DIAGNOSIS AND PROGNOSIS OF CAUDA EQUINA SYNDROME PRODUCED BY PROTRUSION OF LUMBAR DISK

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Groups

This paper draws attention to the poor prognosis for complete recovery of function in some patients with cauda equina syndrome due to compression by prolapse or extrusion of lumbar disk. The study of a comprehensive group of these patients in a neurological centre brings out two important points (1) the relative ease of arriving at an early diagnosis; and (2) that early operation is an essential prerequisite for improvement in prognosis.

Clinical Case Material

During a six-year period, 139 of the patients with "lumbar disk syndromes" admitted to the Maida Vale Hospital, under the care of Mr. Valentine Logue, were submitted to laminectomy. Many of them were operated on by me; 13 were suffering from cauda equina compression. The present paper concerns this group of 13, as in these cases there is maximal disturbance of neurological function, and delay in diagnosis and operation results in incomplete recovery of this function.

TABLE I.—*Clinical Case Material*

Patients operated on for "lumbar disk syndrome" during 6-year period ending May 31, 1957	139
Laminectomies performed	142
Compressing disk lesions found and removed	138
Neurofibromatous thickening of nerve root	2
"Negative" exploration	2
Posterior rhizotomy	1
Radiculysis	1

TABLE II.—*Analysis of Positive Disk Lesions*

Patients with positive disk lesions	136
Laterally situated lesions	97
Centrally	41
Patients with multiple lesions	2
Total compressing disk lesions	138
Patients with cauda equina syndrome and having central disk prolapse or extrusion	13

Tables I and II show that whereas the cauda equina syndrome occurs in less than one-tenth of the patients in this series, it was present in nearly one-third of those whose disk was central in position.

Manner of Involvement of the Cauda Equina

The cauda equina consists of all the anterior and posterior roots which leave the lumbar theca below the lower end of the spinal cord (opposite L 1-2 disk), so it comprises all the lumbar roots with the exception of the first and all the sacral and coccygeal roots. A "total" involvement of the cauda is most unusual, and such is not implied by the term "cauda equina syndrome." The term is used here to mean compression of the lower part of the cauda equina—as all except two of the compressions were at the L 4-5 level or lower. The two exceptions were at L 3-4 level.

In all cases involvement of the cauda equina at and below the level of the disk compression was incomplete. In some cases there was asymmetry of impairment of function as between the two sides, and in all there were differing degrees of disturbance of "motor, sensory, and sphincter" function.

As used in this paper the terms "motor" and "sensory" refer to limb supply only and "sphincter" refers to both motor and sensory supply to the bowel and bladder and not only to their sphincters. It is used as a convenient clinical term only, and is appropriately qualified in the text where necessary.

Motor Manifestations.—These included symptoms and signs of limb weakness or of weakness of some movement, evidence of hypotonia in the limb or some muscle groups, and diminished or absent reflexes.

Sensory Manifestations.—These comprised symptoms of numbness or various paraesthesiae and objective signs of sensory loss in a dermatomal and myotomal distribution.

Sphincter Involvement.—In respect of bladder function this took the form of difficult micturition or retention of urine; failure of appreciation of the filling of the viscus or loss of urethral sensation; and, in some cases, stress incontinence. The main bowel disturbance was constipation, and, in most cases, lack of appreciation of rectal distension and loss of anal sensation. Loss of sexual power occurred in some patients.

Classification of Patients: Features in Diagnosis

The 13 patients fall into two groups; group 1 comprising two cases (Nos. 1 and 2) and group 2 11 cases (Nos. 3 to 13).

Group 1.—In these two patients the syndrome developed slowly—over a period of six years in Case 1 and of one year in Case 2, remaining incomplete in both. There was no history of preceding injury or lifting strain; at operation only a diffuse backward bulge of the disk was found; and the prognosis was better than in the other cases.

