Challenging vascular compression as a cause of trigeminal neuralgia

TO THE EDITOR: As the treatment of patients suffering from trigeminal neuralgia (TN) is a significant portion of my practice, I have always looked forward to Dr. Burchiel's contributions to the literature. In their recent paper, Lee et al.² (Lee A, McCartney S, Burbidge C, et al: Trigeminal neuralgia occurs and recurs in the absence of neurovascular compression. Clinical article. J Neurosurg 120:1048–1054, May 2014) state that “compression of the trigeminal nerve by a blood vessel...is still thought to be the most common cause for TN,” but they also state that “the hypothesis that TN is caused by neurovascular conflict must be challenged.” They note that 99.94% of individuals with trigeminal neurovascular compression (NVC) do not have TN.

It is clear that not all cases of TN are caused by vascular compression. However, anecdotally, it is also clear to me that sometimes trivial, unnamed, small vessels cause TN. I suspect that these vessels “fly under the radar” of many surgeons intraoperatively and are also undetected by even the best MRI. Just because vascular compression was not seen on preoperative imaging or was not visualized intraoperatively does not mean that it was not present. Neither the tools nor the surgeon is 100% accurate. Despite these imperfections, however, neurosurgeons can treat the majority of patients with TN (those who do not have multiple sclerosis or a posterior fossa or trigeminal tumor) using the Jannetta procedure. Most stop the medication they were taking for TN.

Without a doubt, vascular compression can lead to cranial nerve dysfunction. Is there still doubt about the cause of hemifacial spasm? Absolutely not.

Use the authors’ logic on our spine patients. Approximately 60% of MRI studies performed in patients older than 60 years who have never had low-back pain, sciatica, or neurogenic claudication are significantly abnormal.¹ Do we now challenge the notion that sometimes these abnormalities lead to painful conditions that are relieved by surgical intervention?

Rather than proposing a challenge to the vascular compression hypothesis, perhaps the challenge is to perform a better vascular decompression or to obtain better MRI studies. Perhaps the challenge is to find yet another cause of TN. Surely there are undiscovered factors in the TN story. Challenging accepted dogma may be healthy, but challenging Peter Jannetta’s work that has led to the successful treatment of so many grateful patients seems off-base. It would be a shame to unnecessarily reignite some of the past vitriolic discussion against Dr. Jannetta's landmark contributions to neurosurgery and patients with TN.

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RESPONSE: We thank Dr. Kalia for his analysis of our work and would like to comment on the underlying theme of his letter: the tenet that NVC is, with minor exceptions, the underlying cause of TN.

Using his standard, “just because vascular compression was not seen on preoperative imaging or was not visualized intraoperatively does not mean that it was not present,” approaches a religious conviction. We submit that if NVC cannot be imaged or visualized, it is not there. The fact that MRI or MR angiography (MRA) agrees so strikingly with the surgical findings validates the premise that nothing is being “missed” intraoperatively. As an aside, I agree with Dr. Kalia that hemifacial spasm (HFS) appears in many ways to be the exemplar of NVC producing a cranial nerve disorder. In practice, we have never seen a patient with HFS who did not have NVC at the root of the facial nerve. As imaging has improved over the past decade, we have also never seen an MRI or MRA study with 3D reconstruction that did not clearly demonstrate NVC. Clearly, NVC seems to play an essential, and universally detectable, role in this disorder.

Microvascular decompression is the single most important surgical advancement in the treatment of TN in the past 6 decades. It remains the most successful surgery, long term, for this disorder. Nevertheless, the success of this surgery should not be a pretense to stop thinking about this condition. If Gardner and Jannetta had not challenged conventional thinking, they would not have made their seminal contributions to the treatment of TN.

