INNITUS is a purely subjective symptom of poorly understood etiology, which is associated with many pathological conditions, such as vascular anomalies, metabolic disturbances, neurological conditions, whiplash, traumatic injuries to the head, temporomandibular joint syndrome, anxiety neuroses, psychosomatic disorders, and many others. The treatment of tinnitus, at present, is mainly palliative, with neurotomy or destructive surgical treatment used only in some intractable cases. However, most patients with tinnitus are simply told by their doctor that they will just have to learn to live with it. Neurovascular compression (NVC) of the eighth cranial nerve has also been reported as a cause of tinnitus, but few reports describe the characteristics of this condition, making it very difficult to diagnose tinnitus correctly. Furthermore, it is almost impossible to predict whether tinnitus will resolve after neurovascular decompression (NVD), even if the patient presents with signs strongly suggestive of NVC. The reason for the lack of progress in research on tinnitus caused by NVC of the eighth cranial nerve is the absence of an ideal experimental model with which to study the characteristics of the condition and the effectiveness of NVD.

**Object.** The authors sought to clarify the clinical characteristics of tinnitus resulting from neurovascular compression (NVC) of the eighth cranial nerve.

**Methods.** The authors explored the eighth cranial nerve in the cerebellopontine cistern during neurovascular decompression (NVD) of the facial nerve in 10 patients with hemifacial spasm who suffered from incidental tinnitus on the same side. The diagnosis of NVC of the eighth cranial nerve was confirmed in all patients. This condition was found in only seven of 114 patients with hemifacial spasm alone, indicating that NVC of the eighth cranial nerve is one of the causes of tinnitus (p < 0.001, chi-square test). The tinnitus resolved or was markedly improved after NVD of the eighth cranial nerve in eight patients (80%). Both pulsatile and continuous tinnitus responded well to NVD. All patients experienced various degrees of sensorineural hearing disturbance, but other neurotological examinations provided poor diagnostic value.

**Conclusions.** It is the authors’ opinion that sensorineural hearing loss and positive findings on magnetic resonance imaging are the most reliable evidence for the presence of tinnitus caused by NVC of the eighth cranial nerve.

**Key Words** • neurovascular decompression • eighth cranial nerve • tinnitus

We have performed NVD in more than 300 patients with hemifacial spasm and trigeminal neuralgia and during the course of these treatments we realized that some patients complained of unilateral tinnitus on the same side as the spasm. We therefore decided to explore the eighth cranial nerve when we performed NVD of the facial nerve (seventh cranial nerve) in patients with hemifacial spasm who also complained of tinnitus. In this paper we report the results of exploration and NVD of the eighth cranial nerve in those patients undergoing primary NVD of the seventh cranial nerve.

**Clinical Material and Methods**

**Patient Population**

Between July 1974 and December 1996, we performed NVD of the seventh cranial nerve in 142 patients with hemifacial spasm, 10 of whom (seven women and three men; age range 43–73 years) also complained of unilateral tinnitus on the same side as the spasm. Patients with hemifacial spasm sometimes experience tinnitus that is synchronized with their facial spasms. The tinnitus is caused by vibration of the eardrum as a result of irritation.
Neurovascular decompression of the eighth nerve for tinnitus

