Abstract. Some patients have vertigo that is more or less constant, associated with varying degrees of nausea, and only relieved by bedrest. This disorder, named disabling positional vertigo (DPV), was found to be caused by a blood vessel or vessels compressing the eighth cranial nerve in its intracranial portion, and it can be relieved by microvascular decompression (MVD) of the nerve. Important in the differential diagnosis of DPV are a detailed history, the results of audiometry (10 to 15 dB interaural threshold difference or a small mid-frequency notch), acoustic middle ear reflex response testing (may be abnormal), and recordings of brainstem auditory evoked potentials (BAEP). BAEP in such cases show increased conduction time in the auditory nerve and/or prolonged latency of wave V recorded from the contralateral ear, possibly the result of brainstem compression. Abnormalities on vestibular testing often do not reflect the severity of the illness. Forty-one patients who underwent MVD to treat DPV in one year at the author's institution have been followed for 4.5 to 5.5 years. By self-evaluation, 20 had excellent and 10 good results of the operation. The success of this procedure is even higher today, since it was found that very small blood vessels, including veins, can cause DPV; thus all vessels touching the nerve are now managed. Complications of MVD are rare. The most frequent, hearing loss, occurred in only one patient in this series. (Keio J Med 40 (3): 146-150, September 1991)

Key words: vertigo, microvascular decompression operations

Introduction

Symptoms of persistent and severe vertigo are difficult both to diagnose and to relieve because of their many possible causes. We have identified a special group of patients with vertigo and nausea so severe that their daily life activities are severely restricted, and we have labeled their disease entity disabling positional vertigo (DPV). We have also shown that the symptoms these patients have are caused by vascular compression of the eighth cranial nerve close to the brainstem, and that microvascular decompression (MVD) can be effective in relieving the symptoms in the majority of patients.

Vascular Compression Syndrome

Vascular compression of the cranial nerves in the posterior fossa can cause symptoms of both hyperactivity and progressive loss of function. The most common disorders caused by vascular compression of cranial nerves are trigeminal neuralgia (TN), hemifacial spasm (HFS), and glossopharyngeal neuralgia (GPN), and many reports have documented that MVD relieves the symptoms in a very high percentage (over 90% for hemifacial spasm, 85 to 90% for trigeminal neuralgia) of cases. It is also known that vascular compression only causes symptoms of pain or facial twitching when the compressing blood vessel or vessels are in contact with the part of the nerve that is covered by central myelin (root entry/exit zone, REZ); compression of the more distal portion of the respective nerve causes less-specific, if any, symptoms.

The transition zone between peripheral and central myelin (the Obersteiner-Redlich zone) for the eighth cranial nerve is inside the internal auditory meatus, and thus vascular compression can occur at any point along the entire intracranial portion of the nerve. Vascular
compression of the eighth nerve causes the specific symptoms of vertigo, spinning, and nausea. In addition, there are frequently subtle symptoms of involvement of adjacent cranial nerves. Patients with DPV have more or less constant vertigo that is associated with a varying degree of nausea, from a queasy feeling to nausea associated with vomiting. Their symptoms increase with physical activities and may abate with complete bedrest. Often their symptoms are worse when they hold the head in a certain position. In this respect the symptoms of patients with DPV differ from those of patients with such disorders as Meniere's disease, in which well-defined attacks of vertigo, aural fullness, and increasing tinnitus are followed by periods with no symptoms of vertigo at all. The symptoms of DPV are also distinctly different from those experienced by patients with benign paroxysmal positional nystagmus (BPPN), whose attacks of vertigo are also triggered when they place the head in a certain position but whose symptoms and accompanying nystagmus decrease with repeated repositioning of the head. The symptoms of DPV also differ from those of such disorders as perilymphatic fistula, vestibular neuritis, and tumors in the posterior fossa or brainstem.

In about 60% of the more than 400 patients with DPV we have treated, the onset of the disorder was acute, and in that respect DPV resembles vestibular neuritis (VN). However, patients with VN recover gradually over time, depending on the age of onset, and they are usually asymptomatic after a few months. Also their symptoms respond to administration of vestibular suppressant medications, which is not the case for patients with DPV. In fact, vestibular suppressant medications make the symptoms of DPV worse, and the only medication that seems to reduce vertigo in patients with DPV is diazepam (Valium).

Patients with DPV may improve somewhat after the onset of severe vertigo, but they usually continue to be dizzy and nauseated, and because of that they adopt even more restricted lifestyles: They stop driving, become unable to do simple household chores, and over time they come to rely on other people to help them with activities of daily life.

