isation from atheromatous plaques in the aorta (Chester and Banker 1986) or from occlusive disease of the blood vessels in the thigh. There was no clinical evidence of peripheral vascular disease in our patient.

The nerves of the lumbar plexus pass through the psoas muscle, and in our case there was total sensory and partial motor palsy of the femoral, obturator, genitofemoral, and lateral femoral cutaneous nerves. The lumbosacral trunk, ilioinguinal and iliohypogastric nerves appeared to be spared. We speculate that a compartment syndrome developed secondarily to the infarction, and that the partial recovery may have been due to the release of pressure. We conclude that muscular infarction in the psoas may cause an acute lower motor neurone palsy of the lumbar plexus.

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OSSIFICATION OF THE POSTERIOR LONGITUDINAL LIGAMENT AND FLUOROSIS

B. SUBBA RAO, V. R. TARAKNATH, V. N. SISTA

There is only one report of ossification of the posterior longitudinal ligament (OPLL) in patients suffering from fluorosis. Deshpande, Dinakar and Reddy (1976) mention an association with fluorosis in 14 of 26 cases. OPLL is not mentioned in two reviews of fluorosis (Jolly 1981; Reddy and Reddy 1987).

Patients. In our neurological service, we saw 15 cases of OPLL in one year, all presenting with spinal compression. They accounted for about 1.5% of all spinal cases. Ten of them had associated fluorosis. During the same period 17 cases of fluorosis with spinal compression were seen. The OPLL was confined to the cervical region in the five non-fluorotic cases, but in six of the ten fluorotic cases it also involved the dorsal and lumbar regions (Fig. 1). All the cases of OPLL presented with spinal compression, and with loss of about 50% of the sagittal canal width. The spinal stenosis was considerable, the OPLL further diminishing the canal already narrowed by fluorosis. Figure 2 shows a lateral view of the cervical spine in a patient with OPLL and fluorosis. The appearance of the ossified ligament was similar in fluorotic and non-fluorotic cases.

Treatment. Decompressive laminectomy in three patients produced further deterioration while expansive laminoplasty in three gave some relief of spasticity in only one.

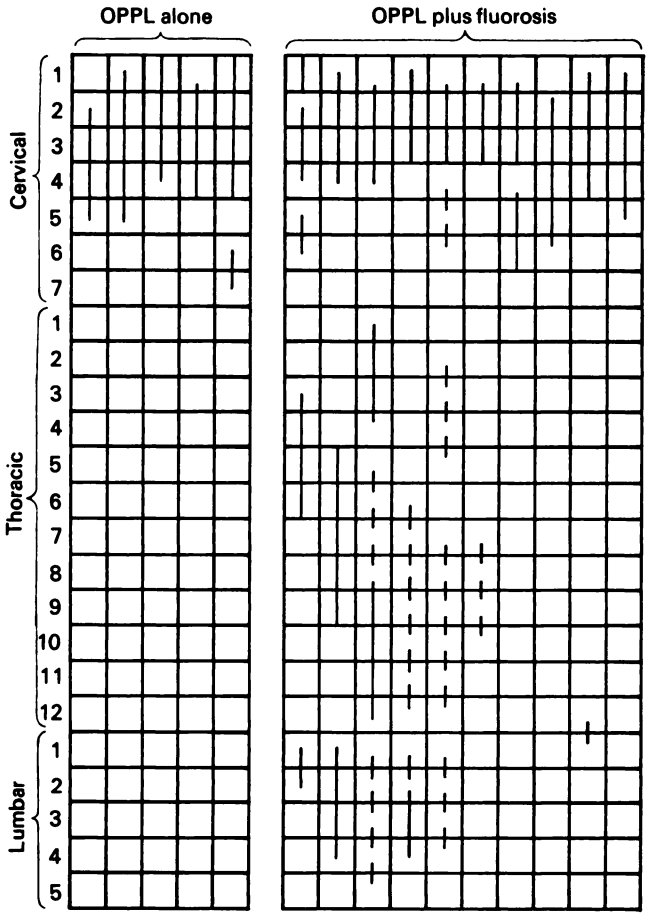

Fig. 1

Discussion. OPLL in its classical form is confined to the cervical spine and is not associated with any other osseous anomaly. Once thought to occur mainly in Japanese patients it is now being seen frequently in India

B. Subba Rao, MS, MCh, Professor of Neurosurgery
V. R. Taraknath, MD, DM, Assistant Professor of Neurology
V. N. Sista, MS, Tutor in Neurosurgery
Departments of Neurosurgery and Neurology, Guntur Medical College, Guntur, India 522 004.

