

DIFFERENTIAL DIAGNOSIS OF CAUSES OF PAIN IN THE LOWER BACK ACCOMPANIED BY SCIATIC PAIN*†

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THE SYMPTOM OF BACKACHE with referred pain along the course of the sciatic nerve has for many years attracted the attention of orthopedic surgeons and industrial surgeons everywhere. Looking back at the evolution in the diagnosis of this condition over a period of 35 years, one cannot help but remember the old days when the diagnosis of "railroad spine" was made in those cases where an injury occurred at the onset of backache, which was not infrequently followed by sciatic pain. This diagnosis was due to the inability of the medical profession to comprehend the underlying pathologic changes which can happen, and which do happen in the lower back. Little effort had been made, judging from the literature, to assign the symptom of pain to any real lesion.

Goldthwaite¹ then propounded his theory of strain, subluxation, or disease of the sacro-iliac joint. As a result of his work much interest was aroused in the subject, and for a few years the literature was filled with the advocacy and defense of this theory. The next great step was made by the contribution of Willis,² who reported anomalies in 7 per cent of 748 anatomic specimens. He found marked variation in the normal anatomy of the lower spine, particularly at the lumbosacral junction. Willis called attention forcibly, and conclusively, to these variations in the mechanical structure which could account for many of the symptoms and combinations of symptoms exhibited in the lower back. These anatomic variations might also affect the emerging nerves and the ligaments supporting this portion of the spine. His findings rationalized certain conclusions made previously on a clinical basis regarding the causes of low back pain.

In 1916, I stated before the American Roentgenological Society that low back pain and sciatic pain were to be attributed more to strain of the ligaments of the low back than to any other one cause. I pointed out that all the components comprising this part of the anatomy, namely, the fascia, muscles, joints with cartilage and synovia, tendinous attachments, interlacing ligaments, all surround a bony canal through which the peripheral nerves course; that these structures were subject to the same diseases to which like structures in other parts of the body were vulnerable; that inflammatory reaction in these structures was much more prevalent than in like structures in other parts of the body where there was less cause for strain; and that infection, allergic reaction, gout and other forms of toxemia caused by sys-

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temic conditions could and did produce the same symptoms which in recent years have been ascribed to rupture of an intervertebral disk.

If there is one place in the body where a fine differential diagnosis is necessary it is in the low back. Mechanically, the low back is structurally weak for the work it is required to do. It is the fulcrum on which every motion of the body is pivoted. It lies at the junction of a flexible with an inflexible part, namely, the lumbar spine with the sacrum and the ilia. As previously stated, the tissues which compose the low back are subject to the same ills as similar tissues elsewhere in the body. Moreover, the mechanical strain exerted on these tissues is much greater than elsewhere. In addition, more anatomic variations in structure are found in the lumbosacral junction than in any other location. All these factors contribute to making the low back an area where pain can occur with great suddenness and can be caused by various agents.

In the recent literature, neurosurgeons stand out preeminently as contributors to the subject of low back pain accompanied by so-called sciatica. Their approach has been mainly from the standpoint of root pressure from a ruptured intervertebral disk. To one who has been interested in the subject for more than 30 years, and has examined many hundreds of such cases, this viewpoint seems much too narrow. It is not the intention to contend that low back pain is not, or could not be caused by rupture of a disk, with pressure on the roots, but to point out that the same symptoms can be caused through disarrangement or inflammation of other structures. It seems strange that Schmorl³ who dissected more than 10,000 cadavers, found only two cases of traumatic rupture of the nucleus pulposus, whereas our surgeons have operated upon hundreds of cases for this condition in the last five years. Schmorl found various degrees of degeneration of the disk and ligaments in more than 15 per cent of all specimens. These degenerative changes around the lumbosacral joint were 7 per cent greater in men than in women, which would tend to indicate that heavy labor has an effect on the structures supporting the lower spine.

It is 30 years since I performed my first bone graft for immobilization of the lower spine. For 12 years I have made sections of the joints of this region in every case operated upon, and in every case pathologic changes were found in the cartilage of the joints, associated with varying degrees of inflammatory reaction in the capsules and ligaments adjacent to or extending from the joints. When one considers that the joints at the lumbosacral junction, for their size, carry approximately ten times the weight per square inch that the knee joints carry, and that the mechanical strain exerted by body weight thrusting downward and forward at the lumbosacral joints puts constant pressure on normally formed joints, it is easy to conceive that in abnormal joints this weight is transferred to the ligaments which, in turn, are required to carry the strain. In either case the strain occurs not on the intervertebral disk in the low lumbar region, but on the ligaments and joints posterior to the spinal canal. In flexion, part of this weight is transferred

