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Neurovascular Compression Syndrome of the Eighth Cranial Nerve. Can the Site of Compression Explain the Symptoms?

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Summary

Considerable skepticism still exists concerning the concept of neurovascular compression (NVC) syndromes of the eighth cranial nerve (8th N). If such syndromes exist, the sites of compression of the nerve must explain the symptoms encountered. We recorded compound action potentials of the cochlear nerve (CCAPs) during neurovascular decompression (NVD) to examine the topography of the three components of the 8th N. The sites of compression of the 8th N in cases of NVC syndrome confirmed at surgery were superimposed on the topography of the CN and vestibular nerve (VN) in order to determine the relationship between the sites of compression and the symptoms. CCAPs were clearly and consistently recorded on the caudal surface of the 8th N along the midline. In patients with vertigo and tinnitus there was vascular compression of the rostroventral (VN) and caudal surface (CN) of the nerve, respectively. In patients with both vertigo and tinnitus, there was compression of both VN and CN. Our findings clearly demonstrate that the symptoms of NVC of the 8th N depend on the part of the nerve that is compressed by blood vessels, and they support the concept of NVC syndrome of the 8th N.

Keywords: Eighth cranial nerve; neurovascular compression syndrome; cochlear nerve; vertigo; tinnitus.

Introduction

Since the eighth cranial nerve (8th N) contains superior vestibular nerve (SVN), inferior vestibular nerve (IVN), and cochlear nerve (CN) components, neurovascular compression (NVC) of the 8th N can produce vertigo, tinnitus, or hearing disturbances, or various combinations of them, unlike vascular compression of the trigeminal or facial nerve. This complicated symptomatology is one of the major reasons NVC syndromes of the 8th N are very difficult to understand. Many physicians, therefore, have an unclear understanding of NVC syndromes of the 8th N, and hesitate to perform neurovascular decompression (NVD). If NVC syndrome of the 8th N is a genuine disease entity, the sites of compression of the nerve must explain the symptoms described above. Jannetta *et al.* [2] reported that the site of compression in cases with tinnitus is more distal than that in cases with vertigo. However, they did not indicate exactly which components of the 8th N were compressed by blood vessels. If a vascular loop compresses a component of the 8th N, it will produce symptoms produced by malfunction of that component. If compression of more than two components occurs, it will produce more complex symptoms attributable to the components affected. However, to our knowledge, there have been no detailed studies of the relationship between sites of compression of the 8th N and associated symptoms. In order to clarify the concept of NVC syndrome of the 8th N, and to clarify the diagnostic criteria and surgical indications, it is of great importance to determine the relationship between sites of compression of the 8th N and symptoms. In addressing this problem, we must initially determine the topography of the cochlear and vestibular components of the cisternal portion of the 8th N. We used electrophysiological techniques intra-operatively to determine the topography of the CN and VN in the cisternal portion of the 8th N, and determined the relationship between the sites of compression and the symptoms in patients for whom NVC of the 8th N was confirmed at surgery.

Patients and Methods

We explored the posterior fossa in 53 patients with a diagnosis of NVC of the 8th N between May 1982 and February 1998. No diagnostic criteria for NVC syndrome of the 8th N have ever been established. All patients, therefore, had undergone a variety of treat-

ments, mainly in neuro-otology clinics, including medical treatment, e.g., with Carbamazepine, drugs to improve microcirculation in the inner ear, steroids or high osmolarity agents, biofeedback therapy, and psychological counselling. Exploration of the posterior fossa was indicated when the symptoms could not otherwise be explained neuro-otologically, when the above-mentioned medical treatments proved ineffective, and when the MRI, CT, air CT or angiography findings suggested NVC. Details of the operative procedure have been described in a previous paper [6].

