TRAUMA: ELEMENTAL LESSIONS. CONTUSION

The term trauma comes from Greek: *“ τραῦμα ”* or the action of wounding and means *“a lesion to the tissue or organs due to a mechanical force”*. Other terms are derived from trauma such as “*traumatized”*: that which suffers trauma and "traumatology". In medicine, this is the study of trauma and its effects.

The mechanical force can be linked to the organism through a solid agent: i.e. a rock, liquid, i.e: a spurt of water; or gaseous: the blast wave after an explosion.

The influence of mechanical forces on cutaneous biology has been examined since 1861 when LANGER first reported the existence of tension lines in the skin of a cadaver. Tissues and cells respond in a wide variety of ways to different mechanical forces: compression, tension, shear stress, osmotic pressure, hydrostatic pressure and gravity. These intrinsic mechanical forces or mechanical stimuli are converted by the cells into electrical, chemical or biochemical signals.

For lesions caused by mechanical force or trauma, the above-mentioned etiological agents i.e.: solids, liquids or gases, transmit mechanical forces extrinsic to the organism combining strong forces of compression, traction and shear stress. These forces have an impact on tissues with kinetic energy, which depends on its mass and speed ($E= ½ m.s^2$).

This energy impact on tissues can cause:

- Morphological alterations and

- Functional alterations.

Morphological alterations__are either deformations or breakage. Deformations are divided into two types. Elastic and Inelastic or Plastic. Elastic deformations are not partial when the force is lifted. Inelastic or plastic deformations are characterized by the tissue deformation when the force is

lifted. Breakage is produced when the resistance of the deformation surpasses the tissue.

In practice, there are two types of tissue morphological alterations found in a trauma.

- Lesion with solution of continuity: CONTUSION, and

- Lesion without solution of continuity: WOUND AND FRACTURE.

Therefore, tissue injury due to mechanical force can produce a contusion (bruise), that is, damage without tissue breakage or damage with tissue breakage. In the later case, if the tissue is soft, the lesion is called a wound, and if the tissue is hard (i.e. mineralized) the lesion is called a fracture (Table 1).

Table 1: Types of trauma Elemental lesions produced by mechanical forces

<table>
<tr><td rowspan="2">Mechanical Energy</td><td rowspan="2">Solid

Liquid

Gas</td><td>Non-rupture tissue</td><td>CONTUSION</td></tr>
<tr><td>Rupture tissue</td><td>WOUND

FRACTURE</td></tr>
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Functional alterations are produced when the mechanical force absorbed by the tissue causes a pathological reduction of vital energy. The degree of tissue vitality after a trauma is either lesser or greater depending on the mechanical force absorbed by the tissue.

Alterations in tissue function are divided into two types:

- Reversible. The tissue recovers homeostasis. The alteration of the function is therefore temporary.

- Irreversible. The tissue suffers a functional alteration from which it doesn't recover. This alteration is associated with cell death and a loss in tissue vitality. The paradigm of this kind of alteration is the lesion with solution of continuity (wound and fracture) in which the tissue is not viable and therefore, needs repair.

The importance of this classification is based on whether there is or isn't a solution of continuity or tissue breakage. This is therefore very important for the surgeon since a wound and fracture normally require immediate anatomical reconstruction.

Three fundamental lesions produced by mechanical force are: contusion, wound and fracture. Likewise, the definition of a wound or fracture is complemented by the degree of contusion suffered by the tissue where the wound or fracture is localized. Therefore, wounds and fractures are usually mixed lesions.

CONTUSION

A contusion is a tissue lesion caused by mechanical force which is a wound where there is no solution of continuity or breakage. A contusion, based on its severity, could be classified in three degrees:

- **First degree, concussion in CNS**. This is characterized by the temporary loss of function. Although it could be associated with pain and mild edema, the dysfunction is reversible, and therefore, full recovery is possible.

- **Second degree, necrobiosis or bruise**. This would occur with ecchymosis, namely with tissue infiltration by red blood cells. The evolution would be ambivalent since cellular and tissue alterations can be reversed or worsened, causing cell death. Thus, the incidence of harmful factors during its evolution, for example, hypoxia and near anoxic environment, favor cell death.

- **Third degree or necrosis**. This is an irreversible lesion since the injury causes cell death mainly by necrosis and the tissue suffers from infarction (Figure 1).