to the bodies of the vertebrae and the intervertebral disks, which act as a cushion and compress at the same time that the inferior articular facets of the fifth lumbar vertebra move upward on the superior articulations of the sacrum. The posterior ligaments are stretched as the articulations slide. If there is normal elasticity in the ligaments and the capsules of the joints, and the joints are smooth, this sliding motion can be made without discomfort. However, if the ligaments are degenerated and have lost their elasticity, or if the joint surfaces are rough and surrounded by small exostoses at the attachment of the capsules and tendons, this motion can cause pain either from overstretching of the ligaments which are not able to withstand the strain put upon them, or by actually causing minute tears of this fibrous structure. Moreover, the joints can catch and lock, because the gliding motion is absent due to erosion.

Everyone is familiar with such occurrences in other joints which are degenerated or eroded. From the pathologic changes and symptoms which occur in other joints, especially the knees, which are much more easily examined, we know that swelling, locking and severe, sudden, sharp stabbing pain occurs. We can feel the crepitation and we can demonstrate the locking and immobility. From our pathologic studies, we know that the changes in the low lumbar joints do not vary from the lesions frequently found in the knee, elbow or hip. The swelling occurring as a result of ligamentous and joint injury is familiar to all practitioners of medicine. In the joints of the low back the pathologic changes do not differ from those of joints elsewhere in the body, except that swelling of the ligaments and the joint capsule can, and frequently does, narrow the exit of the nerves as they emerge from the spinal canal and foramen, the posterior wall of which is frequently formed by the anterior margin of the articular facets. The joint capsule of the articular facet lies immediately posterior to the nerve at its exit and forms part of the posterior wall of the foramen (Fig. 1).

In a cross-section of the spine at the lumbosacral level (Fig. 2) the normal lumbosacral joints lie at an angle of 45° to 60° to the anteroposterior plane of the body. There is, however, a great variation in the normal, and it is extremely difficult in many individual cases to judge from the roentgenograms whether these articulations furnish adequate support on one or both sides. What is normal for one person may not be considered normal for another. Roentgenograms should be taken from various angles before a decision is made whether the mechanical support between the vertebrae is adequate. One must take into consideration not only the anteroposterior but the horizontal plane of the joints, because as the patient stands the lumbar curve is increased and a joint which looks almost perpendicular in the sitting position will become almost horizontal in the erect position, depending upon the amount of tilt in the pelvis (lordosis) in standing. The more horizontal the articulation, the more strain is put upon the ligaments which support it and the low spine, in general, in the flexed position. Instead

