stockinet vest, the bony prominences, such as the iliac crest, being well padded with adhesive felt, known as elephant plaster.

Conclusion

Briefly, therefore, the treatment of an acute severe sciatica should be complete rest in bed, with local anodyne liniments and cataplasma kaolini, and sometimes local heat by infra-red rays. Analgesic drugs must be given, such as aspirin, phenacetin, and caffeine tablets, with or without codeine or opium, and even morphine or heroin injections may be required for a few days. Sleep may be obtained by the use of $7\frac{1}{2}$ grains of medinal with two veganin tablets. Massage must never be used in the acute stage. Later in the more chronic stage massage may help, and galvanism, diathermy, or local heat should be tried. If these fail, massive injection of the nerve or epidural injection by novocain and saline, and extension by weights attached to anklets, with counter-extension at the axilla, should be tried. If these give only temporary relief, it may be advisable to fix a plaster from the axillae to the trochanters so that the patient may be able to get about and attend to a certain amount of business.

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INTERVERTEBRAL DISK LESIONS AS CAUSE OF SCIATICA*

BY

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(WITH SPECIAL PLATE)

A symposium such as this, which brings before us the experience of various men gained from the laboratory, the operating theatre, and the office or dispensary, is a valuable method of integrating our expanding knowledge concerning sciatica. It gives me particular pleasure to open the discussion, and I am deeply sensible of the honour conferred upon me.

For our purposes we may define sciatica as a symptom characterized by the complaint of pain in the posterior thigh and postero-lateral calf. You will note that I do not call it neuralgia, neuritis, or radiculitis, nor do I state that it is pain in the course of the sciatic nerve. It is presumably peripheral pain in the regions supplied by the sciatic nerve, due to irritation of the nerve or of any of the roots of the lumbo-sacral plexus which form the nerve. It must be obvious to all but charlatans and quacks that there are many causes of sciatic pain, the mere enumeration of which is tedious. The actual pathological change present in many, perhaps most, of the clinical cases of sciatica is unknown. It is quite probable that further advancement in our knowledge of this condition will come chiefly from increased experience of the actual pathology present and not from theoretic considerations or didactic therapeutic tests. A search of the literature reveals a plethora of cures and a sad dearth of know-

ledge as to what is being cured. There is one group of patients whose sciatica we have come to recognize as being due to the pressure of intervertebral disk tissue displaced posteriorly against one or more roots of the cauda equina. It is of this pathological entity that I wish to speak.

Anatomy

A normal intervertebral disk is composed of a semi-fluid gelatinous central nucleus pulposus which is encircled by a ring of dense coarse fibrous tissue, the annulus fibrosus. The annulus is intimately attached to the cartilaginous end-plates of the vertebrae immediately above and below each disk and also blends with the strong anterior and posterior longitudinal vertebral ligaments. Each intervertebral disk has a certain internal pressure which prevents its collapse when the superincumbent weight is placed upon it. The total amount of spinal mobility is the summation of the mobility of each of the intervertebral disks.

The function of the disk is to permit mobility of the spine, to cushion the central nervous system from trauma, and to transmit the body weight from one vertebra to another. Schmorl has made exhaustive studies of the pathological changes which occur in intervertebral disks. Extrusion of the nucleus pulposus may occur in any direction, following weakening of its surrounding tissues by developmental defect, disease, or trauma. If the cartilaginous end-plate is weakened, protrusion may occur into the vertebral body. Such lesions can often be detected by radiographs. (See Fig. 5, Special Plate.) So far as is known, these protrusions never cause radiating pain. If the annulus fibrosus is weakened by disease or trauma, peripheral protrusion of intervertebral disk tissue will occur, sometimes slowly, as a developing hernia, sometimes very rapidly, as a traumatic rupture. Schmorl found that small posterior protrusions into the spinal canal occurred in about 15 per cent. of spines examined at necropsy. He felt that they were of no particular clinical significance. Kocher in 1898 and Middleton and Teacher in 1911 reported cases of traumatic posterior intervertebral disk ruptures of sufficient size to cause paraplegia.