Forty-three of the 53 patients were followed up for 2 years or more, and they are the subjects of this study. Three of the 43 patients had vertigo alone (group V), and NVC was confirmed in 2 of them. Twenty-one patients had vertigo and tinnitus with or without sensorineural hearing disturbance (Group VT), and 17 had NVC. Nineteen patients had tinnitus with or without sensorineural hearing disturbance (Group T), and 12 had NVC (Table 1). A clinical summary of the 31 patients who had NVC is listed in Table 2. In the most recent 23 patients, we tried to record compound action potentials of the cochlear nerve (CCAPs), and succeeded in 20 patients. After completion of NVD, a monopolar recording electrode was placed on the surface of the 8th N, and CCAPs were recorded from 10 to 15 points. Five recording points each were aligned across the nerve at the most distal, the middle, and the most proximal portions (Fig. 1). More anatomically detailed recording (9 to 10 recording points in one array) was also attempted in some cases. To avoid damaging the nerve as a result of blind manipulation, we did not record action potentials from the other side of the 8th N. We initially used a gold ball monopolar electrode 0.4 mm in diameter, and later used a fine wire electrode with a 'U' shaped tip for more detailed recording. The reference electrode was placed in a nearby muscle. An alternating click sound of 90–95 dB was used to stimulate the ear on the oper-

Table 1. *Summary of the Surgical Findings in 43 Cases*

	No. of cases	NVC (+)	NVC (−)
Group V (vertigo)	3	2	1
Group VT (vertigo + tinnitus)	21	17	4
Group T (tinnitus)	19	12	17

NVC (+) neurovascular compression was confirmed at surgery; *NVC* (−) neurovascular compression was not confirmed at surgery.

Table 2. *Clinical Summary of 31 Cases of NVC of the 8th N*

Case no.	Age, sex	Side	Presentation V	T	H	F	Neuro-otological diagnosis	Offending vessels	Operative procedures	Follow up period	Results V	T	H	F
1	61F	R		+	+		−	AICA + PICA	NVD	13y7m		+	+	
3	47M	L	+	+	+		M	AICA	NVD	12y9m	−	−	+	
4	24M	R	+	+			BPPV	AICA	NVD	11y7m	i	−		
5	53M	L		+	+		−	SCA	NVD	11y4m		+	+	
7	53M	L		+			−	AICA	NVD	11y8m		−		
8	53M	R	+	+	+		M	AICA	NVD	11y6m	−	+	+	
9	31F	L	+	+	+		M	AICA + IAA	NVD	11y1m	−	i	+	
10	52F	L	+	+	+		BPPV	AICA	NVD	11y1m	−	+	+	
12	47F	R		+	+		SD	AICA	NVD	10y9m		−	i	
13	54M	R		+	+		SD	AICA	NR	10y8m		+	+	
14	68F	R		+	+	+	NVC	PICA	NVD	10y8m		−	+	−
15	42F	R	+	+	+		M	PICA	NVD	10y7m	−	+	+	
16	46M	L	+	+	+		M	AICA	NVD	10y6m	−	−	+	
17	46F	R	+	+	+		M	Ch. plx.	CR	10y5m	−	+	+	
18	38F	R	+				BPPV	AICA	NVD	10y4m	−			
21	53F	L		+	+	+	NVC	AICA	NVD	9y10m		−	+	−
23	43M	L	+	+	+	+	NVC	AICA	NVD	9y7m	−	−	+	−
25	64M	L	+	+	+		M	AICA	NVD	9y1m	−	+	+	
29	49M	L	+	+	+		M	Vein	VR	7y10m	−	i	+	
30	50F	R		+	+	+	NVC	AICA	NVD	7y4m		−	+	−
31	67F	R		+	+	+	NVC	AICA + PICA	NVD	7y3m		−	d	−
33	73F	L		+	+	+	NVC	VA	NVD	6y5m		−	+	−
34	44M	R	+	+	+	+	NVC	Vein	VR	6y2m	−	−	+	−
35	52M	L	+	+			VBI	AICA	NVD	5y9m	−	−		
36	48M	L	+	+	+		−	VA	NVD	4y10m	−	i	+	
38	42M	R	+	+			M	AICA	NVD	3y9m	−	−		
39	58M	L	+	+			M	AICA	NVD	3y4m	i	−		
40	61M	L		+			−	VA	NVD	3y1m		+		
41	42F	R	+				VIB	VA	NVD	3y	−			
42	45M	L		+			−	AICA + Vein	NVD + VR	2y3m		−		
43	44F	L	+	+	+		M	AICA + Vein	NVD + VR	2y	−	i	+	

