Vascular Compression is the Cause of Trigeminal Neuralgia

Peter J. Jannetta

Trigeminal neuralgia is discussed from the standpoint of etiology, pathophysiology, and surgical treatment. The author presents the hypothesis of cranial nerve vascular compression as the cause of trigeminal neuralgia from a historic perspective, with a comprehensive review of the literature from its earliest efforts through the present. **Key words:** *trigeminal neuralgia, microvascular decompression, cranial nerves, constriction, pathologic*

A review of the literature concerning trigeminal nerve vascular compression is interwoven with the personal experiences of the author and his colleagues, in a critical analysis of vascular compression as causative of this painful affliction of the face. Published works are examined and included to add pertinent information to this discussion, even if they were felt to be erroneous or simplistic in approach.

This review is primarily but not necessarily chronological. The early contributions by neurosurgical pioneers, the limited development of these ideas because of inadequate technology, and the independent observations made by applying the surgical microscope to problems of the cerebellopontine angle are discussed. I will also examine the subsequent development of an organized system of clinical pathological correlation, the development of risk factors, and the various stages of discourse before acceptance of a new idea.

The first report in the literature of cranial-nerve vascular-compression syndrome was described by Cushing in 1910.[15] Cushing presented the acute pathological situation of sixth cranial nerve palsy due to severe stretching by the posterior circulation arteries in cases that increased intracranial pressure. All neurosurgeons understand compression of the oculomotor nerve by aneurysms of the posterior communicating artery. This is another pathological situation, although an ectatic artery (cirsoid aneurysm) can cause the same compression. These can be considered loss of functional syndromes.

In 1925, Dandy[17] published a preliminary report concerning section of the sensory root of the trigeminal nerve at the pons in two patients with trigeminal neuralgia. Dandy was one of the few neurosurgeons of his era who could safely operate in the cerebellopontine angle. The general inability to operate in this region without major morbidity persisted in most centers and by most surgeons for decades, although there were some important exceptions. By 1929, Dandy[18] enlarged his series and broadened his thinking and began to discuss the question of abnormalities in the cerebellopontine angle, especially vascular abnormalities as potential causes for trigeminal neuralgia. He persisted in this thinking and by 1934[19] showed real evidence that vessels and occasionally tumors or scars were possibly causative of trigeminal neuralgia.

In a series of papers, Gardner and his group[22,25–27] verified Dandy's observations. Unfortunately, Gardner was not able to find offending vessels in as many patients as Dandy. Although Gardner discussed the mechanism of trigeminal neuralgia, extrapolated elegantly to hemifacial spasm, he was not able to show vascular compression in even 50% of his patients. There were two reasons for this: first, he had no magnification capabilities and second, he did not inspect the root entry zone of either the fifth or seventh cranial nerves.

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It is a central truth of science and medicine that ideas that precede the technology to prove or disprove them lie fallow. In 1966, Olafson et al.[72] demonstrated that the lesion associated with trigeminal neuralgia in patients with multiple sclerosis was located in the root entry zone of the nerve. Lesions anywhere else in the trigeminal system did not cause trigeminal neuralgia. This finding has been frequently misquoted. Gardner in 1968[23] wrote a resumé of his lifelong experiences with the operative treatment of trigeminal neuralgia. He had previously discarded the concepts of vascular compression to show that trigeminal neuralgia was caused by a higher petrous ridge on the side of the pain.[23] This theory was disproved. In his 1968 chapter,[23] Gardner retrospectively associated trigeminal neuralgia with both vascular compression and a higher petrous bone. Gardner and Dohn in 1966[24] associated the trigeminal neuralgia and hemifacial spasm with Paget's disease without correlating it with vascular compression. This curious and undoubtedly spurious correlation was never accepted.

