LOW BACK PAIN TREATED BY MANIPULATION

A CONTROLLED SERIES

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The patient suffering from low back pain provides one of the commonest problems encountered by the general practitioner. Working hours lost through this form of disability must add up to a vast total. The possible causes of backache are legion, and accurate diagnosis may be difficult. In 1951 the Mayo Clinic reported a series of 2,000 cases in which the cause of chronic back pain was investigated, and in 20% no definite diagnosis was reached.

Patients complaining of acute low back pain, however, form a definite clinical group. The onset is often related to strain or trauma. The lesion in such cases must be in the region of the intervertebral joints and adjacent structures. Various controversial opinions are held concerning the exact nature of these lesions and are expressed in such terminology as fibrositis, muscle strain, sacro-iliac strain, slipped disk, etc.

Manipulation as a method of treatment for such cases has been recommended by a few clinicians but condemned by the majority, although it is regularly practised, with apparent satisfaction to the patients, by the medically unqualified manipulator. To the best of our knowledge, no attempt has been made to compile a controlled series to settle the dispute. In this preliminary report we have not entered the controversy on diagnosis; we have simply sought to establish whether or not manipulation has a place in the treatment of recent low backache. Certain established concepts of the causation of low back pain are, however, worthy of mention, as their consideration has led to the form of this investigation.

The normal spine, with its series of interlocking bony units and intervertebral disks, is a resilient structure. The adoption of the upright posture and resultant curvatures provide areas where stress and strain are maximal. Boyle (1954) showed that spondylosis in the dorsal and lumbar spine most commonly affected the joints between the seventh and eighth dorsal and the third and fifth lumbar intervertebral bodies. Degenerative disk changes are common in these areas and begin early in life, leading to fibrosis, desiccation, fissuring, and possible protrusion of the cartilaginous or pulpy portions of the disk. Cyriax (1945) has postulated that minor protrusions bulging the posterior longitudinal ligament, not severe enough to cause nerve-root pressure and sciatica, can cause pain; and that this latter is due to pressure on the adjacent dura mater. Though derangement of the intervertebral joints is undoubtedly responsible for much acute lumbar pain, it is obvious that the pendulum has swung too far and diagnosis of a "disk lesion" is apt to be made too readily. Tears of adjacent muscles and ligaments may occur, or avulsion fractures of transverse and spinous processes. Spondylolisthesis, the presence of transitional vertebrae, and other minor

congenital abnormalities may predispose to stress and strain injuries of the lower lumbar spine by altering local mechanics. Willis (1929) found the fifth lumbar vertebra partially sacralized in 4.7% and completely sacralized in 1.5% of 1,500 skeletons. Partial lumbarization of the first sacral vertebra in 1.2% and complete lumbarization in 4.1% of the same series were discovered.

Crisp (1952) stressed the possibility of injury to apophyseal joints and nipping of synovial fringes as a cause of acute lumbar pain which is amenable to dramatic relief by manipulation.

Whenever manipulation is considered one must always be aware of the rarer and more serious causes of low back pain. These must be excluded by careful clinical and radiological examination. Such causes include osteomyelitis, tuberculous caries, ankylosing spondylitis; fractures, vertebral collapse, and other sequelae of trauma, osteoporosis, or neoplasm; Paget's disease, intraspinal lesions, and pain referred from pelvic structures. These conditions are, of course, absolute contraindications to treatment by manipulation.

Method

It was decided to divide into two groups, without selection, all patients presenting with recent low back pain. One group was treated by manipulation of the lumbar spine, without anaesthesia, according to the methods described by Cyriax (1950). The other group was treated by bed rest, the use of a lumbar pillow, and analgesics. All patients were advised on the posture to be maintained while standing and sitting, and told to avoid spinal flexion and especially bending and lifting. It was of course essential that there should be no contraindication to manipulation in any of these cases, as determined by full clinical and radiological investigation. Any patient complaining of pain radiating below the buttock, or with neurological signs such as diminished reflexes in the lower limbs, muscle weakness, or paraesthesiae, was rejected for the purpose of the present investigation. A detailed history, based on a carefully considered plan, was taken in every case and a full clinical examination made by one of us. The lumbar spine and pelvis were x-rayed in all cases. The information was transferred to a series of punch cards, facilitating the future assessment of each case. Each patient was seen at weekly or shorter intervals for assessment of signs and symptoms indicating improvement or deterioration. The basic features noted in the history and examination were as follows.

