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Adhesions of the Ventral Lumbar Dura
An Adjunct Source of Discogenic Pain?

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An analysis of the frequencies and locations of lumbar ventral dural adhesions was undertaken to ascertain their possible role in enhancing the low-back pain consequent to lumbar disc herniation. Data derived from a previous investigation, conducted for a different purpose, were incorporated with that of the presented study to provide a more extensive statistical base. Adhesions judged sufficient to possibly cause additional nerve disruption on separation were found in 16% of the specimens at the L3–4 level; 40% at the L4–5 level; and 36% at the L5–S1 level. Dural tissue segments, taken from the forcefully separated more firmly fixed adhesions found only at the L4–5 level, microscopically showed disruption of neurovascular bundles containing branches of the sinuvertebral nerve where they coursed between the adherent dura and the posterior longitudinal ligament. It was assumed that forced separation of adhesions during disc protrusion could add to perceived discomfort. [Key words: lumbar dura, adhesions, discogenic pain, posterior longitudinal ligament]

DURING THE ROUTINE REMOVAL of the spinal cords and their entire dural coverings from anatomic cadavera, it was noted that the ventral surface of the dura often was adherent to the opposing structures in the lower lumbar spinal canal. As these fixations were distinct from the expected oblique attachments provided by the ligaments of Hofmann[11] a series of specimens were subsequently dissected to determine the true incidence and locations of these adhesions and their possible role as an adjunct source of low-back pain when disrupted by the protrusion or extrusion of a herniated nucleus pulposus.

MATERIALS AND METHODS

Fifteen randomly selected (nine female; six male) cadavers ranging from 62 to 88 years of age were dissected for detailed observations of the lumbar dural adhesions. In nine of these, all of the laminas were removed from the dorsolumbar and first two sacral segments. The dura was opened middorsally and the lower spinal cord and nerve roots were removed, after which the dura was carefully elevated in a cranial to caudal direction by blunt dissection, except where a scalpel was used to section the thecal extensions at the intervertebral foramens and in the cases where the ventral dura was firmly fixed. The locations and degrees of adhesion were observed and recorded. For six specimens, the entire lumbosacral column was removed *en bloc*, frozen, and sectioned longitudinally on a band saw. The lateral aspects of these specimens then were fixed to a board with the sectioned surface parallel to the horizontal plane. Silk sutures were attached to the cut free edge of the ventral dura, drawn dorsally in the same plane as the sectioned surface and passed over a metal rod, where they were pulled vertically by attached 60-g weights. This arrangement provided a better observation of the dural relations to the ventral spinal canal, and the more consistent traction of the sutures better illustrated the relative degrees of the dural fixations, as shown in Figure 1.

Sections of the ventral dura from the sites of the firm adhesions were removed, refixed in 10% formalin, stained in Sudan Black, and then cleared in a solution of 11 parts tricresyl phosphate and two parts tributyl phosphate for transillumination and microscopic examination.

After the above procedures were completed, the literature search uncovered a previous study of these same adhesions by Blikra[1] in Oslo in 1969. He had attempted to determine the causal factors involved in his two cases of intrathecal extrusion of the nucleus pulposus. Because his observations were derived from 40 autopsy specimens, it was decided to pool our data with his series, and thus provide a broader statistical base, from which more accurate percentages could be determined. As the noted degrees of fixation were primarily based on a subjective estimation in both sets of data, additional benefits of this approach would be the diminution of the effects of individual bias, as well as reducing the possibility that one of the series may reflect some geographic or ethnic peculiarity.

RESULTS

Considering the relatively small number of cases observed in the present study, the results were quite consistent with the findings reported by Blikra as shown in the tabulation of the observations in each series (Table 1). Thus, the percentages derived from the combined data and shown in Figure 2 may be considered a fairly reliable indication of the frequency and locations of the dural adhesions in the general population.

