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The Radiologic Assessment of Trigeminal Neuropathy

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The clinical and radiologic records of 76 patients with trigeminal neuropathy and an abnormal imaging study (CT and/or MR) were analyzed retrospectively. The trigeminal nerve (cranial nerve V) was divided into proximal (brainstem, preganglionic, gasserian ganglion, and cavernous sinus) and distal (extracranial V_1, V_2, and V_3) segments. Lesions were organized according to segments and correlated with the type and distribution of clinical symptoms or signs. The purpose of the study was to (1) determine the efficacy of clinical localization of cranial nerve V lesions, (2) compare CT and MR for cranial nerve V imaging, (3) develop an MR protocol for effective cranial nerve V imaging, and (4) construct a differential diagnosis by anatomic segment for lesions of cranial nerve V. Clinical localization was found to be extremely inaccurate. CT was not as sensitive as MR for lesions involving the basal cisterns and skull base and will not detect the most common brainstem lesions (small infarcts and multiple sclerosis plaques). The MR protocol developed does not rely heavily on clinical localization. On the basis of lesions found in this series, a differential diagnosis by segment was developed.

Patients with cranial nerve V symptoms should undergo MR imaging according to the protocol provided in this article. CT is not as effective as MR in imaging some cranial nerve V segments. Clinical localization is inaccurate.

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The trigeminal nerve (cranial nerve V) is the largest of the cranial nerves, serving both sensory and motor functions to the scalp and face. From its most peripheral branches to its central projections in the cerebral cortex, the trigeminal nerve and its central projections follow a protracted course through the complex anatomy of the face, base of skull, brainstem, thalamus, and cerebral cortex. In the past, clinical symptoms and signs were considered an accurate means of localizing lesions along this complex course. With the advent of CT, and more recently MR imaging, the radiologist is better able to evaluate the entire intra- and extracranial course of cranial nerve V.

Previous reports involving the fifth cranial nerve have focused on specific anatomic areas [1–7], specific types of symptom complexes [8–11], or specific types of lesions [12–21]. Except for a single study in which multiple cranial nerves were evaluated [22], no study approaches cranial nerve V imaging from the perspective of the clinical radiologist. That is, given a patient with cranial nerve V symptoms, how does clinical localization help tailor the imaging examination, where and what are the lesions causing these symptoms, and what is the best method for imaging the patient.

In this study, the clinical and radiographic records of 76 patients with cranial nerve V symptoms and positive CT and/or MR studies were reviewed. The principal purpose of this study was to (1) determine the efficacy of clinical localization, (2) compare CT and MR for cranial nerve V imaging, (3) establish the most efficient MR imaging protocol, and (4) construct a differential diagnosis for lesions of cranial nerve V by anatomic segment.

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Materials and Methods

The radiologic and clinical records of 76 patients with cranial nerve V symptoms and pathologically proved CT and/or MR examinations were reviewed. Pathologic proof was obtained by surgical biopsy (primary tumors), typical clinical course (metastases, multiple sclerosis, and infarcts), or further radiologic workup (vascular malformations). Lesions were organized anatomically according to their site of origin. Each lesion was then correlated with the distribution and type of clinical signs and symptoms.

In order to objectively localize lesions along cranial nerve V, the nerve was subdivided into proximal and distal portions. The proximal portions were defined as intracranial and included the brainstem and central cortical projections, the preganglionic (prepontine) segment, the gasserian ganglion, the cavernous sinus portion of the first and second divisions of cranial nerve V (V_1 and V_2), and the short intracranial segment of the third division (V_3). The distal portions were defined as the extracranial peripheral divisions.

The majority of contrast-enhanced CT studies were performed on GE 8800 or 9800 scanners with bolus injection of 50 ml followed by rapid infusion of 150 ml of 60% iodinated contrast material. Standard head examinations consisted of contiguous 8-mm axial slices, with 5-mm slices in the posterior fossa. Standard face and neck examinations were performed with 5-mm contiguous axial slices. In selected cases, 5-mm coronal images were obtained through the base of the skull. The majority of MR studies were performed on a GE Signa 1.5-T scanner. After initially trying a variety of protocols, the protocol in Table 1 was established and used for the majority of MR scans. A typical scan without supplemental views took approximately 35 min.

Anatomy

The trigeminal nerve is the largest of the cranial nerves and has both sensory and motor functions. It is associated with and innervates the structures derived from the first branchial arch. Specifically, the trigeminal nerve mediates sensation to the scalp; the face; and the ectodermally derived mucous membranes of the nasal cavity, sinuses, and mouth. Motor innervation travels with V_3 to the four muscles of mastication (masseter, temporalis, and medial and lateral pterygoid), the mylohyoid muscle, the anterior belly of the digastric muscle, and the tensor tympani and tensor veli palatini muscles.