Neurological surgeons have recognized and removed tissue arising from intervertebral disks and pressing on the spinal cord or nerve roots, and have called these tumours chondromas or ecchondromas. It is quite probable that most of these tumours were not true new growths, but were actually displaced intervertebral disk tissue. With the advent of localizing diagnostic procedures, particularly the use in the subarachnoid spaces of iodized oil opaque to the roentgen ray, we have begun to appreciate that these posterior protrusions are not uncommon lesions and that many patients whose sciatica was previously unexplained, or who had been treated for sciatic scoliosis or lumbo-sacral or sacro-iliac strain, were in fact suffering from pressure on one or more roots of the cauda equina caused by displaced intervertebral disk tissue. We have had over eighty cases verified by operation at the Massachusetts General Hospital. Posterior ruptures of intervertebral disk tissue may occur at any level, but seem to occur most frequently in the cervical and lumbar regions. The disk between the fourth and fifth lumbar vertebrae is by far the most common site. This is probably due to the fact that this represents the apex of the anterior lumbar curve and the point of maximum stress from the superincumbent body weight. About 50 per cent. of the lumbar lesions occur at this disk; 35 per cent. at the lumbo-sacral disk. It is obvious, I think, that the symptoms and signs of a ruptured lumbar

* Read in opening a discussion at a joint meeting of the Sections of Neurology and Psychological Medicine, and Orthopaedics and Fractures at the Annual Meeting of the British Medical Association, Plymouth, 1938. Material from previously published articles by Dr. J. W. Mixer, Dr. A. O. Hampton, and the author has been freely adapted for this paper.

intervertebral disk will vary greatly, depending on its location and size. The largest protrusions cause complete occlusion of the dural sac with the usual signs and symptoms of paraplegia, partial or complete, with sensory motor and reflex changes, and partial or complete sphincter paralysis. There should be no difficulty in diagnosis or localization of such lesions. The smaller disk protrusions that press on only one or two roots are the ones which present difficulties in diagnosis and which may cause sciatic pain without demonstrable neurological changes.

TABLE

Location of lesion:	Per cent.
Disk between L4-5	50
" " L5-S1	35
" " L3-4 }	10
" " L1-2 }	5
" " L2-3	
History of injury:	
(a) Immediately preceding disability.. .. .	50
(b) Latent period preceding disability	30
Total.. .. .	80
Type of injury:	
Lifting a heavy weight	50
Falls from a height	
Twisting strain	30
Miscellaneous	
No known injury	20
Pain:	
Constant from onset until operated upon.. .. .	60
Remissions and relapses	40
Areas to which pain was referred (unilateral 80 per cent.; bilateral 20 per cent.):	
Posterior and lateral thigh	95-100
Postero-lateral calf	90
Lumbo-sacral region	70
Gluteal and sacro-iliac regions	65
Lateral border of foot.. .. .	5
Signs:	
Positive Lasègue (limitation of straight leg raising)	100
"Sciatic scoliosis"	60
Reversal of lumbar curve (kyphos or flat back)	90
Local tenderness over the lumbar spinous processes	50
Radiating pain on coughing, sneezing, or jugular compression	40
Neurological signs:	
Absent or diminished ankle-jerk	50
Sensory change in the extremity (anaesthesia or hypo-aesthesia)	35
Motor changes (weakness or paralysis)	15
Sphincter disturbance	5
Lumbar puncture:	
Total protein content above 40 mg. per 100 c.cm.	85
Total protein content below 40 mg. per 100 c.cm.	15
Dynamic evidence of block, whether partial or complete	10
Cytological changes in spinal fluid.. .. .	rare

This table is a statistical summary of data obtained from a study of the records of over 100 verified cases of lumbar intervertebral disk ruptures into the spinal canal.