R Right; *L* left; *V* vertigo; *T* tinnitus; *H* sensorineural hearing disturbance; *F* hemifacial spasm; *M* Ménière's disease; *BPPV* benign paroxysmal positional vertigo; *VBI* vertebro-basilar insufficiency; *SD* sudden deafness; *NVC* neurovascular compression; *AICA* anterior inferior cerebellar artery; *PICA* posterior inferior cerebellar artery; *SCA* superior cerebellar artery; *IAA* internal auditory artery; *Ch. plx.* large choroid plexus; *VA* vertebral artery; *NVD* neurovascular decompression; *NR* neurectomy; *CR* resection of choroid plexus; *VR* vein resection; *i* improved; *d* temporary deterioration; + symptom presents; − symptom resolved.

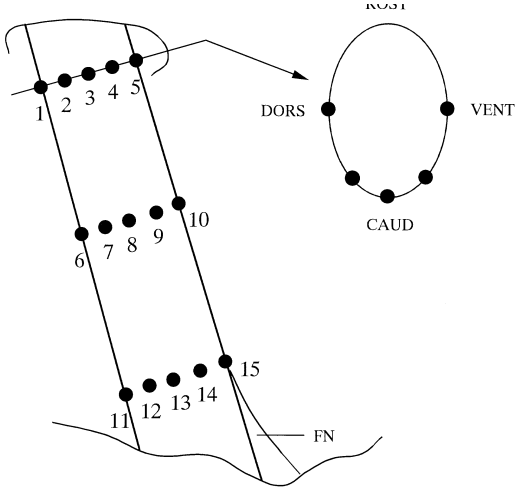

Fig. 1. Recording points on the surface of the 8th N which is viewed via a retrosigmoid approach (right side). *CAUD* Caudal; *ROST* rostral; *VENT* ventral; *DORS* dorsal; *FN* facial nerve

ated side at a frequency of 10 c/s. Ten responses were summed and averaged with a filter setting of 100–1500 Hz. The total recording time was less than 1 min. The differences in amplitude of the CCAPs at each recording point were statistically analyzed by ANOVA, and a p value < 0.05 was considered significant.

We superimposed the vascular compression sites on the topographic map of the 8th N in patients in whom NVC was confirmed at surgery in order to determine the relationship between the sites of compression of the 8th N and the symptoms.

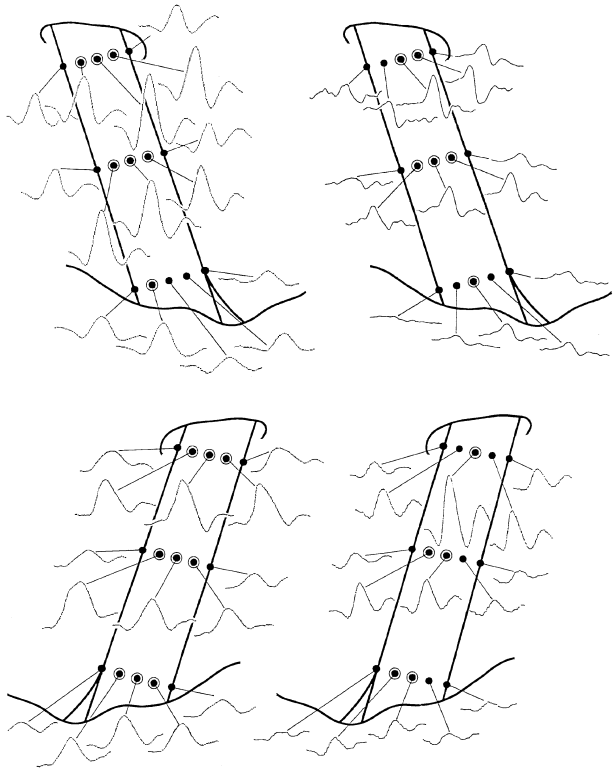

Fig. 2. Typical CCAP recordings in 4 patients. *Top* Right 8th N in 2 patients; *bottom* left 8th N in 2 patients. Circled dots indicate the points from which the largest CCAP amplitude was recorded, and are along the midline of the nerve

