The distal and F wave latencies in both the median and ulnar nerves were delayed. F waves could not be elicited in the peroneal or tibial nerves. Median SCV did not improve. The patient was discharged with no disability in activities of daily life four months after admission.

The patient's serum was tested at intervals for antiganglioside antibody activities by enzyme linked immunosorbent assay (ELISA).¹ GM1a, GM2, GM3, GD1a, GD1b, GT1a, GT1b, and GQ1b were prepared from bovine brain.² The antigenic solution contained 20 pmol/50 µl of each ganglioside. Each patient's serum was tested in triplicate at 1:100 dilution, and binding detected with horse radish peroxidase conjugated goat antihuman IgM or IgG antibody (Jackson ImmunoResearch Laboratories, Inc, West Grove, PA, USA) at 1:1000 dilution. On admission, the serum reacted with both GQ1b and GT1a at a dilution of 1:3200. These two antibody activities, expressed as optical densities, fell to 1/2 in parallel with her improvement, but they could still be detected in her last blood sample (at 16 weeks). IgG antibodies against the other gangliosides were not detected at any time, nor were IgM antibodies to any ganglioside detected.

To determine whether these two antibodies react independently with each ganglioside or react with a common epitope sharing with GQ1b and GT1a, an absorption study was performed using GQ1b or GT1a coated polystyrene beads as described previously.¹ The titre of anti-GQ1b IgG antibody decreased when preincubated with GT1a coated polystyrene beads, and vice versa for the anti-GT1a IgG antibody. Based on these results, we hypothesised that these antibodies reacted with a common epitope sharing GQ1b and GT1a.

Chiba *et al*³ found anti-GQ1b IgG antibodies in patients with Miller Fisher syndrome and also with Guillain-Barré syndrome with ophthalmoplegia. They showed immunochemically that GQ1b was rich in the paranodal regions of the human oculomotor, trochlear, and abducens nerves. These anti-GQ1b IgG antibodies also reacted with GT1a. Yuki *et al*⁴ found anti-GQ1b IgG antibodies in eight patients with acute paresis of the extraocular muscles but without ataxia. These results suggested that anti-GQ1b IgG antibodies had a part in the development of ophthalmoplegia. We have also detected anti-GQ1b and anti-GT1a IgG antibodies in six patients with Miller Fisher syndrome and two with Guillain-Barré syndrome accompanied by ophthalmoplegia. We also found that these antibodies reacted with a common epitope sharing GQ1b and GT1a (unpublished data). Kimura *et al*⁵ have already reported a patient with acute relapsing sensory dominant polyneuropathy without ophthalmoplegia who had an anti-GQ1b IgG antibody which did not react with GT1a. Further clarification of the relation between anti-GQ1b IgG antibody and the development of ophthalmoplegia in Miller Fisher syndrome or Guillain-Barré syndrome is required.

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Can trauma alone to the trigeminal root relieve trigeminal neuralgia? The case against the microvascular compression hypothesis

Idiopathic trigeminal neuralgia (ITN) is increasingly regarded as being due to microvascular compression of the trigeminal sensory root, either by an artery or a vein close to the brainstem.¹ Yet vascular contacts are found in entirely asymptomatic cases, no vascular contacts are found in some asymptomatic patients, and, most importantly, ITN suddenly switches off, even for years, only to return later, in the face of continuing vascular compression.² Despite this, microvascular decompression produces immediate virtually complete (98-100%) relief in 82% of the cases and 64% after 10 years.³ Adams has suggested that, ITN being a hyperfunctional disorder of the brainstem, microvascular decompression "produces chronic trauma to a sensitive zone of the cranial nerve... by the dissection necessary and by the manipulation required microvascular decompression produces sufficient trauma to achieve interference of normal functioning of that nerve", thus dampening the abnormal brainstem activity responsible for ITN.² Failure to achieve an initial result or early recurrence would imply insufficient surgical trauma.² We evaluated this hypothesis in our patients submitted to microvascular decompression. Forty one out of 410 patients with ITN have had keyhole microvascular decompression in the posterior fossa from December 1986 to February 1996 at the neurosurgical pain relief unit of the University of Turin. Twenty six patients showed pronounced arterial or arterovenous compression, three distally (group 1), three mild or disputable contacts (group 2), two slight and three pronounced venous compressions (group 3), five with arachnoiditis, two with a sharp root kink, four distally (group 4). In four cases (group 5), no anomaly whatsoever could be found, despite adequate magnification and careful exploration; vessels were noted close to the root, but no contact or groove could be seen. Thus the root was gently "massaged" with a microdissector; no other manoeuvre was attempted. These four patients included one woman and

three men, with a mean age of 59 (range 54-66). All had typical ITN which at some point could no longer be controlled by drugs at adequate dosage. In all other cases, the vessels were separated from the root by interposing Surgicel; the arachnoiditis was dissected to free the root and, in one patient with slight venous compression partial (25%) rhizotomy was also carried out. In several patients, "massage" of the trigeminal root was performed.

