Trigeminal neuralgia and hemifacial spasm result from mechanical compression of the cranial nerves by surrounding vessels. The compressing lesions are usually ipsilateral to the symptoms.\(^6,11,12\)

Trigeminal neuralgia and hemifacial spasm have been reported as false localizing signs of a mass in the contralateral posterior cranial fossa, although they are extremely rare and the underlying pathophysiological mechanisms remain to be elucidated.\(^2,4,8,9,13,14,17,21,23\) Because there are few reports of patients who have undergone exploratory decompression surgery of the affected cranial nerves, there are few pathoanatomical descriptions of the involved nerves.

We present two cases of trigeminal neuralgia and one case of hemifacial spasm caused by space-occupying mass lesions in the contralateral posterior fossa. Both patients with trigeminal neuralgia underwent decompression surgery of the cranial nerve and the patient with hemifacial spasm underwent tumorectomy. Based on our operative findings, we postulate that the angulation and distortion of the cranial nerve by brainstem displacement and also the thick and strongly adherent arachnoid membrane surrounding the nerve played an important role in the manifestation of the false localizing signs.

**Case Reports**

**Case 1**

This 41-year-old man was hospitalized on April 2, 1987 with a 2-year history of right facial pain. Neurological findings were otherwise normal. Noncontrast-enhanced computerized tomography (CT) scanning revealed a questionable shift to the right of the fourth ventricle and a tight prepontine cistern (Fig. 1 left). A diagnosis of trigeminal neuralgia was made and the patient underwent surgery via a right suboccipital approach. The brainstem and cerebellar hemisphere appeared to bulge, and the space between the petrous bone and the cerebellar hemisphere and the brainstem had narrowed. The fifth cranial nerve was kinked due to compression by the brainstem, whose axis was tilted and distorted by pachyntic arachnoid adhesion. The adhesion was resected to straighten the nerve. There was no apparent involvement of vascular structures. The patient’s symptoms disappeared immediately postoperatively. However, postoperative magnetic resonance (MR) imaging revealed a dumbbell-shaped extraaxial mass in the left cerebellopontine angle (Fig. 1 right). It was totally removed in a second operation. On histological examination, the tumor was identified as a neurinoma originating from the left trigeminal nerve.
At follow-up review 8 years postsurgery, the patient was alive without any evidence of recurrence of the tumor or neuralgia.

**Case 2**

This 56-year-old woman had a 3-year history of left trigeminal neuralgia. She was hospitalized on March 19, 1987. In 1981 she had experienced left facial hypo- or dysesthesia and underwent total removal of a right tentorial meningioma at another institution (Fig. 2 upper left). However, her symptoms did not disappear completely and her left facial dysesthesia progressed to typical tic dououreux 6 years after the tumorectomy. A CT scan obtained at our institution revealed that the right cerebellar hemisphere was invaginated at the cavity of the bone defect and the brainstem was displaced and rotated in a direction opposite to that noted before. (Fig. 2 upper right). Decompression of the left trigeminal nerve was performed. The space between the brainstem and the petrous bone was markedly narrowed, and the fifth cranial nerve was squeezed by these structures and appeared kinked and distorted due to pachyntic arachnoid adhesion around the nerve (Fig. 2 lower). The distortion of the nerve was corrected by arachnoid resection. There was no vascular involvement.

At follow-up review 8 years after her second operation, the patient was alive and without evidence of recurrent neuralgia.

**Case 3**

This 55-year-old man was admitted to our hospital in December, 1993 with progressive gait disturbance and a 4-month history of left hemifacial spasm. Neurological examination revealed left facial spasm, right progressive hearing disturbance, and cerebellar ataxia. A CT scan demonstrated that his brainstem was rotated and displaced to the left by a mass in the right cerebellopontine angle (Fig. 3). The mass was diagnosed as a neurinoma of the eighth cranial nerve, and it was totally resected via a right suboccipital approach. On pathological review the tumor was diagnosed as an acoustic neurinoma.

The left hemifacial spasm gradually decreased postoperatively and finally disappeared 3 months postsurgery with no recurrence during the 18-month follow-up period.

**Discussion**

The pathogenesis of trigeminal neuralgia and hemifacial spasm is thought to result from paroxysmal ephaptic transmission due to mechanical compression at the root entry/exit zone of the cranial nerves and neurovascular compression of the fifth and seventh cranial nerves is believed to be a major cause of these conditions. Other pathoanatomical factors in these clinical entities are tumors, aneurysms, arteriovenous malformations, and arachnoidal adhesion.