In 1967, unaware of Dandy and Gardner's work, I reported an early experience with the anatomy of the trigeminal nerve with selective section for pain and vascular compression in patients with trigeminal neuralgia.[34,35] I demonstrated that blood vessels, large and pathological or small and normal, were causative of trigeminal neuralgia. Surgeons had been able to demonstrate radiographically and see intraoperatively the larger and pathological blood vessels (aneurysms, arteriovenous malformation, and ectatic arteries). The smaller ones, more commonly causative of trigeminal neuralgia, were not readily visible but were there and were responsible. After all, bacteria have caused infection since time immemorial but it has only been since we could see them and grow them in culture that the germ theory of disease became operant. I extrapolated the findings for trigeminal neuralgia to hemifacial spasm when I saw a patient with this problem and performed the first microvascular decompression of a cranial nerve in a patient with hemifacial spasm in June 1966.[36–40] The first microvascular decompression for trigeminal neuralgia was performed by myself and Rand (on a private patient of his) 2 months later.[36–41]

Other surgeons published early papers concerning functional anatomy of the trigeminal nerve, with selective section of the nerve using microsurgical techniques. Among these were Provost and Hardy in 1970,[79] Petty in 1976,[74] and Petty and Southby in 1977.[75] Apfelbaum compared percutaneous radiofrequency trigeminal neurolysis and microvascular decompression of the trigeminal nerve for treatment of trigeminal neuralgia.[1] In this important contribution, he stated independently that the quality of life was better for patients with microvascular decompression and that although the mortality rates were less with radiofrequency lesions, the problems of anesthesia dolorosa and other sensory perversions were significant in patients who had undergone radiofrequency lesions.

By 1978, a number of investigators were critically evaluating, accepting, and using microvascular decompression techniques for trigeminal neuralgia.[8,10,12,57,58,60,80,82] Also by 1978, the new paradigm was actively straining the older concept of idiopathic versus symptomatic trigeminal neuralgia.[56] Shenkin, in all innocence, criticized the concept of vascular compression.[86] By this time, in addition to our own patient series, a significant number of surgeons had also published papers verifying our findings that vascular compression was always present, even in tumors, except in cases of multiple sclerosis. They agreed that microvascular decompression operations could be performed safely, with excellent quality of life, minimal morbidity and mortality, and better results for pain relief.[53,54,67,69,73,81,84,92,94,95] It should be remembered that in the 1970s, quality of life was improving throughout the entire field of neurosurgery, including the treatment of trigeminal neuralgia. Although the original observations on vascular compression or trigeminal neuralgia had been in the United States, neurosurgeons in other countries published the major papers in the late 1970s. Through the early 1980s, Leopold et al.,[59] Voorhies and Patterson,[93] Wilkins,[96] Burchiel et al.,[13] and Apfelbaum[2,3] added to the literature. Apfelbaum further showed that prolonged trigeminal neuralgia was less likely to be relieved by microvascular decompression.[4] This lack of relief has been presumably linked to advanced physiologic and morphologic changes in the nerve that were less likely to revert to normal after relief of the pulsatile vascular compression.

Fukushima simplified the operative exposure of retromastoid craniectomy by making the incision and craniectomy smaller.[21] This limited exposure is more feasible in a brachycephalic oriental head where the posterior fossa is flat and shallow and the cranial nerves course in a more mediolateral than anteroposterior direction. Another significant observation made independently by both Apfelbaum[4] and Niemeyer,[70] was that increased operative experience dramatically raised the percentage yield of causative vascular compression in patients with trigeminal neuralgia. This was the case with Dandy's original observations and it is the case today.

Barba and Alksne[7] demonstrated in 1984 that a

prior destructive operation was detrimental to a good result in hemifacial spasm, with good results dropping from 93% to 46% in these patients. A question regarding the significance of arterial versus venous compression was posed by Piatt and Wilkins.[76,77] Apparently unaware of Portnoy's demonstration that veins pulsate inside the intact skull,[78] these investigators questioned whether venous compression could cause trigeminal neuralgia and performed nerve section if only venous compression was found at operation.

