Neurovascular Compression in Cranial Nerve and Systemic Disease

PETER J. JANNETTA, M.D.

As we age, our arteries elongate and our brains "sag." As a consequence of these processes, redundant arterial loops and bridging or intrinsic hindbrain veins may cause cross-compression of cranial nerve root entry zones in the cerebellopontine angle. This pulsatile compression can be seen to produce hyperactive dysfunction of the cranial nerve. Symptoms of trigeminal or glossopharyngeal neuralgia (somatic sensory), hemifacial spasm (somatic motor), tinnitus and vertigo (special sensory) and some cases of "essential" hypertension are caused by these vessels compressing cranial nerves V, IX–X, VII, VIII, and left X and medulla oblongata. Using microsurgical techniques, the symptoms may be relieved by vascular decompression, findings and results in 695 patients are briefly reviewed and correlated. A chronic primate model of "essential" hypertension is briefly described.

From the Department of Neurological Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania

DESPITE THE WORK of Dandy[5–7] and of Gardner[9–11] regarding vascular and other abnormalities in problems of cranial nerves, there was little acceptance of this concept until recently. Several reasons for this lack of acceptance may be given. 1) Incomplete verification by others: few neurosurgeons ventured safely and comfortably into the cerebellopontine angle. 2) Relatively primitive technology: lighting and instrumentation were not highly sophisticated. Magnification techniques were not used outside the laboratory except for ear surgery in some centers. 3) Inadequate documentation of findings: magnification photographic techniques were not available. 4) Rare definitive treatment: Dandy sectioned involved nerves except when tumors were present. Gardner alone performed vascular decompression in a few patients.

More recently, with the development of microsurgical techniques in neurologic surgery, of documentation of findings by photographic and videotape techniques, and of operative procedures aimed at treating the cause of various symptoms definitively; clarification and elaboration of the cause in a variety of apparently diverse but truly similar entities have been achieved. In this paper, the synthesis of a concept of hyperactivity of sensory, special sensory, motor, and autonomic function in cranial nerves V, VII, VIII, IX–X will be discussed. Mechanical compression of the root entry zone of cranial nerves, usually pulsatile, vascular and a result of the aging process is shown to be associated with and causal of trigeminal neuralgia (TN), hemifacial spasm (HFS), tinnitus and vertigo, glossopharyngeal neuralgia (GPN), and essential hypertension. Further, these entities, which are symptoms and not diseases, as first introduced regarding TN and HFS by Gardner,[11] can be reversed by vascular decompression of the appropriate cranial nerve root entry zone (REZ).

Patient Material

The patient population to be considered consists of 695 patients, 423 women and 272 men, aged 15 through 79 years, all but ten of whom were operated on at Presbyterian-University Hospital between 1971 and 1979. All patients had severe and disabling symptoms which were refractory to medical therapy or recurred after prior operation. All patients underwent retromastoid craniectomy and microvascular decompression of the appropriate cranial nerve root entry zone using microsurgical techniques. Documentation of intraoperative findings was by 35 mm color slides, 16 mm motion picture films, or color videotapes.

Trigeminal Neuralgia

This group consisted of 411 patients, 239 women and 172 men, aged 19 to 79 years, with classical trigeminal neuralgia. The pain was located on the right side in 246 patients, the left side in 143 patients, and bilateral in 12 patients. The pain was usually lower

Presented at the Annual Meeting of the American Surgical Association, Atlanta, Georgia, April 23–25, 1980.

Reprint requests: Peter J. Jannetta, M.D., Professor and Chairman, Department of Neurological Surgery, University of Pittsburgh, School of Medicine, Pittsburgh, Pennsylvania 15261.

Supported in part by grants from the Sarah Scaife foundation and the National Institutes of Health grant #5RO1 HL 24097-25SRC.

0003-4932/80/1000/0518 $00.90

and/or central facial in location, with 40 patients having pain over the entire V_{1-2-3} distribution and only eight patients having pure V_1 pain.

Hemifacial Spasm

This group consisted of 229 patients, 150 women and 79 men, aged 15–76 years, but usually presenting in the sixth decade, with a mean duration of symptoms of five years. Twenty-five patients had undergone previous neurolysis, crush, alcohol injection, or parital section.

