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Topographic relations between the neural and ligamentous structures of the lumbosacral junction: in-vitro investigation

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Abstract Transient or persistent alterations in the L5 lumbar nerve root have been described as a complication after the reduction of spondylolistheses. In a retrospective analysis of our own patients, we observed a higher incidence of transient motor and sensomotor deficits after single-sitting anatomic correction of Mayerding grade IV anterolistheses and spondyloptoses. These findings are consistent with those of other authors. The deficits pertained to muscles in the innervation range of the L5 nerve root, without there being any evidence of intradural root damage or nerve compression. In vitro studies have shown distraction and translation of the nerve root to be pathogenetically relevant mechanisms, leading to damage during intraoperative reduction. Additional alteration of neuronal structures may be caused by ventral parts of the iliolumbar ligament complex during the reduction maneuver. In order to reveal extradural constrictions of the L5 nerve root that might predispose towards intra- or perioperative damage, we examined anatomic specimens to determine the course and neighboring relationships of the lumbosacral plexus, especially in relation to pelvivertebrogenic ligamentous connections. In addition to the

morphologic considerations, we conducted translation tests, which were designed to simulate changes in shape and size of the epineural layer, as well as in vitro measurements of the resulting pressure on the nerve. In addition to a range of variations in the attachment of the iliolumbar ligament complex, which was always located dorsally to the nerve roots, we found a ligamentous connection formed by connective tissue between the sacrum and the fifth lumbar vertebral body on the caudal margin of the ligament apparatus in 14 out of 30 specimens. Its course was constantly ventral to the L5 nerve root, which was also adherent to the periosteum of the sacrum distal to this constriction in one-fifth of the specimens. The average pressure exerted on the nerve root during the distraction and translation process was over 30 mmHg in the area of this lumbosacral ligamentous connection, at a distance of greater than 20 mm. When the translation distance was further increased, perineural fatty tissue was discharged, due to increasing perineural pressure.

Keywords Spondylolisthesis · Spinal fusion · Spinal nerve roots · Lumbosacral region · Nerve compression syndromes

Introduction

Peripheral neurologic deficits have been described by several authors [7, 33, 39] as a complication after the surgical reduction and fusion of unstable segments of the lumbar spine with spondylolisthesis. Lehmer and co-workers [18] pointed to the increased vulnerability of the L5 nerve root. Several retrospective studies have postulated a correlation between the incidence of such complications and the intraoperative reduction distance [5]. Transient neurologic deficits are reported by Freeman and Donati [8] and Harris and Weinstein [11] as having an incidence of over 50% after the anatomic repositioning of the articulation path of grade IV spondylolisthesis or spondyloptosis. In contrast, with a smaller ventral displacement of less than half the width of a vertebral body or in-situ fusion of the segment, the rate of such complications is much lower, at around 20% [15]. However, intradural nerve damage has not been demonstrated either using imaging procedures or in sporadically performed revision surgery. For this reason, Noack and Kirgis [24] suspected an extraforaminal constriction of the nerve root caused by parts of the iliolumbar ligament. While numerous investigations have been conducted on intradural variations of the lumbar nerve roots and their neighboring relationships [12, 16, 17, 22, 32], there are hardly any clinical or anatomic studies concerning variations in their extradural course.

Clinical aspects

In the period 1990–1995, 64 primary monosegmental interventions with anatomic reduction and single-sitting, ventral, intercorporeal fusion of the L5/S1 level were performed on young adult patients (aged 18–35 years) in our department, due to spondylolisthesis vera or spondyloptosis. All patients received dorsal transpedicular fixation (Fixateur interne) combined with ventral intercorporeal fusion using tricortical iliac crest bone grafts. In eight patients, after initially normal findings, we observed motor or combined sensorimotor deficits within the first 72 h postoperatively, which were all attributable to a lesion of the L5 nerve root. The relative incidence increased significantly ($P < 0.001$) in relation to the preoperative baseline findings. There were also striking quantitative differences. The two pareses observed in patients with Mayerding grade I or II baseline findings were limited unilaterally and, in the assessment of the extensor hallucis longus muscle, achieved a score of at worst 3/5 according to Daniels and Worthingham [6]. In both cases, they were completely reversed within 12 weeks. After surgery on higher-grade malpositions (Mayerding grade IV and spondyloptosis), two unilateral and four bilateral pareses occurred, of which three were clinically more pronounced than 3/5 according to Daniels (minimal 0/5). During the acute traumatic phase, a drop in nerve conduction velocity