Natsysgunam et al.,[68] from Rhoton's laboratory, described the anatomy of the operative approach to trigeminal neuralgia, utilizing the vernacular of Rhoton's anatomical descriptions of the cerebellopontine angle structures. In 1989, Keet[49] reiterated the point that the surgeon performing microvascular decompression should be experienced. Klein et al.[51] in 1989 described their nuances of operative technique, demonstrating once again that the microsurgical supralateral cerebellar exposure to the trigeminal nerve is an improvement over the Dandy pericerebellar exposure.

A series of papers using magnetic resonance imaging (Tash et al.,[88] and Wong et al. in 1989,[98] Harsh et al.,[32] and Baldwin et al., in 1991,[6] and Sens and Higer[85]) demonstrated that the newer imaging techniques (especially magnetic resonance angiography) could be useful in identifying vascular compression in a majority of patients with trigeminal neuralgia.

Furthermore, it should be noted that several postmortem studies have also investigated neurovascular relationships and verified findings of vascular compression.[29–31,66]

Bederson and Wilson[9] reported 250 cases of trigeminal neuralgia treated using the cerebellopontine angle exposure. They made the decision, as had Wilkins,[96] that minor distortion by compressing blood vessels was possibly not significant and that a partial nerve section should be performed. They amplified Apfelbaum's earlier finding that trigeminal neuralgia of long duration was less amenable to treatment. Ten percent of their patients experienced little or no pain relief, 12% had recurrence "at an average of 1.9 pain-free years after operation," and recurrence continued at the rate of approximately 2% per year. The presence of a previous percutaneous radiofrequency lesion was associated with a significantly greater incidence of fifth nerve complications and a worse outcome after posterior fossa exploration. Because of this finding, the authors recommended that percutaneous radiofrequency rhizolysis be reserved for patients who have failed posterior fossa exploration or are not candidates for open operation. In this situation, I prefer percutaneous glycerol rhizotomy (again, in experienced hands) to radiofrequency lesions because of the much lower incidence of sensory aberrations in the former.

Some interesting minor causes of trigeminal neuralgia include a primitive trigeminal artery or its variants (Morita et al.[65]) and arteries transfixing the nerve (Tashiro et al.,[89] Hutchins et al.[33]). Babu and Murali[5] reported a patient with an arachnoid cyst of the cerebellopontine angle manifesting as contralateral trigeminal neuralgia. A number of progressively larger series have now shown minimal morbidity and little or no mortality and a better quality of life after microvascular decompression. These include series by Dahle et al.,[16] Meglio et al.,[61] Zhang et al.[99] (with an 89.5% cure rate), and Klun et al.,[52] who has done partial central rhizotomy in addition to microvascular decompression.

Kondoh et al. described a case of contralateral trigeminal neuralgia caused by a chronic calcified subdural hematoma[55] that distorted the brainstem. The trigeminal neuralgia was relieved after the removal of the subdural hematoma, and it can be presumed that this was due to relief of vascular compression by changes in the neurovascular relationships, as the brainstem distortion was relieved in both of these patients. An intramedullary cavernous angioma located in the upper cervical spinal cord caused trigeminal neuralgia in a 45-year-old woman, which was relieved by removal of the angioma, according to Saito et al.[83] Familial trigeminal neuralgia was described by Kirkpatrick[50] and Coffey and Fromm.[14] A tortuous elongated contralateral-vertebral artery looping across the midline was shown to cause trigeminal neuralgia and facial spasm (tic convulsif) (Grigoryan et al.[28]). Tsubaki et al.[90] showed that 7 patients out of a huge series of 1,257 operative patients had a cryptic angioma of the cerebellopontine angle, causing vascular compression of the trigeminal nerve, which was not seen on preoperative computed tomography scans. Angiography showed a small vascular stain in one patient. Presumably benign extraaxial cerebellopontine angle tumors were reported by a number of investigators to cause ipsilateral vascular compression (Michelucci et al.,[63] Metzer,[62] and Olgeznev[71]). Many years ago, Gardner and Dohn[24] showed Paget's disease in association with trigeminal neuralgia, but of course they could now show that this was causal and it probably was not. An association with fibrous dysplasia was seen in one 72-year-old woman in a case reported by Bollen[11] in 1990. Although the fibrous dysplasia involved the area of Meckel's cave, I would presume that the

patient's problem was caused by associated vascular compression, as were the others.