CAUSES OF PAIN IN LOWER BACK

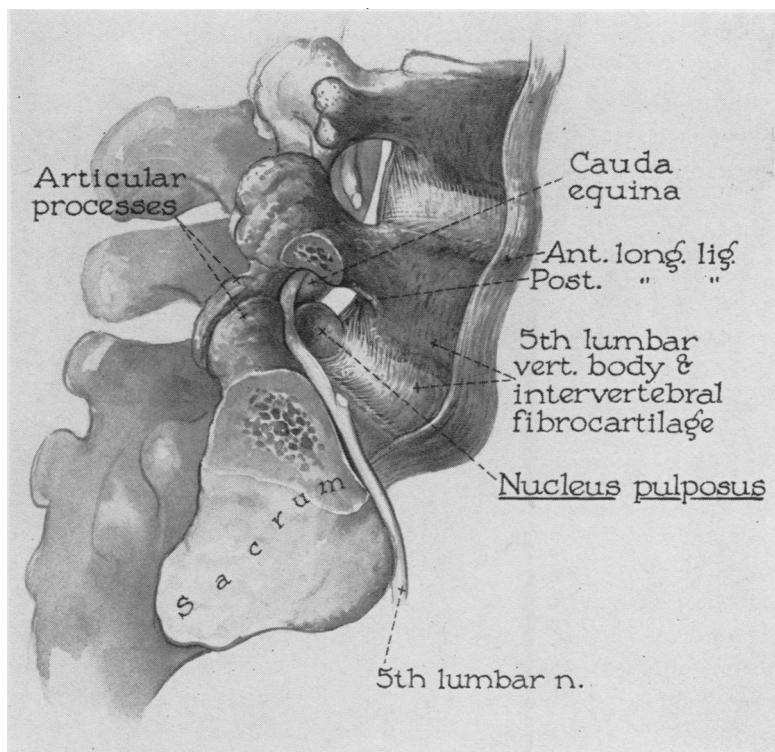


FIG. 1

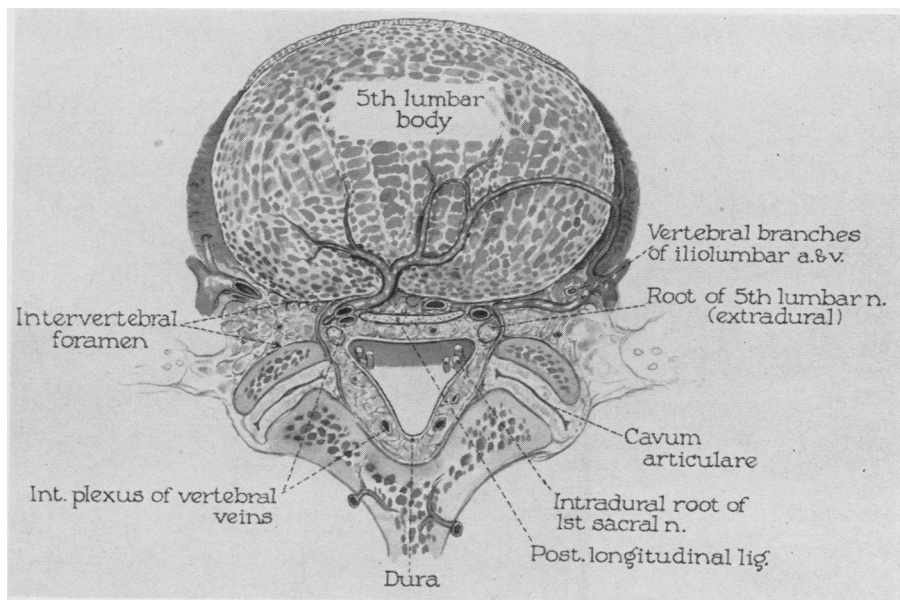


FIG. 2

of the articular facets sliding up and down on each other, they slide from a posterior to an anterior position.

The angle of the joints may also vary on opposite sides. It is not infrequent to see in the same patient a perpendicular joint on one side and an oblique or horizontal joint on the other (tropism). In such a case the perpendicular joint will act as a pivot and the vertebra will rotate on the more or less horizontal joint, frequently producing pain in the more rigid perpendicular joint because of the rotary stresses.

The integrity of motion depends on many structures as well as upon the plane and angle of the joints. The interspinous ligament, the joint capsules, the ligamentum flavum and the posterior longitudinal ligament must maintain the relative position of stability unless the articular processes are properly formed to accept the downward and forward thrust of the weight of the body in the upright or flexed position. The muscles are called into play to support and produce motions which are willed by the patient. Any interference with function of any of these tissues, or any pathologic changes existing, can cause pain in the back which may be followed by radiation down the leg, depending on whether swelling or inflammation occurs around or near the exit of a nerve.

Anatomic study of a cross-section at the fifth lumbar vertebra shows that the fifth lumbar nerve usually makes its exit from the dura just above the level of the fourth lumbar disk. This is the rule in each vertebra above this point. The nerve passes downward and outward to enter the foramen. At the point of exit at the fifth lumbar foramen it is well below the disk. It lies immediately in front of the anterior part of the fifth lumbar inferior facet or lumbosacral articulation, just in front of the anterior part of the joint capsule. The posterior longitudinal ligament, which forms the anterior boundary of the neural canal, extends three-fourths of the way across the width of the canal. Entering the vertebra just lateral to the longitudinal ligament is an internal plexus of veins. The posterior longitudinal ligament is a fibrous structure and is one of the sturdiest parts of the annulus. It blends with the annulus and forms the posterior wall. The nucleus pulposus is situated just a little anterior to the posterior edge of the vertebral body (Fig. 3). The posterior and lateral wall of the neural canal is formed by the ligamentum flavum, which is attached to each vertebral arch above and below. The ligamentum flavum is cupped out at each intervertebral foramen where the nerves exit.

The measurements of these foramina in our specimens varied considerably. The average size was 7 Mm. from front to back in both the fourth and fifth lumbar openings. The measurement between the pedicles in the fourth lumbar averaged 19 Mm., and between the pedicles of the fifth lumbar, 12 Mm. The size of the ganglion in both the fourth and fifth lumbar vertebrae averaged about 7 Mm. (Fig. 4). It will be seen by these measurements in ten fresh specimens that the distance between the pedicles of the fifth lumbar is considerably narrower than in the fourth, and that the

diameter of the foramen is only slightly larger than the diameter of the ganglion. Inasmuch as the joint capsule and ligamentum flavum are continuous and almost inseparable by dissection at this point, it may readily be appreciated that any swelling or inflammation around the foramen could narrow the canal sufficiently to cause pressure upon the nerve root. Moreover, any inflammatory condition within the joint or any swelling or thickening of the ligamentum flavum in conjunction with the joint capsule could spread by continuity to the root of the nerve.