There are four central brainstem nuclei: (1) the *main sensory nucleus*, which mediates tactile sensation, (2) the *spinal nucleus*, which mediates pain and temperature, (3) the *motor nucleus*, which provides motor innervation, and (4) the *mesencephalic nucleus*, which mediates proprioception (Fig. 1A). These nuclei lie predominantly in the tegmentum of the lateral pons, along the anterolateral aspect of the fourth ventricle, at the level of the root entry zone of the trigeminal nerve. From this area of the pons, the mesencephalic nucleus projects cephalad into the midbrain to the level of the inferior colliculus, while the spinal nucleus extends caudally to the level of the second cervical vertebra. The secondary central projections are the prominent ventral (crossing) and minor dorsal (noncrossing) trigeminal thalamic tracts. Both tracts terminate in the ventral posteromedial thalamic nucleus. The most central projections connect the ventral posteromedial thalamic nucleus to the central gyrus of the cerebral cortex.

The large sensory and smaller motor root exit via the lateral pons as a common trunk that runs anteriorly and superiorly through the prepontine cistern. This is referred to as the *preganglionic segment*. Throughout its course in the preganglionic segment and gasserian ganglion, the trigeminal trunk is somatotopically organized, with the maxillary division (V_2) between the mandibular (V_3) (inferior) and the ophthalmic (V_1) (superior) divisions. As the trunk enters the pons (the root entry zone) the organization is reversed, with the V_1 and V_3 divisions exchanging positions [24]. The motor root remains inferior to the sensory root throughout.

The main trunk of cranial nerve V enters Meckel cave through an opening in the dura, the porus trigeminus (entrance to Meckel cave). The nerve carries its dural covering with it into Meckel cave. The leptomeninges also follow the nerve, resulting in a CSF-filled subarachnoid space, the *trigeminal cistern*, surrounding the nerve within Meckel cave.

The *gasserian ganglion* (trigeminal ganglion, semilunar ganglion) lies in Meckel cave and contains the cell bodies of the afferent sensory fibers, excluding those that mediate proprioception. Distal to the gasserian ganglion, the trigeminal nerve trifurcates into its three principal branches, the *ophthalmic* (V_1), *maxillary* (V_2), and *mandibular* (V_3) nerves (Fig. 1B).

TABLE 1: Spin-Echo MR Imaging Protocol in Trigeminal Neuropathy

Variable	Protocol
Patient preparation	Place as far into head coil as possible
Localizing scan	Sagittal T1-weighted, 800/30 (TR/TE), two acquisitions; thickness/skip = 5.00 mm/2.5 mm
Brainstem and central projection scan	Axial T2-weighted, 2000/30,80, one acquisition; thickness/skip = 5.0 mm/2.5 mm
Cisternal, skull base, and extracranial V_1, V_2, and proximal V_3 scans	Axial (two acquisitions) and coronal (four acquisitions) T1-weighted, 800/30, scans from mid pons, including orbit and maxillary sinus; thickness/skip = 3.0 mm/0 mm (axial) and 3.0 mm/1.5 mm (coronal)
Supplemental scans	
If V_3 involved	Extend axial T1-weighted scan from skull base to inferior mandible; thickness/skip = 5 mm/1 mm
When gadolinium used	Repeat T1-weighted axial and coronal imaging

Note.—Matrix size = 256 × 256 for all scans; field of view = 24 cm for sagittal sequences and 20 cm for all others; cardiac gating is used for all scans; all T2-weighted scans have flow compensation.