in combination with increased latency and a higher incidence of polyphasic action potentials in the L5-innervated muscles was demonstrated in all cases by electromyography and neurography [14], which would be consistent with both neurocontusion and axonotmesis [40].

Objective and clinical relevance

The objective of this study was to investigate the course of the L5 nerve root distal to the neuroforamen with regard to possible compression points. Of particular interest was the iliolumbar ligament, as well as the ligamentous tissue connections between the fifth lumbar vertebral body and the sacrum, which are to be interpreted partly as belonging to the iliolumbar ligament and partly as independent structures [2, 19, 27]. Rydevik and co-workers [38] distinguished mechanical deformation, impairment of the microcirculation and the formation of an intraneural edema as pathophysiologic causes of peripheral nerve lesions. The latter part of the present study focused on determining whether and to what extent, on non-fixed cadavers, the pressure and traction forces exerted on the L5 nerve root during the distraction and translation process are liable to cause such lesions.

Materials and methods

In the first part of this study, we conducted a topographic anatomic investigation of the variation of lumbosacral transition on formalin-mounted section blocks from 15 randomly selected cadavers, aged between 57 and 76 years. These were obtained in the frozen state and were limited proximally by the intervertebral space L2/3,

Fig. 1 Preparation drawing. Ventral preparation of the lumbar vertebral body blocks, indicating the iliac vessels as reference points. Measurement markings are shown in the frontal and sagittal plane, with the anterior edge of the promontory as a reference point

Fig. 2 Course variations of the iliolumbar ligament and lumbosacral ligament complex. The difference relevant to a consideration of the repositioning process is the positional relationship to the L5 nerve root. While the iliolumbar ligament (rows 1–4) is always located dorsally to the root, the lumbosacral ligament runs ventrally to this nerve root (row 5)

distally by the femoral necks and laterally 20 mm either side of the iliosacral joints. The two sides of 15 spinal blocks were each examined separately, producing 30 specimens for measurement. In order to describe the course of the ligamentous structures accord-

Fig. 3 Insertion variations of the pelvivertebragenic bands (circles iliolumbar ligament complex, triangles lumbosacral ligament complex)

ing to the course of the L5 nerve root, measurements were performed on the basis of two lines drawn along the frontal and sagittal planes. The reference point was the ventral limit of the promontory at the level of the terminal line of the pelvis (Fig. 1).

In a second part, distraction and translation experiments and pressure measurements were conducted on unmounted (non-fixed) section blocks from four randomly selected cadavers. Since specimens with anterolisthesis were not available, we used lumbosacral transitions with a physiologic form in accordance with the experimental design of Petraco et al. [30]. The test method selected was an adaptation of that described by Hedtmann [13] for mechanical measurements of the motor segments. The L5/S1 motor segment was neutralized by ventral incision of the anterior longitudinal ligament and the fibrous ring and removal of the intervertebral disk and dorsal ligament apparatus. In order to ensure that mobility was as unimpaired as possible during the repositioning process, the dorsal parts of the vertebral bodies were also partially removed. Long-threaded screws were inserted through the pedicles of the L5 and S1 vertebral bodies, which were then connected with internal fixator systems according to Dick. The distraction and translation distances were determined in relation to the ventral limit of the promontory, as in the topographic part of the study. In order to determine the pressure exerted on the nerve root, a measuring catheter was placed between the perineurium and the pelvivertebragenic or sacrovertebragenic ligament apparatus and connected to a continuously operating monitor (Stryker). Four repositioning maneuvers were performed per spinal block side. The endpoint of the test was defined as the achievement of a translation equal to half the width of the vertebral body, corresponding to a mean repositioning distance of 20 mm. In order to simulate greater distances, the pedicle screws were then reinserted using polymethylmethacrylate bone cement (PMMA-Pallacos) reinforcement, and the test was repeated until the system gave way.