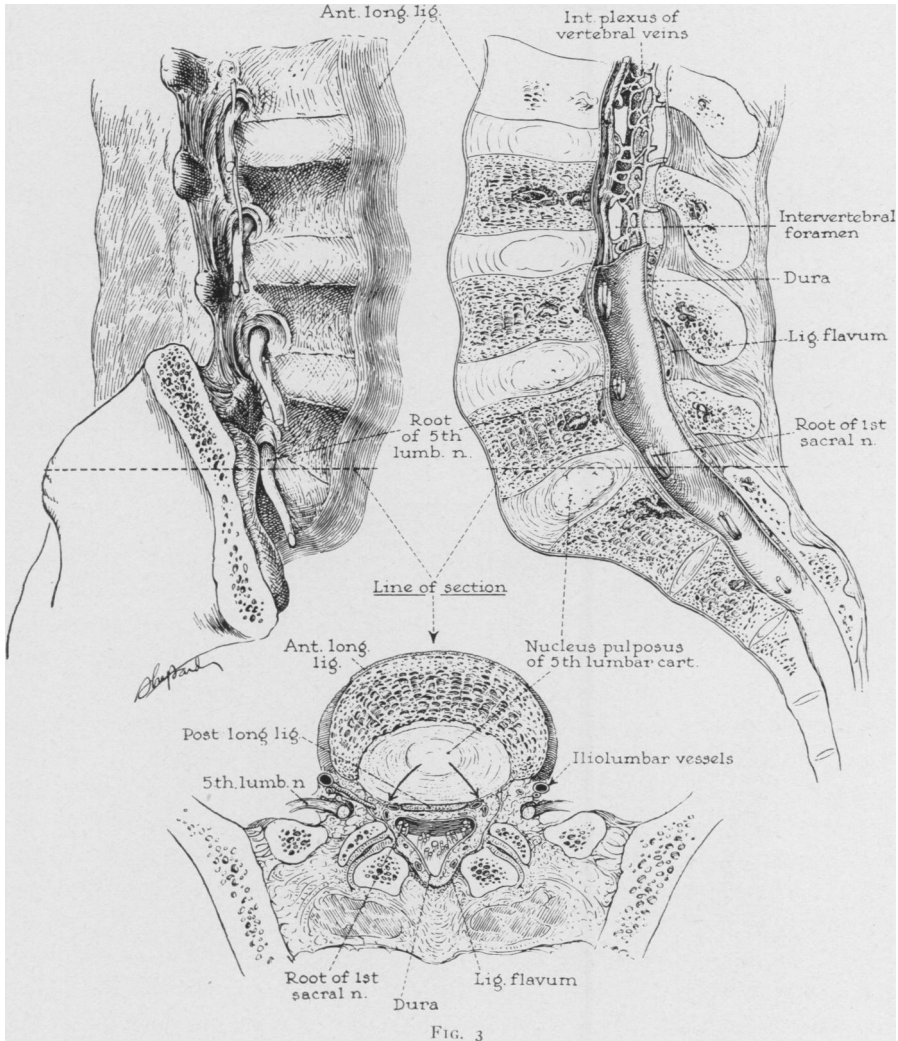


FIG. 3

Personally, I have never seen a case with pain referred to the sciatic distribution, disappearance of reflexes, or neurologic symptoms referable to interference with nerve function, appear in less than seven days after injury to the low spine, excepting in two instances where there was severe

