst when a pulse of pressure passes through them. This is especially true of the heart in diastole when it is filled with blood, stomach, second and third portions of the duodenum, urinary bladder and aorta. The liver and spleen are easily ruptured due to the low cohesive strength of their parenchyma. Blunt force trauma to the abdomen can cause profound intraparenchymal lacerations, which are not visible on the diaphragmatic or posterior surfaces, but can contain sufficient blood to cause hypovolemic shock and death. Intraparenchymal laceration of the liver or spleen can lead to subcapsular hematomas, which can rupture within minutes to days causing hypovolemic shock and death.

Another unanticipated effect of a laceration is tissue damage with complete tearing occurring long after the trauma was inflicted. Non through and through lacerations of the heart, aorta, liver, spleen and gastrointestinal tract are most likely to cause this complication. Although, the laceration is incomplete, the tissue has been weakened, thus, hours to several days later the tissue of the injured area fails allowing for bleeding
and in the case of the gastrointestinal tract, most especially the stomach and duodenum, allowing for escape of gastrointestinal contents into the peritoneal cavity. A close examination of lacerations can provide much needed information in both criminal and civil issues. This is especially true when a manner of death of homicide is being considered. The question to be decided is whether the blunt force traumatic injury was induced by a fall or a blow. If the traumatic injury was produced by a moving object the probability of a homicide must be considered. Reconstruction of how a blunt force traumatic injury was inflicted, such as a laceration of the scalp, may be particularly important in those cases in which the injury caused or contributed to death. Often the instrument used to inflict the scalp laceration(s) did not strike the head directly perpendicular to the surface, which often causes one edge of the laceration being torn away from the skull. This edge may be torn back as a hinged flap or it may be simply undermined. The direction in which the force responsible for the laceration(s) was traveling can often be recognized by comparing the two edges of the wound. As an example, if the impact responsible for the laceration of the scalp was produced by a club, which was traveling downward, the lower margin of the wound is likely to show more bruising and to be more extensively lacerated and undermined than the upper. If however, the force was applied in an upward direction as compared to the position of the head, such as would be the case if the wound were produced by falling on a hard object such as a bare concrete or hardwood floor, the reverse would be true. The medico-legal implications of such observations are obvious.
Approximately 75% avulsion of the scalp

Avulsion injury of the leg
In many cases, abrasions, contusions, and lacerations frequently are seen together. A single instrument may cause a contusion with one blow, a laceration with another, and an abrasion with another blow. Having said that, a single blow can produce all three blunt force traumatic lesions. The victim may be stationary and the instrument used to inflict the traumatic lesions be in motion or vice versa, for any of these types of injuries to occur.

**Fractures:** From a forensic standpoint there are two fractures, which you will most commonly deal with, ‘simple or closed’ and ‘compound or open.’ The ‘simple fracture’ refers to a fracture of the bone with intact skin overlying it, whereas a ‘compound fracture’ refers to a fracture in which one or both ends of the fracture have perforated the overlying skin.

There are several factors, which will determine whether a bone will fracture, with one of the most important factors being age of the victim. For example, in an infant or child, bone, most especially the skull, can undergo substantive deformation due to blunt force trauma without fracturing. The ribs are another example of bone, which can undergo great deformation due to blunt force trauma without fracturing. This ability to undergo deformation without fracturing is because the costal cartilage has not calcified in infants, children and young adults. Calcification of the costal cartilages follows gender related patterns and is generally not radiographically evident until after 30 years of age. Thus, more force may be absorbed by the ribs, with more temporary deformity, during the application of force, than that found in the older adult.

Another example of age playing a role in the induction of fractures is the older individual, male and female, although more often female, who develop osteoporosis. Such fractures in this age group can occur with minimal application of force.