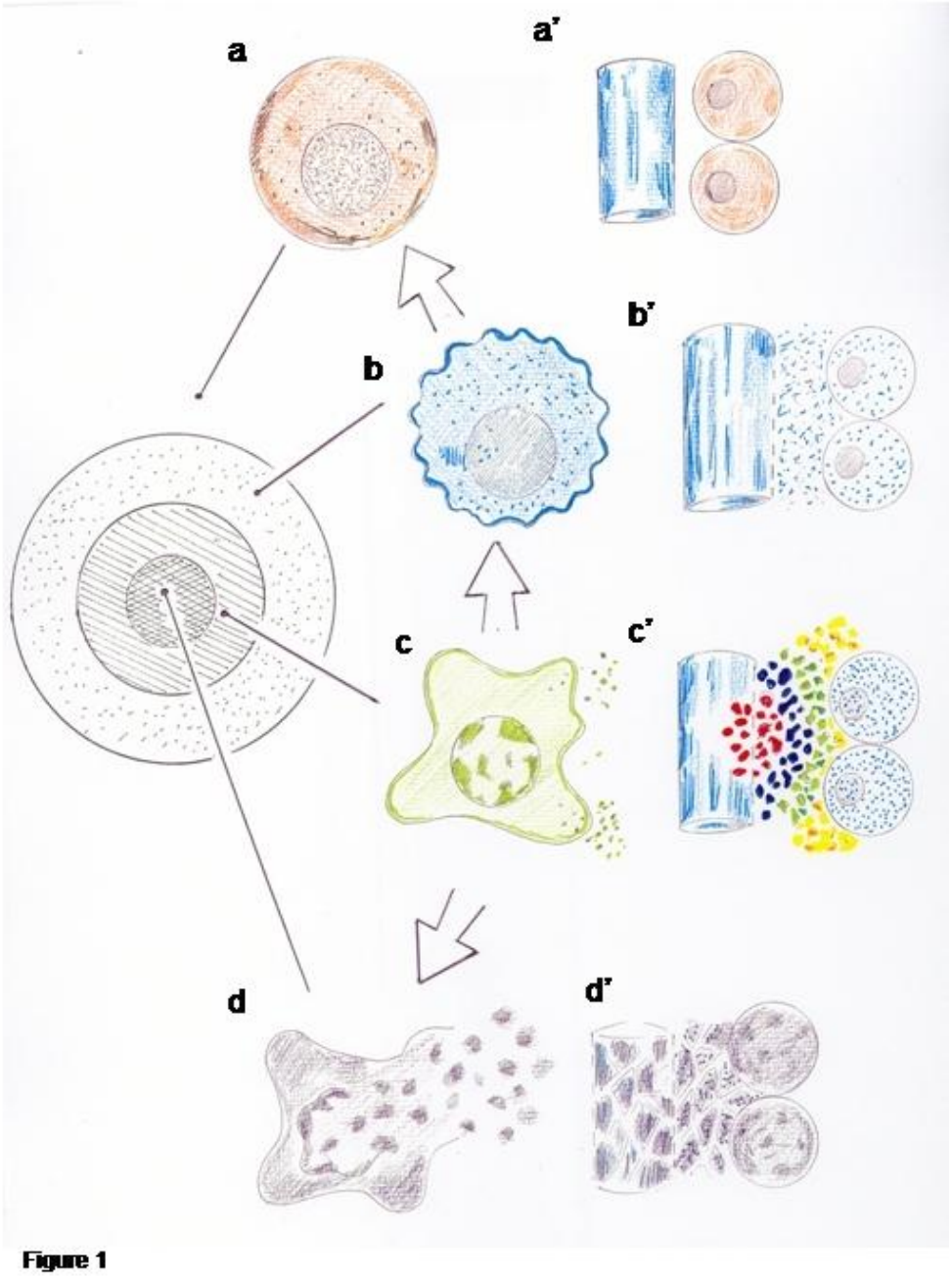

Figure 1. ***Degrees of severity in the contusions***. *Mechanical injury without breakage produced by blunt etiological agents are made up of concentric areas of different degrees of severity. For example, in the third degree contusion, the area of necrosis is surrounded by an area with a second-degree contusion or bruise, which at the same time, surrounds an area with a first-degree contusion or concussion. From the cellular point of view, the first-degree*

contusion is a reversible injury. Alterations consist in the formation of small plasma blebs. In the second-degree contusion, a fusion of the blebs is produced and plasma membrane permeability increases. In the third-degree contusion, cell death is produced by necrosis. From the tissue point of view, edema, namely, swelling of tissue due to excess interstitial fluid, is produced in the first-degree contusion; ecchymosis would be associated with edema in the second-degree contusion; an infarction would be produced in the third-degree contusion.

Tissue alterations

Tissue alterations produced in the contusions correspond to the inflammatory response induced by mechanical aggressions.