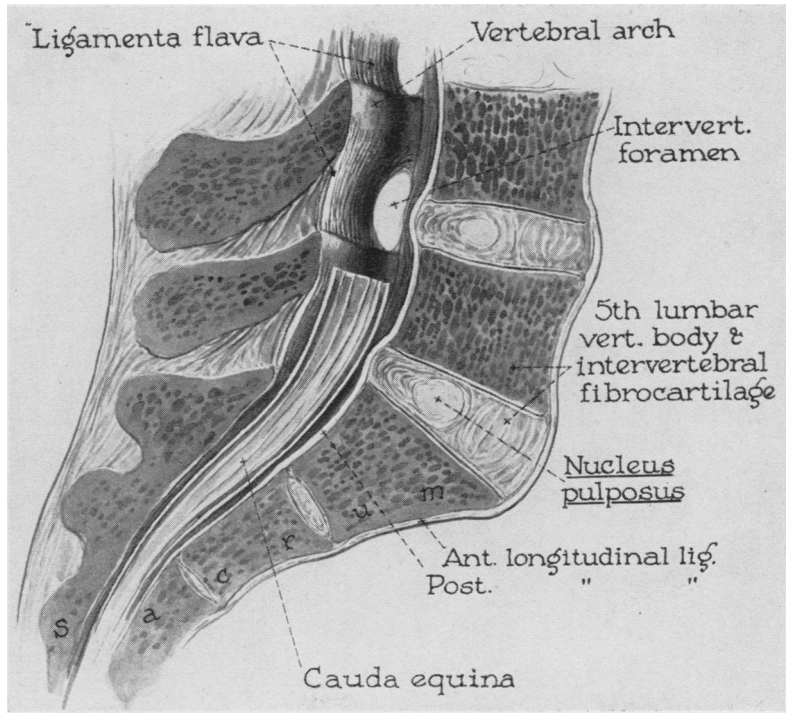


FIG. 4

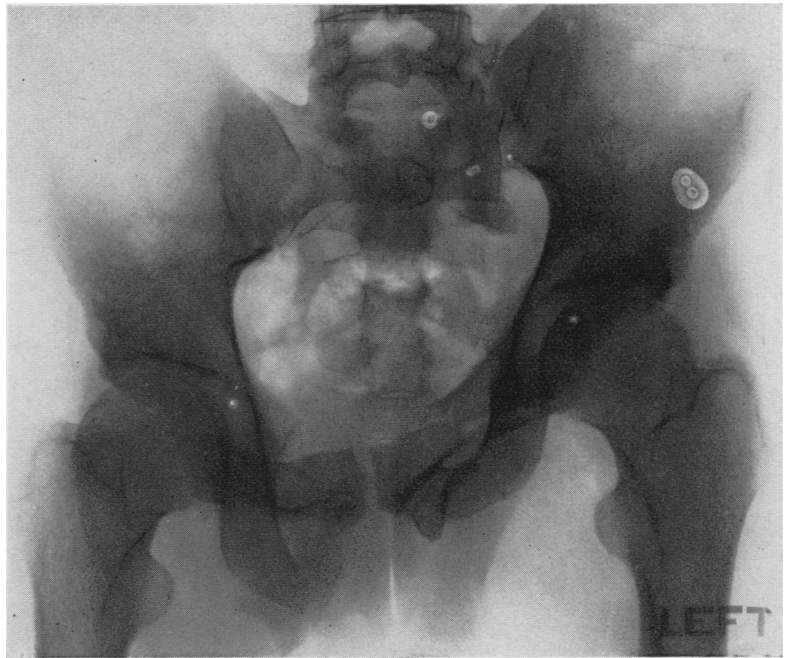


FIG. 5

injury (Fig. 5). In these two cases fracture and upward displacement of the ilium were present. Both patients had exactly the same symptoms referable to the sciatic nerve. I have seen similar symptoms many times in conjunction with low back pain where there was no roentgenologic evidence of pressure or injury to the nerve which exits from the low lumbar region.

In fresh specimens we were able to inject the capsule of the fourth and fifth lumbar joints and narrow the exit of the nerve by as much as 2 Mm. This narrowing was caused by mechanical distension and stretching of the capsule (Fig. 6). The injection was made with thorotrast in order to obviate any possibility of the material breaking through the capsule without roentgenologic evidence of such leakage. If it is possible to cause narrowing of 2 Mm. from simple pressure within the joint, it is quite evident that swelling within the joint, plus thickening of the capsule by inflammation and/or the ligamentum flavum, could cause narrowing of the foramen to a point where serious pressure might be exerted on the nerve. Schmorl³ called attention to the fact that more than 15 per cent of his 10,000 cases showed degenerative process in the ligaments and tissues around the low spine, which means inelastic, thickened, hardened tissues.



FIG. 6

We have previously demonstrated experimentally that arthritis with exostoses can be produced by trauma alone⁴ (Fig. 7). This can be done repeatedly by weakening the support of a joint and causing it to bear cross-strain while the animal is active. Exostoses form around the edge of the joint and the cartilage degenerates progressively due to the often repeated slight traumata constantly administered to the weakly supported joint. These joints become roughened at the weight-bearing point, and this roughness extends toward the edges of the joint. The cartilage wears away at the point of greatest trauma and exostoses develop at the edge of the joint.

These pathologic changes are the same as are commonly seen in the spines of individuals who have done heavy work over a long period of time. When the joints become rough they do not glide normally. The rough margins serve as friction surfaces to the worn and degenerated cartilage.