TABLE 1
Clinical summary of patients with HFS who suffered incidental tinnitus*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Side of Responder</th>
<th>Type of Caloric Nystagmus</th>
<th>SRT in dB†</th>
<th>Hearing Loss in dB</th>
<th>History of Tin</th>
<th>History of HFS</th>
<th>Follow Up (yrs)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68, F</td>
<td>rt</td>
<td>lp</td>
<td>n</td>
<td>30 (30)</td>
<td>40–60 at 4–8 kHz</td>
<td>&lt;6 mos</td>
<td>&gt;3 yrs</td>
<td>12.1</td>
<td>resolved</td>
</tr>
<tr>
<td>2</td>
<td>53, F</td>
<td>lt</td>
<td>lp</td>
<td>n</td>
<td>35 (35)</td>
<td>30–40 at all ranges (30 at all ranges)</td>
<td>&lt;3 yrs</td>
<td>10 yrs</td>
<td>11.3</td>
<td>resolved</td>
</tr>
<tr>
<td>3</td>
<td>43, M</td>
<td>lt</td>
<td>dc</td>
<td>n</td>
<td>df</td>
<td>30% at 110 dB</td>
<td>&lt;8 mos</td>
<td>1 yr</td>
<td>11.0</td>
<td>markedly improved</td>
</tr>
<tr>
<td>4</td>
<td>50, F</td>
<td>rt</td>
<td>hc</td>
<td>n</td>
<td>n</td>
<td>no data</td>
<td>3–4 yrs</td>
<td>2 yrs</td>
<td>8.8</td>
<td>unchanged</td>
</tr>
<tr>
<td>5</td>
<td>67, F</td>
<td>rt</td>
<td>lp</td>
<td>n</td>
<td>25 (25)</td>
<td>20–40 at all ranges (20–40 at all ranges)</td>
<td>&lt;3 yrs</td>
<td>&gt;3 yrs</td>
<td>8.7</td>
<td>resolved</td>
</tr>
<tr>
<td>6</td>
<td>73, F</td>
<td>lt</td>
<td>lp</td>
<td>n</td>
<td>40 (40)</td>
<td>60–70 at 4–8 kHz</td>
<td>3–4 yrs</td>
<td>10 yrs</td>
<td>7.8</td>
<td>resolved</td>
</tr>
<tr>
<td>7</td>
<td>44, M</td>
<td>lt</td>
<td>lc</td>
<td>d</td>
<td>25 (20)</td>
<td>15–30 at all ranges (10–15 at all ranges)</td>
<td>&lt;1 yr</td>
<td>7 yrs</td>
<td>7.6</td>
<td>resolved</td>
</tr>
<tr>
<td>8</td>
<td>70, F</td>
<td>lt</td>
<td>lp</td>
<td>n</td>
<td>25 (20)</td>
<td>50 at 8 kHz (30 at 8 k Hz)</td>
<td>1.7 yrs</td>
<td>1.7 yrs</td>
<td>4.5</td>
<td>unchanged</td>
</tr>
<tr>
<td>9</td>
<td>66, M</td>
<td>lt</td>
<td>hc</td>
<td>n</td>
<td>40 (30)</td>
<td>25–40 at all ranges (20–30 at all ranges)</td>
<td>2 mos</td>
<td>10 yrs</td>
<td>1.8</td>
<td>resolved</td>
</tr>
<tr>
<td>10</td>
<td>65, F</td>
<td>rt</td>
<td>hc</td>
<td>d</td>
<td>30 (25)</td>
<td>30–40 at 8 kHz (15–25 at 8 kHz)</td>
<td>8 yrs</td>
<td>8 yrs</td>
<td>1.0</td>
<td>resolved</td>
</tr>
</tbody>
</table>

* D = decreased; df = delayed from wave f; ETT = eye-tracking test; hc = high-pitched continuous; HFS = hemifacial spasm; lc = low-pitched continuous; lp = low-pitched pulsatile; n = normal; OKN = optokinetic nystagmus; tin = tinnitus. Data for the side contralateral to the tinnitus are given in parentheses.
† Hearing intensity (dB) needed to obtain 50% hearing discrimination.

of the stapedius within the seventh cranial nerve. Patients with this type of tinnitus were excluded from the present study. All 10 patients included in this study who were undergoing NVD for hemifacial spasm consented to exploration of the eighth cranial nerve and subsequent NVD if NVC was confirmed. We analyzed the preoperative characteristics of the tinnitus, the surgical findings, and the results of the operation in these 10 patients. All patients underwent neurotological examinations, including pure-tone audiography, speech perception threshold (SRT), loudness balance, short-increment sensitivity index, Békésy's audiometry, tympanometry, and auditory brainstem response (ABR) for cochlear function, as well as examination for spontaneous nystagmus, optokinetic nystagmus, the eye-tracking test, and testing of caloric responses for vestibular function. The patients also underwent neuroradiological examinations including magnetic resonance (MR) imaging, computerized tomography (CT) scanning, angiography, and air CT of the posterior fossa. However, the four patients studied most recently underwent only MR imaging, which is noninvasive and the most reliable imaging technique to date for the diagnosis of NVC.6 To determine how many patients without tinnitus had NVC of the eighth cranial nerve, we reviewed the surgical records and videotapes of surgery in 132 patients who had hemifacial spasm only. The results were compared with those of patients with hemifacial spasm and ipsilateral tinnitus. The follow-up period after NVD of the eighth cranial nerve ranged from 1 to 12.1 years (mean 7.5 years) and consisted of follow-up interviews conducted by telephone with each patient.

