Editorial

Results of microvascular decompression for trigeminal neuralgia

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Dr. Burchiel has been one of the most careful contemporary students of trigeminal neuralgia (TN) and the results of “microvascular decompression” (MVD) for this condition. In an attempt to clarify the classification of this disease, he has introduced the terms, “Type 1 TN” to denote trigeminal pain with a preponderance of shock-like pain and “Type 2 TN” to denote a preponderance of constant pain. Although this classification, based on “preponderance” of one type of pain over the other, is short of being precise, it at least introduces some uniformity in the definition of this condition. Before Dr. Burchiel’s classification, many clinicians, including me, reserved the term “typical TN” or “tic douloureux” to patients with shock-like pain described variably as “electric,” “lancinating,” “searing,” and so forth, clearly limited to the distribution of the trigeminal nerve, and who generally had abrupt onset of their pain syndrome (“memorable onset”), trigger points, and pain-free intervals and who responded at least initially to anticonvulsant medication. We would allow within this strict definition of typical TN a gradual development of a more constant form of pain underlying the continuing repetitive lancinating stabs of pain. We have felt that strictness in this definition of typical TN is important because only this type of TN responds predictably well to MVD. Of course, this belief has been challenged by many including one of my mentors, Peter Jannetta. The problem is that many clinicians interested in the field have been more inclusive and less strict in defining “typical” TN and have allowed a variable degree of constant pain or the absence of 1 or more of the typical features described above, in their definition of “typical TN.” Dr. Burchiel’s classification gives us more uniform criteria on which to base outcome studies of the treatment of this condition.

In this paper, Dr. Burchiel and his colleagues studied retrospectively, but carefully, the results of MVD and correlated them with a variety of factors. The results are important although not entirely surprising. The overall message is that patients with Type 1 TN fared considerably better at both short-term and long-term (36-month) evaluation. In fact, the type of pain (Type 1 vs Type 2) was the only significant predictor of outcome on multiple regression analysis. As one would expect, there were trends towards better outcome in patients with shorter preoperative duration of symptoms, presence of trigger points, positive response to anticonvulsant agents, pain-free intervals, and memorable onset of pain. Clearly, all of these, except for the duration of symptoms, are characteristics that most of us associate with “typical” TN. Arterial compression rather than venous compression or lack of compression also showed a trend towards better outcome which does not surprise me. It would be interesting to have seen a more detailed analysis of the intraoperative findings in Type 1 versus Type 2 TN and how they correlated with outcome, but the authors have indicated that they have studied this information and will report it in the future. In our own early experience with MVD, arterial compression correlated positively with excellent long-term results.

As carefully as this study was conducted, I have some minor concerns and criticisms that the authors may clarify in their reply to this editorial. Only 121 out of 179 patients surveyed responded. Could there be a bias in the results, in that perhaps patients who had a bad result were less likely to respond? The authors excluded from analysis 9 patients who underwent MVD and had a history of multiple sclerosis (MS). Do they undertake MVD in patients with MS or was this diagnosis arrived at only retrospectively after the operation was performed? There were patients included in this series who never had lancinating pain and whose pain was constant from onset. Do the authors include such cases in their definition of “trigeminal neuralgia”? The importance of this article is that it confirms, in a well-designed retrospective study, previous observations in the literature that the more “typical” the syndrome, the more likely it is that the condition will respond to MVD. Furthermore, a quick look at Fig. 1 shows that the results of MVD for patients with Type 2 TN (predominance of constant pain) are not very good, with only 25% of these patients having an excellent result (no pain off medication) at 36 months. Whether the patients with results categorized by the authors as “good” (an additional 39% of the patients with Type 2 pain were in this category) truly had a “good” result or not would depend on the subjective interpretation of the authors’ definition of this outcome category (“mild or intermittent pain controlled with low-dose medication”). The authors conclude that MVD “re-
mains a very effective treatment for TN of both types” and whether one agrees with this conclusion or not would depend on whether one tends to look at the glass as “half full” or “half empty”—that is, “25% of the patients with Type 2 TN are free of pain without medications and an additional 39% are improved” or “only 25% of the patients with Type 2 TN had an excellent result.”

I personally tend to agree with the authors that, because MVD is a relatively safe operation—which in the expert hands of the authors resulted in no major complication, no hearing loss, and no facial paralysis—it is worthwhile to consider it in patients with Type 2 TN. However, within this category, I would tend to be selective and be more inclined to recommend MVD only to those patients with Type 2 pain who had “typical” features at least at the beginning of their disease and who continue to have sharp paroxysmal pain in spite of a variable degree of constant pain at present. I would not offer MVD to patients who do not have at least some lancinating pain, and Fig. 3 reinforces this opinion in that it appears that only 2 patients in this category had a long-term excellent result. Incidentally, a study published since the authors submitted this paper appears, at least superficially, to support these authors’ conclusion that the presence of constant pain should not deter the clinician from offering MVD to appropriate patients with TN. In that recent article, patients with variable degrees of constant pain did as well as those who did not have constant pain. However, the authors included only patients whose syndrome started as typical TN with only episodic pain who later on in the progression of the disease may or may not have developed some degree of constant pain in addition to continuing episodic pain. This criterion for consideration of MVD is practically identical to mine.