The symptoms and signs of a ruptured lumbar intervertebral disk are so constant and characteristic that the presumptive diagnosis can often be made before radiological studies are made by the use of lipiodol. The typical case is a vigorous man in his thirties who was perfectly well until, while lifting a heavy weight, he felt something "snap" in his low back and had immediate pain in the lumbo-sacral region. Some time thereafter he developed "sciatica" in one leg, which he describes as a deep-seated pain beginning in the buttock, radiating down the posterior thigh, the postero-lateral aspect of the calf, and occasionally going into the lateral border of the foot. Coughing and sneezing, bending forward to tie the shoes, and lying face down in bed cause marked increase in the radiating pain. If an uncomfortable position is maintained for a time the leg tingles and the

foot may "go to sleep." There have been two or three separate periods of disability, the first ones relieved by rest in bed; the last one, however, has yielded to no conservative measures, including back-strapping, heat, bed rest, plaster casts, corset, and osteopathic manipulation. On examination the patient is found to stand with his trunk thrust forward and to one side, with most of his weight borne on the non-painful leg—"sciatic scoliosis." The normal lumbar lordosis has been lost, and in its place is a fixed reversal of the lumbar curve, with prominence of the spinous process of the third, fourth, and fifth lumbar vertebrae. All motions of the lumbar spine are considerably restricted by fixed involuntary muscle spasm. The patient's finger-tips cannot possibly be made to touch the floor if the knees are kept straight. The fixed list of the spine and pelvic tilt are uninfluenced by a lift under either foot. In the sitting position the trunk can be flexed at the hips with ease, so that the chest approaches the knees, but observation reveals that the lumbar spine remains in its fixed position and goes forward *en bloc*. The list of the lumbar spine may not be as noticeable, but side bending remains restricted. (Fig 1, Special Plate.)

The patient gets into bed or on to the examining-table with extreme care. Turning over on the table is an ordeal, and he is unable to lie face down until a fat pillow is placed beneath the abdomen. In that position on palpation there is found slight but definite local tenderness in the mid-line between the fourth and fifth lumbar spinous processes or at the lumbo-sacral junction. There may be a little tenderness at the sacro-sciatic notch on the affected side. Inspection reveals some atrophy of the buttock, thigh, and calf of the painful side. The ankle-jerk is usually absent on the painful side, normal on the other. The other reflexes are all normal. No definite sensory or motor changes are made out. Straight leg raising can be carried through an arc of 70 degrees on the non-painful side and 20 to 30 degrees on the painful side. An attempt to force the leg upward beyond this point causes sharp increase in the sciatic pain. A history and physical examination similar to this suggest strongly that the diagnosis is posterior rupture of one of the lower lumbar intervertebral disks. The usual antero-posterior and lateral radiographs are of little diagnostic aid. They may be entirely normal, as the displaced disk tissue casts no x-ray shadow and the intervertebral spaces may be normal in width, the volume of the displaced tissue being too small to cause apparent narrowing. A definitely narrowed space between the fourth and fifth lumbar vertebrae is of some diagnostic importance when present. A narrow lumbo-sacral interspace occurs about as frequently as other congenital abnormalities, such as sacralized transverse processes and spina bifida, and should be considered as an incidental finding. The scoliosis and kyphosis noted clinically are also seen on the radiograph.

The next step in diagnosis is the lumbar puncture, which should be done as low as possible, preferably at the lumbo-sacral interspace. The first 2 c.cm. of fluid removed should be sent as a separate specimen for determination of the total protein. After careful manometric tests to rule out dynamic block further fluid is withdrawn for cytological study and for Wassermann and colloidal gold tests. A total protein higher than 35 or 40 mg. per 100 c.cm. is confirmatory evidence that the pathological process is intraspinal. We have a few proven cases in our series which have had a normal total protein, but the average figure has been above 60 mg. per 100 c.cm.

As the lesion is usually at one of the two lowest intervertebral disks, the lumbar puncture needle is seldom

placed caudal to it, and it is rare to find any dynamic block. The "reverse Queckenstedt" test described by Love may be of some diagnostic significance.

Examination with Iodized Oil

The radiological examination after the injection of iodized oil is the most important step in the diagnosis. The lesion can be accurately localized and readily demonstrated on the radiograph. It is necessary that the examination be done with the proper technique. In a series of over 150 cases we observed no permanent ill effects which could be attributed to the use of iodized oil in the spinal subarachnoid spaces. It is important to use oil that shows no deterioration—that is, no liberation of free iodine. It should be of a very light colour. (Fig. 3, Special Plate.)