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References

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The author reports no conflict of interest.
Trigeminal neuralgia and the absence of neurovascular compression

To The Editor: I read with keen interest the article by Lee et al.1 (Lee A, McCartney S, Burbidge C, et al: “Trigeminal neuralgia occurs and recurs in the absence of neurovascular compression. Clinical article. J Neurosurg 120:1048–1054, May 2014) and noted the provocative nature of their title and concluding statement regarding the etiology of trigeminal neuralgia (TN). In their retrospective review of 257 patients assessed over 18 years, they remarked the absence of neurovascular compression (NVC) in 29% and 18% of patients with TN Type 1 (TN1) and TN Type 2 (TN2), respectively, by utilizing MRI, intraoperative observations during microvascular decompression (MVD) surgery, and their own facial pain classification system.1,2 They concluded that “the hypothesis that TN is caused by neurovascular conflict must be challenged.” I respectfully suggest that another interpretation of their data is possible and that their conclusion should be challenged.

The authors have shown a strong correlation between MRI and operative findings, but they reported the absence of NVC in a significant proportion of their patients with TN1 and TN2. They have not, however, assessed the subset of patients with purely episodic pain commonly referred to as “classical TN.” This is an important distinction and should prompt a critical re-evaluation of their facial pain classification system. I suggest that their classifications of TN1 and TN2 facial pain lack specificity by including a significant proportion of patients who do not actually have TN and therefore are not expected to have a causative NVC. For example, their TN2 category is defined as an “idiopathic trigeminal facial pain that is aching, throbbing, or burning for more than 50% of the time and is constant in nature (constant background pain being the most significant attribute).”2 This definition is not compatible with the International Headache Society (IHS) International Classification of Headache Disorders (ICHD)-II 13.1.1 “classical trigeminal neuralgia,” in which pain is purely episodic with a caveat that “dull background pain may persist in some long-standing cases.”2 This has important clinical ramifications, as there is no substantive evidence that patients with predominantly constant facial pain—TN2 according to the authors’ classification—actually have TN or that they will benefit from the surgical interventions that are well established as effective for classical TN.

This line of reasoning also has important implications for their category of TN1, described as “sharp, shooting, electrical shock–like, episodic pain lasting several seconds, with pain-free intervals between attacks.”2 This definition is more in keeping with the IHS ICHD-II 13.1.1 classical TN, but there remains an important distinction. According to the authors’ definition, TN1 may also include patients suffering from additional, superimposed, or concurrent constant facial pain that may be prominent and severe for up to 49% of the day.1,2 The distinction between a level of constant pain for more than or less than 50% of the day has been set arbitrarily, and there is no clear clinical or etiological distinction between patients with TN1 and those with TN2. The TN1 and TN2 classifications also do not differentiate the important subset of patients with purely episodic, classical TN pain and other non-TN idiopathic craniofacial pain conditions.

The IHS ICHD-III (beta version) goes further to differentiate between 13.1.1.1 classical TN, purely paroxysmal, and 13.1.1.2 classical TN with concomitant persistent facial pain, the latter being defined by “recurrent attacks of unilateral facial pain fulfilling criteria for 13.1.1 classical TN and persistent facial pain of moderate intensity in the affected area.” Such a stringent differentiation between the purely episodic pain condition and one with up to a moderate associated constant pain element may be better suited to assessing the correlation between disease (that is, TN) and purported cause (that is, NVC).

The hypothesis that NVC is the root cause of classical TN is supported by two well-established principles: 1) vascular compression of the trigeminal nerve root is present in most patients and 2) atraumatic alleviation of such NVC with MVD surgery is usually successful in providing long-term relief from TN pain. The integrity of this NVC hypothesis is not diminished by the observation that not all patients with NVC develop TN or that technically successful MVD surgery does not always result in permanent disease cure. Clinical experience has indeed demonstrated a robust association between a diagnosis of classical TN and culprit NVC, as demonstrated in carefully reviewed MRI sequences, intraoperative observations, and long-term outcomes following MVD surgery. While MVD surgery pioneered by Jannetta6 does not guarantee a cure for TN, the success of this operation remains the strongest evidence that NVC causes TN. As suggested by Hutchinson in 1905,8 the selection of surgical treatment “should depend upon a scientific classification, based solely upon causes of neuralgia,” and this question appears to have been answered at least for classical TN.

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