My experiences and those of my colleagues have clearly shown that vascular compression of the trigeminal nerve is present in virtually all patients with trigeminal neuralgia.[36–40,42–48] The one major exception is in patients with multiple sclerosis. These patients have an extremely high incidence of trigeminal neuralgia that is said to be not as persistent or severe as in those patients who do not have multiple sclerosis, but nonetheless, they have pain. As demonstrated by Olafson et al.,[72] patients with multiple sclerosis have a multiple sclerosis plaque at the root entry zone of the nerve. And as mentioned previously, a lesion anywhere else in the system does not cause trigeminal neuralgia. The vessels causing trigeminal neuralgia can be single or multiple, large or small, and arterial or venous.

It is important for the reader to appreciate that the cranial nerves cannot distinguish the effects of pulsation of a 50–75 μm artery from that of a 2 cm aneurysm or ectatic artery. This size range of compressing vessels is an important consideration when a surgeon is operating. In over 90% of cases, the vascular compression is clear-cut. The others may be very subtle. Figure 1 shows a number of neurovascular relationships of the trigeminal nerve in trigeminal neuralgia.[45] Generally, rostral compression causes V_3 pain. This is most commonly due to the superior cerebellar artery looping downward and upward around the pons. Caudal compression, which is uncommon, causes V_1 pain. This is most often due to a pontine vein, an aberrant trigeminal vein bridging between the pons and the dura adjacent to Meckel's cave, or by the anterioroinferior cerebellar artery. Ectatic vertebral arteries have been shown to do this at times. Isolated V_2 trigeminal neuralgia is most commonly caused by an aberrant trigeminal vein on the side of the vein, but a medial vessel can cause it as well. It can be located quite distally on the nerve. I have seen offending blood vessels in the lip of Meckel's cave and causative blood vessels due to veins in the dura of the petrous bone at Meckel's cave. Until this vascular compression was relieved, there was no relief of the pain.

The central myelin located inside the portio major may extend some distance from the pons in the portio major. There are approximately 100 separate fascicles entering the central nervous system's cone-shaped central myelin in the portio major. This central cone of myelin can extend out to Meckel's cave or can be a shorter segment. Therefore, compression at, and just medial to, Meckel's cave can cause compression of nonfascicular nerve or of the junction of fascicular and nonfascicular nerve (root entry zone) and cause trigeminal neuralgia (Fig. 2). The motor-proprioceptive fascicles and the intermediate fascicles have a short segment of central myelin, with the junctional area at the brainstem. Compression of the motor-proprioceptive fascicles, which cover the nerve rostrally, medially, and a bit caudally as they rotate from proximal to distal, cause constant focal burning pain that can be termed *atypical trigeminal neuralgia.* This is in contrast to atypical facial neuralgia (atypical face pain). Since we have learned about this, operative results of microvascular decompression for atypical trigeminal neuralgia have improved significantly.

The vessels may be located under the ala of the cerebellum. Vessels can be dislodged by minimal retraction of the ala of the cerebellum for exposure of the nerve. One must look for this phenomenon by always inspecting under the ala of the cerebellum and by allowing the ala to fall back gently to see if the vessel will roll back onto the side of the nerve rostrally. The only monitor (other than visual, which is the best) that we have for effective microvascular decompression of cranial nerve V is that frequently there will be slowing of the pulse when the blood vessel is dissected from the trigeminal nerve at the significant area of compression. This can occur with dissection as the vessel is being manipulated away from the nerve and also can occur with placement of the implant for microvascular decompression.