Eighth Cranial Nerve Dysfunction

This group consisted of 38 patients, 24 women and 14 men, aged 17–69 years. Symptoms of intractable vertigo and tinnitus, with or without hearing loss, were present in 20 patients, of vertigo alone in seven, and tinnitus with or without hearing loss in 11 patients. The patients variously carried the diagnosis of Meniere's disease, vestibular or cochlear Meniere's disease, benign paroxysmal vertigo or vestibular neuronitis. Symptoms were located on the right side in 17 patients and on the left in 21 patients.

Glossopharyngeal Neuralgia

This group consisted of 17 patients, ten women and seven men, aged 30–69 years. Symptoms were located on the right side in ten patients and on the left in seven patients.

"Essential" Hypertension

This group consisted of 28 patients, 20 women and eight men, aged 31–74 years who were operated on for the problems noted above and coincidentally had essential hypertension[26] or developed hypertension as a result of operation (the first two patients). The right cerebellopontine angle was explored in five patients and the left in 23. None of these patients were operated on primarily for their hypertension.

TABLE 1. *Microvascular Decompression in Trigeminal Neuralgia*

Operative Findings	Number of Patients
Arterial	242
Aneurysm	1
Venous	57
AVM	1
Mixed arterial/venous	96
Tumor	15
No pathology	1
Unrecorded	1

TABLE 2. *Hemifacial Spasm 229 Patients*

Operative Findings	Number of Patients
Arterial	210
Mixed	10
Venous	4
Tumor	3
Aneurysm	1
AVM	1

Preoperative Evaluation

Patients underwent various consultations and special tests depending on their diagnosis. All patients had plain skull roentgenograms and complete otovestibular testing. TN patients had somatosensory evoked potentials of the trigeminal nerve and electromyography of the temporomasseter muscles. HFS patients had movies of their facial function and electromyography of their facial nerve innervated muscles. Eight nerve patients underwent further special testing of hearing and balance. All patients had computerized tomography with contrast. General internists, medical neurologists, otolaryngologists, and hypertension specialists saw the patients when indicated. Cerebral angiography was performed in some patients but is no longer done routinely because the information received has not been sufficient to justify the testing.

Operative Technique and Findings

All patients underwent a small (<5 cm diameter, usually <4 cm diameter) unilateral retromastoid craniectomy under general endotracheal anesthesia in the modified sitting (lounging) or more recently the contralateral, lateral decubitus position.[15,17–28] Using microsurgical techniques, the cerebellopontine angle was explored and the appropriate cranial nerve REZ was inspected. The offending blood vessel was tested by mobilization away from the nerve and placement of a small implant of plastic or silicone sponge or felt or autologous muscle. Veins, when found, were treated similarly or coagulated and divided. The occasional benign extra-axial tumor, frequently not diagnosed preoperatively, and usually causing REZ vascular cross-compression was removed. Details of technique have been published previously.[19–24,26]

Operative Findings

Abnormalities of the root entry zone of the symptomatic nerve are collated in Table 1 (TN), Table 2 (HFS), Table 3 (Eighth nerve dysfunction), Table 4 (GPN) and Table 5 ("Essential" hypertension). Several points should be made about the pathology:

TABLE 3. *Eighth Cranial Nerve Dysfunction*

Operative Findings		
Offending Vessel		
Artery		26
cochlear	6	
AICA	5	
PICA	5	
unidentified artery	4	
cochlear + AICA	1	
vertebral	2	
superior cerebellar	2	
AICA + PICA	1	
Vein		8
Artery and Vein		4
Total		38

AICA: anterior inferior cerebellar artery; PICA: posterior inferior cerebellar artery.

1) These abnormalities are quite specific and can be correlated with the location and type of symptom, *i.e.* lower facial TN is usually caused by a blood vessel, most commonly superior cerebellar artery on the rostral side of the nerve, V_1 by a vessel on the caudal aspect of the REZ, and isolated V_2 tic by a vessel on the side of the nerve. Similar correlations are seen in HFS and in cochlear and/or vestibular symptoms in the eighth nerve.
2) Multiple vessels are commonly found and were frequently missed early in this series.
3) Veins which have been coagulated and divided may recollaterize causing recurrence of symptoms.
4) Blood vessels are uncommonly seen cross-compressing the REZ of an asymptomatic cranial nerve inspected at operation when another nerve is being operated on. We do not know why some vascular cross-compression, like much cervical and lumbar disc disease and spondylosis, is asymptomatic.
5) REZ vascular compression is frequently subtle. Well-trained microsurgeons may not appreciate up to 30% of the abnormalities early on in their experience with these procedures. These are "unforgiving procedures"[2] which demand petty attention to detail if one is to make valid observations and operate safely.