(1) Sex. (2) Age group. (3) Number of previous attacks of pain. (4) Site of pain, whether bilateral or unilateral. (5) Radiation of pain. (6) Whether aggravated by coughing or sneezing. (7) Whether pain is experienced on rising from bed. (8) Whether cramps are present. (9) Whether a soft or sagging bed is used. (10) Whether employment is significant (strain). (11) Whether there is a history of associated trauma. (12) Whether improving spontaneously. (13) Whether any leg shortening ($\frac{1}{4}$ in. (1.3 cm.) or more). (14) Lumbar lordosis. (15) Lumbar kyphosis. (16) Scoliosis—adolescent; scoliosis—relevant. (17) Other deformities. (18) Hip-joints. (19) Back flexion restricted. (20) Back extension restricted. (21) Back lateral flexion restricted. (22) Back rotation restricted. (23) Straight-leg raising restricted. (24) Any abnormality of knee-jerks, ankle-jerks, or plantar reflexes. (25) Any muscle weakness in lower limbs. (26) Any loss of sensation in lower limbs. (27) Any sign of sacro-iliac joint disease. (28) Radiological evidence of congenital abnormalities, disk degeneration, or bony disease of any kind. (29) Type of treatment employed. (30) Time in weeks after treatment started before patient is free of signs and symptoms.

As previously stated, the patients in this series were placed alternately on either manipulative treatment or rest in bed and analgesics. Some of them did not carry out instructions or did not report for assessment, and have been discarded from the final series. Thus the original two groups, each of 76 patients (152 patients in all), are unequal in the final figures (76 and 60).

Results

The results of the investigation are given in tabular form. The patients treated by manipulation are referred to as Group I and those treated by rest and sedation as Group II. Age and sex distribution are shown in Table I. It was remarkable that the proportion of males was the same in each group, being 62%.

TABLE I.—Age and Sex Distribution

Age	Group I (76)			Group II (60)		
	Total	M 47	F 29	Total	M 37	F 23
	No. %	No. %	No. %	No. %	No. %	No. %
0-19	2 3	1 2	1 4	2 3	1 3	1 4
20-29	19 25	10 21	9 31	8 13	3 8	5 22
30-59	50 66	33 70	17 58	45 75	29 78	16 69
60+	5 7	3 6	2 7	5 8	4 11	1 4

Table II shows the number of previous attacks of acute lumbar pain in both groups.

TABLE II.—Previous Attacks

No.	Group I (76)			Group II (60)		
	Total	M 47	F 29	Total	M 37	F 23
	No. %	No. %	No. %	No. %	No. %	No. %
1	22 29	15 32	7 24	17 28	11 30	6 26
2 or 3	4 5	3 6	1 3	9 15	6 16	3 13
More than 3	22 29	15 32	7 24	12 20	5 13	7 30
None	28 37	14 30	14 48	22 37	15 40	7 30

Information regarding the site of pain is given in Table III.

TABLE III.—Site of Pain

	Group I (76)			Group II (60)		
	Total	M 47	F 29	Total	M 37	F 23
	No. %	No. %	No. %	No. %	No. %	No. %
Central lumbar	54 71	33 70	21 72	42 70	30 81	12 52
Unilateral	33 43	21 44	12 41	20 33	12 32	8 35
Bilateral	13 17	5 11	8 28	11 18	8 22	3 13
Radiation pain	28 37	17 36	11 38	24 40	15 41	9 39

Other information obtained from the history of the patients is shown in Table IV.

TABLE IV

	Group I (76)			Group II (60)		
	Total	M 47	F 29	Total	M 37	F 23
	No. %	No. %	No. %	No. %	No. %	No. %
Aggravated by coughing	46 60	30 64	16 55	41 68	25 68	16 69
Sagging bed used	14 16	9 19	5 17	17 28	12 32	5 22
Employment significant	25 33	22 47	3 10	20 33	19 51	1 4
Associated history of trauma	39 51	25 53	14 48	27 45	19 51	8 35
Improvement occurring	26 34	16 34	10 34	22 37	14 38	8 35

Information obtained from clinical examination is given in Table V.

TABLE V

	Group I (76)			Group II (60)		
	Total	M 47	F 29	Total	M 37	F 23
	No. %	No. %	No. %	No. %	No. %	No. %
Restricted lumbar flexion	48 63	32 68	16 55	33 55	22 60	11 48
" " extension	27 36	17 36	10 34	26 43	17 46	9 39
Restricted lateral flexion	31 41	18 38	13 45	27 45	18 49	9 39
rotation	16 21	10 21	6 21	11 18	8 22	3 13
Straight-leg raising diminished	39 51	27 57	12 41	26 43	21 57	5 22
X-ray films:						
Congenital abnormalities	9 12	7 15	2 7	9 15	8 22	1 4
Radiological evidence of disk lesion	8 11	3 6	5 17	10 17	6 16	4 17
*Other lesions	17 22	12 25	5 17	21 35	11 30	10 43

* Osteophytosis, osteoarthritic changes, etc.