The problem is not whether there is a blood vessel present, but whether the surgeon is astute enough to find all the significant blood vessels. I have even seen, in two patients, a vein that was inside the portio major, completely buried within nervous tissue running from central to peripheral and obviously causing internal compression of the nerve. This phenomenon was noted when nothing else was found in one of these two patients, and in a recurrence in the other that was actually intractable pain rather than a true recurrence. In this case, the vein was found when I started to section the portio major at the pons.

One can expect as seen in the Table, excellent results of long duration in approximately 80% of good risk patients who have not had a prior destructive operation. This percentage reflects permanent relief. Another 10% will have some pain with no major problems, and the remaining 10% will develop frank recurrence demanding another procedure. This can be a repeat microvascular decompression that is usually easier for the patient than the first operation, although more difficult for the surgeon. The only increased risk of reoperation is that of unexpected numbness. A percutaneous glycerol rhizotomy is our

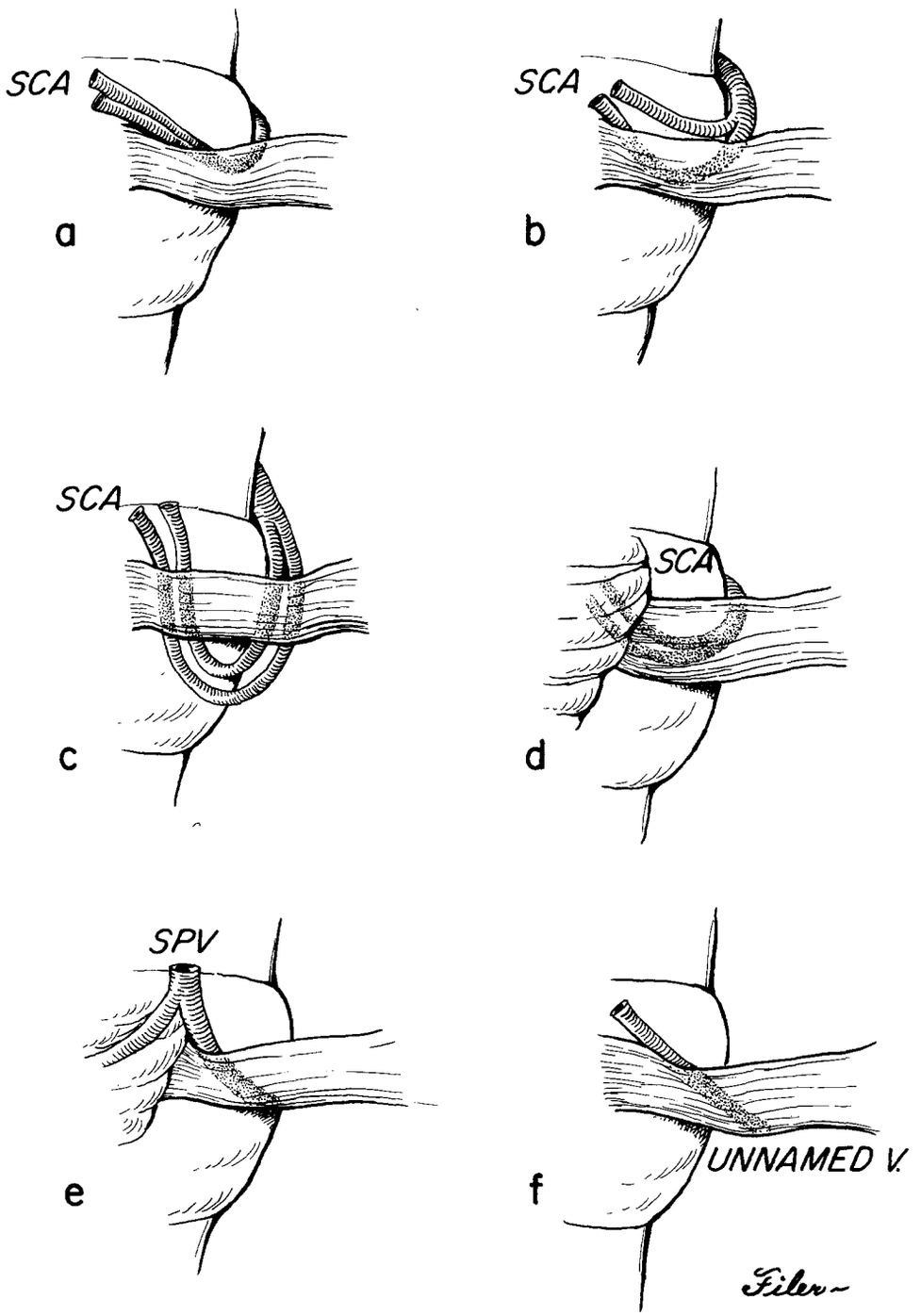

Figure 1. (a, b) Common types of obvious vascular compression in trigeminal neuralgia. Rostral compression is generally found with lower facial neuralgia, lateral or medial compression with V_2 neuralgia, and caudal compression with V_1 neuralgia. (c) Some more subtle types of vascular compression of the trigeminal nerve. These findings may be the sole cause of trigeminal neuralgia or may be found in combination with more obvious compression (a, b). From Jannetta.[45] With permission. *(Figure continues)*