TABLE 4. *Glossopharyngeal Neuralgia*

Offending Vessel	Number
Posterior inferior cerebellar artery	9
Anterior inferior cerebellar artery	1
PICA + vertebral	1
Unidentified small arterial loop	1
No vessel identified	2
Vertebral artery and vein	1
Vertebral artery	1
Vein	1

TABLE 5. *Essential Hypertension*

Offending vessel (all left)		
vertebral artery		13
"normal"	9*	
ectatic	4	
PICA		8†
vertebral and PICA		1
AICA and PICA		1

AICA: anterior inferior cerebellar artery.
PICA: posterior inferior cerebellar artery.
* One questionable, #19, anterior.
† Questionable, #28, very caudal.

Operative Results and Complications

Results of operations are summarized in Tables 6 (TN), 7 (HFS), 8 (Eighth nerve dysfunction), 9 (GPN), and 10 ("essential" hypertension). Complications are summarized in Tables 11 (TN), 12 (HFS), 13 (Eighth nerve dysfunction), Table 14 (GPN).

Several points of interest should be briefly discussed. Repeat operations in TN were sometimes necessary to decompress the nerve satisfactorily. The most common reason for lack of response was a missed blood vessel or vessel that had not been properly decompressed, next was a recollateralized vein and lastly, new late vascular compression due to continuing arterial elongation. Thirty-eight patients take some medi-

TABLE 6. *Microvascular Decompression in Trigeminal Neuralgia*

Results of Operation		Number	Per Cent
Well after one MVD*		328	79.8
Well after repeat MVD		14	83.2
Well after third MVD		1	83.5
Subtotal		343	83.5
Well after MVD & RFL		17	87.6
Well after MVD on med. Rx		38	96.9
Subtotal		398	96.9
Slight pain, no meds		2	0.5
Severe pain		5	1.2
Decreased†		5	1.2
postop	3		
suicide	1		
accident	1		
Status unknown		1	0.2
Total		411	100.0

* MVD: microvascular decompression.
† Three: other patients who were well, have died.

TABLE 7. *Hemifacial Spasm in 229 Patients*

Operative Results	Number	Per Cent
No spasm after one operation	201	87.8
No spasm, second procedure necessary	12	5.2
Partially symptomatic (<25% of preop level)	11	4.8
Failure of therapy	5	2.2

cation, usually small doses of phenytoin to remain pain-free. Two have occasional discomfort without medication. These groups together (total or significant relief) constitute 92.8% of the patients.

Operative deaths in the entire series were from stroke (two patients) and brain stem infarction (two patients): one death in a 79-year-old woman, two in patients with tumors (one a leukemic, was admitted for a radiofrequency rhizotomy and had a 4 cm diameter acoustic; one with a large undiagnosed angioma). We have now had no operative deaths in over 350 consecutive patients.

The major risk in HFS procedures is loss of ipsilateral hearing (table 12), especially in older patients and occasionally occurring two days or more postoperatively. The eighth cranial nerve patients with vertigo and intercurrent unsteadiness have had much better results than those with tinnitus which is much less likely to improve. One older woman in this series sustained an apparent small brainstem infarction two days after operation, leaving her with more severe vertigo than she had preoperatively and an internuclear ophthalmoplegia. She is miserable, and, unfortunately, hateful.

The Hypertension Model

An acute model of pulsatile left medullary compression using a pulsating balloon-catheter powered by a pump, gave results which were statistically significant regarding cardiac output and stroke volume but not for hypertension itself.[38] The brief duration of the studies in an experimental situation attempting to simulate a chronic clinical problem, interference of cardiovascular reflexes by anesthetic agents, and equipment failure (usually balloon breakage) led us to a self-contained system.

In this model (the "balloon-balloon" model) a 5 cm diameter balloon is placed in the thoracic aorta of a baboon through a left axillary arteriotomy. An attached catheter is led subcutaneously over the scapula, where a "Y" connector for injection purposes is in continuity with the catheter. At the other end of the catheter is a 2 cm diameter balloon. This balloon is placed in the subarachnoid space along the left anterolateral medulla via a small retromastoid craniectomy. With placement of air or fluid into the system, contraction of the left ventricle causes compression of the intra-aortic balloon and concurrent expansion of the small balloon in the configuration of an arterial pulse wave. The small balloon functions, therefore, as an artificial artery under control of the heart. The balloon–balloon system is made of Avcothane®.