All patients were relieved by surgery. Follow up disclosed the following recurrences: two in group 1 (follow up 4 months-9 years), none in group 2 (follow up 2.3-5 years), none in group 3 (follow up 1.1-5 years), two in group 4 (follow up 1-6 years), one in group 5 six years postoperatively (follow up 6 months-8 years). Importantly, one of the group 1 recurrences was operated on and no vascular contact whatsoever could be found (partial rhizotomy was elected). The only group 5 recurrence could be controlled by drugs. Whereas this was seen in other groups as well, all four group 5 patients showed transient slight to moderate hypaesthesia in two to three branches after surgery, a hallmark of trauma.

In 1961, Taarnhøj reported that simple manipulation of the trigeminal root by running a nerve hook along the root obtained 60% long term pain free results over a mean of 6.5 years (longest follow up 10 years).⁴ Gardner and Miklos also obtained 67% pain free results over 4.5 years by manipulating the trigeminal sensory root at the point of crossing the apex of the petrous bone,⁵ suggesting that manipulation not at the root entry zone (as suggested by Jannetta), but peripherally at the petrous apex produces as good results as microvascular decompression. As no other report considered the problem, the microvascular compression hypothesis for ITN gained favour. Our data suggest that (1) trauma alone can ensure long term relief; (2) this relief is similar between patients showing microvascular compression and those without; it should be stressed that several patients of the microvascular compression group were "massaged" to ensure a control with the group without.

The trigeminal nerve root is surrounded by many arteries. In 60% of the roots, the trigeminal vessels form arterial rings encircling at least half of the root or its entry zone in asymptomatic patients,⁶ justifying the frequency of vascular contacts at large. Despite a plethora of vessels reported to compress the trigeminal nerve in a recent series,³ initial relief could not be achieved in 18% of the patients. Recently, a group reported electrophysiological data supporting the concept of trauma during microvascular decompression, despite attempts to limit the procedure to simply moving the vessel.⁷ The trauma hypothesis would explain recurrences of successful microvascular decompression in which no new compression is found (for example, our case and see Yamaki *et al*)⁸ at re-exploration.

Adam's contention is being appreciated in the case of hemifacial spasm. Payner and Tew,⁹ discussing their results with microvascular decompression for this disorder, stated that "...perhaps there is truth that the early success in most patients results from minor trauma... to support this theory, additional analysis is needed of the long term follow up in patients without vascular compression and...treated by "manipulation" alone". This is what has been done in this study.

Microvascular decompression entails a risk of death (although this is small) and other serious complications requiring surgery (much more often) in the face of a painful, but benign disorder. Perhaps, "trauma surgery" would be best to ensure equal results with a much lower complication rate. A minimally invasive approach (endoscopy) could be studied in this regard.

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About the original description of cerebellar tonsil herniation by Pierre Marie

Transfalcial and transtentorial herniation and the engagement of the cerebellar tonsils into the foramen magnum are well recognised consequences of increased intracranial pressure. They were discovered through both experimental findings carried out in the 19th century,¹ and pathological and clinical experiences reported in the first decades of this century.² Harvey Cushing (1869-1939) is usually acknowledged as the first clinician who, in 1902, correlated the clinical picture of neurological disturbances and cardiorespiratory failure with the herniation of the cerebellar tonsils into the foramen magnum.³ However, when in his book on pontocerebellar angle tumours Cushing made use of the notion of "cerebellar pressure cone",⁴ he was employing a term first introduced into the English language by Collier,⁵ as Cushing himself acknowledged in an earlier article.⁶

Here we report on the observations of Pierre Marie (1853-1940) that bear on the same issue and antedate those of both Cushing and Collier. On 1 July 1899, during a session of the Société de Biologie, Pierre Marie⁷ reported on two cases of cerebral haemorrhage that resulted in secondary compression of the cerebellum. In the first case, a haemorrhage of the external segment of the lenticular nucleus flattened the superior face of the left cerebellar lobe and shifted the vermis to the right. In the second case, a thalamic

haemorrhage resulted in compression of the superior face of the left cerebellar hemisphere and caused a protrusion of the cerebellar tonsils that "appeared to have engaged the occipital foramen".