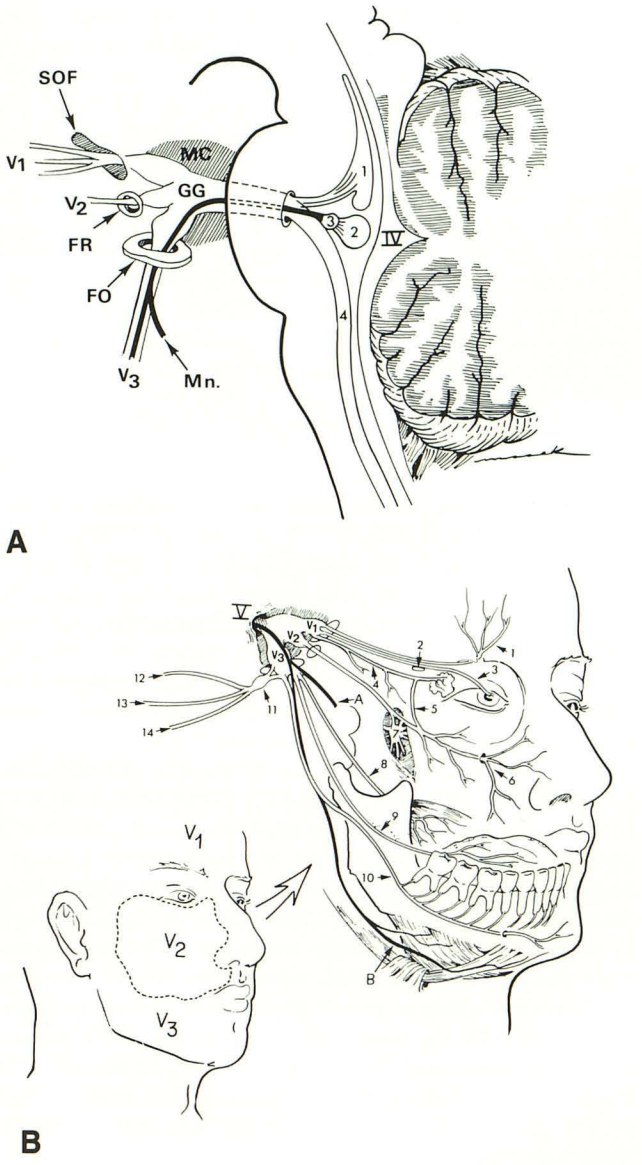

Fig. 1.—*A*, Proximal segments of trigeminal nerve: 1 = mesencephalic nucleus; 2 = main sensory nucleus; 3 = motor nucleus; 4 = spinal nucleus; V_1 = ophthalmic division; V_2 = maxillary division; V_3 = mandibular division; MC = Meckel cave; GG = gasserian ganglion; SOF = superior orbital fissure; FR = foramen rotundum; FO = foramen ovale; Mn. = masticator nerve. Heavy lines are motor divisions; lighter lines are sensory divisions.

***B*, Distal segments of trigeminal nerve: 1 = frontal nerve; 2 = ciliary ganglion; 3 = nasociliary nerve; 4 = lacrimal nerve; 5 = zygomatic nerve; 6 = infraorbital nerve; 7 = pterygopalantine ganglion; 8 = buccal nerve; 9 = lingual nerve; 10 = inferior alveolar nerve; 11 = otic ganglion; 12 = nerve to parotid gland; 13 = nerve to tensor veli palatini muscle; 14 = nerve to tensor tympani muscle; A = masticator nerve; B = mylohyoid nerve; V_1, V_2, and V_3 refer to the facial distribution of the respective trigeminal nerve divisions. Heavy lines are motor divisions and lighter lines are sensory divisions.**

(Reprinted with permission from Hardin and Harnsberger [23].)

V_1 courses in the lateral wall of the cavernous sinus, exiting the skull base through the superior orbital fissure. Within the orbit it subdivides into three major branches, the lacrimal, frontal, and nasociliary nerves. These distribute to and provide sensory innervation to the scalp, nose, and globe. V_1 mediates the afferent aspect of the corneal reflex (Fig. 1B).

V_2 travels near the crease formed between the lateral dural wall of the cavernous sinus and the skull base, exiting the skull base through the foramen rotundum. After passing through the foramen rotundum, the nerve enters the pterygopalatine fossa, where it gives off several branches, including the zygomatic, pterygopalatine, and posterior superior alveolar nerves. The main trunk of V_2 continues anteriorly as the infraorbital nerve, which enters the orbit through the inferior orbital fissure. This nerve travels anteriorly within the infraorbital groove, in the floor of the orbit, and emerges onto the face through the infraorbital foramen. V_2 supplies sensory innervation to the middle third of the face (cheek) and upper teeth (Fig. 1B).

V_3 does not traverse the cavernous sinus, but rather runs along the skull base laterally and exits through the foramen ovale. The motor root bypasses the gasserian ganglion altogether, joining V_3 as it exits the skull base through foramen ovale. As V_3 exits the skull base, it enters the nasopharyngeal masticator space. It then divides into several sensory branches with the principal ones including the buccal, auriculotemporal, inferior alveolar, and lingual nerves. The inferior alveolar nerve enters the mandibular foramen in the ramus of the mandible and travels through the mandibular canal to emerge on the chin at the mental foramen. The sensory branches of V_3 supply sensation to the lower third of the face, tongue, floor of mouth, and jaw (Fig. 1B). In addition to the sensory branches, the motor root running with V_3 has two major branches, the masticator nerve and the mylohyoid nerve. The masticator nerve supplies motor innervation to the masseter, temporalis, and medial and lateral pterygoid muscles, while the mylohyoid nerve supplies the mylohyoid and anterior belly of the digastric muscles.

