SPINE DAMAGE IN THE VERTEBRAL TRAUMA

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Cuvinte cheie: *contuzie, compresie, necroză ischemică*

Rezumat: *În traumatisme măduva spinării poate fi afectată fie prin contuzie, fie prin compresiune. În cazul contuziilor medulare, inițial leziunile măduvei sunt de tip hemoragie şi edem, cu localizare centrală, care progresează rapid transversal, prin lezarea barierei hematice. În a doua etapă apare ischemia hemoragică a substanței cenuşii – necroză hemoragică. În cazul leziunilor prin compresiune se pot produce leziuni directe generate de factorul compresiv, dar şi leziuni secundare (la distanță) generate de ischemie.*

Keywords: *contusion, compression, ischemic necrosis*

Abstract: *Traumatic injury of the spinal cord is possible either by contusion or by compression. In spine contusions, the first injuries that appear are hemorrhage and edema in the central cord, with highly progress due to the destruction of the sanguine barrier. The second stage of the contusion includes hemorrhagic ischemia of the grey substance – hemorrhagic necrosis. In spine compression we can find primary injuries due to the compressive factor but also secondary lesions due to ischemia*

SCIENTIFICAL ARTICLE OF THEORETICAL PREDOMINANCE

The lesions involving the spine are very serious because of the neurological sequelae involved. Knowledge of the mechanisms by which the spinal cord injury occur allows us to choose an optimal treatment algorithm to minimize spinal injuries. The spinal cord is housed in the medullary canal; it is encased by the meninges and is surrounded by the dural sac. It spreads at the cranio-cervical junction level (where it continues with the spinal bulb) to the L_1 - L_2 level. It presents two swelling, at C_5 - T_1 level and T_{11} - L_1 level, regions of nerves innervating the upper limb, lower respectively. The spine is covered by the meninges, a cover well differentiated in three layers: piamater, arachnoid and duramater (1).

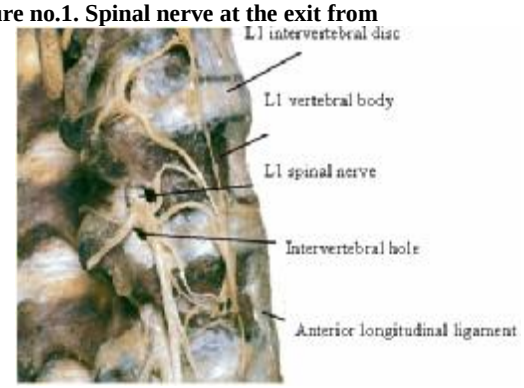
The epidural space is found between bone channel and duramater. Cervical spine epidural space is narrow and contains nerve structures, vascular and connective tissue. In the thoracic region, this space contains blood vessels, nerve roots, spinal ganglia and spinal nerves. In the lumbo-sacral region there are nerves, blood vessels, ligaments and fat to protect the spinal cord while moving.

From the spine channel 31 pairs of cranial nerves goes into the body, and these nerves provide sensitivity and motricity metamerical segmentation, which is useful in the diagnosis of spinal damage location. On the aspect of metamerical distribution of spinal nerves, I will insist extensively in the presentation of spinal cord compression syndromes.

With the exception of C1 and C2 pairs of cranial nerves, all other nerves are leaving the spinal canal through intervertebral hole, each spinal nerve has a previous motor root and a posterior sensory root (see figure 1).

The spinal cord extends up to the second lumbar vertebra. Terminal portion of the spinal level of T_{12} - L_1 is called medullary cone. From the level of L_2 vertebra in the dural sac there are only the lumbo-sacral spinal nerves, included in what is called the "cauda equina" (see fig. 2). At the level of sacral canal, spinal sacral nerve roots form the “filum terminale”.

Figure no.1. Spinal nerve at the exit from



In the cases of spine fractures caused by hiperflexion, spinal cord undergoes a stretching process, which is amortized by the elasticity of the denticulata ligament (2). White substance has a degree of elasticity greater than the gray substance (which is more rigid and more sensitive, having a richer blood microcirculation). In hiperflexion, posterior part of the spinal cord stretches more than the anterior part, which is compressed. Anterior compression may cause direct neural damage or indirect lesions by affecting the intrinsic arterial blood microcirculation.

The longer the time of compression, the higher is the scale and irreversibility of neurological damage. Hence the need for compression agent, regardless of its nature, to be removed within 6 hours after the accident. This can be done by orthopedic or surgical reduction of fractures of the spine in the early hours of the accident. If residual compression after orthopedic treatment persist, surgical treatment of decompression and stabilization of the spine by fixation is required (3).