Group 2.—These cases were characterized by the rapid development of some or all of the features of the cauda equina syndrome. In all cases this syndrome followed an episode of severe pain, and in all save one there was a history of injury, lifting, or twisting strain of the back. At operation the findings were characteristic, and the prognosis was not so good as in Cases 1 and 2. Improvement in results may be expected if the diagnosis is made and definitive surgical treatment carried out earlier. Therefore these cases are discussed in further detail.

Role of Injury or Back Strain.—Injury or back strain was present in all cases in group 2 except Case 3, and in most there were at least two distinct incidents—one before the onset of any back or limb symptoms and again before the onset of the cauda equina syndrome. The injury or strain prior to development of the syndrome was severe in seven patients and mild in two. It is not intended to suggest that such cauda equina syndromes are "traumatic" in origin but that in most cases of this type some lifting or twisting strain of the spine is associated with prolapse or extrusion of part of a disk already degenerated. Such history of injury is mainly of interest in differentiating between disk and tumour as the cause of cauda equina compression. (Jennett (1956) attaches less importance to the role of injury immediately preceding the onset of the cauda equina syndrome. However, such a history was obtained from 10 of the 11 patients.)

Total Duration of All Symptoms.—This varied from 3 months to 15 years, with a mean of 5 years and 11 months. The symptoms consisted solely of back pain

in the lumbar region in six patients, back pain with radiating symptoms in five, and radiating symptoms only in two. In group 1 patients the cauda equina syndrome developed slowly during the whole period of the history (six years and one year respectively). In group 2, only one patient (Case 6) had a sudden onset of the syndrome without premonitory back or lower-limb symptoms. In the other 10 cases back or radiating symptoms preceded the onset of the syndrome by months or years, ranging from 2 months in Case 3 to 15 years in Case 4, giving a mean period for the preceding symptoms of six years and eight months.

Duration of the Syndrome.—The mean duration of symptoms of cauda equina compression in group 2 was 17 weeks. If the two neglected cases (Nos. 6 and 12) are excluded the mean duration was four weeks and two days—varying between five days in Cases 5 and 10, and seven weeks in Case 8. These periods differ markedly from group 1 cases, where the mean duration was 3½ years.

Rate of Development of the Syndrome.—This also showed a marked difference between the two groups. In group 1 the syndrome developed in a gradual manner, and in group 2 it came on relatively very quickly. The following criteria have been used to determine the presence of the syndrome: time of onset of urinary retention, numbness in the lower limbs, or (in those cases without sphincter or sensory symptoms) definite weakness of the legs. With these criteria it has proved possible to make an estimation, accurate within a few hours, of the time during which the cauda equina syndrome became established. Table III brings out the shortness of this period relative to the duration of the syndrome. It was 12 hours or less in five cases, 7 days or less in five, and in only one (Case 6) was the duration more than a week.

Time of Onset of Radiating Symptoms.—This was easier to establish with certainty and was taken to include radiating lower-limb pain, numbness, or tingling paraesthesiae. Table III shows that such symptoms

occurred within a few minutes in 6 cases, within a few hours in two, and in less than a week in three.

Clinical Features

Most of these are brought out in Table IV. Only the more important features are discussed here.

Group 1.—The main impact of the compression in both patients was on the sensory roots and on the fibres to the bladder. There was no involvement of motor function in Case 2 and it was very slight in Case 1. Sphincter involvement was slight in both cases.

Group 2.—These cases fall naturally into two subgroups—with and without sphincter involvement. **Patients with sphincter involvement:**—In respect of the bladder this took the form of difficult micturition or