One ampoule (5 c.cm.) of iodized poppy-seed oil is injected into the subarachnoid lumbar canal. A satisfactory examination cannot be made with smaller quantities. A delay of a few hours to two or three days between the injection and the radiological examination allows time for the oil to fill the root sheaths and permits better visualization of any defect in filling. It may be necessary to give the patient appropriate drugs for the relief of pain so that he can co-operate in the examination. The equipment for the examination consists of a tilting table for screening and a quick change-over switch so that instantaneous films can be taken. The usual equipment may, however, be used with fairly good results in the examination of the lumbar area. The advantages of films taken in this way cannot be overestimated. Before the examination is begun the patient is allowed to sit up for a few moments in order to collect the iodized oil as one mass in the sacral cul-de-sac. He is then placed facing the table in the upright position, and by manipulation of the table under radiological control the oil is forced to flow slowly up and down the anterior aspect of the subarachnoid spaces when the patient is tilted to the horizontal or the Trendelenburg position. Particular attention is directed towards maintaining the iodized oil in a single mass, and it is possible by this method to place the small quantity of iodized oil in practically any portion of the lumbar canal. Since the lesion to be demonstrated is a small anteriorly placed extradural nodule at the disk levels, the iodized oil must be brought into contact with the anterior dural surfaces, and in order to do this the patient must lie face down on the table. If a questionable filling defect is observed, repeated efforts are made to obliterate it either by turning the patient from side to

side or by repeating the tilting process. A constant filling defect in any region is recorded by serial radiographs taken at various angles of rotation of the patient. The upper dorsal area is not examined when the patient has no symptoms referable to that portion of the spinal canal. It is possible that this fact may account for the infrequency of some of the untoward reactions attributed by others to the use of iodized oil. The examination of the upper dorsal area is, however, carried out, when indicated, in the same manner as the examination of the upper lumbar and lower dorsal areas. Care should be taken to prevent the iodized oil from entering the skull, and this can be done by examining the cervical area with the patient in the face-down and lateral positions only.

Interpretation of Radiographs

A correct understanding of the results of radiological examination is dependent on an accurate knowledge of the anatomy of the cauda equina and the nature of the lesion to be demonstrated. The anatomical considerations are beyond the scope of this paper, and only typical examples of the filling defects produced by the shadow of the contrast medium can be discussed. The posterior rupture of the intervertebral disk which produces unilateral symptoms consists of a rounded nodule 1 cm. or more in diameter. This nodule lies just lateral to the strong central portion of the posterior longitudinal

LEGENDS FOR ILLUSTRATIONS IN SPECIAL PLATE

FIG. 1.—Photographs of a patient with lateral list (sciatic scoliosis) and abnormally flat lumbar spine seen in majority of cases of sciatica due to intervertebral disk rupture.

FIG. 2.—Actual appearance of a ruptured disk at operation. The laminae of two vertebrae have been removed, the dura has been opened, and the roots of the cauda equina retracted, exposing the nodule of disk tissue over which a nerve root is riding.

FIG. 3.—Drawing of x-ray appearance of a normal spine after complete filling of the subarachnoid space with iodized oil. The extradural course of the nerve roots is indicated by the dotted lines. The point of exit of each root can be identified by the axillary pouches marked A. Note that a ruptured disk at L4-5 will press on the fifth lumbar root but cannot involve the fourth root.

FIG. 4.—A ruptured disk may cause a complete block as illustrated by this case. The patient's original complaint was unilateral sciatica. There were only minor motor and sensory changes. She was completely relieved by removal of a large posteriorly displaced disk fragment.

FIG. 5.—A well-defined characteristic filling defect in the lipiodol column at the level of the L4-5 intervertebral disk. Note the obliteration of the axillary pouch. A ruptured disk fragment 1 cm. in diameter pressing upon the left fifth lumbar root was found at operation.

FIG. 6.—Antero-posterior film showing an hour-glass constriction of the lipiodol column which was found at operation to be due to a markedly thickened or hypertrophied ligamentum flavum. The patient's symptoms and clinical examination were very similar to the ruptured disk group.