Results

Figure 2 shows typical recordings of CCAPs in 4 patients. The CCAPs were recorded from the entire surface of the 8th N, but their amplitudes differed considerably both between patients and between recording points in the same patient. Fig. 3 shows differences in average amplitude between the recording points in each electrode array on the surface of the 8th N in 20 patients. The CCAPs in the central and adjacent points (points 3, 2 and 4) in the most distal electrode array had the greatest amplitude. The amplitude of the potentials outside these three points was much lower, and this phenomenon was confirmed statistically. The more proximally the electrode array shifted, the smaller the amplitude became in most cases. The amplitudes of the CCAPs in the central and adjacent points, however, were still the highest in each array.

Figure 4-a shows the topography of the CN in the 8th N based on the results of this study. The CN is located along the middle of the caudal surface of the nerve (gray zone). The blank area indicates the VN (SVN and INV). The borders between the two components were determined from marked changes in CCAP amplitude. We reconstructed the vascular compression site in each case on this topographic map from the operative records, video tapes, and photographs taken during surgery. Figure 4b–d show the sites of compression of the right 8th N superimposed on the topographic map in Groups V, VT, and T, respectively. The sites of compression of the left 8th N are also superimposed on this map. The numbers are the sequential numbers of our 53 patients. Case 13 is not plotted here, since a portion of the 8th N (probably the CN) was extremely stretched by a blood vessel and could not be represented in the figure. Case 17 is also not represented here, since the exact compression site was not determined. In contrast to compression at the caudal side, compression of the ventral surface is very difficult to adequately represent in the two dimensional figures, although it was confirmed in most cases in Groups V and VT at surgery (Figs. 4b, and c). Figure 5 shows photographs of NVC in typical cases in each group. Table 3 shows the main sites of compression of the 8th N in Groups V, VT, and T. The sites of com-

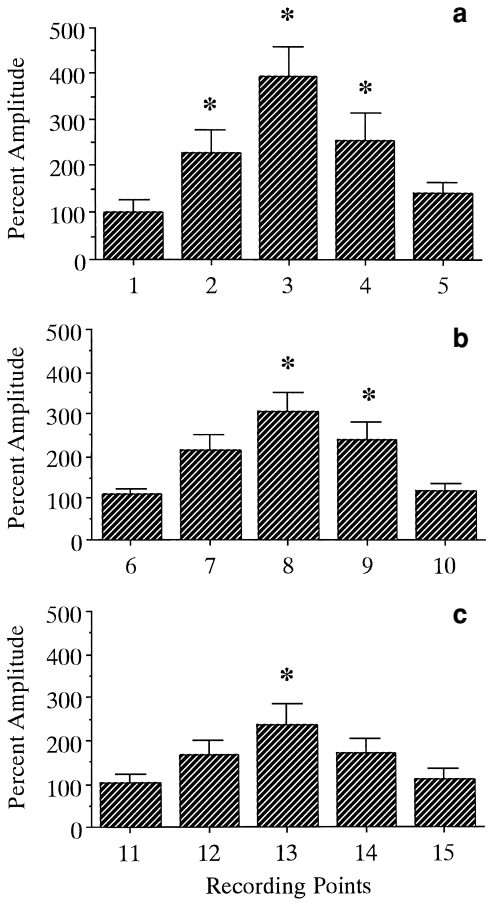

Fig. 3. Differences in average amplitude among recording pints in 20 patients. Each amplitude is expressed as a percentage of the amplitude at point 1 as a control. Each value is given as mean ± SEM. *, $p < 0.05$. Note that the amplitudes at the central and adjacent points are largest in the most distal array. They become smaller and the differences between them decrease as the recording array shifts more proximally, but the central recording point still has the highest amplitude