A problem naturally arises with the designations of "dense" and "firmly fixed" adhesions, as these obviously embrace a continuum, and the point of differentiation between the two is a matter of opinion. However, because the terms were introduced by Blikra, an attempt at some consistency required their continued use. The primary concern of Blikra was to account for a fixation sufficiently firm that the pressure from an extruding nucleus pulposus would rupture the membrane rather than strip it away from the posterior longitudinal ligament and the dorsum of the disc. He identified such possible instances as those in which sharp dissection was required to separate the dura from the posterior longitudinal ligament (PLL). This type of fixation was equated with those in our sectioned series that required forceful traction of a hemostat to effect separation. However, our current concern was not with the few cases of very firm adhesions, but with the greater number of instances in which a relatively dense adhesion fixed the ventral dura sufficiently to elevate the PLL above and below the disc. These could be separated when the membrane was gripped with a hemostat and subjected to a moderate degree of traction. This was judged to occur at multiple levels in six of our 15 cases.

DISCUSSION

Disc rupture with the posterior or posterolateral extrusion of material from the nucleus pulposus may produce pain in two ways. The first is by a neuritis consequent to the traction or compression of the regionally related nerve root(s), and the second is by the direct distortion or disruption of nerves in the connective tissues of the involved motion

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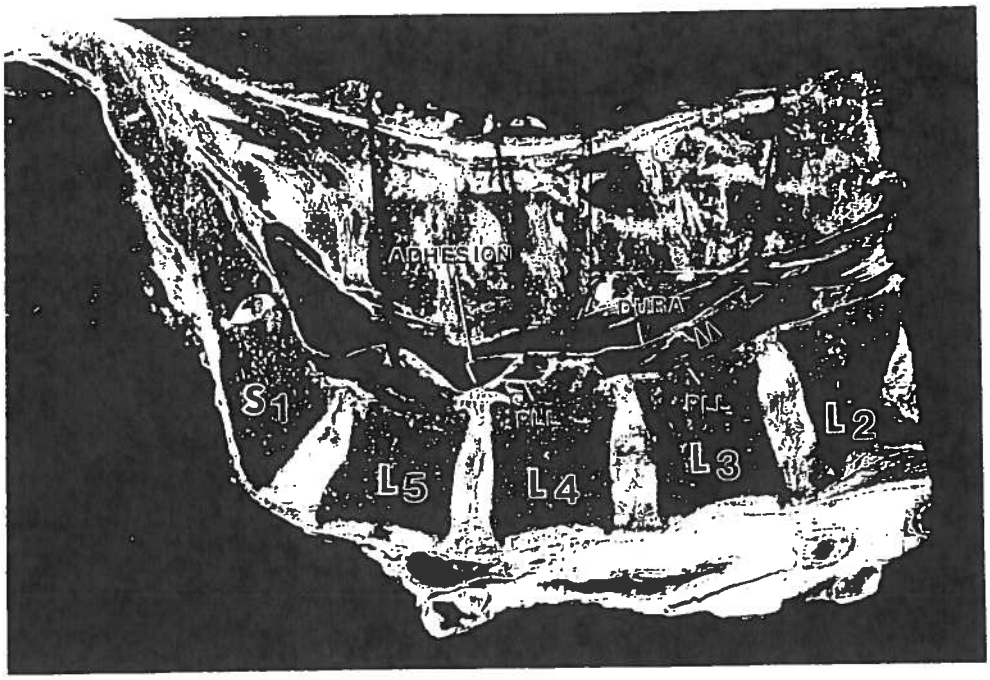

Fig 1. A photograph of a slightly parasagittal section of a lumbosacral spine from a 62-year-old man of the USDSM series. The cut edge of the ventral dura has been elevated by silk sutures that were subjected to 60 g traction by attached weights. Note that the adhesions increase in density dorsal to the bodies of L4 and L5, where they elevate the underlying PLL and its subadjacent veins. This specimen illustrates a firm fixation (labeled "adhesion") to the PLL and the dorsum of the L4–5 disc that was eventually disrupted by traction with a hemostat. The double arrows indicate a thickening of the dura formed by the attachment of a ligament of Hofmann that arose from the dorsolateral PLL over the L3–4 disc.