TABLE IV.—Summary of Clinical Features

Case No.	Sex and Age	Occupation	Main Symptoms and Signs	Period of Sphincter Involvement	Returned to Work	Length of Follow-up
1	M 51	Labourer	M+S+Sph	2 years	3 mths. (L.W.)	6½ yrs.
2	M 48	Chemist	S+Sph	4 weeks	2 " (O.O.)	6 "
3	F 38	Housewife	M+S	—	4 " (L.W.)	3½ "
4	M 52	Shipping manager	M+S+Sph	5 weeks	4 " (O.O.)	3 "
5	M 33	Cinema projectionist	M+S+Sph	5 days	4 " (O.O.)	3½ "
6	M 53	Stock jobber	M+S+Sph	2 years	6 " (O.O.)	4 "
7	M 39	Tulip exporter ..	M	—	3 " (O.O.)	2½ "
8	M 42	Tool-setter	M+Sph	4 weeks	5 " (O.O.)	2 yrs. 2 mths.
9	F 36	Rubber packer ..	M+S+Sph	6 days	22 mths.; not returned	1 yr. 10 mths.
10	M 40	Baker	M+S+Sph	5 "	3 mths. (L.W.)	1 " 9 "
11	F 40	Housewife	S+Sph	2 months	6 " (L.W.)	1 " 7 "
12	M 50	Labourer (Cypriot)	M+S+Sph	10 "	Not known	—
13	M 41	Lathe turner ..	M	—	5 mths. (L.W.)	1 yr.

M=motor; S=sensory; Sph=sphincter; O.O.=original occupation; L.W.=lighter work.

retention in eight patients (Cases 4, 5, 6, 8, 9, 10, 11, and 12); six of these had altered bowel function and five had loss of urethral and anal sensation. Six of the patients with sphincter involvement also had motor and sensory symptoms and signs (Cases 4, 5, 6, 9, 10, and 12); one had sensory manifestations (Case 11), and one motor manifestations (Case 8). **Patients without sphincter involvement:**—There were three patients in this subgroup (Case 3 with sensory and motor features, and Cases 7 and 13 with motor manifestations only). The details of motor and sensory involvement are not shown in Table IV and are not given for the group as a whole; they are, however, described in the case reports.

Factors in Prognosis

Pain

Pain in the lumbar region of the spine and radiating in the distribution of sciatic or femoral nerves is present in most cases with this syndrome, and, while of value in making the diagnosis, requires consideration in prognosis. Pain may be absent from the outset (rare) or may disappear during the course of the illness (sometimes suddenly). This may be due to involvement of motor roots only—or to cessation of conduction in pain fibres. The latter is the more usual cause and is associated with objective sensory changes. Such cessation of conduction may be temporary if the roots are not destroyed, or permanent if there is such destruction. But in a particular patient there is no way of being sure which of these two possibilities exists—other than by the passage of time. Accordingly early operation should be the rule.

A good prognosis should not necessarily be given because of the absence or cessation of pain, as clearly in

TABLE III.—Time Relations and Role of Injury in the Syndrome (Group 2)

Case No.	Total Duration of Symptoms	Injury or Strain		Time Interval Between Pain or Strain and Onset of Radiating Symptoms	Period During which Syndrome Developed	Duration of Syndrome Before Operation
		Preceding Past History	Preceding Cauda Equina Syndrome			
3	3 months	Nil	Nil	12 hours	Within 12 hours	1 month
4	15 years	Yes	Yes	Few minutes	Within 1 week	6 weeks
5	11 months	"	"	" "	Within 12 hours	5 days
6	2 years	"	"	" days	Within 4 weeks	2 years
7	13 "	"	"	" minutes	3 days	1 month
8	12 "	"	"	" "	Within 7 weeks	7 weeks
9	2 "	Nil	"	2 days	12 hours	10 days
10	4 "	"	"	Few minutes	4 days	5 "
11	13 "	"	"	" hours	Within 24 hours	2 months
12	5 "	Yes	"	" minutes	Within 12 hours	10 "
13	3 "	"	"	6 days	6 days	6 weeks

some cases there is, in these circumstances, *progression* of the syndrome and therefore a worse prognosis. For example, in Case 6 there was complete disappearance of the pain (some weeks) after the onset of the syndrome. Had there been persistence of pain after the first operation failed to disclose the disk prolapse, presumably the patient would have demanded earlier reassessment of his problem (there was a two-year interval between the two operations). In Case 13 such improvement of pain did in fact cause delay in referring the patient for operation. In all patients with pain, rapid relief of this pain was obtained by operation.

