

Microvascular decompression may be an effective treatment for nervus intermedius neuralgia

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Dear Sirs,

We read with utmost interest the engrossing paper by Saers *et al.* entitled 'Microvascular decompression may be an effective treatment for nervus intermedius neuralgia'.¹

Since its original description by Wrisberg in 1778, nervus intermedius neuralgia has represented a long-standing and intriguing challenge in the fields of anatomy, surgery and clinical medicine.² Recent intra-operative observations have indicated the presence of unexpected and isolated electromyographic activity of the perioral muscles following electrical stimulation of the nervus intermedius, which can help identify the nerve.^{3,4}

In Saers and colleagues' reported case, they postulated neurovascular compression as an aetiology of nervus intermedius neuralgia, and stated that it was 'of vital importance' to verify possible vascular structures by magnetic resonance angiography.

Although attractive, this theory is in our opinion not fully convincing. The diameter of the nervus intermedius (less than 0.5 mm) and its variable course do not allow confirmation of the problem with diagnostic imaging.⁵ In fact, Saers and colleagues' Figure 1 fails to show the nervus intermedius.

More experimental studies are needed to confirm this neurovascular compression theory (with its parallels to trigeminal neuralgia aetiology). Such research could investigate, for example, the exact location of the nervus intermedius transition zone between central and peripheral myelin, and the relationship of the nervus intermedius to the brainstem.

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References

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Authors' reply

Dear Sirs,

Thank you very much for giving us the opportunity to clarify our manuscript with regard to the above comments.

We thank Drs Alfieri and Strauss for their positive comments regarding this anatomically and pathophysiologically interesting topic. As they point out, the nervus intermedius cannot itself be visualised.

In our reported case, we aimed to establish whether the proximity of a vascular loop to the cranial nerve VII/VIII complex resulted in neurovascular compression; we also intended to emphasise the importance of excluding other causes of neuralgic pain. We agree that the presence of such neurovascular compression cannot be established with absolute certainty. However, in cases of trigeminal neuralgia, management approaches based on the theory of neurovascular compression have produced promising results.

We concur with Drs Alfieri and Strauss that further experimental studies are needed to confirm the compression theory. We hope that our case report will act as a stimulant for future research.

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