In addition to the dominant symptoms of vertigo and nausea, many patients with DPV over time notice symptoms due to involvement of cranial nerves that are adjacent to the eighth nerve. Most frequent is a sharp, pinching ear pain on the involved side, indicating involvement of the nervus intermedius. Almost as frequent is a mild twitch under the eye (indicating involvement of the seventh cranial nerve) or quivering of the orbital muscles. Transient numbness in one of the branches of the trigeminal nerve is also a symptom that can accompany the vestibular symptoms in patients with DPV. Some patients with DPV also notice occasional throat pain on the affected side, or difficulties swallowing — signs of involvement of the glossopharyngeal nerve. A few patients experience a progressive hearing loss on the affected side and tinnitus which, however, seldom is pulsatile in nature.

When a patient with DPV is walking, he/she walks as if inebriated, but with one important difference: He/She drifts to one side only (the affected side). Such patients cannot make a quick turn without making a wide circle or even falling. Most patients summarize these symptoms as feeling "like I had one or two drinks too many."

In the majority of patients with DPV the disorder begins rather suddenly, but in about 40% of patients the symptoms begin insidiously with a light-headed, swimming feeling that worsens to the symptoms just described over a period of years.

**Differential diagnosis of disabling positional vertigo**

The case history is most important in the differential diagnosis of DPV, and considerable time must be spent with the individual patient. Typically, the patients that we see have consulted numerous physicians over the spectrum of medical specialists, which has often left them confused and depressed because they have received much conflicting information and many different suggestions for treatment but have not had relief.

We have found the results of audiometric testing (audiometry and recording of the acoustic middle ear reflex response) and recording of BAEP (brainstem auditory evoked potentials) very helpful in evaluating patients with DPV. BAEP recordings are analyzed in these cases because the blood vessel or vessels that are compressing the vestibular nerve may also compress the auditory portion of the nerve, and such compression will be reflected in changes in the BAEP.

**Using the BAEP in differential diagnosis of vestibular problems**

For recordings of BAEP to be useful in differential diagnosis it is necessary to know the anatomical origins of the various components of the BAEP. Recordings from the intracranial portion of the human eighth cranial nerve have shown that both peaks I and II are generated by the auditory nerve (peak I by the distal portion and peak II by the intracranial (proximal) portion of the nerve).5-8 and peak III is mainly generated by the cochlear nucleus.9 Peaks IV and V have more complex origins and most likely represent activities in the lateral lemniscus (see Figure 1).8

When zero-phase digital filtering10 is used to suppress the noise and enhance the peaks in BAEP recordings and computer programs identify the exact latency and amplitude of each BAEP peak, these recordings can readily be used to facilitate the differential diagnosis of
disorders affecting the auditory nerve. The BAEP recordings from patients with DPV show increased conduction time in the auditory nerve. The interval between peaks I and III often fluctuates in such recordings, and peak II, which is generated by the proximal portion of the eighth nerve, often has an abnormal waveform. These patients also often have prolonged latency of peak V recorded from the contralateral ear, possibly as the result of compression of the brainstem. Peak V is generated by the termination of the lateral lemniscus after the main auditory pathways have crossed over to the other side, and so similar BAEP changes may also often be seen in recordings from patients with small acoustic tumors.

Patients with DPV often have minor abnormalities in their pure tone thresholds — either a 10- to 15-dB difference in threshold between the ears or a small notch in the mid-frequency range, usually at 1500 Hz. We first noted these notches in audiograms of patients with hemifacial spasm (11%) and interpreted these changes in the threshold as representing damage to nerve fibers at the place of compression. The acoustic middle ear reflex response may also be abnormal in patients with DPV. The reflex threshold may be elevated (>95 dB), or the reflex response may be absent when elicited from the affected side (see Figures 2 to 4).

On vestibular testing, patients with DPV are usually found to have a reduced caloric response together with spontaneous nystagmus of the destructive type on the affected side. Some patients with DPV have vestibular test findings that are more subtle and far less severe, such as directional preponderance or positional nystagmus, than would be expected from the severity of their symptoms.

Findings of a Study of DPV

Patients

All patients who underwent operation (MVD) to relieve DPV in one year at our institution were grouped together for evaluation of their progress, and they have now been followed for 4½ to 5½ years. The 32 women
and 9 men in the group range in age from 28 to 64 years. Two patients had bilateral DPV and underwent a second procedure during this study period.

In 2 patients the symptoms of DPV began after a motor vehicle accident, but in the other 39 no predisposing event could be identified. Symptoms usually began between the ages of 25 and 35, and patients had had symptoms an average of 4 to 6 years before undergoing MVD. Only 4 of the 41 patients were employed when they underwent treatment for MVD, and their jobs were classified as sedentary.