Sphincters Involved

In *all* such patients there was incomplete return of normal micturition. In Cases 6 and 12 (syndrome present for two years and for 10 months respectively) automatic bladder function became established. Of all symptoms those of sphincter disturbance are the most important for assessing prognosis.

Sensory Disturbances

In all patients with sensory involvement there was little return of innervation—for example, Cases 1 and 5, in which examinations were made 6½ and 3½ years after the operations. O'Connell (1950) gave an explanation for this persistence of sensory deficit—that is, the compression of the sensory-nerve root occurs central to the ganglion cells, so that regeneration of destroyed fibres cannot take place. The sensory deficit in disturbance of the sphincters is likewise more persistent than motor loss. So that, in the absence of awareness of bladder-filling, emptying of the viscus is possible owing to recovery of power in the bladder detrusor, assisted in the initial stages by manual compression (see report of Case 5).

Motor Involvement Only

There were two patients in this series (Cases 7 and 13) solely with motor involvement. In both a fair return of power took place before leaving hospital, but return to work was delayed for three and five months respectively. In these cases the syndrome was present four and six weeks before operation. Recovery was finally complete in Case 7; in Case 13 there was slight weakness of dorsiflexion of the left great toe one year after the operation.

Duration of Syndrome

If Cases 6 and 12 are excluded, the duration of the syndrome (within the time range of the remaining patients), if considered alone, is less important in prognosis than the presence of sphincter or sensory involvement. However, the duration of the syndrome need not be considered in isolation and should be assessed only in association with the other factors discussed above. In the slowly developing incomplete compression (Cases 1 and 2) the duration was not significant in the prognosis, being 6 years and 1 year and recovery occurring rapidly in both. In the neglected patients (Cases 6 and 12) the long duration of the syndrome with complete loss of voluntary bladder control will result in recurring inflammatory episodes (already present in Case 6) and shortening of life from eventual ascending urinary infection.

In the remaining nine patients the picture is not so clear. In Cases 5 and 10 the syndrome was present for five days before operation, and sphincter and sensory recovery have remained incomplete, whereas in Case 7 early return of function occurred though the syndrome had been present for one month. However, in this patient only motor roots were being compressed.

There can be no certainty on a clinical basis alone what proportion of nerve fibres (sensory, motor, or to sphincters) are destroyed at the initial prolapse or extrusion and in what proportion there is (potentially) only a temporary loss of function from compression or interference with blood supply. Therefore it is important to make the diagnosis and carry out the operation as early as possible.

If, in this series, at the time of operation, there had been some patients with syndromes present for 24 hours or less (and in six the syndrome developed within 24 hours) a more truly significant relation might have been drawn between duration and prognosis.

Incomplete Compression From a Slowly Developing Lesion

Patients with such lesions (Cases 1 and 2) have a relatively favourable prognosis. However, as can be seen from the report of Case 1, recovery is earlier and more complete in respect of motor power and longer delayed, and remains incomplete for sensory and sphincter function.

Case 1

This patient, a man of 51, had been a miner for 15 years and then a heavy labourer for 20 years. He had had lumbago for six years prior to admission, and during this period he noticed progressive weakness of his legs. In addition, he had attacks of increasing pain and numbness which came on while working—starting in his feet, radiating up the legs, and becoming so severe that he had to sit down for relief. The symptoms would then subside and he could resume working. Weakness was associated with these attacks, and in succeeding years the bouts of weakness, numbness, and pain on walking progressed in severity and came on after shorter distances, so that just before admission merely movement about his house brought on the attacks. He had difficulty in micturition and absence of anal sensation for two years.