In forensic pathology, if there is evidence that blunt force trauma may have played a role in the victims demise, radiological exams should be undertaken, because not all fractures show a visible deformity. However, it is important to keep in mind that not all fractures will be visualized with X-rays. Perhaps as many as 20% of linear fractures of bones of the skull are not found. This underscores the importance of doing a complete autopsy in those who have been victims of blunt force trauma.
Compound Fracture left ankle
Noting the presence of fractures is important, for their shape can provide information on the nature of the object, which induced the fracture. This is especially true of fractures of the skull. For example, the fracture defect made by a hammer may resemble the face of the head of the hammer. The shape of the fractures of the skull may also indicate the direction in which the blow was struck. In the case of a chopping instrument, the undermined edge of the fracture defect is the direction in which the lateral force vector is exerted, and the slanted edge is the side from which the force was directed. In the case of fractures of the long bones, such as those in the lower extremities, the direction in which a severe blow was struck can be determined by X-rays or direct examination of the fragments, with the fragments being loosened on the side opposite that from which the force came. As far as fractures of the pelvis, you may be able to determine the direction of the applied blunt force trauma, as for example, fractures of the pelvis occurring directly beneath the primary impact site, as determined by the presence of abrasions, contusions, or lacerations, or a combination of these and fractures of the opposite side of the pelvis. A line drawn across the pelvis to connect both fracture sites will indicate the direction of force, and may give information on the direction the victim was facing at the time of being struck.

The age of a fracture can provide substantive information most especially in child abuse cases. Radiologic and gross examination of the healing fracture site can give you information on the state of healing and therefore an estimation on the time from between the date of the examination and when the injury was inflicted. Microscopic examination, most especially by an orthopedic pathologist can give you good information as to the age of the fracture.

**Pathophysiologic effects of Fractures:** There are two complications, which are of concern to the forensic pathologist: Early and Late. Of the two, it is the early complications, which are of most concern to the forensic pathologist. The early complications occur at the time of the fracture or soon after. The early complications are divided into those, which are local and those which are systemic. Local complications include vascular injury causing hemorrhage, internal or external, visceral injury causing damage to structures such as the brain, lung or bladder, damage to surrounding tissue, nerves or skin, hemarthrosis, compartment syndrome and wound
infection, which are more common in open fractures. Systemic complications include fat embolism, thromboembolism, shock, exacerbation of underlying diseases, such as diabetes or coronary artery disease and pneumonia.

Late complications of fractures, although typically not a concern of the forensic pathologist are divided into local and systemic. The local complications are delayed union, nonunion, malunion, joint stiffness, contractures, myositis ossificans, avascular necrosis, algodystrophy (Sudeck’s atrophy), osteomyelitis and growth disturbance or deformity. The systemic complications are gangrene, tetanus, septicemia, osteoarthritis and fear of mobilizing.

The central portions of long bones contain an admixture of fat and bone marrow, which with a fracture, can gain entrance to the venous circulation and carried to the lungs and possibly other organs. Unlike emboli that arise from thrombi, fat emboli are small and multiple, thus they have widespread effects.

Fat embolism in lung
Fat embolism in pulmonary artery in lung

Interleukin-6 to aid in the identification of fat emboli in the lung
Interleukin-6 acts as both a pro-inflammatory and anti-inflammatory cytokine. It is secreted by T cells and macrophages to stimulate an immune response to trauma, especially burns and fractures. It is an early marker for fat emboli.

Cerebral fat embolism can cause symptoms 1 to 3 days after the occurrence of the fracture and can ultimately lead to death. Typically, pulmonary fat emboli will cause symptoms 14 to 16 hours after fracturing.

There is a syndrome which you may see referred to and that is Fat Embolism Syndrome (FES). Fat embolism syndrome is distinct from fat emboli. Symptoms usually occur 1 to 3 days after a traumatic injury and are manifested by shortness of breath, hypoxemia, neurological (agitation, delirium, or coma), dermatological (petechial rash), and hematological (anemia and low platelets). FES occurs more frequently in closed fractures of the pelvis or long bones. The petechial rash, which usually resolves in 5 to 7 days is said to be pathognomonic for the syndrome, but only occurs in 20 to 50% of cases.

Petechiae on the back that can be seen in FES
Petechiae on the feet that can be seen in FES
The above is a photograph of a coronal section of the brain at the level of the third ventricle and thalami showing multiple fat emboli represented by petechial hemorrhages in a fatal case of FES.

Multiple petechial hemorrhages involving the kidneys

Fat emboli occur in almost 90% of all patients with severe injuries to bones, although only 10% are symptomatic. The risk of FES is believed to be reduced by early immobilization of fractures, especially by early operative correction. There is also some evidence that steroid prophylaxis of high-risk patients reduces the incidence. The mortality rate of FES is approximately 10 - 20%.

