of syringomyelia related to a spinal schwannoma, the possibility of a co-incident relationship is remote. It is true that the MRI will continue to attract notice to more and more cases of double pathology, however, in this case a causal relationship is more likely. This is supported by the observation that the syrinx cavity communicated with the tumour bed and the lack of evidence of communication between the syrinx and the fourth ventricle, so that the absence of any other responsible pathology such as another tumour.

In our case there was no evidence that the syrinx cavity was an extension of the tumour or that it extended into the cord above the tumour and communicated with the syrinx. The presumed localised impairment of vascular supply of the cord related to the tumour could be responsible for the cavitation within the cord. However, we feel that the pathogenesis is best expressed in a similar way to the proposition put by Quencer.1 The longstanding presence of the schwannoma had caused a block in the spinal subarachnoid space obstructing the pulsations. In addition, the tumour itself must have caused local damage and atrophy to the spinal cord around the area it was excavating. After a period of time the altered CSF dynamics caused a hole in the cord. Through this and possibly through the dilated perivascular spaces CSF entered the syrinx cavity. Fluid movement inside the cavity leads to expansion.

We conclude that the syringomyelia in association with extramedullary tumours is now a well recognised condition and must be considered on each occasion where the neurological deficits could not be explained by the position of the tumour. With the availability of MRI this pathology may be more frequently identified.

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Cervical cord compression and femoral neuropathy in hereditary multiple exostoses

Hereditary multiple exostoses is an inherited dysplasia of bone, characterised by the presence of multiple clavicular, knee, and elbow exostoses. The exostoses arise mainly from the metaphyses of long bones, but any bone formed by endochondral ossification may be affected. Patients with this condition usually below average height, and have typical deformities such as ulnar deviation of the wrist, subluxation of the radio-humeral joint, limb length discrepancy, and valgus of the knees and ankles.

The exostoses grow slowly until skeletal maturity, and behave as space occupying lesions. The most frequent local complications are bursa formation, interference with joint function and restrictions of movement. Neurological complications are uncommon, but when they occur most commonly take the form of a peripheral compression neuropathy.

Subcutaneous cervical cord compression in hereditary multiple exostoses is rare. Thirteen cases have been reported from 1843 to the present,1 one of which had in addition a confirmed cranial peripheral neuropathy of the nervous system due to exostoses at separate sites.2 We report a case presenting with a peripheral compression neuropathy of the left femoral nerve caused by a large iliac exostosis, which masked the onset of a spinal paraparesis from an exostosis arising at C2. A 15 year old boy with a family history of hereditary multiple exostoses, presented with painful knees and difficulty in walking. Examination revealed multiple large exostoses involving the shoulders, elbows, and knees and small exostoses at the wrist and ankles. The standing intermalleolar separation was 18 cm. Knee flexion and extension were restricted by large exostoses arising from the distal femoral and proximal tibial metaphyses. A large left sided abdominal mass was present, arising from the left iliac crest extending from the anterior superior iliac spine to the ala of the sacrum with an upper border at the level of the second lumbar vertebra. Excision was performed and histopathological examination confirmed a benign exostosis. Postoperatively the patient had a complete femoral nerve palsy, which recovered rapidly to MRC grade three. Electromyography confirmed a lower motor neuron lesion.

Four months later he was readmitted for correction of his genu valgum. Examination on this occasion revealed a spastic paraparesis, with weakness in both legs. The London reflexes were increased, apart from the left knee jerk, which was still absent. The plantar responses were extensor. Unsus- tained clonus was present at both ankles. Neurological examination revealed a large exostosis arising from the left iliac crest at the MRC grade four level, compressing the dorsal sacral cord. Computed tomography confirmed the presence of a large exostosis arising from the neural arch of C2 (fig). At decompresive laminectomy the exostosis was found to arise from the spinal cord above the tumour.

Figure CT scan of the cervical spine with metrizamide showing the origin of the exostosis from the neural arch of C2 with marked cord compression.