Examination showed marked bilateral wasting of glutei, hamstrings, and calf and small foot muscles, with hypotonicity, weakness, and absent ankle-jerks; cutaneous sensibility was impaired in all sacral dermatomes. There was no evidence of vascular insufficiency in the lower limbs.

Myelography revealed a partial block at L4-5, and at laminectomy Mr. Logue found a large central disk protrusion. This took the form of a diffuse backward bulge of the whole disk and there was no real extrusion of cartilage. The radiating symptoms quickly improved and were no longer present three months after operation. The power also quickly returned to normal in three months. Further recovery was only slight, but when last seen 6½ years after operation he could walk for long distances (for 4 to 6 miles; 6.5 to 10 km.) compared with 20 yards (18 metres) before the operation. He was still having a little difficulty with micturition. On examination at this time there was slight weakness of eversion of both feet. Sensation was more affected, there being marked hypalgesia in S2 and 3 dermatomes and slight loss of pain in the fifth sacral dermatomes.

Comment.—This case illustrates the features of group 1 patients: absence of history of injury or spinal strain, slowly progressive development of the cauda equina syndrome, and good initial recovery.

Case 5

A cinema projectionist aged 33 first noted back pain 11 months before admission after lifting a heavy film projector. A prolapsed intervertebral disk was diagnosed at this time and treated with a surgical belt for three months with relief. Two further episodes of back pain occurred three months and two weeks prior to admission.

Five days before admission and following a jolt on his motor-cycle he noticed a similar pain in his back. On this occasion it persisted and was associated with bilateral sciatica and radiation into the buttocks. During the next 12 hours the pain became very severe indeed, with more widespread radiation into the legs and ankles. Morphine injections were given to relieve it and he was seen in a surgical out-patient department next day, when retention of urine had developed. He was referred back to his own doctor, who relieved the urinary retention by catheter. Constipation developed with the urinary retention and complete numbness in the saddle area. There was very little weakness of the legs.

Examination revealed obliteration of the lumbar lordosis, spasm in the erector spinae muscles, poor and painful spinal movement, and reduction of straight-leg raising to 45 degrees right and left. He had a symmetrical cauda equina syndrome involving all sacral segments on both sides, with hypalgesia in the first and second sacral dermatomes and analgesia in the third, fourth, and fifth dermatomes, including loss of urethral and anal sensation. Wasting, hypotonicity, and slight weakness of gluteal and hamstring muscles were apparent but no weakness of foot movements. Both ankle-jerks were absent. The myelogram showed complete block at the lower border of L 4 vertebral body (Fig. 1), and at operation I removed a disk extrusion measuring 2 by 1 by 1 cm. After operation the return of bladder function was slow, and on discharge from hospital six weeks later manual compression was still needed. After six months the sensation of bladder-filling had not yet returned, manual assistance was still required occasionally, and the saddle anaesthesia showed no obvious change. After one year there was still straining on micturition, troublesome constipation, and little change in sensation. This patient was seen twice more, 1½ and 3¼ years after operation. There had been no further significant change in symptoms, and examination showed the sensory change in the sacral dermatomes as before—that is, analgesia in S3 to 5, hypalgesia in S1 and 2. He had been rendered impotent by the disk extrusion and remained so. He was free from pain.

Comment.—This case illustrates a history of previous back pain, the role of back strain preceding the onset of symptoms of cauda equina compression, and the

short time required for full development of the syndrome. Of interest also is the relative sparing of motor function with marked involvement of sensory, sphincter, and sexual function. In regard to prognosis it should be stressed that, though operation was done within five days, return of bladder function was still incomplete a year later, and sensory impairment was virtually unchanged over three years after the operation. The importance of testing sensation within the sacral dermatomes is well exemplified by this case (this can only be done adequately if the patient is in the prone position).

Case 8

A tool-setter aged 42 was admitted to hospital in November, 1955. He had had recurring attacks of low back pain for 12 years—four or five such attacks yearly, each lasting for some two to three weeks. He had sustained an initial severe jolt to the spine during the battle of Alamein, when he was thrown out of a dugout after a direct hit by a shell. Some of the subsequent episodes of pain he associated with small strains, such as coughing, sitting up, stepping off a kerb, etc.