The association between blunt force trauma and venous thromboembolism is well recognized. The reported incidence of venous thromboembolism following blunt force trauma varies between 7 to 58%. In some studies the mortality rate associated with thromboembolism approaches 50%.

That trauma patients are at risk for deep venous thrombosis and pulmonary embolism has been recognized for almost a century. In 1934 J.S. McCartney suggested that there was an association between trauma and death from pulmonary embolism, and that the association was particularly strong in patients with lower extremity fractures. This observation was followed by a number of autopsy studies that not only confirmed the relationship between injury and thromboembolic events, but also suggested that these
events were rarely diagnosed premortem. These studies stimulated the sentinel work by Freeark and others (1967), who demonstrated venous thrombosis by venogram in 35% of patients with fractures. Thrombus formation was observed within 24 hours of injury and involved both the injured and the uninjured extremity. What was most interesting is the majority of these patients were asymptomatic.

The generally accepted risk factors for venous thromboembolism are: a lower extremity fracture, head injury, a need for ventilation greater than 3 days, the need for major operative procedures, an age >40 years, or the presence of a venous injury.
Thromboembolis showing evidence of re-canalization

**Compression:** This is manifested by the continued prolonged application of force to the whole body or a portion thereof. A classic example of compression is that of the chest, such as produced by the victim working beneath a car, which is elevated by jacks with the car slipping off the jacks, thus, coming down on the person. Typically, such compression of the chest leads to traumatic asphyxia. What must be kept in mind is traumatic asphyxia is not always accidental; infants have died as the result of someone placing their hands on the infant's chest, thus, preventing them from inhaling and exhaling. Some adults have been murdered as the result of an assailant sitting on their chest, preventing inspiration and expiration.

**Hemorrhage:** The amount of blood within the circulatory system is related to the size of the person. The average-sized adult male has a blood volume of 5 to 6 quarts of blood (4800 to 5760 cc). A loss of 1/10 of your vascular volume (480 to 576) does not cause any significant difficulty, such as a blood donation (450 to 500 cc) in a normal adult. A loss of up to 15% of your blood volume (720 to 862 cc) can be compensated for by constriction of your vascular bed with maintenance of blood pressure, normal
respiratory rate, normal capillary refill, normal urine output, normal mental status to slight anxiety and pallor of the skin.

A 15% to 30% loss of your blood volume (720 to 1728 cc) in a normal healthy adult leads to the cardiac output no longer being maintained by arterial constriction, tachycardia >100 beats per minute, increase respiratory rate, blood pressure is still maintained, but there is an increase in diastolic pressure with a narrow pulse pressure. There is evidence of sweating from sympathetic stimulation, the victim appears mildly anxious, shows delayed capillary refill and a urine output of 20 to 30 ml/hour. However, in the elderly with evidence of atherosclerotic and/or hypertensive cardiovascular disease, perhaps complicated by a decrease in kidney, respiratory and liver function, such a blood loss can lead to the victims demise.

A 30% to 40% blood volume loss, (1728 to 2304 cc) especially if sudden and not immediately treated can cause death in a normal adult. Clinically these people will show classic signs of hypovolemic shock manifested by systolic blood pressure of 100 mmHg or less, marked tachycardia of >120 beats per minute, marked tachypnea >30 breaths per minute, decreased diastolic blood pressure, alteration in mental status (confusion, anxiety, agitation), sweating with cool, pale skin, delayed capillary refill and a urine output of approximately 20 ml/hour.

A loss of greater than 40% of the persons vascular volume, particularly if acute, even in a normal healthy adult, must be treated immediately within minutes, otherwise death will ensue. If treatment is delayed the person may survive, but with morbidity. The clinical manifestations are extreme tachycardia >140 beats per minute with a weak pulse, pronounced tachypnea, significant decrease in systolic blood pressure of 70 mmHg or less, decreased level of consciousness, lethargy, and coma, the skin is sweaty, cool and very pale (moribund), absent capillary refill and negligible urine output. A pragmatic point to remember is larger people can tolerate slightly more blood loss than smaller people.