Neurophysiology of cauda equina compression

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In central spinal stenosis there is a gradual narrowing of the spinal canal, leading to reduced space available for the nerve roots of the cauda equina. If the cross dimensions of the spinal canal are reduced beyond critical values, the cauda equina nerve roots will be subjected to mechanical compression, leading to neurogenic claudication. In this presentation, neurophysiological aspects of cauda equina compression in spinal stenosis are summarized.

Anatomy and physiology of spinal nerve roots

Caudal to the level of termination of the spinal cord, the nerve roots form the cauda equina and constitute the anatomical connection between the central and peripheral nervous systems (Figure 1). The cauda equina is located in the cerebrospinal fluid in the subarachnoid space, where there is a strict anatomical pattern of arrangement of the nerve roots from respective spinal segments (Wall et al. 1990) (Figure 2). Laterally, the respective pairs of nerve roots, comprised of a ventral (motor) and a dorsal (sensory) nerve root including the dorsal root ganglion, pass out from the spinal canal through the nerve root canals (Rydevik et al. 1984, Cohen et al. 1990).

The nerve roots of the cauda equina have a microanatomy which is different from that of the laterally located nerve roots in terms of for example connective tissue layers. The nerve roots of the cauda equina have a sparse connective tissue network and they lack diffusion barriers on their surface. The nerve root components in the lateral nerve root canals have a more complex microanatomy with a rich connective tissue stroma including the root sleeve (Rydevik et al. 1984, Olmarker et al. 1992, Rydevik and Holm 1992).

The nerve roots have a vascular supply which comes from both peripheral and central sources (Crock and Yoshizawa 1976, Parke and Watanabe 1985, Olmarker 1991). However, the nerve roots have no regional, segmental blood supply, in distinction to the peripheral nerves (Lundborg 1975). It has been postulated that the cauda equina nerve roots, particularly in their central parts, exhibit a zone of "relative hypovascularity" (Parke et al. 1981). On the other hand, these nerve roots derive some of their nutritional supply via diffusion from the surrounding cerebrospinal fluid. (Rydevik et al. 1990). The surface of the nerve roots in the spinal canal is covered by a thin root sheath, which has been shown to allow an almost free passage of various molecules (Kobayashi et al. 1991, Yoshizawa et al. 1991). The dorsal root ganglion is different from other parts of the nerve root complex, in terms of the microvascular supply which is very rich to the ganglion.

The endoneurium of the spinal nerve roots, which is the compartment in which the nerve fibers are located, has a similar structure to the endoneurium of the

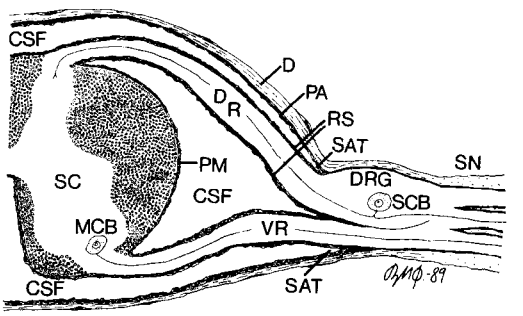

Figure 1. Cross section of a segment of the spinal cord (SC), a ventral (VR) and a dorsal (DR) spinal nerve root. The cell bodies (MCB) of the motor axons, which run in the ventral nerve root, are located in the anterior horn of the gray matter of the spinal cord. The cell bodies (SCB) of the sensory axons, which run in the dorsal nerve root, are located in the dorsal root ganglion (DRG). The ventral and dorsal nerve roots blend just caudal to the dorsal root ganglion, and form the spinal nerve (SN). The spinal cord is covered with the pia mater (PM). This sheath continues out on the spinal nerve roots as the root sheath (RS). The root sheath reflects to the pia-arachnoid (PA) at the subarachnoid triangle (SAT). Together with the dura (D), the pia-arachnoid forms the spinal dura. The spinal cord and the nerve roots are floating freely in the cerebrospinal fluid (CSF) in the subarachnoid space. (From Olmarker K: Spinal Nerve Root Compression. Experimental Studies on Effects of Acute, Graded Compression on Nerve Root Nutrition and Function, With an in Vivo Compression Model of the Porcine Cauda Equina (thesis). Gothenburg University, 1990. Reproduced with permission.)

