Spinal cord tolerance to anteroposterior and lateral compression: experimental study showing differences in electrophysiological response and cord pathological changes

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Background: The avoidance of neurological complications, especially spinal cord injury, is considered one of the most important challenges in spine surgery. The cause of the surgically related neurological complications has been considered multifactorial, comprising different potential factors: cord ischemia, compression by instrumentation, and cord inflection by compression.

Aim: This study was aimed at establishing, by means of neurophysiologic monitoring, the tolerance of the spinal cord to compression (anteroposterior and lateral), and at describing the sequence of changes in the neurophysiologic parameters.

Material and methods: Spinal cord was exposed through a large laminectomy in 13 experimental animals (domestic pigs) with mean weight of 35 kg. Dural sac (T7-T11) was exposed. The width of the dural sac was measured at the level where the compression was going to be performed. A pair of sticks attached to a specifically designed compression device was set up anteroposterior or at both sides of the spinal cord between T8-T9 roots. The sticks were sequentially approximated at a rate of 0.25 mm every 2 minutes to cause progressive cord compression. Epidural catheters were placed cranial and caudal to the compression level, and spinal cord to spinal cord evoked potential (EP), D-wave recordings and somatosensory epidural evoked potential (SEP) were obtained for each approach of the sticks. Once the experiment was completed, the spinal cords were histologically studied to evaluate the neural tissue damage.

Results: The mean width of the dural sac was 7.5 mm. For progressive compression, increasing latency and decreasing amplitude of the evoked potentials were observed after a mean displacement of the sticks of 1.5±1 mm for the motor EP, 1.5±0.7 mm for the cord to cord EP, and 2.5±1.3 for the SEP when provoking an anteroposterior compression; and 2.9±1.1 mm for the motor EP, 2.7±1 mm for the cord to cord EP, and 4.1±1.3 for the SEP when performing the lateral compression. Independently of the direction of the applied compression forces, ischemic lesions were found at the site of cord compression, being more extensive in those cases undergoing anteroposterior compression. Cases with less damage showed axonal lesions expressed by vacuolization and loss of structuration of the myelin sheaths (Fig. 1.a,b). In more hypoxic cases, neurons showed pyknosis and chromatolysis (Fig. 2.a,b).

Conclusions and clinical implication: The spinal cord is more sensitive to anteroposterior compression that to lateral compression. In both cases, cord-to-cord EP and D-wave are the first neurophysiological parameters to detect the injury, whereas the SEPs are less sensitive to compression. D-wave and cord-to-cord EP are equally accurate for the detection of spinal cord damage. These findings have significant relevance to implement the neuromonitoring protocols during spine surgery.