Experiments on three control and three hypertensive preparation animals have been completed. In these studies, cardiac output, pulse rate, stroke volume, peripheral resistance, and arterial blood pressure were measured over periods up to seven weeks. The data, which appears to simulate essential hypertension, although presented at the meeting, are not published here as several of the author's colleagues have participated in these studies and will be coauthors of a paper specifically limited to this experimental model.

TABLE 8. *Eighth Cranial Nerve Dysfunction*

Symptoms	Results of Operation: Relieved	Mild Improvement	Recurred or no Relief	Worse
Vertigo and tinnitus	13	0	6	1
Vertigo	6	1	0	0
Tinnitus	5	0	6	0
Totals	24	1	12	1

TABLE 9. *Glossopharyngeal Neuralgia Personal Series (11 Patients)*

Treatment	
microvascular decompression	9
nerve section	2
Results	
pain free—MVD	6
no relief or recurrence—MVD	2
pain free–section	2
dead—stroke	1

TABLE 10. *Essential Hypertension Vascular Decompression Results of Left Lateral Medullary-IX, X**

Normotensive on on drug therapy	6
Normotensive on diuretics	1
Normotensive for a time, recurred	1
Improvement on same drug therapy	3
No improvement	3
Total	14

* Not attempted in 8 of 22 patients; attempted in 14 patients.

TABLE 11. *Microvascular Decompression in Trigeminal Neuralgia*

Complications	Number of Patients
Permanent cranial nerve deficit	23
Aseptic menningitis	21
Intracranial hematomas	4
Mortality	4
Bacterial meningitis	3
Infarction	3
Pneumonia	2
CSF rhinorrhea	2
Pulmonary embolism	2

TABLE 13. *Eighth Cranial Nerve Dysfunction*

Complications
Temporary
Epidural hematoma
postoperative and n. VI paresis
X cranial nerve paresis
aseptic meningitis
urinary tract infection
Permanent
internuclear ophthmoplegia and worsening of vertigo
deterioration of hearing

Discussion

These cranial nerve symptoms, which are apparently diverse, on analysis from a perspective concerned with the arterial elongation and brain sag seen routinely with the aging process, do indeed appear to be similar. That is, they are all symptoms of hyperactive dysfunction of the cranial nerve, a caricature of their normal function. They are all, gradually or rapidly, accompanied by progressive evidence of loss of function. They are caused almost exclusively by pulsatile vascular compression at the root entry zone, a junctional area between central and peripheral myelin (except in the eighth nerve which contains central nervous system myelin to the internal auditory meatus region), and where there are known defects in the myelin.

These problems have generally in the past been treated by replacing the hyperactive symptoms with loss of function (*i.e.*, pain-numbness, spasm-weakness, *etc.*) using destructive techniques. Definitive treatment is now available in many centers.[1-3,8,14,15,17-29,39,41] Apfelbaum[2] has compared a series of radiofrequency lesions with microvascular decompression in TN. He makes a strong point about the difference in quality of life if the patient does not have a constant reminder of his prior symptoms. This author is biased toward the definitive procedure but feels that radiofrequency rhizotomy is a useful method of treatment, the only question being that of the indications for one procedure versus the other.

Lew and Grant in 1938[30] discussed the role of arteriosclerosis in the origin of TN but placed the theoretical lesion in the thalamus. Early investigators noted abnormalities of the cranial nerves in patients with TN and HFS.[4-7,9-11] Only Dandy[5-7] inspected the trigeminal nerve in a large number of patients using a caudolateral approach and without magnification. The surgical microscope was not applied to clinical neurosurgery until Kurze began to use it in about 1957.

Sunderland[40] and Hardy and Rhoton[16] have done postmortem dissections of the neurovascular relationships at the base of the brain. These careful studies are without clinical correlation. Haines et al.[13,14] have recently performed two postmortem studies with clinical correlation of these relationships about the trigeminal nerve, the latter study a comparison with videotapes from operations for TN. The differences in the two groups of brains are significant. Still, some cadaver brains have apparently significant trigeminal nerve vascular compression without a history of symptoms during life. These data correlate with the progressively common asymptomatic evidence of lumbar and cervical disc disease and spondylosis in the population at large.