False localizing signs of intracranial lesions are defined...
as signs not generally associated with disturbances of function at the site of the lesion.\textsuperscript{1,7} They are reported to occur in 12.5\% to 20\%\textsuperscript{1,3} of all cases with intracranial neoplasms; the majority of these lesions are supratentorial.\textsuperscript{1,7} Cranial nerve involvement in false localizing signs, first described in 1905 by Funkenstein,\textsuperscript{5} occurs considerably less frequently with posterior cranial fossa lesions.\textsuperscript{4,8,15,18,19,21}

Paillas, \textit{et al.},\textsuperscript{19} reviewed 20 cases of subtentorial tumors accompanied by contralateral cranial nerve symptoms, with the fifth cranial nerve being most commonly affected by a contralateral tumor, followed in decreasing order by the seventh, eighth, and third cranial nerves. Most cranial nerve dysfunction presenting as a false localizing sign, however, appears as hypoaactive dysfunction syndrome; hyperactive dysfunction syndrome manifesting as trigeminal neuralgia or hemifacial spasm is extremely rare.\textsuperscript{3,4,8,13,14,18,21}

False localizing hemifacial spasm was first reported by Gardner and Sava,\textsuperscript{6} and Nishi, \textit{et al.},\textsuperscript{13} and Rhee, \textit{et al.},\textsuperscript{21} each reported one patient with hemifacial spasm. To our knowledge, these three are the only other reported cases of hemifacial spasm due to a contralateral infratentorial mass.

We have treated 578 cases of trigeminal neuralgia and 1388 cases of hemifacial spasm using microvascular decompression (MVD). The present two cases of trigeminal neuralgia represent 0.4\% of our trigeminal neuralgia cases and the case of hemifacial spasm discussed here represents less than 0.1\% of our hemifacial spasm cases. Of the trigeminal neuralgia patients, our Case 1 is the first reported case with contralateral trigeminal neurinoma and our Case 2 is unusual because the symptoms were not really caused by the contralateral tumor.

\textit{Mechanisms of False Localizing Signs of the Cranial Nerves}

Although the pathophysiology of false localizing signs in cases with a posterior fossa mass remains controversial, there is general agreement that mechanical displacement and/or distortion of the brainstem leads to anatomical transformation of the cranial nerve (Fig. 4).\textsuperscript{3,4,8,9,13,14,18,20,23} The main factors associated with the pathophysiology of false localizing cranial nerve signs due to a contralateral infratentorial mass are shown in Table 1.

Cushing\textsuperscript{2} and Paillas, \textit{et al.},\textsuperscript{19} posited the presence of a sharp angulation of the trigeminal nerve root at its point of entry into the tentorial foramens as a mechanism of false localizing trigeminal neuralgia, whereas Parker\textsuperscript{20} and Hamby\textsuperscript{9} posited compression of the root between the brainstem and the petrous temporal bone or the free edge of the tentorium. O’Connell\textsuperscript{18} suggested that displacement of the brainstem by the tumor results in a slackening of the horizontally directed nerves (seventh, eighth, ninth, tenth, and eleventh), whereas the anteriorly directed nerves (fifth and sixth) are rendered tense and stretched around the lateral margin of the dural foramens or compressed by it.

Regardless of the direction, the rotation or displacement of the brainstem may result in mechanical changes affecting the cranial nerve, for example, compression, stretch-

\begin{table}[h]
\centering
\begin{tabular}{|l|}
\hline
\textbf{Factors in the pathophysiology of false localizing cranial nerve signs} \\
\hline
indirect: size, nature, \& location of the tumor \\
anatomical direction of the cranial nerve \\
individual variations in the size or shape of the posterior fossa \\
direct: stretching or kinking of the cranial nerve \\
compression of the cranial nerve against the dura or bone structure \\
by the brainstem \\
arachnoid adhesion with or without vascular compression \\
\hline
\end{tabular}
\caption{Table 1}
\end{table}
ing, or angulation. As a matter of fact, in such cases, CT scanning and/or MR imaging clearly demonstrate displacement and/or distortion of the brainstem.4,8,10,16,17,21,23,24 Although the direction of brainstem rotation is various or equivocal, a common anatomical finding is that the pre-pontine cistern on the side contralateral to the mass is very tight.

The clinical course of trigeminal neuralgia in our Case 2 is interesting and may shed some light on the underlying mechanisms. The initial symptom was facial hypo- or dysesthesia with the tumor located at the contralateral cerebellar convexity. The symptom subsequently manifested as typical trigeminal pain when the cerebellar hemisphere became invaginated at the bone defect after tumor resection. Our is the only reported case in which a false localizing sign changed from hypoactive to hyperactive. The reported pathophysiological findings, however, apply primarily to dysfunction of the trigeminal nerve, but not to its hyper- or hypoactive dysfunction. The finding on the CT scan was curious because it demonstrated that the rotational direction of the brainstem after tumor resection was opposite to that noted before the first surgery. Intraoperative findings indicated that this change in the rotational direction of the brainstem may have changed the condition of the affected trigeminal root from stretched to angulated, resulting in compression of the nerve between the brainstem and petrous bone. We suggest that the direction of the displacement is individually determined by the shape of the bone structure of the posterior fossa. Among anatomical configurations, we posit that the most significant factor leading to false irritative cranial nerve dysfunction is displacement rather than rotation of the brainstem toward the side contralateral to the tumor. Such displacement may result in compression and/or angulation of the affected nerve rather than stretching or traction, thereby inducing contralateral trigeminal neuralgia and hemifacial spasm.