Preoperative testing results

Preoperatively all patients underwent several imaging studies, both computed tomography (CT) and magnetic resonance imaging (MRI), all with negative findings. All patients also underwent pure tone and speech audiometry with recorded lists of phonetically balanced (PB) words, recording of middle ear reflex responses, and recording of BAEP responses to 2-kHz tone pips and in some patients also click sounds. Table 1 summarizes the abnormal results of audiometric testing, recording of BAEP, and vestibular testing.

Table 1 Summary of Abnormal Test Results in 41 Patients Who Underwent MVD for DPV

<table>
<thead>
<tr>
<th>Surgical findings</th>
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<td>At operation in all patients a blood vessel was seen clearly to be in contact with the vestibular, and also often the auditory, portions of the eighth nerve. Frequently, the offending blood vessel or vessels were found to be located under the flocculus, which had to be dissected off the nerve for the surgeon to be able to move the offending blood vessel off the eighth nerve. In many cases the offending blood vessel had left a groove in the nerve.</td>
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<tr>
<td>Results of surgery</td>
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<tr>
<td>It is always difficult to assess the efficacy of a procedure in patients with vertigo. Thus whether the results of</td>
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vestibular testing are normal or abnormal does not pro-
vide a reliable guide to the degree of improvement a
patient may have after such a surgical procedure. We
therefore chose to use the patients' own assessments of
improvement as the basis for our evaluation of the
surgical results. We know from experience that the first
symptom of DPV that is relieved by MVD is the nausea.
The vertigo subsides gradually, more gradually the longer
the patient has had the symptoms. Most patients increase
their activities within a few weeks and usually full recov-
ery is reached between 3 and 6 months after surgery.

Twenty patients of the 41 patients in our study group
reported excellent results of MVD: They returned to
work, driving, and activities such as tennis. Ten patients
noticed clear improvement in symptoms of DPV but
now and then have brief episodes of vertigo, especially
when they are under stress. We consider these patients
to have good results of MVD. Three of
these last 9 patients had signs and symptoms of bilateral
compression, but elected not to have a second operation
during this study period. In no patient was DPV worse
after MVD than before.

The 73% success rate for MVD to treat DPV in these
41 patients may seem low, but in view of the degree to
which these patients were disabled before the operation,
we regard the results as very positive. More recent
results show a greater percentage of improvement with
this type of surgical procedure, probably for several
reasons but notably because it was learned from electro-
physiological recordings made during MVD operations
for HFS that even very small vessels, including small
veins, can cause nerve compression effects.12 This know-
ledge was applied to MVD operations of the eighth nerve
for DPV, and now all vessels, even the smallest ones, are
lifted off the nerve and soft Teflon padding is placed
between the nerve and the vessels. Veins, small or large,
are either moved off the eighth nerve or coagulated
and divided.

Complications of surgery to relieve DPV

Generally, postoperative complications of MVD for
DPV are rare, and permanent neurological deficits are
very rare. None of the 41 patients reported upon here had
any permanent neurological deficit, although 1 patient
had a temporary paresis of the superior oblique muscle
which resolved in 6 to 7 months and 1 patient had vocal
cord paresis which resolved in 3 months. Hearing loss is
the most frequent complication of MVD for DPV, but
even this complication is rare. One patient in this series
suffered total loss of hearing on the operated side, but
the vertigo did not worsen.

References

2. Möller MB, Möller AR, Jannetta PJ, Sekhar LN: Diagnosis and
surgical treatment of disabling positional vertigo. J Neurosurg
64: 21–28, 1986
3. Möller MB: Results of microvascular decompression of the eighth
5. Möller AR, Jannetta PJ: Compound action potentials recorded
intracranially from the auditory nerve in man. J Exp Neurol 74:
862–874, 1981
6. Möller AR, Jannetta PJ: Interpretation of brainstem auditory
evoked potentials: Results from intracranial recordings in humans.
7. Möller AR, Jannetta PJ: Auditory evoked potentials recorded
from the cochlear nucleus and its vicinity in man. J Neurosurg
59: 1013–1018, 1983
8. Möller AR, Jannetta PJ: Neural generators of the auditory brain-
stem response. In: The Auditory Brainstem Response, Jacobson
9. Möller AR: Use of zero-phase digital filters to enhance brainstem
auditory evoked potentials (BAEPPs). Electroencephalogr Clin
10. Möller MB, Möller AR, Audiometric abnormalities in hemifacial
11. Möller AR, Jannetta PJ: Monitoring facial EMG responses during
microvascular decompression operations for hemifacial spasm.
Bluestone CD, Brackmann DE, Krause CJ, eds, Mosby Year