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

normal pressure, with one cell per c.mm. and a protein content of 32 mg. per 100 ml. The blood Wassermann reaction was negative. The electroencephalogram showed rather excessive low amplitude, slow activity.

At operation (Mr. N. O. K. Gibbon) on February 28 a single plum-coloured growth the size of a pea was found in the head of the pancreas and removed without undue difficulty. Convalescence was uneventful. Post-operative hyperglycaemia was not observed, the fasting blood-sugar values falling between 95 and 114 mg. per 100 ml. A glucose-tolerance test done ten days after operation gave the following results: fasting, 95 mg.; half an hour after 50 g. glucose, 130 mg.; 1 hour after, 120 mg.; 1½ hours after, 100 mg.; 2 hours after, 100 mg. per 100 ml.

Section of the tumour revealed an insulinoma, which was thought to be benign. The patient was discharged, symptomless, on March 20. When seen in the out-patient department six weeks later he felt very fit. He was working regularly and was symptom-free. The nystagmus had disappeared, but the muscle-wasting was unchanged.

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Foster Kennedy Syndrome Produced by Myelomatous Deposits in the Skull

The complete Foster Kennedy syndrome comprises an association of anosmia, primary optic atrophy and central scotoma, and contralateral papilloedema. The essential features of the syndrome are the atrophy and papilloedema, either or both of the other two components often being absent. The syndrome was first described in detail by Foster Kennedy (1911). It is usually produced by olfactory groove or sphenoidal meningiomas or by frontal-lobe tumours (Walsh, 1947). Its occurrence in any other condition is a rarity, and it is accordingly commonly seen in the 40–60 age group. Clearly, the syndrome may be due to any space-occupying lesion near to the olfactory tract.

The case described below is exceptional in that the compression was due to myelomatous deposits. Multiple myeloma also occurs most frequently after the age of 40 (Snapper, 1949). In this case the patient was unusually young, and the manner in which the case presented was more suggestive of a cerebral tumour than of myelomatosis.

CASE REPORT

A male steel worker aged 34 was first seen on July 30, 1953, complaining of generalized aches and pains which he had had for six months. These had at first affected his back and pelvic region. Later his shoulders and neck were involved. After these pains had been present for four to five weeks he developed severe frontal headaches. All these symptoms, with the exception of the headache, disappeared spontaneously five to six weeks prior to admission to hospital.

For five to six weeks before admission he had noticed that his left eye was beginning to protrude. There was no associated deterioration of vision. At the same time he developed intractable vomiting, which occurred 10 to 15 times a day. He also complained of general weakness, loss of weight, and recent nocturnal frequency of micturition. On inquiry he admitted that his food had recently become tasteless.

On examination he was clinically anaemic, but otherwise appeared in fair general health. There was marked proptosis and epiphora of his left eye. The patient had no sense of smell. There was neither diplopia nor ophthalmoplegia. The left fundus showed primary optic atrophy with

an occasional blotchy haemorrhage near the disk. In the right fundus the whole disk was ill-defined, the veins were prominent, and the complete lack of contour and blurring of the edge of the disk indicated a moderate degree of papilloedema. In addition several blotchy haemorrhages were present, mostly near the disk. The visual fields were normal, but the visual acuity of the left eye was diminished to 6/9, while that of the right eye was 6/6. He was tender over the fifth and sixth ribs on the left side. No further abnormalities were found.

Several laboratory investigations were carried out. The urine contained Bence Jones protein, but showed no other abnormality. The blood urea was 155 mg. per 100 ml. and his urea clearance was 15% of average normal standard clearance. The findings in the serum were: alkaline phosphatase, 13 K.A. units; acid phosphatase, 3 Gutman units; total protein 8.8 g. per 100 ml. (albumin 5.7 g., globulin 3.1 g.); sodium, 135 mEq/l.; potassium, 6.5 mEq/l.; alkali reserve, 18 mEq/l.; chloride, 88 mEq/l.; calcium, 12 mg. per 100 ml. The peripheral blood examination showed: haemoglobin, 51% (7.5 g. per 100 ml.); red cells, 2,500,000 per c.mm.; C.I., 1; leucocytes, 6,000 per c.mm. (neutrophils 3,000, eosinophils 60, lymphocytes 2,340, monocytes 600); no plasmacytes were seen; platelets, 250,000 per c.mm. (Lempert); E.S.R. (Westergren), 124 mm. in one hour.

Marrow smears from the right and left iliac crests and from a radiologically evident myelomatous deposit in the left clavicle showed gross infiltration with cells of the plasmacytic series, consistent with a diagnosis of myelomatosis.

Pentamidine isethionate was given by intramuscular injection on alternate days to a total of 4 g. over a period of six weeks. The patient also received deep x-ray therapy, 2,500 r depth dose to the anterior fossa of the skull, between August 12 and 28.

After these measures the proptosis and papilloedema disappeared, whilst the vomiting gradually ceased. It was at first thought that the vomiting may have been due to chronic renal failure, but as the blood urea level did not fall concurrently with the decline in vomiting it was concluded that the latter was caused by the raised intracranial tension.

COMMENT

This case presents several unusual and interesting features. Multiple myelomatosis in a man of 34 is rare. The youngest in Bayrd and Heck's (1947) series of 83 cases was 37. Though myeloma deposits are often found in the skull, producing the characteristic radiological appearance, it is relatively rare to find the tumour extending beyond the confines of these bones. This was observed in only two cases in the series quoted above. Accordingly, only exceptionally is intracranial tension raised in myelomatosis. Snapper (1949) states: "Although myeloma so often involves the skull, no convulsive seizures have been observed and the optic disks never show evidence of increased intracranial pressure." In the present case there were definite signs of raised intracranial tension—namely, papilloedema, retinal haemorrhages, and vomiting. Lumbar puncture was thought to be inadvisable in these circumstances, and so no direct measurement of the C.S.F. pressure was made. The only component of the Foster Kennedy syndrome that was lacking was the central scotoma.

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