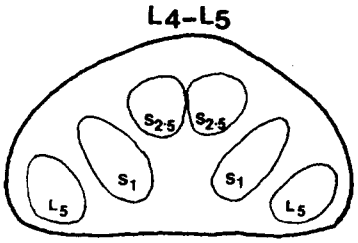

Figure 2. The intrathecal nerve root organization. Schematic presentation of individual roots at the L4-5 cross-sectional disc level showing the L5 root in the anterolateral position. The S1 root is displaced medially forming a diagonal layer (V configuration). The S2-5 roots remain dorsal to the midline. (From Wall E J, Cohen M S, Massie J B, Rydevik B, Garfin S R. Spine 1990;15:1244-1247. Reproduced with permission.)

peripheral nerves (Gamble 1964). However, the amount of total collagen in nerve roots has been determined to be 5 times less than in peripheral nerves, and about 6 times greater than in in the spinal cord (Stodieck et al. 1986). Available data indicate that there are no lymphatic vessels inside the endoneurium of nerve roots and peripheral nerves (Sunderland 1978).

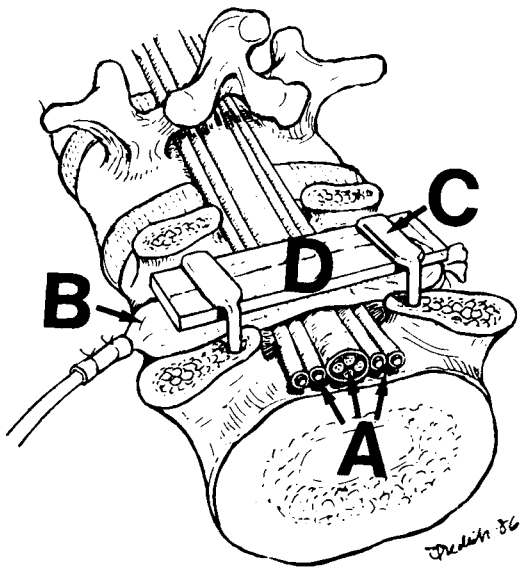

Figure 3. Schematic drawing of experimental model. The cauda equina (A) is compressed by an inflatable balloon (B) that is fixed to the spine by two L-shaped pins (C) and a Plexiglas plate (D). (From Olmarker K, Rydevik B, Holm S. Spine 1989; 14: 579-583. Reproduced with permission.)

Effects of nerve root compression

The pathophysiology of cauda equina nerve root compression has been analyzed experimentally over the last few years (Olmarker 1991, Rydevik et al. 1991, Pedowitz et al. 1992). In a porcine model, graded compression of the cauda equina is induced by an inflatable balloon which is secured to the lower sacro-coccygeal spine after one ore two level laminectomy (Figure 3). Using this model, Olmarker and colleagues have shown that impairment of venular blood flow in terms of congestion in the microcirculation was induced already at pressures as low as 10 mmHg (Olmarker et al. 1989a). Nutritional impairment of the nerve roots in terms of reduced solute transport, studied by 3H-labeled methyl glucose, showed that there was an overall reduction of the nutrition by about 55% at 50 mm Hg compression (Olmarker et al. 1990). Such impairment of nutrition might be related to the formation of intraneural edema (Olmarker et al. 1989b). Nerve root inpulse propagation has also been evaluated in the same experimental cauda equina compression model. During a 2 hour compression period, a critical pressure level for impairment of impulse conduction is present in the range 50–75 mmHg compression. Higher pressure levels (100–200 mmHg) may induce a total conduction block with varying degrees of recovery following compression release. It has also been shown, in this model, that the sensory fibers seem to be slightly more susceptible to compression than the motor fibers (Rydevik et al. 1991, Pedowitz et al. 1992).

The above mentioned functional changes induced by controlled graded cauda equina compression, are also dependent on factors such as the systemic blood pressure. The threshold for impulse propagation impairment is lowered by experimental hypotension and elevated by hypertension (Garfin et al. 1990, Lind et al. 1992). These observations indicate the pathophysiological significance of an adequate blood supply to the nerve roots in conjunction with various compression disorders.