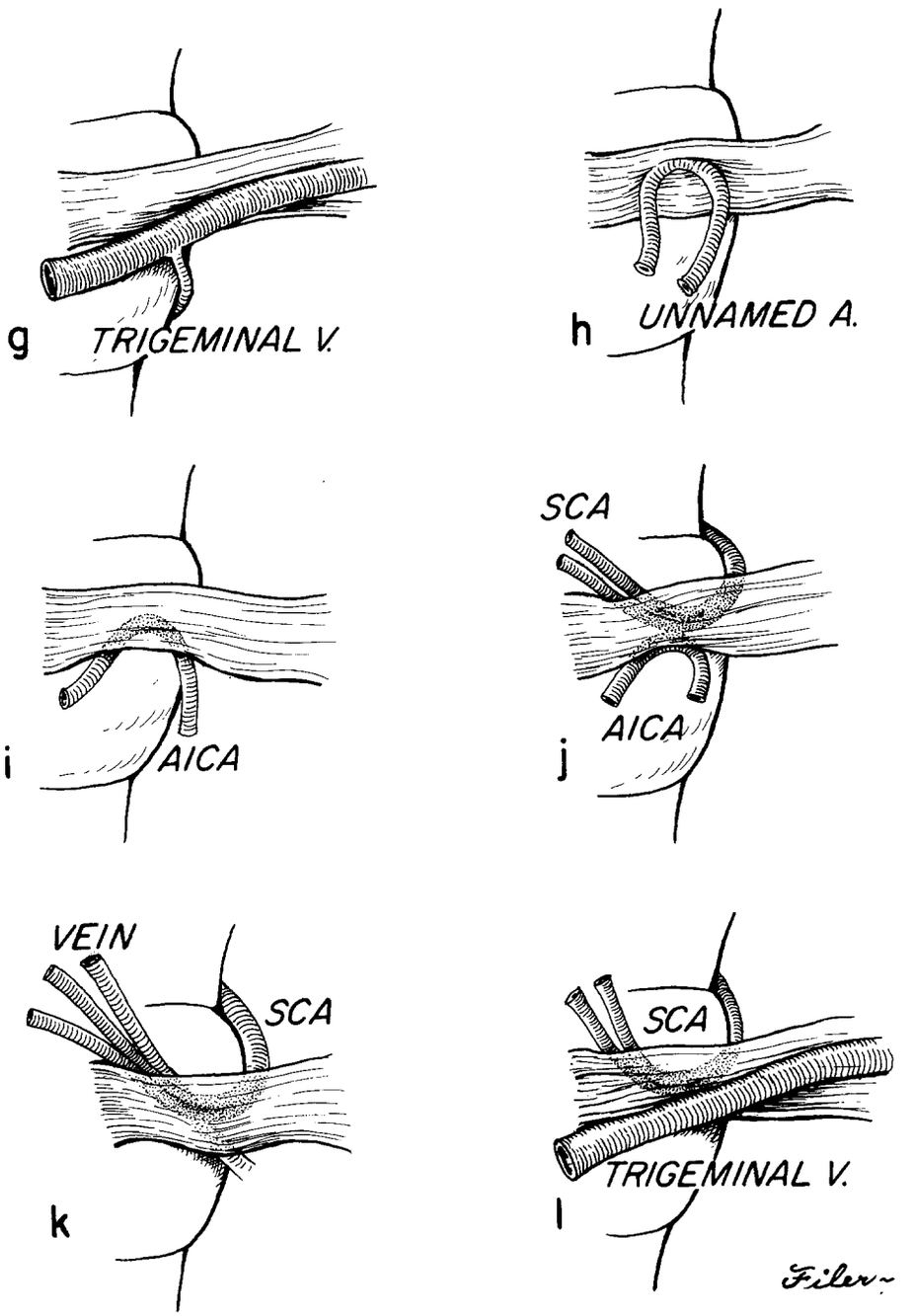

Figure 1. (*Continued*)