Results

A total of 76 patients were imaged. They were 14–88 years old, though all except eight were over the age of 30 years.

Table 2 lists the distribution of lesions according to their location. The peripheral divisions were involved most often (49%), followed by the preganglionic segment (18%) and brainstem (18%), gasserian ganglion (8%), and cavernous sinus (7%). In 40% of patients with malignant peripheral lesions, there was perineural tumor spread to the gasserian ganglion (Figs. 2 and 3).

In Table 3, the distribution of symptoms is listed according to the location of the lesion. In general, the distribution of symptoms did not help localize the lesion. For example, 13 of 37 patients with peripheral lesions, in which single-division involvement would be expected, presented with multiple-division symptoms. Similarly, six of 14 patients with brainstem lesions, in which involvement of all three divisions would be expected, presented with symptoms in only one or two divisions.

In Table 4, the types of presenting symptoms or signs are listed according to lesion location. Pain and numbness were somewhat useful in localizing lesions, as this symptom complex was present almost exclusively in patients with peripheral lesions. Trismus was seen only in patients with malignant lesions of the masticator space.

Overall, 34% of patients had CT, 45% had MR, and 21% had CT and MR. Early in the series, only CT was available. When MR became available, many patients were scanned with both CT and MR. For peripheral lesions, both techniques

TABLE 2: Type and Distribution of Lesions Causing Fifth Cranial Nerve Symptoms

Location/Type	No. (%)
Brainstem	
Multiple sclerosis	4
Glioma	4
Stroke	3
Metastasis	1
Cavernous angioma with hemorrhage	1
Syringohydrobulbia	1
Total	14 (18)
Preganglionic segment	
Vascular compression	4
Arteriovenous malformation	3
Meningioma	2
Epidermoid	2
Acoustic neuroma	1
Metastasis	1
Surgical sectioning	1
Total	14 (18)
Gasserian ganglion	
Metastasis	3
Trigeminal schwannoma	3
Total	6 (8)
Cavernous sinus	
Cavernous carotid aneurysm	3
Metastasis	2
Total	5 (7)
Peripheral divisions V_1–V_3	
Neurofibroma	1
Spindle cell skin carcinoma	1
Tongue squamous cell carcinoma	1
Peripheral divisions V_1 and V_2	
Nasopharyngeal squamous cell carcinoma	1
Sphenoid wing meningioma	1
Neurofibroma	1
Peripheral divisions V_2 and V_3	
Malignant salivary gland tumors	4
Lymphoma	2
Lip squamous cell carcinoma	1
Poorly differentiated skin carcinoma	1
Rhabdomyosarcoma	1
Peripheral divisions V_1 and V_3	
Metastasis	1
Peripheral division V_2	
Nasopharyngeal squamous cell carcinoma	2
Skin squamous cell carcinoma	1
Maxillary sinus squamous cell carcinoma	1
Chondrosarcoma	1
Sphenoid mucocele	1
Maxillary sinusitis	1
Malignant salivary gland tumor	3
Malignant schwannoma	2
Lymphoma	2
Osteomyelitis	1
Abscess	1
Nasopharyngeal squamous cell carcinoma	1
Oropharyngeal squamous cell carcinoma	1
Ewing sarcoma	1
Chondrosarcoma	1
Metastasis	1
Total	37 (49)

Note.—Lesions in the V_1 peripheral division are classified under cavernous sinus.

were equally effective in displaying the full extent of the abnormality. For proximal lesions, MR showed a definite advantage in detecting and displaying the full extent of the lesion, particularly in the brainstem, basal cisterns, and skull base (Figs. 4 and 5). Because of this, and because clinical information proved of little use in localizing lesions, later studies were done only with MR.

During the time period of the study, 20 patients with trigeminal neuralgia (tic douloureux) were referred for MR. Five of these patients had positive examinations and were included in this series. One had multiple sclerosis (Fig. 6), another had an arteriovenous malformation (Fig. 7), two had vascular compression (Fig. 8), and a fifth had maxillary sinusitis.

Discussion

Patients with trigeminal neuropathy present with a wide variety of symptoms including facial pain, numbness, masticator muscle spasm and weakness, trismus, and trigeminal neuralgia. Lesions producing these symptoms may occur anywhere along the protracted course of the fifth cranial nerve from its distal facial ramifications to its nuclear columns in the brainstem. Accurate and efficient radiologic evaluation of these lesions requires focused imaging coupled with precise anatomy-directed image interpretation.