Results

In only three specimens (10%) did the iliolumbar ligament present as a morphologically clearly definable, single-component structure. In the majority of cases, the ligament appeared as a spreading, connective tissue complex, which could be differentiated into two (9/30), three (14/30) or four (4/30) main bands (Fig. 2). While the iliac attachment of the ligament complex was consistently located on the ventral or cranial surface of the iliac bone, the total of 79 insertion points on the spine varied considerably. Most often, these were the lateral surfaces (21/79)

Fig.4 **A** Photographic and schematic images showing the topography of the connection of the bands of iliolumbar (IL.L.) and lumbosacral (LS.L.) ligaments between the fifth lumbar vertebral body and the sacrum and positional relationship to the dorsal course of the L5 nerve root. **B** Photographic and schematic images of the IL.L. complex, the LS.L. complex, and nerve roots LIV/1, LIV/2, LV, and LIII, showing adhesion (Ad) of the L5 nerve root above the terminal line of the pelvis

and the transverse processes (33/79) of the fifth lumbar vertebral body and, less often, the fourth (15/79) and third (3/79) lumbar and the first sacral vertebral body (7/79) (Fig. 3). Topographically independently of the iliolumbar ligament complex, 14 out of 30 of the specimens (46%)

had a connection between the ventral surface of the fifth lumbar vertebral body and the sacrum (Fig. 4A). The positional relationship towards the L5 nerve root can be seen as a major morphologic difference compared with the iliolumbar ligament complex. While the latter was located

Fig.5 Correlation between the repositioning distance and subligamentous pressure ($n=32$ tests)

dorsally to the lumbar plexus, and only extended up to the L5 neuroforamen in three specimens, the lumbosacral ligamentous tissue connection of the L5 nerve root was always located ventrally (Fig.4A). This consisted of one (9/14) or two (5/14) bands. The root itself was traced from its connection with the caudal part of the L4 nerve root to the lumbosacral trunk, and beyond the terminal line into the small pelvis. A striking feature in 6 out of 30 specimens were adhesions between the perineurium of the nerve root and the periosteum of the sacrum. These were located between the caudal margin of the pelvivertebragenic ligaments and the point of insertion of the nerve root IV/2, and were not manually detachable

(Fig.4B). Eight tests were conducted up to a mean distance of 19 mm (range: 16–24 mm). After additional fixation of the screws with bone cement, the maximum translation distance was increased up to 29 mm. The subligamentous pressure exerted on the L5 nerve root remained consistently below 20 mmHg in all tests up to a displacement distance of 15 mm. When the mean distance was extended to 20 mm, the pressure increased to an average of 28 mmHg. Pressures of over 30 mmHg were measured in all translations of greater than 22 mm (Fig.5). Peridural fatty tissue was discharged below the lumbosacral connective tissue connection at a pressure level of 35 mmHg (Fig. 6). If the dorsal movement of the fifth lumbar vertebral body was continued, macroscopic deformities of the nerve became visible.

Discussion

Analysis of the course of the iliolumbar ligament revealed a greater variability than that described in the literature [34, 45], especially with regard to the width and proximal extent of the ligament complex [31]. However, a causal significance regarding clinically relevant root compression can be ruled out for the lower, lumbar nerve roots. For one, in few individuals does the ligament complex extend far enough distally to affect the course of the root, although its dorsal localization would appear more important. In the correction of an anterior vertebral displace-

Fig.6 Discharge of perineural fat (arrow) in a partially fixed specimen (repositioning distance 29 mm, pressure 39 mmHg)

ment, this results in either a uniform movement of the nerve root and iliolumbar ligament complex, or an increase in the distance between the two structures, but not a reduction.