Operative findings in one case each have been reported by Hamby9 and by Gardner and Sava.6 Hamby demonstrated that the fifth cranial nerve root ran downward, not upward and forward, and that its lower end was tightly compressed between the pons and the incisura of the tentorium. Gardner and Sava found that the seventh cranial nerve appeared to be squeezed between the petrous bone and pons because of displacement of the latter structure by an expanding lesion on the opposite side. In our Cases 1 and 2, the fifth cranial nerve was squeezed between the brainstem and petrous bone, appearing kinked and angulated, and the angulated fifth cranial nerve axis was encased by pachyntic arachnoid adhesion surrounding the nerve.

The question arises as to why hemifacial spasm with contralateral tumor is much less frequent than trigeminal neuralgia, although classic hemifacial spasm is seen as frequently as trigeminal neuralgia. One tenth of the cases of trigeminal neuralgia are due to an ipsilateral posterior fossa tumor,12 whereas hemifacial spasm due to an ipsilateral subtentorial tumor is extremely rare (0.3%-0.6%).21 We suspect that because of the different originating sites from the brainstem between the fifth and seventh cranial nerves and because of the difference in the angle of these nerves to the brainstem, the seventh cranial nerve is less frequently affected by contralateral mass lesions.

Factors Underlying the Manifestation of False Localizing Signs

O’Connell18 and Paillas, et al.19 reported that the size, consistency, histology, and location of the mass play a role in the incidence of false localizing signs. The ratio of meningiomas to acoustic neurinomas in the cerebellopontine angle is 1:15.5 to 1:23.22 The number of meningiomas, however, appears to be disproportionately high (approximately 70%) among all causative tumors in the false cranial nerve dysfunction syndrome.8,19 The predominance of meningiomas may be attributable to the fact that they grow more slowly than acoustic neurinomas without a special localizing sign. Therefore, meningiomas are diagnosed later and may produce false localizing signs more frequently. In our second case of trigeminal neuralgia, the symptom was not ascribable to the contralateral tumor, but rather to the shift of the cerebellar hemisphere and brainstem due to invagination of the contralateral cerebellar hemisphere at the site of the bone defect 6 years after tumor removal. We propose that such a chronic but progressive change in the anatomy of the posterior cranial fossa may result in distortion of the brainstem and thus in the elicitation of symptoms, irrespective of the size or consistency of the mass.

According to Snow and Fraser25 and Haddad and Taha,8 the rotation of the brainstem shifts the basilar artery or a loop of its branches close to the trigeminal nerve at its root entry zone, thereby inducing typical trigeminal neuralgia. This arterial loop hypothesis may be borne out in some cases. In approximately 10% of cases seen at our institution, trigeminal neuralgia was due to ipsilateral tumor, and in some patients the fifth cranial nerve was compromised by the tumor and not by an arterial loop. If an arterial loop were the main mechanism underlying false trigeminal neuralgia and hemifacial spasm, then hemifacial spasm with contralateral tumor should occur at approximately the same rate as false localizing signs. However, vascular involvement was not apparent in either of our cases of trigeminal neuralgia, suggesting that neurovascular compression plays a minor role in the development of false localizing signs.

We have performed microvascular decompression in a large number of typical trigeminal neuralgia cases in which the trigeminal nerve axis was tilted and distorted by thick pachyntic arachnoid adhesion without apparent neurovascular compromise at the root entry zone. In such cases, complete remission cannot be obtained without resection of the arachnoid adhesion to correct the distortion of the fifth cranial nerve. In both of our cases with trigeminal neuralgia, total resection of the arachnoid adhesion completely abated the neuralgia despite the marked narrowing of the cerebellopontine cistern. This strongly suggests that arachnoid adhesion may be a very important contributing factor.

Operative Strategy

The optimum operative strategy for the treatment of false localizing trigeminal neuralgia and hemifacial spasm is controversial. Hamby9 and Haddad and Taha8 pointed out that the contralateral side must be explored first to avoid disastrous surgical results, because the space around
False trigeminal neuralgia and hemifacial spasm

the cranial nerves is extraordinarily tight in the presence of a contralateral tumor.

Our current strategy for the treatment of false localizing signs such as trigeminal neuralgia or hemifacial spasm is to remove the contralateral mass lesion first and to wait for the disappearance of the symptoms. If they persist, the affected nerve is explored. In such cases it is of utmost importance to resect the thick, adherent arachnoid membrane around the nerve completely to straighten the axis of the cranial nerves, especially in cases in which vascular involvement of the target cranial nerve is not apparent.

References