In this report we examined the clinical and radiologic records of 76 patients in order to address the following questions. First, how accurate is the preradiologic clinical evaluation in localizing the lesion affecting the fifth cranial nerve and can it be used to focus the imaging process to precise regions along the course of cranial nerve V? Second, what is the segment-by-segment unique differential diagnosis of lesions causing trigeminal neuropathy? Third, what is the role of radiologic examination in patients presenting with trigeminal neuralgia? Finally, does the more expensive technology of MR provide any advantages over CT in this patient population? After analysis of the imaging data collected in this study, a suggested optimum MR imaging protocol was devised for patients with trigeminal neuropathy.

In this series, clinical findings were extremely inaccurate for lesion localization. In particular, the distribution of clinical findings (Table 3) did little to localize a lesion. Single- or multiple-division clinical involvement was seen with lesions in all locations. Clinical patterns that could be identified were related to the type of symptoms (Table 4) and included the combination of pain and numbness, which occurred almost exclusively with peripheral lesions, and trismus, which occurred only in patients with malignant lesions of the masticator space. Other authors [5, 10] have noted the variable presentation of patients with cranial nerve V symptoms, but there has been no satisfactory explanation for why proximal lesions, such as those in the brainstem and preganglionic segment, clinically spare certain divisions. For the radiologist, this lack of clinical specificity means that all segments of clinically involved divisions must be imaged from their brainstem origins to their peripheral endplates.

Table 2 provides a differential diagnosis, by segment, for lesions involving cranial nerve V. Generally, lesions remained

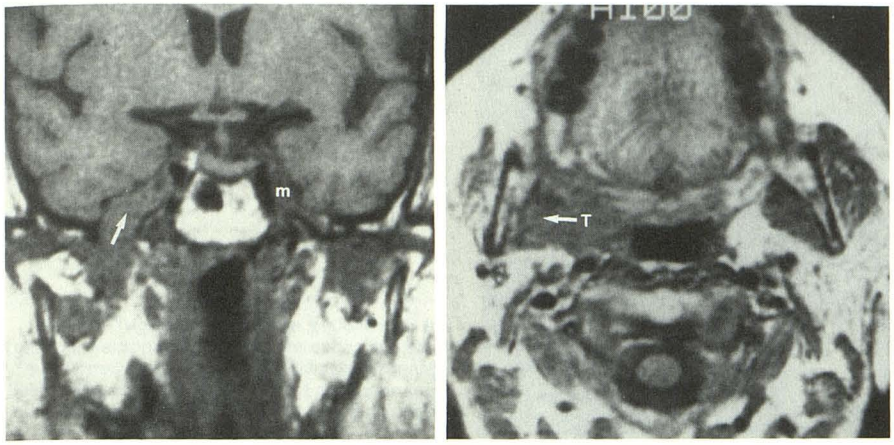

Fig. 2.—Perineural tumor spread in a patient with right V_3 pain.
***A*, Coronal T1-weighted image (600/20) shows tumor extending from nasopharyngeal masticator space, through foramen ovale (*arrow*), and into Meckel cave. m = Meckel cave on normal side.**
***B*, Second axial acquisition through mid oropharynx reveals clinically occult submucosal squamous cell carcinoma (T) of faucial tonsillar crypts. Tumor had invaded adjacent masticator space (*arrow*) and spread perineurally along V_3 to level of gasserian ganglion in Meckel cave.**

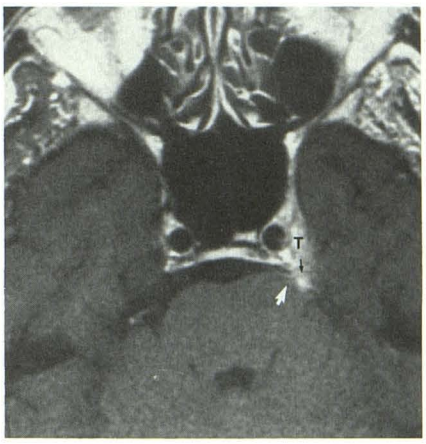

Fig. 3.—Perineural tumor spread in a patient previously treated for spindle cell carcinoma of the skin. Gadolinium-enhanced axial T1-weighted spin-echo image (600/20) shows perineural tumor spread (T) along V_2 to gasserian ganglion and further spread along preganglionic segment to root entry zone of cranial nerve (*black arrow*). Presumed subtle brainstem invasion is seen as hyperintense strands extending from preganglionic segment into pons (*white arrow*).