The presence of a ligamentous tissue connection between the fifth lumbar vertebral body and the sacrum should be considered a pathogenetically relevant finding. This structure has been described in several publications [2, 10, 11, 19, 23, 27, 44] under the term “lumbosacral ligament” or “lumbosacral junction”. Leong et al. [19] consider its main biomechanical function to be the passive stabilization of lumbosacral transition. Based on mechanical investigations, Chow et al. [4] demonstrated a stabilization effect on flexion, extension, axial rotation and lateral bending movements. According to the observations of Briggs and Chandraraj [2], in most cases the band runs from the transverse process of the fifth lumbar vertebral body to the promontory. In contrast, the attachment to the anterolateral part of the vertebral body, which we observed more often, is also described as the most common variation by Bullough and Boachie Adjei [3]. There is also controversy regarding the origin and assignment of this band connection. The majority of authors [2, 19, 27] assume the presence of a constant structure, which is to be assigned to the iliolumbar ligament, and is located ventrally to the L5 nerve root only as a course variation in one-third of cases. This incidence is consistent with our observations. On the basis of embryologic investigations, Uthoff [44] demonstrated that the band connection is already present in the fetus, and concluded that it is an inconstant structure that is independent of the iliolumbar ligament. The clinical relevance of this connection as a possible instigator of nerve root irritation has thus far only been investigated by Briggs and Chandraraj [2] with regard to chronic compression. On 9 out of 65 cadavers, they found signs of pressure damage on the L5 nerve root, and especially an increased fibrosis of the epi- and perineurium on the side of the root facing the ventral ligament band. In investigating postreduction damage, individuals with this course variation are of primary interest to us, since dorsal displacement of the fifth lumbar vertebral body against the fixed sacrum can press the nerve against its bony bed. Furthermore, the periosteal adhesion of the L5 nerve root itself, observed in several specimens, appears to be of considerable pathophysiologic importance. If one assumes an intradural course of the lumbar nerve roots of between 60 mm (L1) and 170 mm (S1), the free displacement distance of the corresponding axons described by Breig and Marions [1] is reduced by fixation to a total length of well below 300 mm. Lundborg and Rydevik [20] demonstrated in animal experiments that the extension of a peripheral nerve by as little as 8% already results in impairments of intraneuronal microcirculation. When the length was increased by more than 15%, the authors observed a complete cessation of blood flow. This result is consistent with the observations of Dyck et al.

(cited in Rydevik et al. [44]), who observed a loss of conductivity within a spinal nerve at an extension of over 15% of the original length. This difference is significant, since an 8% strain tolerance on a distance of 200 mm would produce a capacity of only 16 mm. Compared with the idea of free axonal mobility, this results in a considerable injury potential. In practice, a distraction of more than 16 mm is improbable, even in the correction of high-grade malpositions. Distraction injury can probably therefore be ruled out as a sole explanation of the pareses observed.

For a discussion of the repositioning tests and their transferability to in vivo scenarios, the following limitations apply:

1. In vitro investigations must be considered an imperfect method with regard to the pathomechanism on which the nerve damage is based. All of the clinical pareses we observed occurred after symptom-free intervals. The experimental design presented cannot reproduce the pathophysiologic chain, but can only describe the factors that may lead to this chain.
2. Our investigations were conducted on cadavers with a physiologic spinal form. The question of whether and to what extent the topographically described connective tissue connections are altered if a spondylolisthesis is present must therefore remain unanswered.
3. A final limitation is that we were unsuccessful in simulating repositioning by a complete vertebral body width, equivalent to grade IV spondylolisthesis, using the experimental design described. The mechanical properties of the motor segment we removed certainly also play a role in in vivo repositioning and the description of the forces that occur. The approximately linear connection between the repositioning distance and pressure is, therefore, not necessarily to be understood as a model that is valid in vivo.