The attack which culminated in his cauda equina syndrome began seven weeks before admission. On getting out of bed one morning and on attempting to gain the erect position he had a sudden pain in his back, with immediate radiation down both legs to the feet accompanied by a numb sensation. Acute retention of urine developed later, and he was admitted to another hospital and treated for acute prolapsed intervertebral disk. Urinary retention proved to be of short duration. He was treated by catheter and was later referred to another hospital under the care of a neurologist. Examination there showed wasting of calf and anterior tibial muscles of both legs, with hypotonicity and marked weakness resulting in bilateral foot-drop and loss of ankle-jerks. Disturbance of cutaneous sensibility was minimal—hyperpathia to pin-prick in all the sacral segments but no actual loss of sensation.

Myelography revealed a large anteriorly situated lesion opposite the L 4-5 disk space. Mr. Logue felt there had been a massive extrusion of the L 4-5 disk, and on December 3 carried out laminectomy. An extensive prolapse of this disk was found, with several large necrotic fragments of cartilage within it. The disk protrusion was estimated to be the size of a marble and had produced a tight compression of the lower part of the lumbar theca and cauda equina.

The post-operative and subsequent course was favourable. The position became static about five months after operation.

Since that time he has had minor complaints only—very slight pain in the back and radiating tingling sensation from feet to knees. He was last seen 2 years and 2 months after operation, when there was almost complete return of power—there still being slight weakness of extension of the left ankle and hallux. The ankle-jerks were still absent and there was hypaesthesia in L 5-S 1 dermatomes bilaterally.

Comment.—This case illustrates the role of injury, the long preceding history, and the suddenness of onset of the cauda equina compression. The severity of involvement of the motor fibres, with almost complete escape of sensory roots and of bladder function, is of interest, especially in view of the tight compression found at operation. The relatively good prognosis with almost full recovery in five months is worthy

FIG. 1.—Myelogram showing complete block to downward flow of ethyl iodophenylundecylate opposite L 4-5 disk.

of comment, even though the lesion had been present for seven weeks before operation.

Case 13

This patient, a metal lathe turner aged 41, doing heavy work, had had occasional episodes of lumbago for years.

One morning, doing a twisting movement, he felt "something pop in my back." There was a little mild pain, which built up during the course of the morning and became so severe that at lunch-time he returned home and went to bed. The pain continued for six days, when for the first time he noticed weakness of his legs. This weakness progressed rapidly and was accompanied by cramps in his calves. Later the pain became intense, radiating down the whole of the right leg and to a less degree down the left leg. His legs were so weak that he had to be helped to bed. His doctor visited him the next day and diagnosed a prolapsed disk. There followed a fortnight of delay before admission to hospital. The pain was severe during this period and was aggravated by coughing or sneezing.

In hospital he was put on fracture boards for two weeks and then encouraged to get up for a further week. His pain improved greatly but the legs remained weak. Other sensory symptoms or signs did not appear and the sphincters were not involved. After about a month he was seen by a neurologist, who arranged for immediate transfer to the Maida Vale Hospital in February, 1957.

Examination revealed a flaccid paraparesis involving gluteal, hamstring, and especially flexor and extensor groups of both legs. There was slight weakness of the quadriceps on both sides, but the knee-jerks were present. The ankle-jerks were absent. There were no sensory signs.

A myelogram revealed an almost complete block at the lower border of L3, and at laminectomy an extruded portion of disk (5 by 0.5 cm.) was removed. This was curled up and pressing back into the theca and had gone from the L 3-4 disk through a small hole in the thinned annulus and posterior common ligament. There was no disturbance of bladder or sensory function post-operatively. Recovery of motor-power was relatively rapid and on discharge from hospital five weeks after the operation he was able to walk, though with a waddling, shuffling gait. However, he did not return to work for five months and did not regain full use of the legs until nine months after operation. When last seen, one year post-operatively, there was very slight weakness of dorsiflexion of the left great toe as a remnant of his neurological disability.