TABLE 3: Distribution of Clinical Signs According to Lesion Location

Distribution of Symptoms	Lesion Location				
	Brainstem	Preganglionic Segment	Gasserian Ganglion	Cavernous Sinus	Peripheral
V_1–V_3	7	4	2	1	1
V_1 and V_2	2	2	1	0	3
V_2 and V_3	2	4	2	1	9
V_1 and V_3	0	0	0	0	0
V_1	0	0	0	2	–[a]
V_2	1	1	0	1	7
V_3	1	1	0	0	16
Not known	1	2	1	0	1
Total	14	14	6	5	37

Note.—The distribution of clinical signs was determined by history and physical examination.
[a] See cavernous sinus.

TABLE 4: Presenting Symptoms or Signs by Lesion Location

Symptom or Sign	Lesion Location				
	Brainstem	Preganglionic Segment	Gasserian Ganglion	Cavernous Sinus	Peripheral[a]
Pain	2	6	1	1	8
Numbness	11	5	3	3	13
Pain and numbness	0	0	0	1	11
Other	0	1[b]	0	0	1[c]
Not known	1	2	2	0	4

[a] In five patients with malignant lesions, trismus was part of the symptom complex.
[b] Hyperactive jaw reflex was the only cranial nerve V manifestation in a patient with a large cerebellopontine angle meningioma.
[c] Jaw weakness and trismus were the only cranial nerve V manifestations in a patient with a deeply invasive, mixed malignant minor salivary gland tumor extending from the base of the skull to the angle of the mandible.

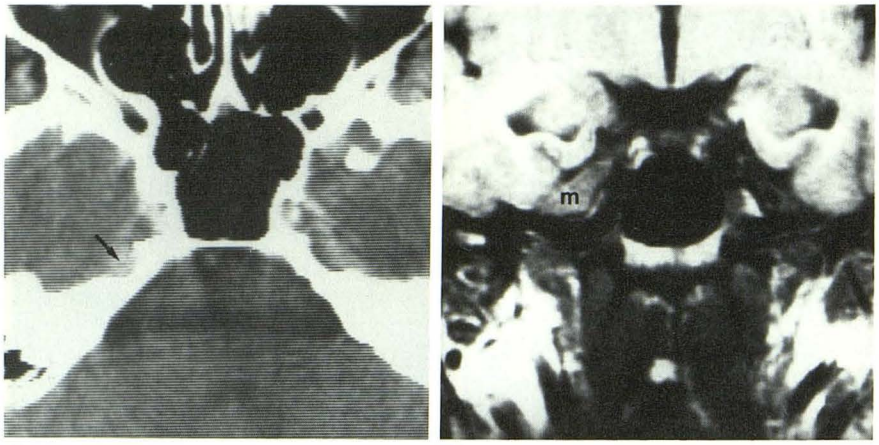

Fig. 4.—Breast carcinoma metastasis to right gasserian ganglion in a patient with right V_2 and V_3 numbness.

A, Enhanced axial CT scan shows subtle area of enhancement at porus trigeminus (*arrow*), which was interpreted as normal.

B, Coronal T1-weighted spin-echo image (800/20) through Meckel cave shows large metastatic deposit (m).

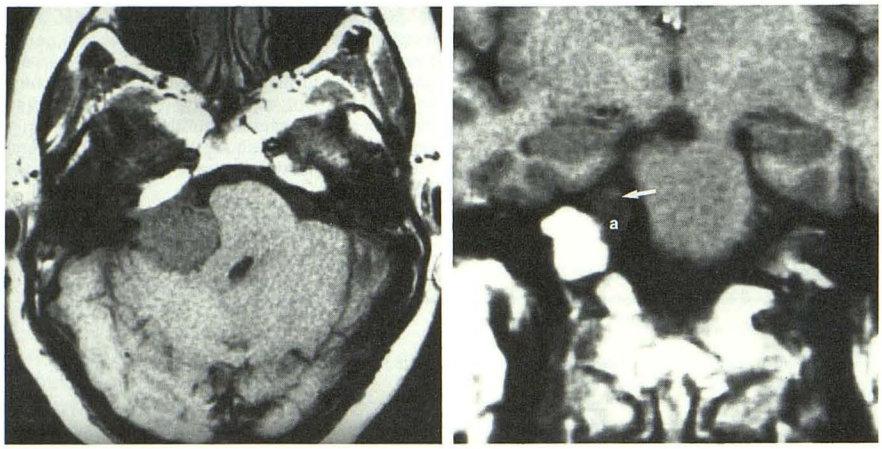

Fig. 5.—Acoustic neuroma in a 46-year-old woman with right sensorineural hearing loss, loss of taste, and right V_1 and V_2 numbness.