Table 1. Combined Observations of Two Series

Series	*Oslo*	*USDSM*	*Total*
No. of spines examined	40	15	55
Levels with dense adhesions			
Disc L_2-L_3	0	1	1
L_3-L_4	7	2	9
L_4-L_5	16	6	22
L_5-S_1	15	5	20
Firm fixation at L_4-L_5	8	2	10

This table combines the observations of Blickra (Oslo series) with those of Parke and Watanabe (USDSM series) to provide a total sample of 55 spines. Those designated as firm fixations were included in the listing of dense adhesions at the L4-5 disc level. The Oslo series were all autopsy specimens, whereas those at the University of South Dakota School of Medicine series were all anatomic cadavera.

The extent and locations of the neural sources of the low-back pain associated with disc herniation still lack definitive confirmation. It is obvious that tearing of the anular lamellas could be a primary source of nerve stimulation, but this was initially discounted, as nerve fibers in the anulus were difficult to demonstrate. However, the works of Bogduk et al.[3] Malinsky,[8] and Yoshizawa et al[14] have offered evidence that the outer third of the anulus is innervated, and is, most likely, the initial contributor to the so-called discogenic pain. There is a consensus[6,9,12] that the PLL is a highly innervated structure, and the intimate relationship of its dorsolateral expansions over the corresponding surfaces of the disc ensure its distortion and/or disruption by bulging of the anulus or the protrusion of the nucleus pulposus. As the varieties of nerve endings demonstrable in the PLL include the fine free terminals and the small clavate endings that are conventionally regarded as pain receptors, it is very probable that, in addition to the disc, this structure is one of the major sources of discogenic pain.

Heretofore, however, the nerves related to the spinal dura have not

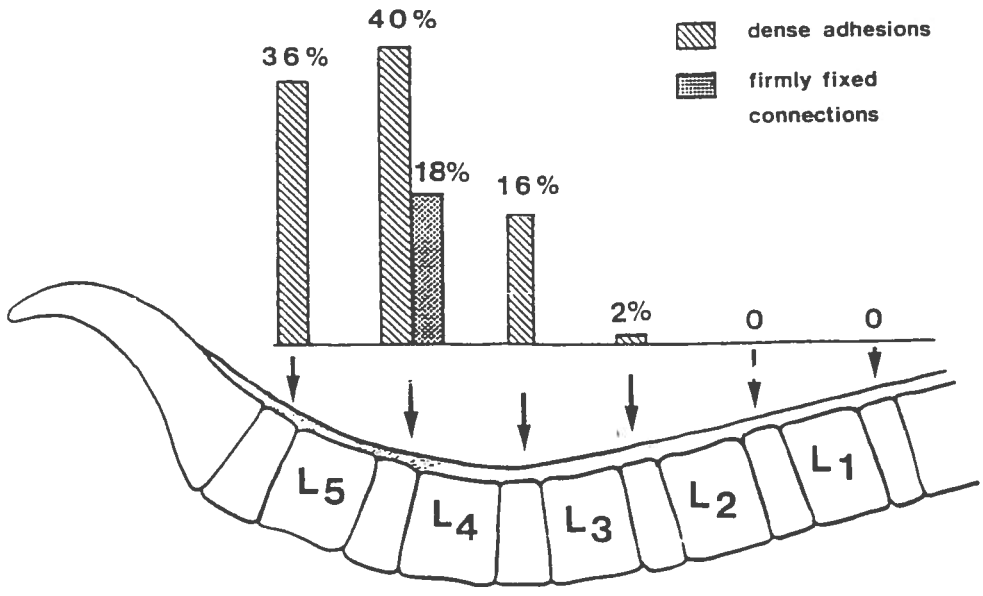

Fig 2. A graph-enhanced schema illustrating the percentage of frequencies and locations of occurrence of the ventral lumbar dural adhesions noted in the combined series listed in Table 1. Adhesions designated as firmly fixed were those in which the dura, the PLL, and the outer laver of the anulus were fibrously bound into an apparently single layer that could be separated only by forceful traction or sharp dissection.