This interference with smooth motion in the joint can cause catching and stabbing pain in the back, thereby creating muscle spasm and pain, exactly as seen in other joints similarly affected. We are all familiar with crepitation, thickening of the capsule, and swelling in the joints which can more easily be examined. There is no reason to suppose that the joints of the low lumbar spine differ in their pathologic reaction to various stimuli, such as trauma,

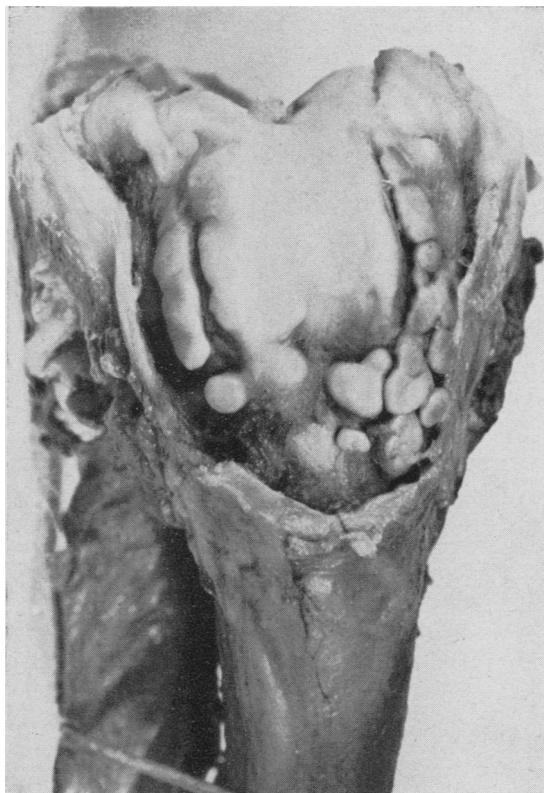


FIG. 7

infection, toxemia and allergic reaction. These may all be contributing causes in the degenerative process which occurs at an area well known to be a point of great mechanical stress. The degenerative process, when it occurs in a disk, may cause marked narrowing of intervertebral space, associated with collapse of the disk and irritation of the surrounding soft parts and bone (Fig. 8). When this happens, the annulus is pressed backward toward the canal and narrows the anteroposterior diameter of the canal. At the same time the foramen through which the nerve passes is diminished in size. Roentgenologic evidence of narrowing of the canal from front to back does not confirm the diagnosis of pressure on the canal or on its contents. That this irritation can be long-standing is shown in Figure 7 by the eburnation of the posterior lower third of the fifth lumbar vertebra. The area of osteosclerosis

is more marked at the posterior part of the body where the greater weight is borne. Although the fourth lumbar intervertebral space is somewhat decreased there is no sclerosis, because the weight is borne across the diameter of the bone. It is only at the point of greatest pressure that the most marked reaction is seen.

The anteroposterior diameter of the canal is ample to accommodate such increased bulging from in front without actual pressure on the contents.



FIG. 8



FIG. 9

The exits and course of the nerves through the foramen also vary. In one of the ten fresh specimens there was a double outlet laterally for the fifth lumbar nerve, the nerve dividing into two branches before it made its exit from the spine (Fig. 9). In another case the nerve divided just outside the ganglion; one branch passed through the foramen, the other ran down under a broad ligament to its exit one-half-inch below that of the upper branch. The lower exit was extremely narrow and the nerve was held firmly by the overlying broad ligament (Fig. 10-A). In another specimen (Fig. 10-B) the foramen was very narrow on the side of entrance of the nerve and widened at the exit, its perpendicular diameter being about one-half its anteroposterior diameter (Fig. 11).

It is quite apparent that variations in the path of the nerves are frequent not only in the foramen but at the exit from the foramen. Where the nerve is bound down, or the space through which it emerges is narrow, as in the specimens just described, a very little overstrain or inflammatory process in the ligaments through which the nerve makes its exit could cause definite