Comment.—This case illustrates the selective involvement of the cauda equina and the more favourable prognosis when the nerve supply to the sphincters and the sensory roots are not involved clinically. The relative freedom from pain and lack of bladder and sensory disturbance may have had a bearing on the lamentable delay in arranging transfer for operation.

Case 9

This patient aged 36, was a housewife and part-time worker in a rubber factory. Two years previously she had a single attack of right sciatica. Ten days before admission to hospital she suffered sudden low backache. This cleared for a day, then returned and persisted. After four days it was associated with radiating pain in the right lower limb. On the same day retention of urine was noted. Next day tingling paraesthesiae and weakness appeared in both feet. For two weeks immediately preceding this episode she had been nursing her bedridden mother.

Examination revealed symmetrical motor and sensory involvement of the cauda equina with flaccidity and weakness of all muscles below the knees, less marked wasting, and weakness of gluteal and hamstring muscles. Marked diminution of all forms of sensation was found in all the sacral dermatomes. Urinary retention and

constipation were evident. A myelogram revealed a complete block opposite the body of L4.

At operation I removed two large necrotic fragments of disk, lying free in the spinal canal. These had extruded from the L 4-5 disk and had extended up the canal to lie above the disk level.

About a month after operation micturition had become relatively normal. Power returned to the limbs much more slowly and there was virtually no change in sensation. When seen 1 year and 10 months after operation she had not returned to her employment in the rubber factory and required the assistance of a stick and leg-iron for walking. On examination the proximal muscle groups (gluteal and hamstrings) had regained their bulk and power. The distal muscles were very weak. Sensation was virtually unchanged from the pre-operative state.

Comment.—This case illustrates the poor prognosis in some cases. It is especially so when the sphincters and sensation are involved in addition to motor loss.

Discussion

Diagnosis of the cauda equina syndrome can be made by evaluation of the history and by clinical examination. When such a lesion is suspected judicious inquiry should be made for symptoms indicating compression of lumbar or sacral nerve roots. The most important of these are: weakness of muscles below the knees, impairment of skin sensation in the saddle area, difficult micturition, and radiating symptoms. Radiating symptoms consist of pain, tingling, and other paraesthesiae or numbness. Constipation may go unnoticed if it represents little change in the patient's usual bowel habit.

Examination will be directed especially towards the spine, lumbar and sacral skin and muscle segments, and the bladder. The only way of examining the sacral segments adequately is with the patient prone. Even small degrees of wasting of the buttocks will be apparent; loss of tone, weakness of the gluteal muscles, and sensory loss in the sacral dermatomes can be readily demonstrated. One or both ankle-jerks are absent in most of these cases. A distended bladder not uncommonly escapes casual observation.

The cauda equina syndrome is only very rarely complete—in disk cases, only the lower lumbar, sacral, and coccygeal roots being involved—and is often asymmetrical in regard both to right and left sides and to manner of involvement of "motor," "sensory," and "sphincter" supply. The most common, and remediable, cause of the syndrome is central prolapse of a lower lumbar intervertebral disk. In most of such cases there is a long history of lumbar back pain—constantly or more commonly as attacks of lumbago—or radiating symptoms, and often a back strain due to twisting or lifting, sometimes slight in degree and apparently trivial, immediately before the onset of the symptoms of cauda equina compression.

There have been surprisingly few articles in the British medical press on this syndrome. O'Connell (1950), in a review of 500 surgically treated cases, found compression of the cauda equina in only 2%. He wrote: "The only absolute indication for operation is the sudden development of severe neurological disturbance as a result of involvement of the cauda equina by a large protrusion." A paper by Jennett (1956) reported 25 cases of the syndrome culled from about 1,000 verified disk lesions over a period of 18 years. These two papers contain reviews of the more important publications on the subject up to 1956.