In brief, in *the first-degree contusion*, inflammatory pain is caused by extracellular matrix mild damage in which its pathogeny seems to be phase-specific. Thus, after the initial electrical phase with upregulation of ionic channel expression in the nociceptive circuits that causes spontaneous neural firing, the

following would be an immune phase, with cytokines, chemokines and autacoids derived from glial and immune cells, acting as pain mediators and modulators. Lastly, neurotrophic factors, including nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF) and neurotrophins 3 and 4, would be associated with structural neural remodeling. However, analgesia quickly takes place, through the descending inhibition of pain and the key mediators involved are noradrenaline, serotonin and endogen opioids. In this first-degree contusion, the increase of endothelial permeability produces mild interstitial edema and the lymphatic circulation is simultaneously activated.

The second-degree contusion initiates its evolution with inflammatory pain, edema and ecchymosis, also called a bruise, due to its blue color, from the Latin word *"cardinus"* or bluish, which explains its purple color. Ecchymosis means that the red blood cells are the first blood cells to infiltrate the interstitial space post-trauma. The initial dark blue color of the ecchymotic lesion comes from the carboxyhemoglobin, which is the result of the bounding of carbon monoxide to hemoglobin. Then, the release of hemoglobin into the interstitial space is a phenomenon associated with hemolysis. Hemoglobin, released from red blood cells, is the major source of heme for bile pigment.

Hemorrhage-associated macrophages (Mhem) ingest erythrocytes, which are then trafficked to the lysosome. Within the lysosome, heme is released causing *“heme stress”* which is associated with oxidative stress. However, heme stress causes phosphorylation of activating transcription factor 1 (ATF-1) and its translocation to the nucleus. This drives gene transcription and leads to heme disposal into ferritin (heme oxygenase-1 (HO-1)/Fe pathway). Biliverdin (green) and bilirubin (orange and yellowish) are then released to diffuse out the cell.

The microsomal enzyme HO catalyzes the oxidative degradation of free heme, and generates carbon monoxide (CO) ferrous iron (Fe^{2+}) and biliverdin. Two distinct HO isoforms exist: an inducible form, HO-1, and the constitutively expressed HO-2. Accumulating data demonstrates that the HO enzymes execute anti-inflammatory, anti-apoptotic and anti-proliferative functions through the effector molecules generated by heme-catabolism. Biliverdin is almost instantaneously converted into bilirubin by biliverdin reductase, and the fee iron is rapidly scavenged by con-induced ferritin. Bilirubin has a number of new and interesting biochemical and biological properties. In addition to having a protective role against oxidative stress, bilirubin has anti-apoptotic and anti-mutagenic properties. Therefore, the increase in the production of bilirubin in the bruised tissue is considered to have beneficiary effects as an inflammatory modulator. Although CO, Fe^{2+}/ferritin and biliverdin/bilirubin affect different biological processes, including the resolution of inflammation, the executed effects, however, depend on the generated amounts and the microenvironment.

The second-degree contusion is an evolutive lesion, whether it returns to normal or progresses towards cell necrosis.

The third-degree contusion is characterized by the presence of cell necrosis. When a mechanical force acts on the tissue, especially if this occurs through a blunt etiological agent, an abrupt crushing is produced removing blood from the tissue. This bloodless tissue is white. If there is continuous crushing of the tissue, the tissue becomes damaged and this could possibly lead to sphacelation. Thus, in a third-degree contusion, the tissue suffers a crush injury with vasospasm, endothelial damage and thrombosis. In essence,

the formation of “no-reflow phenomenon” is characteristic of the tissue in a third-degree contusion. Therefore, the phenomenon is caused by microvascular spasm, swelling of endothelial cells, increased capillary permeability, interstitial edema, micro-thrombi, neutrophil adhesion and local acidosis.

Necrosis, is largely represented as an accidental form of cell death with simultaneous disruption of multiple pathways. Necrosis is derived from the Greek word “necros” for corpse and is characterized by oncosis, or swelling, and the formation of plasma membrane blebs, which in contrast to the apoptosis blebs, are devoid of organelles. A cellular feature of necrosis is plasma membrane rupture, which occurs during a “metastable stage” resulting in bleb rupture. Until recently, necrosis has often been viewed as an accidental and uncontrolled cell death process. Nevertheless, growing evidence supports the idea that necrotic cell death may also be programmed. The most relevant mechanisms culminating in cell necrosis correspond to:

- Mitochondrial dysfunction and ATP depletion.

- Loss of intracellular ion homeostasis with osmotic swelling.

- Hyperplycolisis and overproduction of lactate.

- Oxidative stress, with excessive formation of reactive oxygen species.