Surgical Technique

With the patient placed in the lateral position, an approximately 2.5 × 2-cm craniectomy was performed just behind the origin of the mastoid process. The cerebellum was gently retracted from the caudolateral to the rostro-medial direction to expose the seventh and eighth cranial nerve complex. The entire length of the eighth cranial nerve in the cerebellopontine cistern was carefully examined after completion of NVD of the seventh cranial nerve, and NVD of the eighth cranial nerve was performed if NVC was detected. The offending vessels were gently displaced away from the nerve and attached to the adjacent dura mater by using a small piece of oxycellulose soaked in a bonding agent (Biobond; Yoshitomi Pharmaceutical Industries, Osaka, Japan) or fibrin glue. In case of venous compression, the offending vein was coagulated and sectioned to free the eighth cranial nerve. To prevent postoperative hearing disturbance, special care was taken not to touch the eighth cranial nerve and not to stretch the anterior inferior cerebellar artery (AICA) or internal auditory artery. If any arteries exhibited vasospasm as a result of surgical manipulation, papaverine hydrochloride (40 mg dissolved in 10 ml of physiological saline) was applied to them. The ABRs were monitored continuously during the entire procedure, and if waveform V decreased in amplitude and became obscured, surgery was temporarily discontinued until it regained its normal shape. After completion of NVD of both nerves, the dura was closed in a water-tight fashion, and the bone defect was filled with bone chips.

Results

Types of Tinnitus

Five patients had pulsatile tinnitus, and its tone was low pitched in all five. The other five patients had continuous or monotonous tinnitus, which was high pitched in four and low pitched in one (Table 1). The duration of the tinnitus was quite different from that of hemifacial spasm, except in two cases. The intensity of the tinnitus varied considerably from patient to patient. In some patients it did not affect their daily life, although they were always aware of it and it became annoying when they were in a quiet environment. Others suffered very much and had received various kinds of medical or psychological treatment, but to no avail. One patient (Case 10 in Table 1) also had occasional rotatory vertigo lasting up to 30 minutes and was diagnosed as having Ménière’s disease at an ear,
nose, and throat clinic. She was referred to our department of neurosurgery for treatment of hemifacial spasm.

No bruits were detected around the ears or neck in any of the 10 patients. None of them had a history of hyper-tension, diabetes, or noise exposure.

Hearing Loss

All patients had various degrees of sensorineural hearing loss bilaterally, but their hearing was more severely affected on the side of the tinnitus, with a hearing loss ranging from 15 to more than 90 dB. Four patients had flat-type hearing loss, and the other six had high-frequency hearing loss. Most patients could not discern their degree of hearing loss or how long they had been having trouble hearing. The SRT was usually not greatly affected even if hearing was mildly disturbed. A patient with a hearing loss of more than 90 dB had only 30% speech discrimination ability at 110 dB (Case 3). The SRT was not tested in one patient because her hearing loss was too great (Case 4). In two patients, ABRs on the affected side showed prolonged wave I latency, but in the rest of the pa-tients there was no difference in latency between the affected and unaffected sides.

Vestibular Function

None of the patients had spontaneous nystagmus, and the results of the eye-tracking test were within the normal range. In one patient the caloric response was mildly decreased objectively, and in two it was decreased so slight-ly that it was detected only by the patients themselves.

Neuroradiological Findings

In the early portion of this series we performed angiography and air CT scanning of the cerebellopontine cistern, but NVC of the eighth cranial nerve was not confirmed in any of the cases. Magnetic resonance imaging was per-formed in the four patients seen most recently, and NVC of the eighth cranial nerve was diagnosed by neuroradiol-o-gists in all four of them (Fig. 1).