been suspected of contributing to the discomfort experienced with disc herniation. Most probably this has been due to the incognizance of the previously described adhesions. Gray's Anatomy[5] notes that the ventral spinal dura is attached to the PLL by fibrous slips, especially in the caudal end of the spinal canal. As these slips, the ligaments of Hofmann, increase in length and the obliquity of their attachment in the lumbar region, they would offer little resistance to a dural configural accommodation to the bulge of herniated disc material. The texts fail to mention that there is an additional system of fine collagenous strands, collectively resembling areolar connective tissue, that also joins the ventral dura to the PLL. These fibers offer little resistance to the elevation of the dura in the upper regions of the spinal canal, where they are sparse, but as they increase in number and thickness as the lower lumbar region is approached, they offer, in at least one third of the cases, an increasing resistance to the dural separation. These fibers are well illustrated in Figure 1, where they can be seen to be rather few and scattered at the L2 level, but increase in density toward the lower lumbar segments. It will be noted that dorsal to the L2 and L3 vertebral bodies the dura was elevated without lifting the PLL. However, dorsal to L4, the density of the fibers increased sufficiently to elevate the PLL and its subadjacent veins. These latter structures are also known to be highly innervated. A similar situation prevails over the dorsum of L5 and its disc. Note that, in this specimen, the same degree of traction fails to separate the dura from the L4–L5 disc and ligament. As this had to be separated by greater traction with a hemostat, it was judged as typical of the more firmly fixed ventral dural adhesions evident in approximately one out of six cases. Should one visualize a dorsal protrusion of disc material with sufficient pressure to elevate this adhesion and stretch the connective tissue strands extending above and below it, a considerable connective tissue disruption becomes apparent. When this information is related with what is known about the distribution of the branches of the sinuvertebral nerve relative to the PLL and the dorsal surface of the disc, then extensive nerve fiber stress becomes obvious. Bogduk and Twomey[3] and Parke[9] have shown from their dissections that each sinuvertebral nerve enters the intervertebral foramen and divides into superior and inferior branches. The inferior branch sends fibers to the ligaments and dorsal disc surfaces at the level of the nerve entrance, whereas the superior branch sends fibers to the disc one segment above. Thus, two levels of sinuvertebral nerves would have fibers that intermingle among the connective tissue strands that bind the adherent dura to the PLL over a single disc level. Throughout most of their course, these branches run between the dura and the PLL and provide fine filaments to the PLL, the periosteum, and the overlying dura. It is, therefore, not surprising that samples taken from these elevated sections of adherent dura showed the disruption of neurovascular bundles, as illustrated in Figure 3. It is not known at this point whether the nerves stained were dural ramifications or fibers that were fixed in the adhesion on their course for distribution elsewhere. In either case, the essential concept derived here is that branches of the sinuvertebral nerves pass through these adhesions, and their forced separation would amplify the degree and extent of nociceptive fiber disruptions. Thus, they would most likely contribute to the low-back pain consequential to the protrusion and/or extrusion of a lower lumbar disc. The inspection of a lateral view of a myelogram showing a frankly protruded L4–5 and its obviously elevated dura should enhance the appreciation of this assumption.

As only one in three individuals may have lumbar dural adhesions sufficiently dense to resist dural elevation, the notorious variability of the subjective discomfort reported by a series of patients with almost identical objective signs of pathology may be due partly to the presence or absence of these connections.

The irregularity in the occurrence and degree of fixation, and the mode(s) of origin of the lower lumbar ventral dural adhesions, raise some interesting academic questions. Teng and Papatheodorou,[13] who studied 444 cases of disc herniation showing dislocation of the extruded fragments, found on 41 occasions that the sequestered material perforated both the anulus and the PLL, and in three of these instances, they penetrated the dura. They analyzed these last cases with another five found in the literature and noted that, in all but one of the eight patients, the dural perforation occurred at the L4–5 disc level. These authors concluded that irritation from the pathologic disc or previous surgery produced the adhesions. Unfortunately, their excellent study was conducted 5 years before the publication of Blickra's analysis[1] of the frequent adhesions in the nonpathologic spine.