A number of points regarding clinical hypertension may be made which may be of interest especially from

TABLE 12. *Hemifacial Spasm in 229 Patients*

Cranial Nerve	Deficit		Number of Patients
Trigeminal	Hypalgesia		1
Facial	postop weakness		9 (6 persist)
	postop late onset weakness		7 (1 persists)
Acoustic	Hearing loss		18
	deaf	8	
	profound	8	
	mild	2	

Cranial nerve deficits (23 patients).

TABLE 14. *Glossopharyngeal Neuralgia (Personal Series—11 Patients)*

Complications	Number of Patients
Death, hypertensive crisis	1
Temporary hypertension	1
Decreased palatal and gag reflexes	
MVD (temporary)	2
section	2

the perspective of neurovascular compression as an etiologic factor.

- High (or low or normal) blood pressure is a measurement, not a disease.
- A lucid literature exists regarding renal artery stenosis, pheochromocytonia and other endocrine tumors, based on understanding of the etiologic process in these diseases.
- A literature of similar clarity regarding the patient population with "essential" hypertension, where the etiology has been obscure, does not exist.
- For years surgeons successfully treated "essential" hypertension in selected patients by interrupting the autonomic nervous system peripherally.
- Most antihypertensive drugs have major action in the central nervous system, and in the area impinged upon by the artery.
- Hypertension augments arterial elongation, a self-perpetuating system correlating with clinical progression.
- The autonomic distribution of the vagus nerves is asymmetrical.
- Although the common finding in patients has been left lateral medulla vascular compression, it is conceivable that arterial loops could cause hypertension by pulsatile compression anywhere from midline and paramedian forebrain to upper thoracic spinal cord, for some cardiovascular control is located throughout these regions. The left anterolateral medulla and vagus nerve are mechanically available to vascular compression by elongated vertebral or posterior inferior cerebellar arteries.
- Left anterolateral medullary arterial compression has not been noted in over 50 nonhypertensive patients.
- Several neurosurgeons have now made independent observations corroborating the author's findings.[42,43]

The hypertension experimental work[27,39] must be considered preliminary insofar as full proof of the Henle-Koch postulates regarding etiology are concerned until the present series of chronic animal experiments using the "balloon-balloon" model are extended. The broadening of the concept of hyperactive cranial nerve sensory, motor and special sensory dysfunction to include hyperactive autonomic (vagal nerve) dysfunction appears reasonable and further clinical and laboratory experience will presumably verify or refute the many questions which are naturally raised by this apparently real phenomenon. This author, on the basis of the findings noted here, raises the question, with some temerity, of a concept of the origin of a growing series of problems associated with the aging process and specifically with vascular deterioration and its primary mechanical and subsequent morphological and pathophysiologic effects on control of various cranial nerve and systemic autonomic function.

Acknowledgments

The author would like to thank Mr. D. Bissonette and Mrs. E. Narvin for their extensive work in data compilation.

References

1. Alksne J. Neurosurgical Society of America, Colorado Springs, 1977. (Abstract)
2. Apfelbaum RI. A comparison of percutaneous radiofrequency trigeminal neurolysis and microvascular decompression of the trigeminal nerve for the treatment of tic douloureux. Neurosurgery 1977; 1:1:16–21.
3. Becker DP. What's new in neurological surgery. Bull Am Coll Surg 1978; 63:1:23–27.
4. Campbell E, Keedy C. Hemifacial spasm: a note of the etiology in two cases. J Neurosurg 1944; 4:342–347.
5. Dandy WE. The treatment of trigeminal neuralgia by the cerebellar route. Ann Surg 1932; 96:787–795.
6. Dandy WE. Concerning the cause of trigeminal neuralgia. Am J Surg 1934; 24:447–455.
7. Dandy WE. Trigeminal neuralgia and trigeminal tic douloureux. *In* Lewis' Practice of Surgery. Volume 12 Hagerstown, WF Prior Company, 1946. pp. 167–187.
8. Dujovny M, Osgood CP, Faille R, et al. Posterior fossa AVM producing hemifacial spasm: a case report. Angiology 1979; 30:425–432.
9. Gardner WJ. The mechanism of tic douloureux. Trans Am Neurol Assoc 1953; 78:168–173.
10. Gardner WJ, Miklos MV. Response of trigeminal neuralgia to "decompression" of sensory route, Discussion of cause of trigeminal neuralgia. JAMA 1959; 170:1773–1776.
11. Gardner WJ. Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. J Neurosurg 1962; 19:947–957.
12. Haines SJ, Martinez JA, Jannetta PJ. Arterial cross-compression of the trigeminal nerve at the pons in trigeminal neuralgia. J Neurosurg 1979; 50:257–259.
13. Haines SJ, Jannetta PJ, Zorub DS. Microvascular relations of the trigeminal nerve. An anatomical study with clinical correlation. J Neurosurg 1980; 52:381–386.
14. Fabinyi GCA, Adams CT. Hemifacial spasm: treatment by posterior fossa surgery. J Neurol Neurosurg Psychiatr 1978; 41: 829–833.
15. Hankinson HL, Wilson CB. Microsurgical Treatment of hemifacial spasm. West J Med 1976; 124:191–193.
16. Hardy DG, Rhoton AL, Jr. Microsurgical relationships of the superior cerebellar artery and the trigeminal nerve. J Neurosurg 1978; 49:669–678.
17. Jannetta PJ. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. J Neurosurg 1967; 26(11):1159–162.
18. Jannetta PJ. Microsurgical exploration and decompression of the facial nerve in hemifacial spasm. Curr Topics Surg Res 1970; 2:217–220.
19. Jannetta PJ, Hackett ER, Ruby JR. Electromyographic and electron microscopic correlates in hemifacial spasm treated by microsurgical relief of neurovascular compression. Surg Forum 1970; 21:449–451.