The results of treatment are given in Table VI.

TABLE VI

	Group I (76)			Group II (60)		
	Total	M 47	F 29	Total	M 37	F 23
	No. %	No. %	No. %	No. %	No. %	No. %
Well in 1 week	38 50	24 51	14 48	16 27	11 30	5 22
With Lasègue's sign initially	20 26	15 32	5 17	10 17	8 22	2 9
Well in 3 weeks	28 37	18 38	10 34	20 33	13 35	7 30
" " " " " "	1 1	0	1 3.5	7 12	5 13	2 9
Persistent symptoms and signs after 6 weeks	9 12	5 11	4 14	17 28	8 22	9 39
With Lasègue's sign initially	3 4	2 4	1 3	6 10	3 8	3 13
With congenital abnormalities	1 1.3	1 2	0	2 3	2 5	0
With radiation of pain initially	3 4	1 2	2 7	7 12	3 8	4 17

Discussion of Results

From the information obtained, we have selected certain groups of figures which we regard as of significant value. Statistical analysis has not yet been applied, because the present preliminary figures are inadequate for this purpose.

The distribution of male to female patients was 3:2. Most patients were in the age group 30-59 years and had suffered no previous attack or only one previous attack of pain. About 70% of each group complained of central lumbar pain. Unilateral and bilateral lumbar pain were much less common (see Tables I, II, and III).

There was no obvious percentage difference between male and female patients except in the group of "significant employment." As one might expect, there was a great predominance of males. Aggravation of pain by coughing occurred in about 65% of each group and each sex. The use of a sagging type of mattress, often a feather bed, may be an important factor, being noted in about 20% of all cases. Trauma might be expected to play an important part, and about 50% of the patients in each group gave such a history, with disability coming on within a short period of time (see Table IV).

The commonest signs found on examination were painful restriction of lumbar spinal flexion, occurring in about 60% of each group, and diminished straight-leg raising predominantly on one side, which occurred in about 50% of each group (see Table V).

There was a higher percentage of congenital abnormalities present than in the general population, by comparison with Willis's findings, especially of sacralization or lumbarization of L5-S1 vertebrae. This suggests that such malformations make the back mechanically unsound and prone to lesions causing backache in later life (see Table V).

Of the patients manipulated 50% were free of signs and symptoms at the end of one week as compared with 27% in the control group (see Table VI). At the end of six weeks only 12% of Group I were suffering from signs and symptoms, while 28% of Group II were still affected.

Perusal of that Table as a whole shows how simple manipulation of the lumbar spine, in the type of case described, results in freedom from symptoms and disability more quickly than does rest. It should be remembered, moreover, that the spinal manipulations were carried out by both physiotherapy students and trained staff, and the figures thus include many cases treated by relatively unskilled therapists.

It may be noted that of the patients in each group who were well after one week, over half had diminished straight-leg raising originally. If a Lasègue sign is taken to indicate a disk lesion, albeit of slight degree, then manipulation had helped to relieve it.

Of the patients in each group who still had signs and symptoms after six weeks, only a small proportion had Lasègue's sign originally on examination. It may also be noted that few of these patients had congenital abnormalities.

A relatively high number of this resistant group had radiation of pain initially, and this may be an important symptom in predicting response to treatment in either group.

Conclusion

In the patient with acute low back pain in whom the probable lesion is derangement of an intervertebral joint, including minor protrusion of a disk, and in whom irritation or pressure on adjacent structures occurs, then treatment by manipulation, without anaesthesia, seems a logical method of gaining relief from signs and symptoms.

Summary

A group of 152 patients suffering from acute low back pain were selected at random and treated either by manipulation or by bed rest and analgesics.

Full clinical and radiological examination was carried out in every case, and progress, whether improvement or deterioration, was noted at weekly intervals.

Of the patients manipulated, 50% were symptom-free and discharged within one week and 87% were discharged within three weeks.

Of the patients treated by rest in bed and analgesics, 27% were symptom-free and discharged within one week and 60% within three weeks.

After six weeks, 12% of the patients treated by manipulation, and 28% of the patients treated by rest in bed and analgesics still suffered pain and disability.