Correspondence to Professor B. Subba Rao.

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(Deshpande et al 1976; Jayakumar et al 1987). An association with other diseases causing skeletal hyperostosis is now recognised, including diffuse idiopathic skeletal hyperostosis (DISH). Resnick et al (1978) observed OPLL in 50% of cases of DISH, and Tsuyama (1984) in 24%. Fluorosis causes extensive narrowing of the spinal canal and the root exit foramina; OPLL may cause further spinal stenosis. Probably because fluorosis produces more diffuse changes, our results of surgery in patients with fluorosis have been disappointing.

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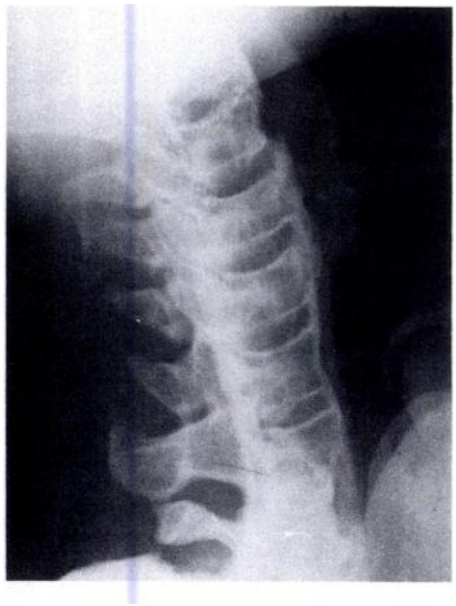

Fig. 2

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SUPRASCAPULAR NERVE ENTRAPMENT IN AN ARTHRODESED SHOULDER

LENNART SJÖSTRÖM, BENGT MJÖBERG

Suprascapular nerve entrapment is rarely considered as a cause of shoulder pain. We report relief of pain in an arthrodesed shoulder by decompression of the nerve.

Case report. An epileptic man in his 20s developed painful post-traumatic arthritis after delayed diagnosis of a posterior dislocation of his left shoulder. Four years later he had a successful arthrodesis and his pain was relieved.

One year after the operation he complained of aching pain in the scapular region radiating down the lateral side of the arm to the elbow. The pain was constant but aggravated by use of the arm and the patient had been unemployed for two years. Repeated radiographs confirmed that the arthrodesis was sound.

The diagnosis of entrapment of the suprascapular nerve was considered, because tenderness was maximal over the suprascapular notch. Injections of local anaesthetic and steroid gave prompt pain relief for one to two weeks. Electromyography showed denervation potentials in the supraspinatus but not in the infraspinatus.

At operation, through an incision just above the spine of the scapula, the transverse ligament was excised and the notch widened with a rongeur (Rask 1977). The nerve appeared normal. The pain was immediately and completely relieved: three weeks later the patient returned to heavy manual work in a timber yard and was still painfree after one year.

Discussion. The most common cause of suprascapular nerve entrapment in the suprascapular notch is direct trauma to the shoulder (Kopell and Thompson 1976; Laulund et al 1984; Hadley, Sonntag and Pittman 1986). The nerve has no cutaneous afferent fibres and the pain is therefore a deep, dull ache which may be made worse by shoulder movements, particularly adduction of the extended arm across the body. There is often wasting of the spinati muscles and local tenderness over the notch. The diagnosis may be confirmed by a suprascapular nerve block and by electromyography. Repeated nerve blocks with or without steroids may be curative but operative decompression usually gives permanent relief (Laulund et al 1984; Hadley et al 1986).

Another rare cause is frozen shoulder, which necessitates increased scapulothoracic movement which can produce a neuropathy in individuals with a narrow suprascapular notch (Kopell and Thompson 1976).

L. Sjöström, MD, Orthopaedic Surgeon
B. Mjöberg, MD, PhD, Associate Professor
Department of Orthopaedics, Uppsala University Hospital, S-751 85 Uppsala, Sweden.

Correspondence to Dr L. Sjöström.

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