20. Jannetta PJ. Trigeminal and glossopharyngeal neuralgia. Curr Diagnosis 1971; 3:849–850.
21. Jannetta PJ. Neurovascular cross-compression in patients with hyperactive dysfunction symptoms of the eighth cranial nerve. Surg Forum 1975; 26:467–468.
22. Jannetta PJ. Microsurgical approach to the trigeminal nerve for tic douloureux. *In* Krayenbuhl H, Maspes PE, Sweet WH (eds.) Progress in Neurological Surgery. S Karger, AG Basel, Switzerland, Volume 7 (Pain—its neurosurgical management), 1976. pp. 180–200.
23. Jannetta PJ. Treatment of trigeminal neuralgia by suboccipital and transtentorial cranial operations. Clin Neurosurg 1977; 24:538–549.
24. Jannetta PJ, Abbasy M, Maroon JC, et al. Hemifacial spasm: etiology and definitive microsurgical treatment. Operative techniques and results in forty-seven patients. J Neurosurg 1977; 47:321–328.
25. Jannetta PJ. Observations on the etiology of trigeminal neuralgia, hemifacial spasm, acoustic nerve dysfunction and glossopharyngeal neuralgia. Definitive microsurgical treatment and results in 117 patients. Neurochirurgia 1977; 20:145–154.
26. Jannetta PJ. Microsurgery of cranial nerve cross-compression. Clin Neurosurg 1979; 26:607–615.
27. Jannetta PJ, Gendell HM. Clinical observations on etiology of essential hypertension. Surg Forum 1979; 30:431–432.
28. Laha RK, Jannetta PJ. Glossopharyngeal neuralgia. J Neurosurg 1977; 47:316–320.
29. Lazar M. Trigeminal neuralgia: recent advances in management. Texas Med 1978; 75:45–48.
30. Lewy FH, Grant FC. Physiopathologic and pathoanatomic anatomic aspects of major trigeminal neuralgia. Arch Neurol Psychiatr 1938; 40:1126–1134.
31. Loeser JD. What to do about tic douloureux. JAMA 1978; 239: 12:1153–1155.
32. Maroon JC. Hemifacial spasm, a vascular cause. Arch Neurol 1978; 35:481–483.
33. Maroon JC, Lunsford LD, Deeb ZL. Hemifacial spasm due to aneurysmal compression of the facial nerve. Arch Neurol 1978; 35:545–546.
34. Petty PG. Arterial compression of the trigeminal nerve at the pons as a cause of trigeminal neuralgia. Inst Neurol Madras Proc 1976; 6:93–95.
35. Petty PG, Southby R. Vascular compression of the lower cranial nerves: Observations using microsurgery, with particular reference to trigeminal neuralgia. Aust NZ J Surg 1977; 47:3.
36. Petty PG, Southby R, Siu K. Vascular compression: cause of trigeminal neuralgia. Med J Aust 1980; 1:166–167.
37. Rhoton AL, Jr. Microsurgical neurovascular decompression for trigeminal neuralgia and hemifacial spasm. J Fla Med Assoc 1978; 65:425–428.
38. Ruby JR, Jannetta PJ. Hemifacial spasm: ultrastructural changes in the facial nerve induced by neurovascular compression. Surg Neurol 1975; 4:369–370.
39. Segal R, Gendell HM, Canfield D, et al. Cardiovascular response to pulsatile pressure applied to ventrolateral medulla. Surg Forum 1979; 30:433–435.
40. Sunderland S. Neurovascular relationships and anomalies at the base of the brain. J Neurol Neurosurg Psychiatr 1948; 11: 243–247.
41. Weidmann M. Surgical treatment by microvascular decompression of the trigeminal nerve root. Med J Aust 1979; 2:628–630.
42. Kurze T. Personal communication.
43. Fein J, Frishman. Neurogenic hypertension related to vascular compression of the lateral medulla. Neurosurgery 1980; 6: 615–622.