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Medical Memoranda

Amyotrophy Due to Hypoglycaemia

Neurological disturbances often occur in hypoglycaemia. Transient aphasia and hemiplegia are not uncommonly seen in hypoglycaemia due to insulin overdosage. More profound disturbances may occur, particularly in cases of islet cell tumour, when considerable diagnostic difficulty may be encountered. Lawrence *et al.* (1942) have described in detail the changes in the brain in six fatal cases of hypoglycaemia. There was widespread degeneration and necrosis of the nerve cells, particularly in the cortex, putamen, and caudate nucleus, with slighter changes in the brain-stem and cerebellum. The changes are probably due to failure of the normal cellular oxidative processes from glucose lack, although vascular factors may play a part. Rarely, lesions of the lower motor neurone, probably on the same pathological basis, may occur. Moersch and Kernohan (1938) mention loss of the anterior horn cells from a segment of the lumbar cord in an account of the changes in the nervous system from a fatal case of hypoglycaemia. Tom and Richardson (1951) describe a case of islet-cell adenoma, undiagnosed in life, which showed severe muscle atrophy of the legs. Histological examination of the spinal cord from this case showed destruction of the anterior horn cells. Roxburgh (1954) mentions wasting of the small hand muscles in the report of an islet-cell tumour in a child of 5½ years. Other cases have been described by Lidz *et al.* (1949) and Silfverskiöld (1946). The latter described two cases under the title of "polyneuritis hypoglycemia." There is no record concerning the degree, if any, of recovery, but in the

case recorded by Lidz the mental state and ataxia improved after removal of an islet-cell adenoma, but the muscle-wasting remained unaltered.

CASE REPORT

The patient was a married man aged 28 years. His past medical history was uneventful and there was no family history of nervous disease. He worked as a fitter in a large factory, but was not exposed to danger of poisoning by heavy metals or organic compounds. For at least twelve months prior to admission to hospital he complained of feeling weak, tremulous, and "queer" after any undue exertion. This often happened at his work, particularly just before lunch, after which he felt quite well again. There were times when he was unable to concentrate on his work, and his memory became rather capricious. During a holiday in June, 1953, he was found unconscious one morning and admitted to the local hospital. On examination he was found to be conscious but aphasic and there were signs of a right hemiparesis. The blood sugar concentration was 30 mg. per 100 ml., and he recovered fully and promptly following an intravenous injection of 20 ml. of 50% glucose. As he was on holiday he was given a high-fat, high-protein, low-carbohydrate diet and told to report to his doctor on his return home. His doctor referred him to Walton Hospital on August 13, 1953. He was then feeling better and refused in-patient investigation. He remained well until January, 1954, when his symptoms recurred. His wife stated that it became very difficult to wake him in the mornings until he was given a sweet drink. He had short episodes of mental confusion terminating in an attack of sweating, and was amnesic for these episodes. Both he and his wife realized that taking sugar prevented these symptoms, and he eventually carried sugar with him.

He was admitted for investigation on February 8, 1954, with the provisional diagnosis of spontaneous hypoglycaemia. He was tall and spare of build; his colour was good; and examination of the chest, heart (blood pressure 130/70 mm. Hg), and abdomen showed no abnormality. He gave the impression of being a little slow mentally. The pupils reacted normally and no abnormality was seen in the fundi; ocular movements were full, but there was nystagmus on extreme lateral gaze in both directions. There were no cranial-nerve palsies. The musculature of both shoulders and upper arms appeared to be a little wasted, but there was no detectable weakness. Both hands showed marked wasting of the intrinsic muscles, the interossei and hypothenar groups in particular. There was a little weakness of the extensors of the fingers. The tendon reflexes in the arms were brisk and equal and there was no subjective or objective sensory loss. Co-ordination was normal. The abdominal reflexes were present and equal. There was symmetrical wasting of both quadriceps, with marked fasciculation and fibrillation. The glutei and hamstrings were powerful, but there was some weakness of dorsiflexion of the left foot with a little associated wasting of the anterior tibial muscle group. The knee- and ankle-jerks were brisk and equal, and the plantar responses flexor. There was no objective sensory loss, and tests of co-ordination were normal.

Three successive fasting tests resulted in hypoglycaemia after periods of approximately twelve hours: blood-sugar values were 40, 40, and 35 mg. per 100 ml., and administration of intravenous glucose resulted in prompt recovery on each occasion, thus fulfilling Whipple's criteria. A glucose-tolerance test following a diet containing 350 g. of glucose for three days gave the following figures: fasting, 85 mg.; half an hour after 50 g. glucose, 100 mg.; 1 hour after, 85 mg.; 1½ hours after, 70 mg.; 2 hours after, 50 mg. per 100 ml. The haemoglobin was 93% and the white cell count 11,000 per c.mm., the differential count being normal. Liver function tests, including serum bilirubin, thymol turbidity, and serum alkaline phosphatase, were normal. X-ray films of the chest, skull, and cervical spine showed no abnormality. Lumbar puncture revealed clear fluid under