Higher stiffness of gray matter and richer intrinsic vasculature explains the predominance of central lesions in

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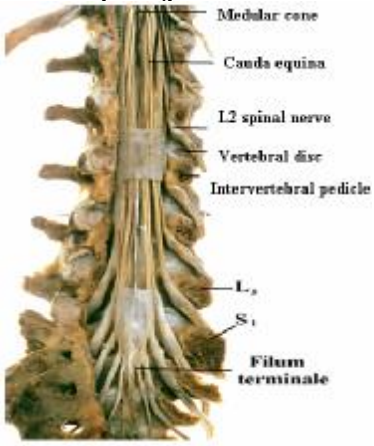
fractures caused by hyperflexion. This means that, in hyperflexion injuries, major changes will be in the centrally located gray matter. In hyperextension injuries, a more or less extended central syndrome appears, or an anterior compression syndrome.

Figure no. 2. Cauda equina (part of the dissection)



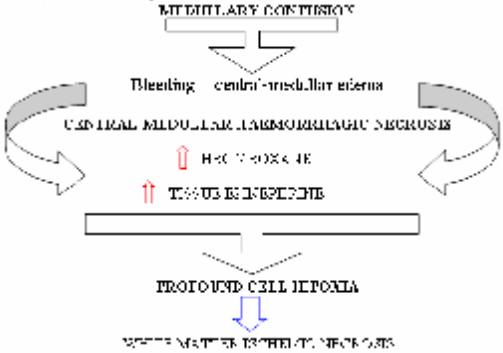
In fractures caused by hyperextension, the spinal cord is compressed between the postero-inferior angle of the superjacent vertebra and the vertebral arch of the superjacent vertebra, the marrow being caught as in a clamp - pinces traumatic mechanism (4). And in this case, the lesions appear centrally, at the level of the gray matter. Note that in order to produce neurological damage in fractures caused by this mechanism, it requires that the lesion to be larger and contain two or three levels or a change in the spinal canal calibre (congenital or acquired stenosis of the spinal canal) to pre-exist.

Basically, the spinal cord may be affected either by contusion or compression.

Myelopathy by contusion

In the first hour after trauma, the spinal cord lesions are haemorrhagic and edemic, and centrally located, progressing rapidly transversally, damaging the hematic barrier. In the second phase, the hemorrhagic ischemia of the gray matter appears - haemorrhagic necrosis. The central- medular hemorrhagic necrosis rapidly progresses transversally and longitudinally. The vasoconstriction installs, reducing blood flow in white matter and gray one, with secondary ischemia and increased thromboxane and tissue epinephrine. Biochemical changes associated to ischemia causes profound cell hypoxia. Two hours after the injury, ischemic necrosis of white matter appears - which is secondary to the first injuries - and this is done by secondary lesion extension, according to the intensity of the initial injury and the possibilities of compensating for the intrinsec blood flow.

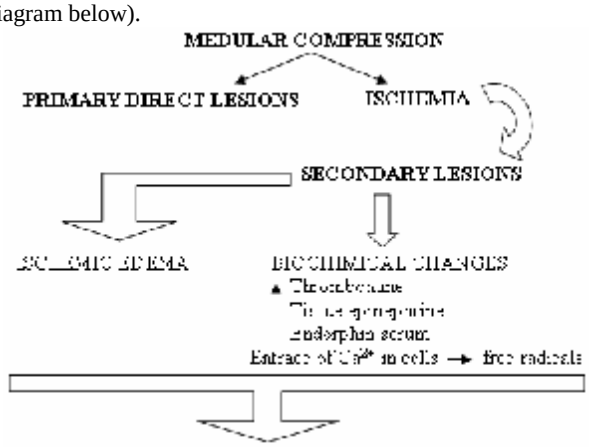
Ischemic lesions in the white matter leads to a poor prognosis (see the diagram below).



Myelopathy by compression

The lesions are triggered by mechanical compression factor, which causes ischemia. At the white matter level, microcirculation lesions (capillary rupture) do not appear, but the aspect of ischemic edema does. This is highlighted in the first hour, and it reaches the climax in the first 8 hours after injury. Removing the compression factor becomes crucial in the first 8 hours after injury, being the main therapeutic element in this case.

The spine is less sensitive to ischemia than the brain. What characterizes the spine is that it is more sensitive to ischemic time taking than its intensity. Local ischemia leads to hypoxia and to the inevitable death of axons, with neuronal depopulation. The damaged cells release leuco-tactical factors (thromboxane) and endogenous amines (epinephrine). In conditions of hypoxia, the level of serum endorphins and vasoactive factors (responsible for the loss of intrinsic self-regulation flow) rises, thus increasing ischemia, hypoxia and neuronal suffering. Basically, through compression, can be produced direct (primary) damage generated by the compression factor, and also secondary lesions (at distance) caused by ischemia. Hypoxia promotes the entry of Ca^{2+} into cells and activates the phospholipases and the chain of reactions that contribute to the production of free radicals. Free radicals are the source of lipid peroxidation, contributing to the destruction of cells, both neurons and capillaries in the spinal cord in the gray and white matter (5). Resulting cellular hypoxia may be exacerbated by systemic hypotension or hypoxemia (see diagram below).



Conclusion: These physio-phatological knowledge are the basis of therapeutic principles in spine trauma: emergency reduction of movements resulting from a spinal injury, detecting and removing any compression factor, regulating and stabilizing blood pressure and stabilizing the enzymatic disorder.

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