Invited Commentary

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As the authors write, trigeminal neuralgia (TN) is a severely painful condition that typically arises in otherwise healthy people in late middle age.[1] The underlying pathophysiologic mechanisms responsible for the paroxysmal pain of TN remains unknown,[2] despite extensive research directed towards the study of neurophysiology of the trigeminal system. Research has implicated central mechanisms in patients with TN; others have argued for a peripheral mechanism. Clinical observations supporting a peripheral mechanism include occurrence of TN as the manifestation of a mass lesion (tumor, dolichoectatic basilar/vertebral artery, or aneurysm) compressing the preganglionic trigeminal root in the posterior or middle cranial fossa.[3] However, Jannetta affirms that TN is a painful condition caused by an abnormality of the root entry zone of the trigeminal nerve, and that the abnormality may be associated with cross-compression by an artery loop that has imposed upon the nerve as a result of vascular elongation secondary to the aging process, or by a cross-compressing vein, or by both.[4-6]

The treatment options for patients with TN include conservative and surgical ones. One of the surgical treatment options is micro-vascular decompression (MVD) – of which the authors’ papers deal[7] with- for patients with typical TN and less than 65 years. The basis for this procedure is that there is persuasive evidence to show that TN is caused by demyelination of trigeminal sensory fibers within either the nerve root or, less commonly, the brainstem. In most cases, the trigeminal nerve root demyelination involves the proximal, CNS part of the root and results from compression by an overlying artery or vein. Some large sequential case series from specialist centers report MVD rendering over two thirds of patient’s pain free at 10 years and with one per cent experiencing facial numbness.[8-9] Complications are rare in good hands, but are reported as severe as brain stem and cerebellar infarction secondary to coagulation of a vein[10] or as a two per cent chance of permanent ipsilateral hearing loss.[10] A not very well known, but relatively common (in up to 18%), complication of MVD for TN is the intraoperative occurrence of the trigemino-cardiac reflex (TCR) first described by the senior author.[11] The TCR is a well-recognized phenomenon consisting of bradycardia, arterial hypotension, apnea, and gastric hypermotility during manipulation of one of the sensory branches of the trigeminal nerve.[12] TCR was defined
as a drop in mean arterial blood pressure (MABP) and the heart rate (HR) of more than 20% to the baseline values before the stimulus and coinciding with the manipulation of the trigeminal nerve.\[11\] Schaller could for the first time demonstrate that a central stimulation of the trigeminal nerve during MVD leads to the TCR\[13\]. One may suggest that other complications of MVD – like the ipsilateral hearing loss – may be also influenced by the TCR\[14\] underlying its importance of prevention. After cessation of the manipulation, HR and MABP return (spontaneously) to levels before the stimulus.\[13\]

In conclusion, MVD is a very effective treatment for TN patients who are intolerant or refractory to the medical measures. There are some complications with the procedure which can rarely be fatal (like cerebellar and brain stem infarction). The TCR, observed in patients with skull base surgery and operations in the cerebello-pontine angle, is also a common complication with MVD and has to be known not only by anesthetist\[1\] but also by every surgeon performing MVD.

References