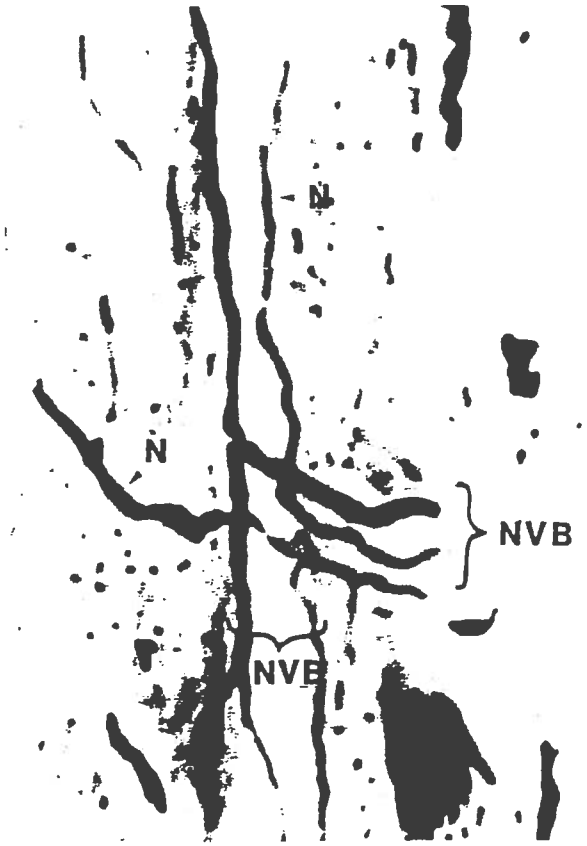

Fig 3. A photomicrograph of a cleared segment of dura that had been stripped from its adhesion at the L4–5 disc level, where the plane of separation may also include fibers from the PLL. The tissue was stained in a polypropylene solution of Sudan Black that has an affinity for lipids in the myelin sheaths of nerve fibers. Those seen here accompany cleared small blood vessels, forming a neurovascular bundle (NVB), which passed between the fused layers of the adhesion. The specimen was cleared in a solution of tricresyl/tributyl phosphates, mounted between glass, and transilluminated. The nature of these fibers is unknown, excepting the fact that they are branches of the sinuvertebral nerve passing between the dura and the PLL that have definitely been disrupted by the separation of the adhesion.

During development, the dura had a variable relationship with the more rapidly lengthening spinal canal, similar (but in a lesser degree) to the relative ascension of the spinal cord. This differential growth accounts for the increasing caudal obliquity of the ligaments of Hofmann and also indicates that the described adhesions must develop after the dura and vertebral column have acquired their definitive topographic affinities. Because the majority of the adhesions can be attributed neither to a developmental origin nor a regional disc pathology, what can account for their irregular but frequent occurrence, and

why do they have a very marked predilection for the dorsum of the L4–5 disc? A possible answer may involve the greater angulation of the spinal canal that frequently occurs at the L4–5 intervertebral level. In sagittal or slightly parasagittal sections of the lumbar spine, it was noted that there often is an abrupt increase in the degree of the lumbar lordosis at this point. This is made more noticeable by a more pronounced buckling of the ligamentum flavum opposite the L4–5 disc that reduces the anteroposterior dimensions of the lateral recesses of the canal and produces a distinct "kink" or constriction in the corresponding level of the dura that is often visible in myelograms. The dorsal dura and the more dorsally positioned nerve roots are bent around this angle, whereas the ventrally positioned roots and the ventral dura tend to "bowstring" across the greater arc of the curvature (Refer to Figure 1 for a visual comprehension of these relationships.). It is this latter phenomenon that may account for the dural adhesions. With spinal flexion and extension, a freely movable dura would exhibit a range of excursion relative to the underlying PLL, and an induced adhesion eventually would fix the ventral dura to reduce the motion and the tendency to bowstring.

Because contrast-enhanced peridurography[4,7] or CT epidurography[10] should provide excellent methods of visualizing lumbar ventral dural adhesions, it is somewhat surprising that the cited descriptions of the lumbar epidural spaces failed to make any special reference to ventral dural adhesions. However, considering that the reports of the dimensions of the ventral peridural space provided by Lewit and Sereghy[7] for all lumbar and the first sacral levels showed 0 mm as the minimum extreme for each observed series at each level, it is quite likely that ventral dural adhesions were observed, but the variability of their occurrence and location obscured their significance. Nevertheless, an extensive series of contrast-enhanced visualizations of the lumbar ventral peridural space, with specific attention directed to dural adhesions, should provide a more accurate account of their frequencies.

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