basic pathology to neuropsychological sequelae of treatment makes the book a valuable addition to the library of those who not only remain focussed on their own share of management of human intracranial germ cell tumors but want to broaden their perspective of this very complex disease.

Prof. Manfred Westphal, Hamburg, Germany

Letter to the Editor

Transient Mutism After Brain Stem Infarction (ref. Acta Neurochirurgia 1999, 141: 209–213)

Sir:

I enjoyed reading the article by Miyakita *et al.* [5] regarding transient mutism after brain stem infarction. In our review of the literature, we did not include cases in which mutism had been experienced after brain stem surgery [2], since Rekate *et al.* [7] hypothesised that the lack of long tract findings or cranial nerve dysfunction favoured a purely cerebellar origin. However, I subsequently had a case of cerebellar mutism following the excision of an exophytic brain stem glioma [3]. In the literature, we were able to find only 4 patients with a brain stem lesion in whom mutism had developed [1, 4, 6]. As far as I know, the first case of cerebellar mutism after brain stem infarction was reported by D'Avanzo *et al.* [1] in 1993. He was a 48-year-old man with two ischaemic areas in the brain stem on magnetic resonance images. His mutism lasted for 16 weeks. However, ischaemic cerebrovascular disease of childhood particularly involving the brain stem is very uncommon. Miyakita *et al.* [5] presented a well-documented case of cerebellar mutism caused by brain stem ischaemia.

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Referring to the Posterior Fossa Cranioectomy and Tonsillar Resection in Order to Treat Chiari I Malformation with Syringomyelia

Dear Sir,

In the Guyotat, Bret, Jouhanneau, Ricci and Lapras review which was issued in your journal [1], it is reported the treatment of posterior fossa craniectomy was performed on 75 patients suffering from Chiari I malformation/syringomyelia. Furthermore, the following was added: in order to restore a "normal" cerebrospinal fluid (CSF) circulation at the craniorrhachidian joint and prevent the relapses when preserving cervical subarachnoid spaces, a third ventricle shunting in 16 patients, a syringosubarachnoid shunting in 9 patients and as innovation, a cerebellar tonsillar resection in 8 patients were performed.

Although several authors have commented on cerebellar tonsils relating to Chiari I malformation/syringomyelia [2], none of the above mentioned procedures addresses the fact that the narrowness in CSF circulation generated by cerebellar tonsils is the cause of Chiari I/syringomyelia complex. According to my point of view, these narrowings are a consequence of an abnormal shifting of the lower part of the cerebellum by (neuraxis) traction due to the presence of a filum terminale syndrome [3].

My opinion is that the advantage of the posterior fossa craniectomy is twofold. First, it improves the narrowing in the cerebrospinal fluid spaces. Second, it liberates the obstacles to the caudal traction of the neuraxis which together with the paradoxical fact that the cerebellar lowering into the vertebral canal when the posterior fossa is surgically enlarged [5], favours a traction mechanism for yielding an abnormal stress at the neuroaxis [3].

So, I think cerebellar tonsillar resection is not required since patients need liberation of the axial stress on the neuraxis by performing a filum terminale resection [6]. In cases where an adequate result has not been obtained due to significant prolapse of the cerebellar tonsils, posterior fossa craniectomy and C1 laminectomy would then be the most appropriate approach.

Keywords: Malformation Chiari; syringomyelia; scoliosis; etiology; treatment.

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Comment

We have read with much interest the comment about our paper "Syringomyélia associated with type I Chiari Malformation. A 21 Years Retrospective Study on 75 Cases treated by Foramen Magnum Decompression with a Special Emphasis on the Value of Tonsils Resection".

The author of the comment argues about the hydrodynamic theory and its variations, debated but usually accepted, and proposes another theory with the filum terminale playing the leading role. According to this theory an overstretched filum terminale would be responsible for excessive traction on the neural axis, thus causing descent of cerebellar tonsils and medullary ischaemia which is the origin of the associated syringomyelia.

The association of Chiari I malformation and syringomyelia with an overstretched filum terminale, an anatomical variation of tethered cord, may not be uncommon within complicated dysraphisms. This association however is far from being a constant, as demonstrated by the whole spine MRI studies which are commonly used for the radiographic evaluation of a Chiari malformation with syringomyelia.

The author's suggestion, based on his theory of the stretched filum terminale, is to cut this structure. In his paper (in Rev Neurol 1997; 25: 523–530), he reports the results of a series of 4 patients treated by this technique. In our opinion this series is too small to draw conclusions.

We are interested in the tethered cord syndrome in adults [1]; it is uncommon that section of the filum terminale leads to the ascent of the conus medullaris. Therefore, it is even less likely that it determines an ascent of the cerebellar tonsils, all the more so because the role of the dentate ligaments in keeping the spinal cord in place increases in a caudal-cranial sense.

In our opinion, the section of filum terminalis does not seem to be the procedure of choice to propose first in the treatment of Chiari I malformation with syringomylia.

J. Guyotat

Reference

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