In summary, the pressure exerted on the nerve root is consistently over 30 mmHg in the area of the lumbosacral connective tissue connection once a repositioning distance of between 22 and 25 mm is achieved, and increases further if the dorsal movement is continued. Petraco et al. [30] simulated the extension strain on the L5 nerve root under repositioning conditions in vitro using synthetic cord on cadaver segments after the removal of all soft tissues. Upon dorsal displacement by less than half a vertebral body width from the physiologic form of the lumbosacral transition, they only observed small tractional forces, leading to extension of the synthetic cords by less than 5 mm. This was also largely independent of other variable factors like sacral tilting or the degree of lordosis. When the simulation was continued, there was an exponential increase in the tractional forces, especially in the final range of the reposition, equivalent to an articulation path of more than 75% of the vertebral body width. The fine anatomic structure of the nerve root is decisive for the

Fig. 7 Schematic presentation of the pathophysiologic mechanism discussed, which leads to damage to the L5 nerve root. It is characterized by a combination of traction (*arrows*) as a result of distraction accompanied by fixation (FX.) of the nerve root and pressure from the ventrally running lumbo-sacral ligament (L.S.). The combined effect is to exert an adhesion-related limitation on the nerve's evasive path. By taking into account posttraumatic edema formation with consecutive impairment of microcirculation, the model is consistent with clinically observable latencies between the end of surgery and the occurrence of clinically manifest damage

extent and the course of its vulnerability. The individual fascicles are embedded in the epineurium and surrounded by the perineurium. In addition to its protective mechanical function, the high degree of vascularization of this mantle contributes to the metabolism of the peripheral nerve [29, 41, 42]. Several authors [9, 25] have described deformations of the nerve fascicle and their occurrence in the range of an exerted pressure of 30–50 mmHg, which is consistent with our observations. Forces acting directly on the nerve surface lead to more pronounced displacement than indirectly exerted forces [26]. Ochoa [25]

found a deviation of the nerve fibers, particularly in the marginal areas of the compression. After animal experimental induction, as an electron microscopic correlate of these deformations, he described a focal displacement of the nodes of Ranvier. Physiologically, these nodes were striking as zones of segmental demyelination, with a functionally resulting local conduction block. Rydevik investigated pressure-related changes in intraneuronal blood flow in vivo on the tibial nerve of the rabbit. Venous stasis occurred above a pressure of 20–30 mmHg, and the microcirculation ceased completely above a pressure level

of 60–80 mmHg [35, 36, 37]. While endoneural vessels display a barrier function under physiologic conditions similar to that of the blood-brain barrier, epineural vessels allow the diffusion of proteins and macromolecules [28]. As a reaction to pressure, they react with elevated permeability and edema formation [28, 34]. As an indirect effect of vascular compression, the impairment of axonal transport may be due to the edema-related increase in endoneural fluid pressure [20]. An equivalent limitation of flow was induced experimentally [21] at pressures of 30 mmHg.

Conclusion

In conclusion, it can be expected that a pressure of more than 30 mmHg exerted directly on a nerve root will damage it mechanically and by impairing its metabolic function. Our findings suggest that this pressure can be achieved or exceeded by a combination of distraction and subsequent dorsal displacement of the fifth lumbar vertebral body by more than 20 mm, if certain anatomic factors are also present. The individual risk of damage, or its degree, may be dependent upon the presence of variables like a connective tissue fixation or crossover of the nerve root and increases in relation to the displacement distance (Fig. 7). Damage-inducing factors present in individual cases may be potentiated by an increase in intraneuronal

tension. At the same time, as regards the integrity of the axons, an injury at a specific point can impair structure and function at other points [43]. It is doubtful whether a causal elimination of the structures described in the sense of preventive neurolysis would be practicable, due to their position directly below the iliac vessels and their bifurcation. A possible surgical consequence may therefore be a limitation of the repositioning distance to a maximum of 20 mm, possibly leaving a low-grade malposition, or in situ fusion. If the goal of surgery remains the anatomic correction of dorsal alignment, this consideration implies reduction in several sittings. Resection of the fifth lumbar vertebral body should be stressed as a possible surgical solution if spondyloptosis is present. By removing the anterior and, especially, antero-lateral parts of the vertebral body, the proximal origin of the lumbosacral ligament complex is eliminated, and thus a pressure-related nerve lesion avoided. Another advantage of this procedure is the considerable reduction of the translation distance.

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