FIG. 7.—Antero-posterior and lateral films showing irregular filling and partial block which was found at operation to be due to chronic adhesive arachnoiditis. The defect is not directly opposite the intervertebral disk, and appears to be intradural.

ligament and is directed towards the lamina of the vertebra, usually just opposite the articular facet. It produces an indentation or filling defect in the column of iodized oil, best seen in the antero-posterior view. Unless the defect is very large its presence is obscured in the lateral view by the dense iodized oil lying in the normal side of the canal. The defect is often crescentic or dog-nose in appearance. (Fig. 5, Special Plate.) Minor pressure defects only seen in the lateral view probably represent the normal physiological bulge of the intervertebral disk and have no clinical significance.

Large ruptures may produce a complete or nearly complete block to the flow of the iodized oil, and the appearance may simulate that of an intradural tumour. (Fig. 4, Special Plate.)

Ruptures at the level of the lumbo-sacral disk are more difficult to demonstrate than those higher up, as the dural sac is narrower and smaller here than elsewhere, and so only a small filling defect may be produced by a relatively large protrusion which lies laterally and exerts pressure chiefly on the extradural portion of the nerve root.

Clinical Course and Operative Treatment

The pathological studies of Schmorl have shown that the normal intervertebral disk mechanism is often disrupted and that extrusion of disk material may and does occur in many patients without causing symptoms. It is only when sufficient disk material is displaced posteriorly into the spinal canal to cause one or more roots of the cauda equina to be pinched that symptoms arise. It is doubtless true that many patients with this lesion are relieved by conservative measures. I have under observation one patient with a disk lesion, proved by lipiodol examination, who is perfectly comfortable if the spine is immobilized in a back brace or plaster jacket, but is incapacitated by severe pain if the support is discarded. If the pressure on one or more nerve roots is marked there will eventually be degeneration of the pain fibres and this symptom will disappear. As Foerster has shown, this may entail no peripheral anaesthesia, as section of at least two adjacent roots is necessary if anaesthesia in the dermatome is to be demonstrated. Manipulation of the back or forced straight leg raising under anaesthesia might relieve the pain of a ruptured disk by stretching and paralyzing the pain fibres of the involved nerve root. There is danger, however, of precipitating a paraplegia by injudicious manipulation. This has occurred in two of our cases, one manipulated by an osteopath, the other by a qualified practitioner who thought he was dealing with a sacro-iliac subluxation.

Laminectomy without removal of the protruding mass of disk tissue might relieve symptoms by taking away one limb of the "nut-cracker" which is pinching the nerve root. Spine fusion, if done in a pain-free interval, might prevent recurrence of symptoms and, indeed, has probably been done many times on unrecognized cases of intervertebral disk lesions. Laminectomy and removal of the ruptured protruding portion of the disk is certainly the logical treatment for this condition if conservative non-operative methods fail. (Fig. 2, Special Plate.)

The operative removal of a ruptured disk requires laminectomy followed by a transdural or extradural removal of the disk fragment. If the lesion has been accurately localized by radiological examination the laminectomy need sacrifice no more than one or two laminae on one side of the spinous processes. The articular facets are saved if possible. The dura is usually opened in order to inspect the nerve roots and to remove the iodized oil. The ruptured fragment of disk tissue is generally found lying free beneath the posterior longitudinal ligament and compressed into a rounded mass, which can be seen to displace one or more nerve roots. Usually the most lateral root, anchored at its point of exit from the dura, rides directly over the mass. The root pressed upon may be flattened, and above the point of pressure there may be engorgement of the blood vessels and swelling of the root due to oedema. The larger ruptures may show complete occlusion of the dural sac and lack of pulsation of the dura below the point of pressure. When the posterior longitudinal ligament is incised the mass can be grasped by an Allis forceps and withdrawn. It tends to unroll, and looks like a piece of fascia or tendon when removed. A sinus which leads into the depths of the intervertebral space can always be demonstrated at the site of removal. When the extruded mass is taken away the displaced roots are seen to fall back into normal position.

There is a definite problem as to whether the spine should be fused over the laminectomized area. If an

articular facet has been sacrificed, and if the patient is one who will on recovery lead an active life, we feel that the spine will be stronger if bone chips, supplemented with an osteoperiosteal graft removed from the tibia, are used to bridge the defect. This procedure need not prolong the operation more than a few minutes, as the tibial graft can be removed by a second team at the same time as the laminectomy is being done. The wound is closed in the usual fashion, after careful haemostasis, without drainage. If there has been no fusion the patient is kept recumbent in a firm bed for two or three weeks. If fusion has been done the patient is kept recumbent for four to eight weeks and a brace or jacket is continued for three to six months. Proper physiotherapy aids restoration of correct posture and muscular tone.