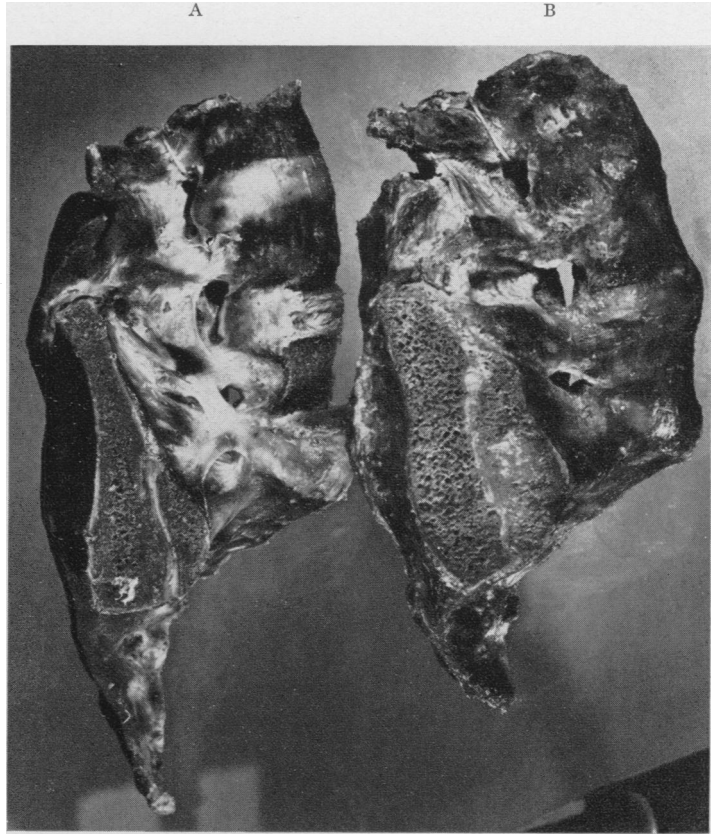


FIG. 10

symptoms of pressure on the involved branch. It is true that rupture of a nucleus pulposus could cause pressure upon a nerve where it leaves the dura and passes down to the vertebra below to gain entrance to the foramen. At this point the nerve is pressed into the corners of the triangular canal laterally. The nucleus is wedged between the root and the dura and presses the root toward the lateral surface of the canal, while the dura is pressed toward the median line (Fig. 12). If this happens the symptoms should be easily traceable to pressure upon this particular root, and there should be no question about its localization on physical examination. The reflexes should be interfered with and the pain should be referred to the area supplied by this root. The pain should be consistently localized to one place, and visual-

ization (of which I do not approve) should show a filling defect outside the dura (Fig. 13). If there is a rupture of the disk it can be plainly seen as a protruding mass outside the dura.

If it is necessary to make a crucial incision through the annulus in order to disclose the nucleus, then any existing symptoms are not due to a disk,

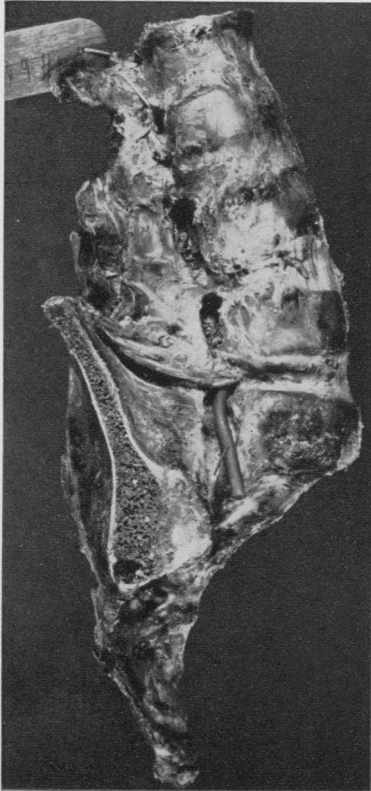


FIG. 11

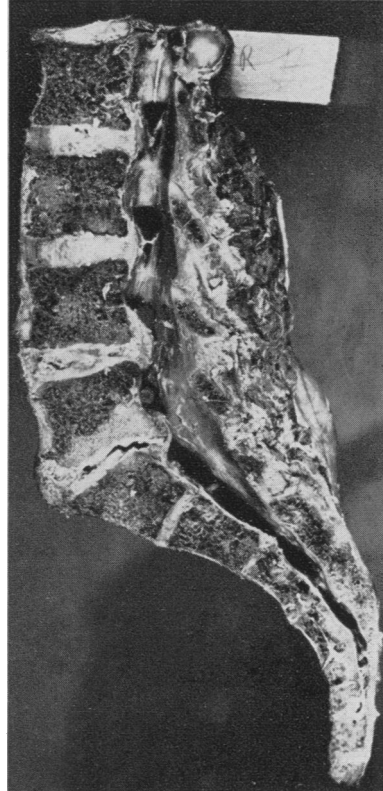


FIG. 12

hidden or herniated. It is always possible through an incision in the annulus to curet or otherwise bring out the material which forms the disk. No incision should be made in the annulus at the time of exploration. If there is a hernia of the disk it lies on the anterior surface of the canal between the dura and the annulus through which it has ruptured; further exploration is unnecessary. Should the annulus be punctured by trauma which forces the disk out, or by an instrument at the time of operation, this causes collapse of the intervertebral space. If the center of the disk degenerates or is allowed to escape through rupture in the annulus, the remainder of the disk may be forced backward into the canal, as shown in Figure 8. Collapse of the disk with narrowing of the space between the vertebrae throws the articular facets between the vertebrae into a distorted position; the angle may be

changed or they may override. This, in itself, puts strain on the joints, the capsules and the supporting ligaments, and will eventually produce traumatic inflammatory reaction which will necessitate furnishing support for the joints.