- Sustained increases in intracellular calcium and activation of degradative hydrolases, including proteases, phosphorylases and endonucleases, and

- Degradation of cytoskeletal proteins with disruption of cytoskeletal integrity (Figure 2).

Tissue necrosis is accompanied by complete release of cellular constituents into the extracellular environment, a pathological phenomenon that can elicit a significant inflammatory response. Contusion can be produced by a direct mechanism, when the traumatic biochemical force impacts the contused tissue, or by an indirect mechanism, when the lesion is produced at a distance from where the extrinsic biomechanical force impacts an organism, e.g. rapid acceleration or deceleration or intense changes in pressure as occur in blast exposure.

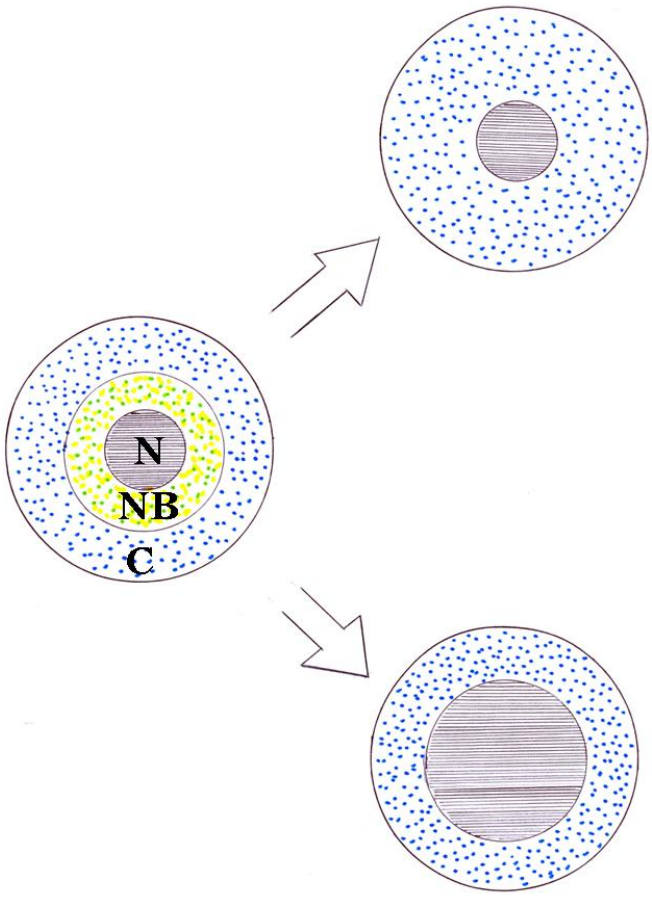

Figure 2. *When kinetic energy from a focal impact is delivered to the surface of the skin, the energy is distributed within the elastic tissue in a three-dimensional gaussian-like distribution, with the epicenter (third-degree contusion or necrosis) receiving the peak energy and surrounding regions receiving progressively less energy with distance both laterally and deeply For heuristic purposes, we divide the three-dimensional continuum from the epicenter outward into three distinct regions: - Third-degree contusion or necrosis; - Second-degree contusion or necrobrosis, and- First-degree contusion, shock, shocked tissue or concussion (i.e. traumatic brain injury). In all three regions, enough kinetic energy is deposited to have important biological consequences, whether reversible or irreversible. In the second-degree contusion area, the amount of energy deposited is not enough to produce immediate necrosis, but is enough to activate mechanosensitive molecular processes, related with inflammation, particularly NF-κB and AP-1, thereby initiating a series of events that later will lead to the delayed necrosis or hemorrhage. However, the mechanosensitive molecular processes initially activated could possibly revert, in which case this necrobiotic area would return to normal. Ischemia is a main factor that causes, in the post-injury period, the second-degree contusion to evolve progressively to secondary necrosis or hemorrhage.*

Diagnosis

* Signs and symptoms.

* The contused tissue has no structural pathology and will therefore not have any abnormality on a CAT scan or magnetic resonance imaging (MRI).

* Diagnosis testing must value the degree of tissue dysfunction and metabolic damage or the degree of viability of the injured tissue.

Treatment

* Posture treatment. Reducing edema favors vascularization and reduces ischemia.

* Pain during the acute symptomatic period can be treated with analgesics, such as acetaminophen and non-steroideal anti-inflammatory drugs (NSAIDs).

Aspirin should be avoided due to theoretical concerns of increased bleeding risk.

* Debridement or resection of devitalized tissue.

Complications

* Injury from secondary ischemia from edema or damaged and friable capillaries.

* Infection from necrotic tissue.

* Intraparenchymal hemorrhagic evolution.

* Post-contusion syndrome. Long-term persistence of signs and symptoms.

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