Discussion

Dr. Robert W. Rand (Los Angeles, California): Dr. Jannetta has introduced a unique and original concept of medulla oblongata vascular compression by the vertebral artery as one of the causes of hypertension. Further, he has demonstrated this neurovascular compression phenomenon can be surgically treated in certain instances by microvascular decompression. This experimental and clinical research has historic roots, as he mentioned, in the surgical treatment of the syndromes of trigeminal neuralgia and hemifacial spasm by Drs. Walter Dandy and W. James Gardner, respectively.

I would like to review briefly these operations. The original drawings by Dr. Dandy in the late 1920s depict his evidence that the looping cerebellar arteries can compress and distort the trigeminal nerve and thus cause tic douloureux or major trigeminal neuralgia.

Dr. Jannetta has expanded and popularized this concept of the neurovascular compression syndrome of Dandy as a mechanism of the cause of trigeminal neuralgia. About 103 of 206 instances described originally by Dandy were caused by some type of vascular compression of the trigeminal nerve. In addition to the arteries, Dandy also described venous neurocompression. As Dr. Jannetta has pointed out, not many surgeons believed that Dr. Dandy was correct at that time.

Dr. W. James Gardner in 1959 was the first surgeon to put an absorbable sponge between the compressing cerebellar artery and the distorted trigeminal nerve. The first patient treated by Dr. Gardner remained completely free of tic douloureux without neurologic loss for five years. Subsequent patients treated by Gardner were also relieved of the pain.

It has been 14 years since, with Dr. Jannetta, I operated on my first patient using microneurosurgical decompressive techniques of the trigeminal nerve. The patient remains completely free of tic douloureux and without sensory loss. Additional patients, like those of Dr. Gardner, have been successfully treated at UCLA in this manner by subtemporal transtentorial and suboccipital operations.

Dr. Gardner went on to work on the problem of hemifacial spasm, as Dr. Jannetta has indicated, using similar neurovascular decompression procedures he developed for trigeminal neuralgia. In 1961 Dr. Gardner's first group of seven patients with hemifacial spasm successfully underwent his neurovascular decompression operation without having facial palsy, and yet being relieved completely of the hemifacial spasm.

Dr. Brooke Roberts (Philadelphia, Pennsylvania): To those of us who are not neurosurgeons, this concept is almost completely new. I had been aware of Dr. Jannetta's work with regard to tic douloureux and some of the neuralgias of the cranial facial nerves, but this concept of hypertension explained on a similar basis is new and an extraordinarily exciting concept.

New concepts often have difficulty being accepted. It is fascinating when a group of diseases or entities whose cause is poorly or completely understood are brought together to show by a common cause that they can be explained and to have them explained in such a simple way.

What percentage of hypertension, so called essential hypertension, caused by this remains to be seen? Some of it is unquestionably true from the results which we have heard. I look forward to more work from this quarter.

Dr. William S. Blakemore (Toledo, Ohio): I have watched Dr. Jannetta's work for a number of years. It is true that he has had to go through the criticism of his colleagues to establish the concept of the hyperexcitability of the endocranial portion of the cranial nerves. I wish especially to comment on this newly presented cause for hypertension. It evolves that after 25 or 30 years, when Smithwick and Zintel and Wolferth and others studied surgical treatment of hypertension, which in long-term follow-up studies has better results for the severely hypertensive patient than any medical treatment with ten year follow-up results. We have identified several