When examined microscopically the removed disk fragments may be found to consist wholly of normal nucleus pulposus or a fragment of annulus fibrosus, or of the elements of both. They in no way resemble chondromas or other neoplastic tissue.

Differential Diagnosis

As noted earlier in this paper, the typical case of ruptured intervertebral disk has a rather characteristic history and shows definite physical signs. The difficulty is that the mechanical low back strains often have this identical clinical picture. I have seen cases diagnosed as lumbosacral or sacro-iliac strain after careful examination by various clinicians of the highest repute—cases which were later found to arise from pressure of displaced intervertebral disk tissue on one or more roots of the cauda equina.

Almost every author, in his discussion of the differential diagnosis of low back pain and sciatica, stresses the necessity for ruling out cauda equina tumour, and details the cardinal reflex, motor, and sensory changes which accompany pressure on the cauda equina. A ruptured intervertebral disk may cause enough pressure on the cauda equina to produce obvious paralysis, anaesthesia, etc., but the majority of these cases have protrusions so located and of such size that only one nerve root is pressed upon and the only symptom is referred pain, all objective neurological signs being absent. We cannot accept a negative neurological examination as proof of the absence of this lesion. Neither can we accept the statement that a given case is a "classical picture" of sacro-iliac or lumbosacral strain. In either instance the actual pathology may be intervertebral disk pressure on a nerve root. As Love has said, "There are no physical, neurological, or orthopaedic signs which are found alone in cases of protrusion of intervertebral disks. All of the known signs may also be found in other conditions."

There are certain organic lesions of the low back other than tumours, probably rarer than rupture of an intervertebral disk, that need to be considered in the differential diagnosis. Arachnoiditis, characterized by matting together of the roots of the cauda equina, may cause intractable low back and sciatic pain, which is usually bilateral. Subarachnoid haemorrhage following trauma may be the cause of this condition. Lumbar puncture, if done in the area of fibrosis, will show the dynamic alterations of a complete or incomplete block, or it may be impossible to obtain fluid unless the needle is inserted at a higher level. The lipiodol examination is not characteristic, but an incomplete, very irregular slow filling or a block with an irregular margin is usually noted. (Fig. 7, Special Plate.)

A thickened ligamentum flavum may so constrict the dura as to produce pressure on one or more roots of the cauda equina. Spurling has recently drawn attention to this condition. Can it be due to localized extradural haemorrhage producing fibrosis and thickening of the ligament? Lipiodol examination usually reveals a constricted hour-glass type of filling defect on the posterior and lateral aspects of the spinal canal. (Fig. 6, Special Plate.)

Results

A total of eighty-three cases of lumbar intervertebral disk ruptures producing sciatic pain have been operated on at the Massachusetts General Hospital up to June, 1938. The immediate complete post-operative relief of the sciatic pain is commented upon by practically every patient.

The operation is of considerable magnitude, and should be undertaken only by competent surgeons after a thorough trial of conservative methods or after demonstration of the presence of a dangerous amount of pressure on two or more nerve roots. There have been two deaths in our series; both of these patients had severe irreparable nerve damage from large ruptured disks. There are three known failures. The rest of the patients are either entirely well or are very markedly improved.

Summary

Rupture or herniation of lumbar intervertebral disk tissue into the spinal canal causing pressure on one or more roots of the cauda equina is a well-established clinico-pathological entity, and is the cause of intractable sciatica in many cases. The lesion can be accurately localized by radiological examination after injection of iodized oil into the subarachnoid space. Laminectomy and operative removal of the extruded disk fragment give prompt and complete relief of symptoms in most of the cases.

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ORTHOPAEDIC ASPECTS OF SCIATICA*

BY

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In cases of sciatica a careful eliminative diagnosis leaves a group which is essentially orthopaedic. The physical examination is very important, and represents skilled interpretation of an aggregation of indirect signs which, taken together and supported by an understanding of the local anatomy and bodily statics of the part, can often point to a definite orthopaedic conclusion in the individual case and determine the proper treatment. This is the more likely to be the case if the examiner is able to interpret properly the evidence of stereoscopic films of the low back and pelvic joints.