Double level nerve root compression

It has been shown that central spinal stenosis often is present at more than one spinal level at the same time (Porter and Ward 1992). A combination of central and lateral spinal stenosis is also commonly seen in patients with symptomatic nerve root compression. The pathophysiological effects of double level cauda equina compression has been investigated by a modification of the currently presented porcine cauda equina model, by placing 2 balloons at two adjacent disc levels, which resulted in a 10-mm uncompressed nerve segment between the balloons. In such an experimental set-up much more pronounced impairment of nerve

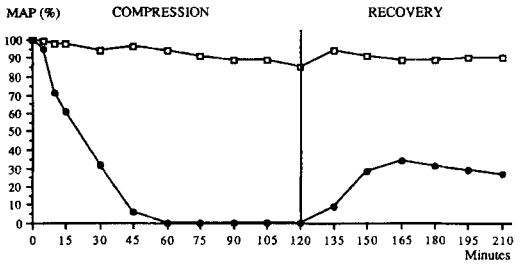

Figure 4. Mean muscle action potential (MAP) amplitude in tail muscles during compression at 50 mm Hg and recovery, with single (□) and double level (●) cauda equina compression. Note that there are much more pronounced effects of double level compression than single level compression. (From Olmarker K and Rydevik B: Clin Orthop 279:35-39, 1992. Reproduced with permission.)

impulse conduction was induced than previously found at corresponding compression levels with single level compression (Olmarker and Rydevik 1992). For instance, a pressure of 50 mmHg in 2 balloons induced a block of nerve impulse amplitude after 60 minutes of compression, whereas 50 mmHg in one balloon showed no reduction (Figure 4). The mechanism behind such functional changes induced by double level compression may be based on the particular local vascular anatomy of the nerve roots. Unlike peripheral nerves, there are no regional nutritive arteries from surrounding structures to the intraneural vascular system in spinal nerve roots (Lundborg 1975, Olmarker et al. 1991, Parke and Watanabe 1985). Compression at 2 levels might therefore induce a nutritionally impaired region between the 2 compression sites. Experimental evaluation of this hypothesis showed that the blood flow in the uncompressed nerve root segment located between 2 compression balloons was reduced by 64% when both balloons were inflated to 10 mmHg (Takahashi et al. 1993).

The overall conclusion from the experimental studies on double level nerve root compression, as related to clinical observations in spinal stenosis patients, is that double level compression induces much more pronounced changes in nerve root nutrition and function than single level compression at corresponding pressure levels.

Chronic nerve root injury and pain mechanisms

Long-standing compression of spinal nerve roots is likely to induce intraneural edema which may be transferred into fibrotic scar tissue within and around the nerve roots (Rydevik et al. 1984, Rydevik and Holm 1992). The thin nerve root sheath on the surface of the cauda equina nerve roots may become thickened, leading to impaired passage of nutrients from the cerebrospinal fluid to the nerve root tissue (Olmarker et al. 1992). In case of central spinal stenosis, the free passage of cerebrospinal fluid at the level of stenosis may also be significantly reduced due to the reduction of the dimensions of the dural sac. The nerve fibers can react to long standing compression by local demyelination and/or axonal degeneration. Such structural nerve fiber injury can, together with the nutritional impairment, lead to ectopic impulse generation (Rydevik et al. 1984) which is likely to be related to pain production in such disorders.

Experimental–clinical correlation

Critical dimensions for the dural sac in the lumbar spine as well as pressure levels acting on the cauda equina nerve roots have been determined by Schönström et al. (1984, 1985). In these experiments, it was demonstrated that the minimal space necessary for the neural elements of the cauda equina, including the dural sac was on an average 77 ± 13 mm^2 at the L3 level. This cross sectional area of the dural sac corresponds to approximately 44% of the normal cross sectional area at this level and was called "the critical size," which means the area at which the pressure starts to build up among the nerve roots of the cauda equina with compression. With further constriction of the dural sac down to 63 ± 13 mm^2 (= about 36% of normal), the pressure among the nerve roots was elevated to 50 mmHg. Constriction of the dural sac to 57 ± 11 mm^2 (=32% of normal) the corresponding pressure level increased to 100 mmHg. However, there was a gradual decrease of this recorded pressure, which seemed to be related to adaptive creep phenomena in the nerve roots. The cross sectional area at which acute pressure increase among the nerve roots is induced, corresponds to the clinically measured cross sectional area in patients with central spinal stenosis (Schönström et al. 1985). However, it should be noticed that this situation is dynamic and there are certain changes in the cross sectional area of the dural sac with for example flexion-extension and axial compression-distraction.

Conclusion

Studies on experimental cauda equina compression have shown that well defined changes in spinal nerve root blood flow, nutritional supply via diffusion from the cerebrospinal fluid and impulse conduction occur at pressure levels which correlate to pressures which are likely to act on the cauda equina in central spinal

stenosis. Double or multiple nerve root compression seems to be of special pathophysiological and clinical significance in spinal stenosis. Radiating nerve root pain is likely to be based on structural changes in nerve fibers such as demyelination, including ischemic factors and other nutritional deterioration.

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