SURGICAL RELIEF FOR SPASTIC TETRAPLEGIA DUE TO CERVICAL CORD COMPRESSION

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The purpose of this paper is to alert the clinician to an unusual, but entirely treatable, cause of progressive spastic tetraparesis. In the management of tetraplegia, in which treatment usually has to be supportive rather than curative, it is important to be aware of any remediable condition that calls for early surgery. Neurological localisation in the upper cervical cord is often imprecise, and simple compression of the cord is often not recognised in the first instance. McRae (1960) analysed sixty-eight cases of bony abnormalities of the foramen magnum amenable to surgical treatment and showed that fifty-three of the patients had been previously diagnosed as suffering from incurable neurological disease. This report underlines the importance of early diagnosis of cervical cord compression.

CASE REPORT

A man aged forty-one had suffered progressive weakness of the right leg since the age of fifteen. Gradually the whole right side became involved in a spastic paresis, and a decade later this spasticity progressed to the left side of the body. He was thought at that time to be suffering from multiple sclerosis and was treated symptomatically. His main disability during the subsequent years was weakness of the right side of the body with severe clonus of the wrist and ankle, and this compelled him to give up his work as a chef in 1967.

In January 1969 a radiograph of the cervical spine revealed a congenital anomaly of the arch of the axis. The lateral, plain radiograph (Fig. 1) showed an abnormal protuberance of
the bone of the posterior aspect of the neural arch. It was noted that the sagittal diameter of the cervical canal was narrowed at this point and tomography confirmed this finding (Fig. 2).

In May 1969 the patient was admitted to the Wessex Neurological Centre. Clinical examination revealed spastic weakness of all four limbs with spontaneous clonus of the right wrist and ankle. The power of dorsiflexion of the right foot was Grade 2 (Medical Research Council grading) and he had worn a toe-raising spring for four years. There were no objective sensory findings and there was no difficulty with micturition. He had a markedly spastic gait and used one walking stick in the right hand. Blood pressure was 120.80 millimetres of mercury.

Examination of other systems, and preliminary investigations including blood Wassermann reaction, full blood count and chest radiograph were normal. Lumbar puncture showed normal pressure. Positive contrast myelography (Dr E. H. Burrows) revealed a partial hold-up opposite the junction of the second and third cervical vertebrae (Fig. 3). Prone and supine films clearly demonstrated that the indentation on the posterior aspect of the dural sac conformed to the congenital anomaly at that level.

*Operation*—Decompressive laminectomy was performed (by Mr John Garfield). The arch of the atlas and the third cervical laminae were normal. The neural arch of the axis consisted of apparently normal laminae with an additional mass of bone immediately beneath them, rather like reduplicated laminae. These “extra” laminae were clearly compressing the theca. The laminae of C.2 and C.3 and the anomalous bone were removed. The theca then assumed a normal shape and a soft rubber catheter would pass with ease both upwards and downwards in the extradural space. Histological examination of the excised fragments showed normal lamellar bone. The patient made an uncomplicated post-operative recovery. There was early subjective increase in the power of both lower limbs and the tendency to spontaneous ankle and wrist clonus lessened considerably in the first week.
Repeat myelography two weeks after the operation showed a free flow of medium at the C.2-3 level and the posterior thecal indentation had disappeared (Fig. 4). Six weeks after the operation plantar-flexion and dorsiflexion of the right foot were Grade 4, and the patient had discarded his toe-raise spring and his walking stick.

DISCUSSION

Congenital anomalies of the cervical spine are not uncommon; they usually result from agenesis, non-union or non-segmentation during the embryonic period. These malformations are often asymptomatic, but narrowing of the cervical canal with cord compression may occur. It is the comparative rarity of such neurological disturbance that merits emphasis on the condition, because the correct diagnosis is unlikely to be made in the first instance. Gradual compression of the upper cervical cord may cause widespread spasticity in the limbs, a picture that is readily confused with multiple sclerosis in its spatial, although not in its temporal, distribution. Burrows (1963) showed that the sagittal diameter of the canal at the level of the axis, in the male, averaged 20.3 millimetres with a normal range of 15 to 25 millimetres. The measured sagittal diameter of the cervical canal at the level of the axis was 7 millimetres in our case, indicating a considerable degree of cord compression.

The congenital anomalies of this region of the cervical spine that are usually encountered include disturbances of development of the dens, non-segmentation of adjacent vertebrae, the Klippel-Feil syndrome, atlanto-occipital fusion and basilar impression. The significance of such abnormalities was reviewed by McRae (1960) and by Epstein (1968), but neither these nor other authors have reported the exact type of abnormality described in our case.

Ossification of the axis is from five primary centres: one for the centrum, one each for the two lateral halves of the neural arch and two for the body of the atlas (the dens). The bone at birth consists of centrum, dens and the two lateral halves of the arch as separate bony pieces joined by cartilage. The bony vertebral canal is not completed until the sixth to seventh year (Frazer 1958), when the centrum and lateral halves consolidate. The anomaly in this case appears to have been a product of exuberant growth of the developing laminae. It is tempting to postulate that symptoms did not begin until the second decade of life because cord compression from the extra bone was not manifested until fusion of the primary centres had closed the neural arch.

Finally, this case emphasises the fact that a compressing lesion of the cervical cord may not produce a diagnostic sensory or motor level. The investigation of any case of spasticity of all four limbs must therefore include a detailed radiological assessment of the cervical spine, so that a remediable cause of a relatively common symptom complex is not overlooked.

SUMMARY

1. A case of cervical cord compression due to a congenital anomaly of the arch of the axis, treated successfully by decompressive laminectomy twenty-six years after the onset of symptoms, is described.
2. The significance of bony abnormalities of the cervical spine as a treatable cause of spastic tetraparesis is stressed.

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REFERENCES