A and *B*, Coronal (*A*) and axial (*B*) T1-weighted spin-echo images (800/20) show large mass extending from right internal auditory canal into cerebellopontine angle cistern. Acoustic neuroma (a) elevates and flattens preganglionic segment near root entry zone of cranial nerve V (*arrow*).

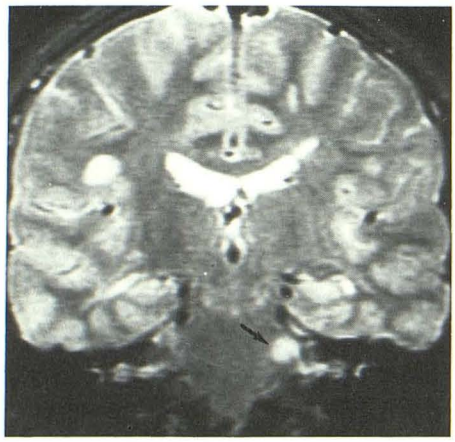

Fig. 6.—Multiple sclerosis in a 15-year-old boy with left trigeminal neuralgia. Coronal T2-weighted spin-echo image (2200/70) shows multiple sclerosis plaques including one in vicinity of left main sensory nucleus of cranial nerve V (*arrow*).

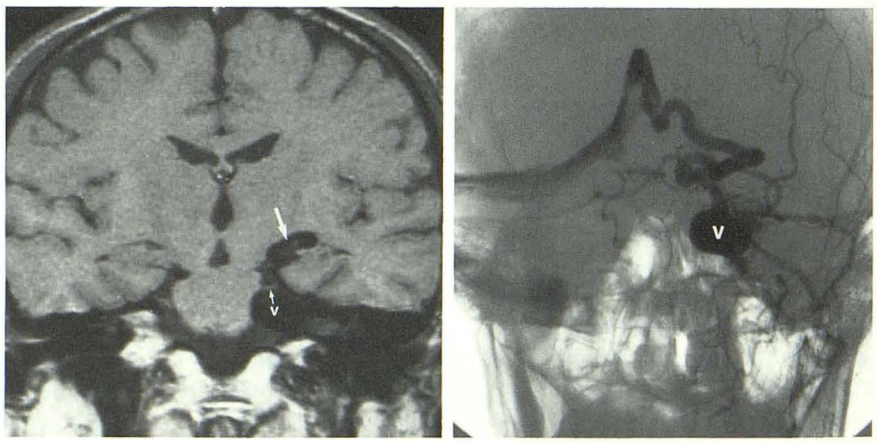

Fig. 7.—Dural arteriovenous malformation in patient with tinnitus and left trigeminal neuralgia.

A, Coronal T1-weighted image (800/20) shows large venous varix (v) elevating and compressing preganglionic segment of left cranial nerve V (*small arrow*). There is also an associated large draining vein (*large arrow*).

B, Anteroposterior angiogram from left external carotid artery injection shows large arteriovenous malformation with associated venous varix (v).

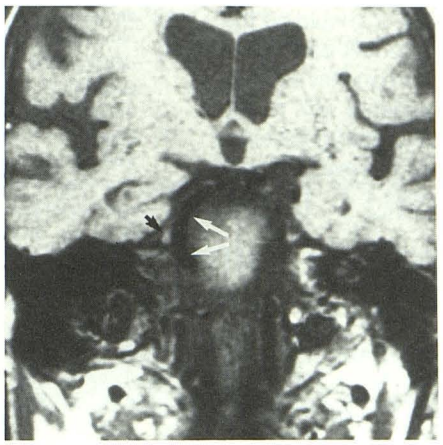

Fig. 8.—Vertebrobasilar dolichoectasia in patient with right-sided trigeminal neuralgia. Coronal T1-weighted image (1000/20) shows dolichoectatic basilar artery (*white arrows*) laterally displacing and compressing preganglionic segment of right cranial nerve V (*black arrow*).

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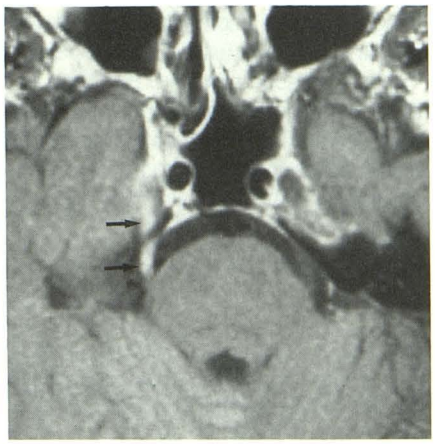

Fig. 9.—Trigeminal neuritis in patient with trigeminal neuropathy. Gadolinium-enhanced axial T1-weighted spin-echo image shows enhancement without enlargement of right gasserian ganglion and preganglionic segment of cranial nerve V. (Courtesy of W. Coit, Portland, OR.)