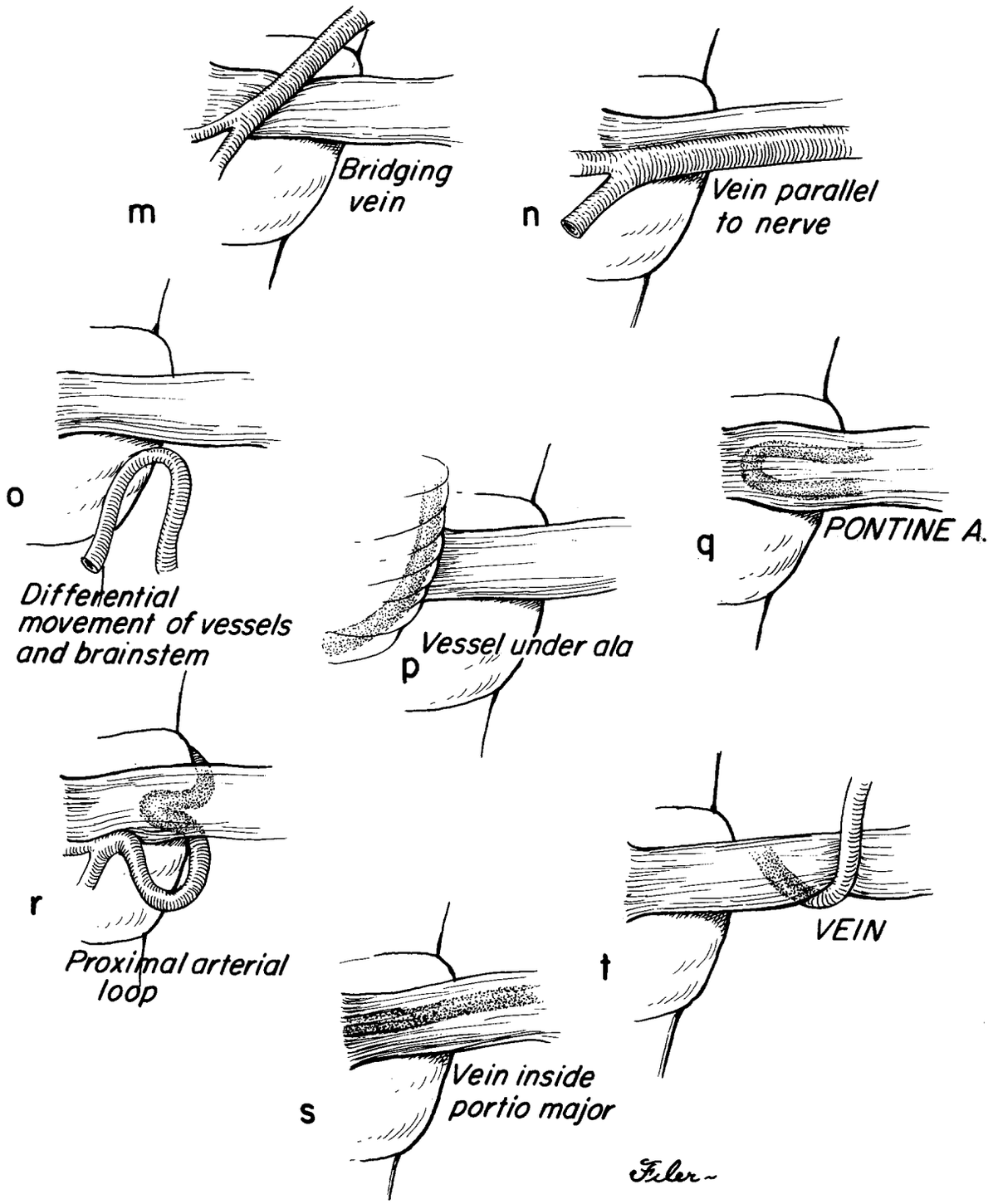

Figure 1. (*Continued*)

Table. Microvascular decompression for trigeminal neuralgia: comparison of operative results in several larger series

Author	Year	No. of Cases	Negative Findings (%)	Incomplete Relief (%)	Incomplete Recurrence (%)
Janetta	1975[36]				
	1976[38]				
	1980[40]	411	0.2	10	10
Apfelbaum	1982[2]				
	1984[3]				
	1988[4]	300	3	18.9	12.9
Bederson and Wilson	1989[9]	252	11.9	5	12
Sindou et al.	1987[87]	113	NA*	8.9	7.9
Piatt and Wilkins	1984[77]	103	8.7	23†	23†
Van Loveren et al.	1982[91]	50	16	4	12
Wilson et al.	1980[97]	50	10	2	14
Burchiel et al.	1981[13]	42	14	19†	—
Mori et al.	1986[64]	33	12	4	NA*
Ferguson et al.	1981[20]	24	2	12	17

* NA, data not included in study report. † Data concerning incomplete relief and recurrence are presented as one value.