Pierre Marie drew attention to the pathophysiological research of Leonard Hill. "In his *Hunterian Lectures* in 1896, he showed that the brain does not transmit the pressure on one point in all directions. Indeed, a clear pressure difference is created between the main cranial cavity and the posterior fossa. According to Hill, the pressure gap is due to: (1) the viscosity of the cerebral substance; (2) the interposition of the falx cerebri and the tentorium cerebelli. This explains why a haemorrhage lesion exerts a localised compression on a particular part of the cerebrum, instead of creating a uniform pressure growth on the whole cerebral mass, as some think. In the present circumstance, the compression is exerted on a cerebellar hemisphere.

Furthermore, according to Leonard Hill "the compression exerted by the haemorrhaged cerebellar hemisphere upon the superior surface of the cerebellum can have very serious consequences. In fact, if the compression is sufficiently strong, the downward push on the cerebellum causes the engagement of its inferior surface into the foramen magnum. In this process the cerebellum takes the form of two cones constituted by the cerebellar tonsils. As a result the medulla oblongata is trapped within the foramen magnum, and if the pressure is strong enough its vessels will be compressed to the point of failing to supply the organ. Hence a severe bulbar ischemia will ensue."⁷

Pierre Marie concluded his communication by considering the possibly severe consequences of cerebellar tonsillar penetration into the occipital foramen and the resulting bulbar hypoperfusion, exactly devising the mechanism of cerebellar herniation. In the next year, Pierre Marie reported the pathological findings of another two cases of raised intracranial pressure.⁸ In the first case with a thalamic haemorrhage the rise in intracranial pressure caused the caudal shift of a cerebellar tonsil, that consequently "engaged into the occipital foramen". The second case was of increased intracranial pressure of unknown origin, disclosed by pronounced hydrocephalus. "Both cerebellar tonsils had shifted into the occipital foramen such as to form a sort of cone enclosing the bulbous for more than half of its circumference." In his conclusions, Pierre Marie suggested that the compression of the medulla could have contributed to the fatal outcome.

A few years later, in 1905, Louis Pierre Marie Alquier (1872-1956) described two further cases of cerebellar tonsils herniation into the occipital foramen.⁹ In Alquier's article the discussion of Jean Athanase Sicard (1872-1929) and Pierre Marie on the topic is reported. Mentioning his personal experience in similar cases, Pierre Marie also made the somewhat surprising suggestion that Alquier's pathological findings might be attributed to a postmortem process. We could find no further contribution by Pierre Marie on this topic in his subsequent scientific production.

Recently, Fisher¹⁰ has pointed out that cerebellar herniation is not necessarily a terminal event, except, in cases in which cerebellar herniation occurs in posterior fossa infarcts, acute subdural haematomas, or during a lumbar puncture. In such cases, acute herniation is justifiably considered among the determinant factors of clinical outcome. The first

clinical description of this important phenomenon was made by Pierre Marie.

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Blepharospasm induced by flunarizine

Flunarizine is a calcium channel antagonist that has been widely prescribed for vertigo, cerebral or peripheral vascular diseases, migraine, and epilepsy.^{1,2} At doses of 10-40 mg/day, it can produce side effects such as parkinsonism or abnormal involuntary movements, especially in elderly people.^{1,2} A case of blepharospasm in a 67 year old woman who had been taking 20 mg/day flunarizine for 18 months has been reported.² However, the cause of the disorder in this instance may be questionable as this patient had also been treated with cinnarizine and her dystonic movements did not disappear 13 months after the discontinuation of both drugs.² We have seen a 30 year old woman who was treated with 10 mg/day flunarizine for migraine prophylaxis. After two months of treatment she developed a progressive blepharospasm which eventually prevented her from reading or watching television. Three weeks after the appearance of symptoms, flunarizine was withdrawn, and the patient made a gradual and complete recovery over the next month.

Dopaminergic, cholinergic, serotonergic, or other poorly understood mechanisms might be implicated in the pathogenesis of blepharospasm.³ Flunarizine has been reported to have antihistaminic, antiserotonergic, and antidopaminergic activities.² This interference with dopaminergic transmission seems to be both complex and particularly