Operative Findings

Neurovascular compression of the eighth cranial nerve was confirmed in all patients. The vessels involved were the AICA in three patients, the posterior inferior cerebel-lar artery (PICA) in two, the vertebral artery (VA) in two, a vein in one, a vein and the AICA in one, and the PICA and the AICA in one (Table 2 and Fig. 2). Both the sev-enth and eighth cranial nerves were compressed by the same vessel in five patients. In two patients the eighth cranial nerve was compressed by two vessels and one of the two vessels was the same as that found to be compressing the seventh cranial nerve. The seventh and eighth cranial nerves were compressed by different vessels in the other three patients. We displaced all the offending arteries from the seventh and eighth cranial nerves, and the veins were coagulated and sectioned successfully.

We reviewed the operative records and videotapes of surgery in 132 patients to determine the frequency of

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**TABLE 2**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Compressing Vessels</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CN VIII</td>
</tr>
<tr>
<td>1</td>
<td>PICA</td>
</tr>
<tr>
<td>2</td>
<td>AICA</td>
</tr>
<tr>
<td>3</td>
<td>AICA</td>
</tr>
<tr>
<td>4</td>
<td>AICA</td>
</tr>
<tr>
<td>5</td>
<td>AICA &amp; PICA</td>
</tr>
<tr>
<td>6</td>
<td>VA</td>
</tr>
<tr>
<td>7</td>
<td>vein</td>
</tr>
<tr>
<td>8</td>
<td>PICA</td>
</tr>
<tr>
<td>9</td>
<td>VA</td>
</tr>
<tr>
<td>10</td>
<td>vein &amp; AICA</td>
</tr>
</tbody>
</table>

* CN = cranial nerve.
Neurovascular decompression of the eighth nerve for tinnitus

Fig. 2. Photographs of typical examples of NVC of the eighth cranial nerve in the cerebellopontine cistern, reconstructed from intraoperative videotapes. Upper: Case 3. The AICA is seen compressing the eighth cranial nerve in its middle portion. The seventh cranial nerve was compressed by the PICA (not shown). Lower: Case 4. The AICA is seen cross compressing the eighth cranial nerve in its distal portion. The proximal part of the AICA was compressing the seventh cranial nerve (not shown). The tinnitus was unchanged after the patient underwent NVD.

NVC of the eighth cranial nerve in the patients with hemifacial spasm alone and were able to obtain precise information concerning the relationship between the seventh and eighth nerve complex and surrounding blood vessels in 114 patients. Arterial cross compression or contact with the eighth cranial nerve was found in six of these patients and venous compression in one, although they did not have any history of tinnitus. The occurrence of NVC of the eighth cranial nerve in patients with tinnitus incidentally associated with hemifacial spasm was significantly higher than in the patients with hemifacial spasm alone (p < 0.001, chi-square test).

Operative Outcome

There was no evidence of hemifacial spasm in any of the 10 patients after NVD of the seventh cranial nerve, and the tinnitus resolved completely in seven patients after NVD of the eighth cranial nerve. In one of the remaining patients the tinnitus diminished markedly, and in two it remained unchanged. One patient (Case 5) experienced a temporary hearing loss of 20 dB, but her hearing gradually recovered to preoperative levels within 3 months. Sensorineural hearing loss did not improve after NVD in any of the patients. The patient who had been diagnosed as having Ménière’s disease (Case 10) has been free from vertigo as well as tinnitus and hemifacial spasm.

Discussion

The diagnosis of NVC of the eighth cranial nerve is rarely made in patients with tinnitus because this condition has not yet been clearly defined, and there are no definite diagnostic criteria or operative indications for it at present. This makes it very difficult to obtain patients’ consent to perform NVD of the eighth cranial nerve for tinnitus, despite the fact that many of those suffering from tinnitus refractory to drug therapy may have NVC. We noticed that some patients complained of ipsilateral tinnitus unrelated to their hemifacial spasm. Ten (7%) of the 142 patients with hemifacial spasm whom we examined had unilateral tinnitus. Examination of those patients proved to be very valuable, allowing us to gather information about tinnitus caused by NVC of the eighth cranial nerve.