We are thankful to Dr. Burchiel and his group for providing us with additional excellent data upon which to base our recommendations for or against MVD in patients with pain in the trigeminal distribution. Of course, it goes without saying that many other factors must be considered—notably age, comorbidities, and whether medical therapy has truly failed. In the future, it is very likely that preoperative studies to visualize the presence or absence of vascular contact with the nerve will become an increasingly important factor in these considerations. Although the authors themselves have contributed in a major way to this area of preoperative visualization of vascular contact by sophisticated MR imaging methodology, I think for the present they would agree with me that a negative study will not deter them from undertaking surgical treatment in a patient with Type 1 TN. Would a study that suggests no vascular contact with the trigeminal nerve deter them from surgical treatment in a case that stands at the end of the spectrum of Type 2 TN, involving mostly constant pain and no other features of typical TN? I am sure that Dr. Burchiel, with the respect that he commands in this field, does not wish to open a Pandora’s box that could encourage less-experienced neurosurgeons to operate indiscriminately in cases involving patients with facial pain in the trigeminal distribution who do not have any of the typical features of TN. Finally, the experience and skill of the surgeon must be honestly considered.

Most neurosurgeons cannot expect to undertake surgical treatment of a large group of patients such as the cohort presented in this paper without significant complications.

References


Response

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We are indebted to Dr. Heros for his thoughtful analysis and kind commentary. We are, in fact, in vigorous agreement with his conclusions. Our study does suffer from its retrospective nature, and it is possible, in fact likely, that our sample may under-represent poor outcomes from MVD. Our study suggests a starting point, not an end point.

At present, the medical literature is devoid of a single natural history study of TN. Lacking this, it is difficult to put the surgical treatment of this disorder into perspective. Our hope is that our retrospective analysis will bolster the use of a more objective terminology to describe TN, and that true natural history studies of the disorder, both before and after surgery, will be forthcoming.

Our analysis of the results of MVD reinforces our conviction that the best prognostic category for a patient to occupy is a clinical syndrome of Type 1 TN and unequivocal arterial neurovascular conflict demonstrated at the time of surgery.

Patients with MS were excluded from this survey. Several years ago, we postulated that patients with symptomatic TN secondary to MS might still be candidates for MVD. At that time, we felt that these patients might develop symptoms of TN due to a combination of demyelination in the brainstem descending tract and otherwise modest vascular compression of the nerve. In our series, all patients with MS who underwent MVD had some degree of trigeminal neurovascular conflict demonstrated by high-resolution MR imaging. Although this small subset of patients unanimously experienced improvement immediately after surgery, more long-term follow-up was disappointing. It is likely that the short-term benefit was simply from manipulation of the nerve at the time of MVD, from a mild rhizolysis effect. We have since abandoned MVD for patients with MS, and do not consider the procedure to be indicated in these patients.

In our schema, patients with spontaneous, but pre-
dominantly constant, facial pain are assigned the category of Type 2 TN. This lumps together patients who have a significant history of lancinating facial pain with those that have never had any episodic pain. This is the logical product of treating all spontaneous facial pain as a variation on the theme of TN—that is, that TN is a continuum from purely episodic to purely constant pain. As a first approximation, this is a reasonable way to conceptualize the disorder. However, we readily admit that Type 2 TN may well be a composite category, containing 1 or more other diagnoses. The results of our study would tend to support this thesis. Perhaps it would make more sense to divide Type 2 TN into 2 subdivisions: cases in which there is some history of lancinating pain (Type 2a), and those in which there is no such history (Type 2b). This would more accurately reflect the prognostic implications of any history of lancinating pain, as Dr. Heros has suggested.

For a disorder that was described more than 300 years ago there are still a disturbingly large number of unanswered questions regarding the true nature of TN. Certainly the physiology of this disorder has fascinated generations of neuroscientists, and yet we still do not have a complete understanding of the mechanism of this unusual pain. Many questions also remain unanswered in the clinical domain. For example, how do we account for patients with the classic syndrome who lack any detectable neurovascular compression on either high-resolution MR imaging or compulsively thorough surgical exploration? This is a situation that we, and many others, face with a low, but uncomfortable, frequency. Furthermore, if we have conclusively discovered and corrected the origin of the patient’s pain, by decompression of the nerve, why do most MVD outcome series, including ours, demonstrate a slow, but statistically relentless recurrence of the pain? If we have “cured” the patient of pathological neurovascular compression, why does the pain come back, often many years after MVD? We are not convinced that the usual explanation of recurrent nerve compression is a likely explanation for this failure of surgical therapy.

Perhaps one “real world” test of an outcome study is whether or not it comports with the experience of a master surgeon. Our hope is that as our efforts in the description of this disorder, and its surgical treatment, unfold, our objective results will parallel the observations of Dr. Heros and other neurosurgeons that have contributed so eloquently to our understanding of TN.

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