immediate reduction of the displaced acetabular fragments in cases of sciatic-nerve palsy. He stated that the nerve was usually stretched over the sharp margin of the tilted fragment and that recovery was jeopardized by delay.

The decision to operate in this case was taken because the large posterior fragment could be fixed more securely with internal fixation than by conservative treatment. If an operation had not been performed the patient might have developed a delayed sciatic palsy due to movements of the fragment or to involvement in the healing fracture. This emphasizes the

need for open reduction in fracture-dislocations of the hip with large posterior fragments of the acetabulum even without clinical evidence of sciatic palsy.

My thanks are due to Mr. R. C. Tatham for permission to publish this case.

REFERENCES

- Armstrong, J. R. (1948). *J. Bone Jt Surg.*, **30B**, 430.
Watson-Jones, R. (1953). *Ibid.*, **35B**, 147.

Medical Memoranda

Involvement of Anterior Horn of Spinal Cord in Infectious Mononucleosis

Brit. med. J., 1965, **1**, 1112

Neurological complications of infectious mononucleosis are rare. In 1946 Wechsler *et al.* reported 556 cases of infectious mononucleosis, in which the incidence of involvement of the nervous system was less than 1%. Hoagland (1960), reporting his experience of 200 cases, mentioned only two with neurological complications. Though in the past paralysis of muscles due to involvement of peripheral spinal nerves has been reported by Richardson (1942), Saksena (1943), and Erwin *et al.* (1959), paralysis due to a lesion of the anterior horn of the spinal cord has not hitherto been reported. The following case report describes this unusual complication of infectious mononucleosis.

CASE REPORT

In September 1964 a 20-year-old skilled labourer developed a sore throat which lasted for three days. He felt increasingly weak and tired for a week after this, but remained ambulant. At the end of this period he was found to have a temperature of 102° F. (38.9° C.) for which he was sent to bed. Within a day or two he developed pain in the middle of his back and in the upper part of his left arm in the deltoid region. He also noticed that the muscles around his left shoulder twitched from time to time. Three or four days after the onset of pain he found that he could not raise his left upper arm above the shoulder. He had been vaccinated against poliomyelitis five years previously, having had the usual three injections of Salk vaccine.

When he was admitted to hospital, two weeks after the onset of illness, his temperature was 100° F. (37.8° C.). There was a faint maculopapular erythematous eruption over the trunk, forearms, and thighs and a moderate degree of enlargement of lymph nodes in the posterior cervical, axillary, and inguinal regions, the cervical lymph nodes being both visible and palpable. The spleen was firm and palpable 3.5 cm. below the costal margin and the liver was soft and palpable 3 cm. below the costal margin. Muscular power was markedly diminished in the left shoulder, abductors (deltoid and supraspinatus) and external rotators (posterior fibres of deltoid, teres minor, and infraspinatus) being affected. Diminution of the left triceps jerk was found. No sensory changes could be detected on repeated examination on different occasions. Muscular fasciculation was never seen.

A blood count showed a haemoglobin of 14.2 g.% ; white blood cells 18,000/c.mm. (neutrophils 9%, lymphocytes 7%, glandular-fever type cells, 84%). The Paul-Bunnell heterophile antibody

test was positive in a 1/1,280 titre of unabsorbed serum, positive in a 1/1,280 titre of serum absorbed with guinea-pig kidney emulsion, and positive in less than 1/10 titre of serum after absorption with ox-blood cells. Liver function tests showed 530 μ mol./hr./100 ml. of serum glutamic pyruvic transaminase (normal range 22–92), 270 μ mol./hr./100 ml. of serum glutamic oxalacetic transaminase (normal range 35–107), and 848 μ mol./100 ml. of serum alkaline phosphatase (normal range 156–576); these results suggested hepatic cellular necrosis. A throat swab did not reveal any pathogenic organisms. A radiograph of the chest was clear. The C.S.F. pressure was 170 mm. and the C.S.F. protein 70 mg./100 ml.; only one lymphocyte was seen per c.mm. Tissue culture failed to demonstrate any virus in the faeces, and virus complement fixation tests were negative in several blood samples.

The absence of sensory changes makes it unlikely that the (prednisolone). All symptoms and signs disappeared within a fortnight, except the motor paralysis of the affected group of shoulder muscles. This partial recovery was probably due to natural healing processes, and it is doubtful if the corticosteroid treatment played any significant part. Despite immobilization and subsequent physiotherapy, at the end of four months the paralysis showed no improvement. The left supraspinatus, infraspinatus, deltoid, and triceps muscles showed atrophy. Power of external rotation and abduction of the shoulder remains nil.

COMMENT

The absence of sensory changes makes it unlikely that the lesion was in the peripheral nerves. In fact, the close similarity of the clinical features of the paralysis to those of poliomyelitis makes it highly probable that it was in the anterior horn cells (or in the anterior root). It is theoretically possible that this was in fact poliomyelitis, coincident with infectious mononucleosis, but the previous immunization with Salk vaccine makes this unlikely. It seems more probable that in this case there was genuine damage to anterior horn cells by the hypothetical virus of infectious mononucleosis.

I am grateful to Dr. John Forbes for his encouragement, criticism, and permission to report this case.

S. K. MUKHERJEE, M.B., B.S., M.R.C.P.ED., D.T.M.&H.,
Medical Registrar, Wrexham Group of Hospitals.

REFERENCES

- Erwin, W., Weber, R. W., and Manning, R. T. (1959). *Amer. J. med. Sci.*, **238**, 699.
Hoagland, R. J. (1960). *Amer. J. med. Sci.*, **240**, 21.
Richardson, J. S. (1942). *Lancet*, **1**, 618.
Saksena, H. C. (1943). *Brit. med. J.*, **2**, 267.
Wechsler, H. F., Rosenblum, A. H., Sills, C. T. (1946). *Ann. intern. Med.*, **25**, 236.