All 10 patients examined had NVC of the eighth cranial nerve, and the results of NVD of this nerve were remarkable. Neurovascular compression was found mainly along the midline of the inferior surface of the eighth cranial nerve, when viewing it through a retrosigmoid craniectomy. By means of monitoring compound action potentials we confirmed that the compression site corresponded mainly to the cochlear nerve. The tinnitus completely resolved or was markedly improved in eight (80%) of the 10 patients after NVD. On the other hand, a review of the operative findings in 114 patients who had hemifacial spasm alone showed that only seven patients had NVC or contact with the eighth cranial nerve. These findings indicate that NVC of the eighth cranial nerve is one of the causes of tinnitus (p < 0.001, chi-square test).

The results of NVD of the eighth cranial nerve for tinnitus reported in the literature are not encouraging. Møller, et al., reported that 56.9% of their patients experienced only slight relief from or no improvement in tinnitus, and the condition had worsened in 2.8% of their patients after NVD. The difference between the results reported by Møller, et al., and our own is probably attributable to differences in the duration of tinnitus. The mean durations of tinnitus in their cases in the slightly improved group and the group showing no improvement were 5.2 and 7.9 years, respectively, whereas the mean duration of tinnitus in our cases was 2.3 years. However, the critical time for curing tinnitus by NVD of the eighth cranial nerve was unclear from the present data because of the small number of patients. More cases will have to be collected to clarify this point.

There have been very few reports on the type of tinnitus caused by NVC of the eighth cranial nerve. Some authors have reported that pulsatile tinnitus is more likely to be caused by NVC of the eighth cranial nerve, whereas others have reported that high-pitched continuous tinnitus is attributable to NVC. Our study showed that both continuous and pulsatile tinnitus responded well to NVD of the eighth cranial nerve.

All 10 patients examined had bilateral sensorineural hearing disturbance of various degrees, with more hearing impairment on the side of the tinnitus in seven of them. Sensorineural hearing disturbance was suggested as a possible initial sign of vertigo caused by NVC of the eighth cranial nerve. This may also be a very early sign of tin-
nitus caused by NVC of the eighth cranial nerve, although the mechanism underlying the bilateral hearing disturbance was not elucidated in the present study. Abnormal ABR findings have been suggested to be very important evidence for the diagnosis of tinnitus caused by NVC of the eighth cranial nerve, but only two of our patients exhibited abnormal ABR findings. The SRT on the side of the tinnitus was normal or only slightly increased in eight patients and markedly increased in two. Most of the results of vestibular function testing were also normal, except for a slightly decreased caloric response in three patients (Table 1). This may have been because the patients selected for the present study had NVC mainly of the cochlear component, with little involvement of the vestibular components.

It is our impression that tinnitus caused by NVC of the eighth cranial nerve cannot be diagnosed based on neurotological findings alone, although we will need to accumulate more information to reach a definite conclusion.

The results of MR imaging were very encouraging. The MR images successfully disclosed NVC of the eighth cranial nerve in all four of our most recent cases. We believe that MR imaging is the best auxiliary tool currently available for diagnosis of NVC of the eighth cranial nerve.

It is noteworthy that six of the 10 patients reported herein were 65 years of age or older and that the tinnitus resolved in five of those six after they underwent NVD of the eighth cranial nerve. This suggests that many cases of tinnitus caused by NVC of the eighth cranial nerve in the elderly population may be hidden, with the condition possibly being attributed to circulatory disturbance or a degenerative process in the inner ear.

The characteristics of tinnitus caused by NVC of the eighth cranial nerve are very poorly understood at present. More information on this type of tinnitus is needed to establish diagnostic criteria for this syndrome. We would like to emphasize that we as neurosurgeons have an opportunity to investigate this type of tinnitus by exploring the eighth cranial nerve directly during NVD of the seventh cranial nerve in patients with hemifacial spasm who also have tinnitus. We believe that evidence obtained in such cases will reveal the characteristics of tinnitus caused by NVC of the eighth cranial nerve and lead to the establishment of diagnostic criteria and indications for surgery.

References


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Address reprint requests to: Hiroshi Ryu, M.D., Ph.D., Department of Neurosurgery, Hamamatsu University School of Medicine, 3600 Handa-Cho, Hamamatsu 431-31, Shizuoka, Japan.

H. Ryu, et al.