9

confined to individual segments of the nerve. The exception was the peripheral segments, in which malignant perineural tumor spread to the gasserian ganglion was common (40%). This type of spread can occur with a variety of lesions, but by far the most common is squamous cell carcinoma of the face (Fig. 2). Mohs and Lathrop [25] and more recently Ballantyne et al. [26] have recognized the importance of detecting this type of spread. In the series of Mohs and Lathrop, nearly two-thirds of the patients with perineural spread had had previous treatment, suggesting that unrecognized perineural spread was a cause of treatment failures (Fig. 3).

Cranial nerve V, as the principal sensory nerve to the suprahyoid neck, serves as the major conduit for perineural tumor spread. In this study, the most common pattern of spread occurred when a malignant tumor in the masticator space traveled along V_3 through the foramen ovale to the gasserian ganglion (Fig. 2). Another common perineural tumor spread pattern [4] was seen along branches of V_2, frequently the infraorbital nerve, to the pterygopalatine fossa and subsequently through the foramen rotundum to the gasserian ganglion. With either pattern, more proximal spread along the preganglionic segment to the brainstem was seen (Fig. 3). Clinical signs do not reliably predict the presence or extent of perineural spread [6, 7, 25, 26]. Because of this, and because of the possible role of perineural tumor spread in treatment failure, all patients with malignant lesions of the masticator space or pterygopalatine fossa should undergo complete cranial nerve V imaging.

At our institution, patients with typical trigeminal neuralgia are usually not referred for imaging studies. However, when patients present with atypical trigeminal neuralgia symptoms or are severely affected despite medical treatment (or if there is a question of multiple sclerosis), it is necessary to evaluate these patients for demyelinating plaques, structural lesions, and vascular compression, which may mimic trigeminal neuralgia [27, 28]. As discussed, MR proved to be useful in this regard (Figs. 6–8).

Only 21% of patients had both CT and MR. Despite this limited number of comparisons, it was evident that MR provided a distinct advantage in the radiologic examination of certain segments of cranial nerve V (Fig. 4). In the brainstem, small infarcts and multiple sclerosis plaques are invisible to CT but quite evident on MR. Similarly, the preganglionic segment is directly visualized with MR, even when it is normal [29], but is rarely seen with CT. Consequently, in the preganglionic segment, MR precisely indicates areas of cranial nerve V compression, better displays the full extent of tumors, and shows the relationship of masses to other important structures such as cranial nerves VII and VIII (Fig. 5).

Early in the series, it became evident that clinical localization was not useful for accurate lesion localization. Consequently, an imaging protocol was developed that did not rely heavily on clinical information and that would provide the best imaging of all segments of cranial nerve V, including those segments not well imaged with CT. The MR protocol in Table 1 was established for this purpose. The only clinical information needed to institute this protocol was a suspicion of cranial nerve V pathology and knowledge as to whether or not the third division of cranial nerve V was involved. If V_3 was involved, the imaging study was extended to the inferior mandibular margin. With this approach, all segments that may be pathologically involved were completely imaged.

Gadolinium enhancement promises to have a place in the evaluation of trigeminal neuropathy. Our experience substantiates this claim. A recent case seen after the closure of this series is shown in Figure 9. This patient with fifth cranial neuropathy showed diffuse enhancement of the preganglionic segment and gasserian ganglion of cranial nerve V without enlargement of the nerve itself. The presumptive diagnosis for this MR finding was trigeminal neuritis. Because of this improved sensitivity to intrinsic (Fig. 9) and perineural (Fig. 3) nerve abnormalities with gadolinium enhancement, we have begun to use Gd-DTPA in the evaluation of all patients with trigeminal neuropathy.

Conclusions

The lack of accurate clinical localization necessitates comprehensive imaging of the fifth cranial nerve in patients with cranial nerve V symptoms. Because MR is superior to CT in

imaging certain segments of cranial nerve V, it should be the primary imaging study. For this purpose, the MR protocol in Table 1 is recommended. Because of the frequency of clinically occult perineural tumor spread, all patients with malignant lesions of the masticator space and pterygopalatine fossa should also undergo a complete imaging evaluation of cranial nerve V.

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