It may be asked why a certain number of patients are relieved of pain when they are operated upon for ruptured nucleus pulposus when no rupture has been found, only to have, later, a recurrence of symptoms. It is my

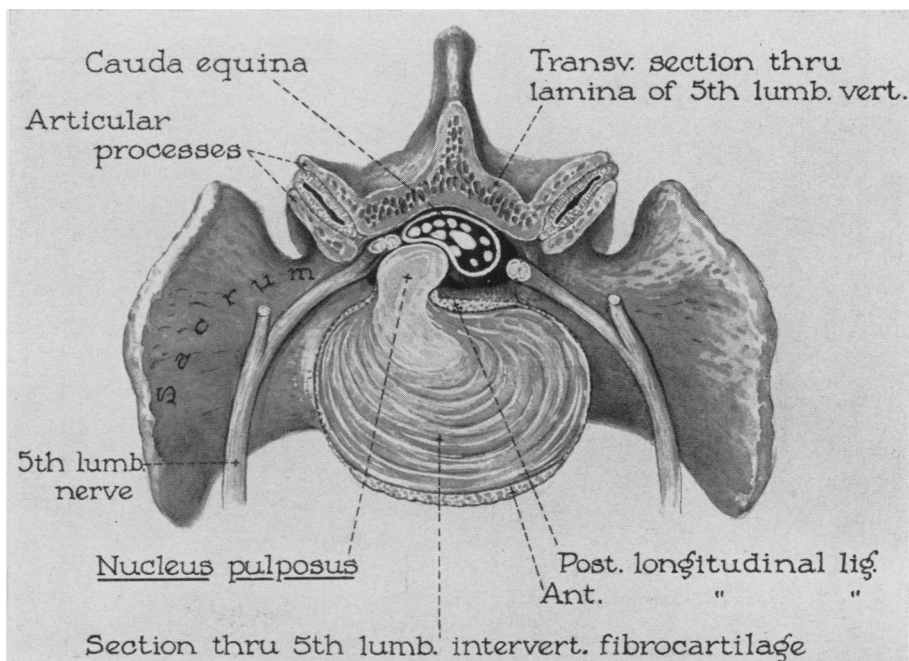


FIG. 13

opinion that the reason lies in the fact that part of the lamina and the ligamentum flavum is removed, resulting in decompression of the probably thick and indurated parts of the joint which is continuous with the ligamentum flavum. Also, in the manipulation of operative procedure, joints which are locked because of roughness, muscle spasm and inelasticity of the ligaments, are relieved. I have seen many patients recover from their symptoms following rest in bed with traction, without any surgical procedure.

It is my opinion, therefore, that the reason for onset of pain after injury to the low back is, in a large number of cases, intimately connected with strain or other injury to the low lumbar ligaments and joints, associated with swelling and inflammation which, in turn, may be caused by minor strains of ligaments or joints already somewhat degenerated, or by lack of mechanical bony support at this point because of malformation.

There are many other contributing factors which cannot be discussed in a communication of this kind because they are not directly related to the

subject matter with which we are concerned. It is sufficient to say that the low back can be, and frequently is, the site of sudden severe pain followed by sciatic pain in days, weeks or months, which may be due to a combination of physiologic degeneration of the supporting tissues, infection, toxemia, arthritis, or a combination of one or more. These cases should not be subjected to operation on suspicion; they should be thoroughly investigated and an attempt made to establish a diagnosis. A large percentage of these patients get well with rest and traction and adequate treatment of the systemic condition which is a contributing factor. The promiscuous operation for and removal of intervertebral disks without definite evidence of root pressure is not justifiable until every other method of treatment has been reasonably tried.

I believe the spine should be permanently immobilized at the time of operation for a ruptured nucleus pulposus, whether or not the rupture is found. If the symptoms have been severe enough and have persisted for a sufficient length of time to warrant operation for their relief, then permanent relief should be afforded the patient by taking the strain off the degenerated tissues at the point from which the pain emanates. This can be done only by permanent immobilization.

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