The number of patients in the present series represents a high incidence relative to the total number of patients suffering from lumbar disk disease. The incidence is also high compared with other series. This may be due to the method of reference of patients with disk disease to the neurosurgical unit of the Maida Vale Hospital. All in this series had been examined by one or more consultants before transfer.

However, the cauda equina syndrome as produced by lumbar disk prolapse or extrusion is not so rare as is generally supposed. In the first nine months' tenure of my present appointment I have had three patients suffering from this syndrome referred to my care. Fig. 2 is a photograph of a "disk sequestrum" removed at operation from the last of these patients.

FIG. 2.—Single extruded fragment of disk comprising bulk of nucleus pulposus.

Mention is required of the association of manipulation and the cauda equina syndrome. Such a manoeuvre precipitated the onset of the syndrome in one patient in this series. If there is any evidence to suggest a central disk prolapse not yet producing a cauda equina syndrome, manipulation should be avoided. The finding at operation in a number of these patients of a thinned annulus and posterior common ligament, and the fact that so many of the frank cases of the syndrome follow relatively trivial traumata—acting on an already diseased disk—indicate the danger of manipulation.

The profound disturbance of function which follows acute central prolapse or extrusion of a lumbar disk is clear from a study of the patients forming the basis of this paper. The slow return of this function, with delay in diagnosis and operation, is also apparent. These lesions do not of themselves endanger life. However, when no relief is afforded, the paralysis of bladder and sensory function leads to ascending urinary infection and decubitus ulcer formation, and so result in a shortening of the life-span. Uraemia or septic absorption from bed-sores brings to an early end a miserable and limited existence.

Summary

In order to improve the prognosis attention is drawn to the need for early diagnosis and operative treatment of patients with the cauda equina syndrome due to lumbar central disk prolapse or extrusion.

Thirteen patients have been chosen for study, being nearly one-third of those having central lumbar disk lesions and one-tenth of those with compressing disk lesions verified at operation and seen over a six-year period.

The 13 patients are classified into two groups. The main diagnostic features are described, the role of injury and the rapid development of the syndrome in most cases being specially stressed.

The manner in which the cauda equina may be involved is described.

Factors in prognosis are mentioned: pain; sphincter, sensory, and motor involvement; and duration of the syndrome. Their relative importance is discussed.

Five illustrative case-histories are given, and the main clinical features of the whole series are tabulated.

The discussion includes remarks concerning the diagnosis and frequency of the syndrome. The possible danger of manipulation is mentioned and allusion made to the plight of the unrelieved sufferer from this condition.

I thank Mr. Valentine Logue for granting me access to his personal notes of these patients and for allowing me to perform many of the operations. I am indebted to Dr. D. Sutton for permitting me to include Fig. 1. My thanks are due to the medical committee of the Maida Vale Hospital for allowing me to publish. I am grateful to Mrs. Davis for secretarial assistance.

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PULSELESS DISEASE, OR TAKAYASHU'S DISEASE

CASE REPORT

BY

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Cases of unilateral absence of pulse due to aortic aneurysm have been known since 1757, when William Hunter described the condition, but bilateral absence of the arterial pulses is exceedingly rare. In 1908 Takayashu reported a peculiar case with cataracta complicata and unusual ocular findings. At the meeting at which the case was described Onishi and Kagoshima reported two similar cases, with the addition that radial pulsation was absent in one of the patients. It was not, however, until the review by Shimizu and Sano (1953) that this pulseless-disease-aortic-arch syndrome, or reversed coarctation, became recognized by Japanese ophthalmologists as a rare but definite entity. Since that time 69 cases have been reported from Japan, and 34 from Europe and America, including several in recent years. The present paper records a further case, the first one from India.

Clinical Signs and Symptoms

Clinically, pulseless disease is characterized by three cardinal signs; the most common is absence of palpable