other choice. I have stopped using radiofrequency procedures because of the higher incidence of serious sensory perversions after this procedure in contrast to the glycerol procedure.

Certainly the weight of the data and those reported by other surgeons with extensive patient series support and corroborate the hypothesis that vascular compression is indeed the cause of trigeminal neuralgia.

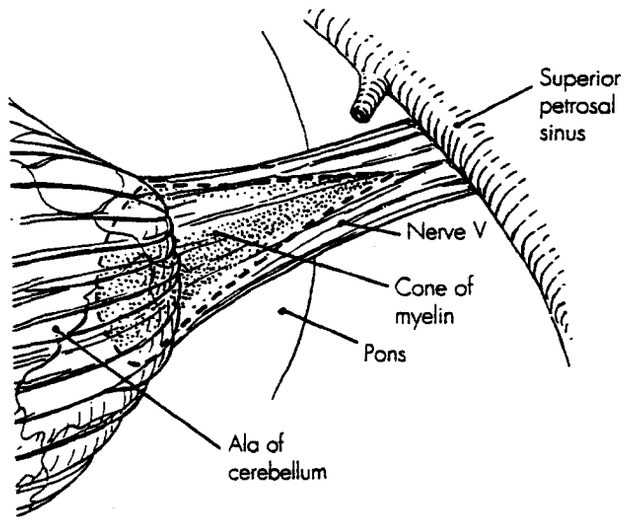

Figure 2. Central myelin extends a considerable distance from the nerve. This implies that the junctional area of central and peripheral myelin can be at, or near, the area of Meckel's cave. The nerve is protected on the rostroanterior aspect by the motor-proprioceptor fascicle. From Jannetta.[47] With permission.

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