Vulnerability of Fifth Lumbar Nerve

Orthopaedists try to explain sciatica largely on the basis of referred pain from the sacro-iliac and lumbo-sacral joints and their supporting ligaments. There is also the consideration which Putti has given to the funicular portion of the fifth lumbar nerve as it lies in the foramen. Situated as it is in this fibro-osseous canal, the fifth root, surrounded by a thick venous plexus, practically fills it. The walls of the canal are bounded posteriorly by the posterior spinal articulation, antero-internally by the intervertebral disk, and postero-externally by the lumbo-sacral and ilio-lumbar ligaments. The dorsal primary division of the nerve turns backwards to supply the sacrospinalis muscle, and in its course runs close to the lateral aspect of the posterior articulation, supplying the joint capsule itself. These relations make it possible to assert that the fifth lumbar nerve is in a position of peculiar vulnerability, as it may be affected by the action of trauma of the low back on the lumbo-sacral articulations and by inflammation of them also. It may also be affected by prolapse of the disk or by change in shape and size of the canal secondary to displacement of the fifth lumbar vertebra on the sacrum or to degenerative changes of the intervertebral disk. The slight margin of safety present in the asymptomatic individual by reason, I consider, of the competency of the normal muscular support is further jeopardized by anatomical variations of the lumbo-sacral region, which either cause increased motion at the expense of stability or, by reason of their asymmetry, produce abnormal stresses. In this connexion it is of interest to note that Steindler found anatomic variations in the lumbo-sacral region in 37 per cent. of a large group of cases of sciatica referred to the orthopaedic service.

Circumstances Leading to Sciatica

It may be submitted, then, that many cases of sciatica have their explanation in the operation of a chain of circumstances consisting of: (1) the special anatomic form of the patient, who is either of the unduly heavy or unduly slender build; (2) functional decompensation of the supporting structures of the low back, mainly muscular, often added to by advancing age, unsuitable occupation, increasing weight, debility, and a toxic factor, and leading to faulty posture with exaggeration of the normal lumbo-sacral angle. Such faulty and imperfect use, combined with degenerative changes in the disks and the presence of

G. Bertoni and B. Melli (*Rif. med.*, 1938, **54**, 1526), who record twelve illustrative cases in patients aged from 12 to 35, state that Donaggio's reaction is performed as follows. If a certain quantity of ammonium molybdate is mixed with an equal quantity of normal filtered urine or cerebrospinal fluid, boiled, filtered again, and a watery solution of a basic aniline dye, such as thionin, is added, the colouring solution is completely precipitated by the molybdate and the fluid above the precipitate soon becomes quite clear. In some conditions, however, especially typhoid fever, the urine and spinal fluid acquire the property of checking or entirely preventing the precipitation of the thionin. This phenomenon has been shown by Donaggio to occur when the fluids in question contain a larger amount of organic colloid substances than normally. In typhoid fever the reaction becomes positive at an early stage and remains so throughout the course of the infection.

* Read in a discussion at a joint meeting of the Sections of Neurology and Psychological Medicine, and Orthopaedics and Fractures at the Annual Meeting of the British Medical Association, Plymouth, 1938.

JOSEPH S. BARR: INTERVERTEBRAL DISK LESIONS AS CAUSE OF SCIATICA
(For Legends see Text)

FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 5

JOSEPH S. BARR: INTERVERTEBRAL DISK LESIONS AS CAUSE OF SCIATICA

(For Legends see Text)

FIG. 6

FIG. 7

EDWARD R. ELKAN: THE XENOPUS PREGNANCY TEST

FIG. 1.—*Xenopus laevis* Daud. Female ovulating after injection with pregnancy urine.

FIG. 2A.—*Xenopus laevis* Daud, female. Note valves on both sides of external opening of cloaca.

FIG. 2B.—*Xenopus laevis* Daud, male. Note absence of valves at external opening of cloaca.

FIG. 3.—Test jar for xenopus pregnancy test. The centre of lid is perforated. The toad sits on a platform: the eggs fall to bottom of jar. Note typical attitude, holding nostrils above surface of water.