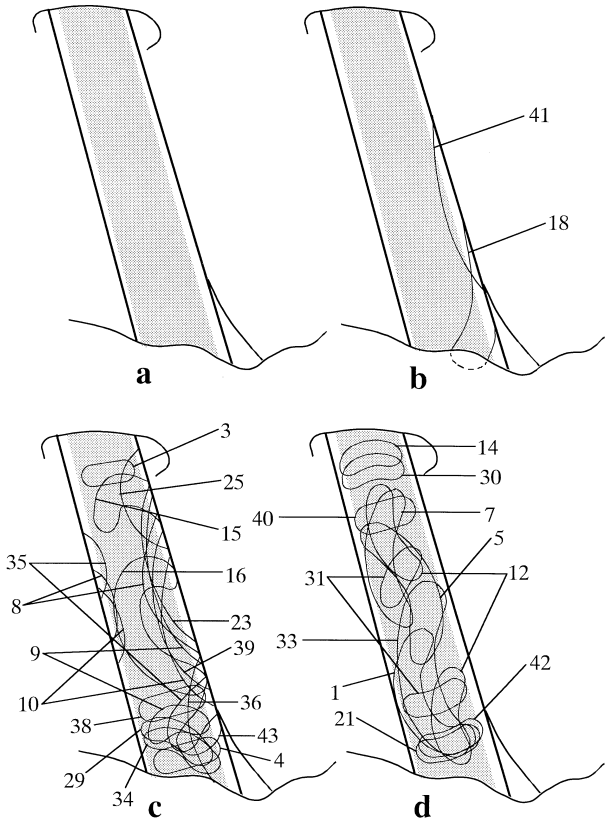

Fig. 4. Sites of compression of the 8th N in 31 patients superimposed on the topographic map of the 8th N. (a) Topography of the CN of right 8th N in the CP cistern as indicated by CCAPs. The CN (gray zone) is located along the midline on the caudal surface of the 8th N. The blank area indicates the VN. The borders between the CN and VN were postulated on the basis of marked changes in the amplitude of CCAPs. (b–d) Sites of compression in Group V (vertigo alone), Group VT (vertigo and tinnitus), and Group T (tinnitus alone), respectively. Note that the compression site is mainly on the VN (ventral aspect of the 8th N) in Group V, on both the CN and VN (caudal and ventral aspects) in Group VT, and on the CN (caudal aspect) in Group T. Compression of the ventral aspect of the 8th N is not adequately described in these two-dimensional figures, although it was confirmed at surgery

pression mainly involved the ventral and rostral surface of the nerve (VN) in Group V, the caudal and ventral surface (CN and VN) in Group VT, and the caudal surface (CN) in Group T. The clinical manifestations were significantly dependent on the sites of compression ($p < 0.001$, chi-squared test).

Discussion

We were able to determine the topography of the CN of the 8th N by recording the CCAPs. The CN is located along the midline on the caudal surface of the 8th N. Based on the position of the CN and marked changes in the amplitude of CCAPs, we were also able to readily determine that the vestibular components (SVN and INV) are located in the rostral part of the nerve. Silverstein and Norell [9] reported an obvious cleavage plane between the CN and VN at the cerebello-pontine (CP) angle in 75% of their surgical cases. We explored the CP angle in 53 patients with a diagnosis of NVC syndromes of the 8th N, but were unable to identify the cleavage plane in any of the cases studied. It is our strong impression that differentiation of the CN and VN during posterior fossa surgery is quite difficult even under the surgical microscope. This impression is supported by those of others [4, 7]. It is therefore impossible to determine with certainty which components of the 8th N are compressed by blood

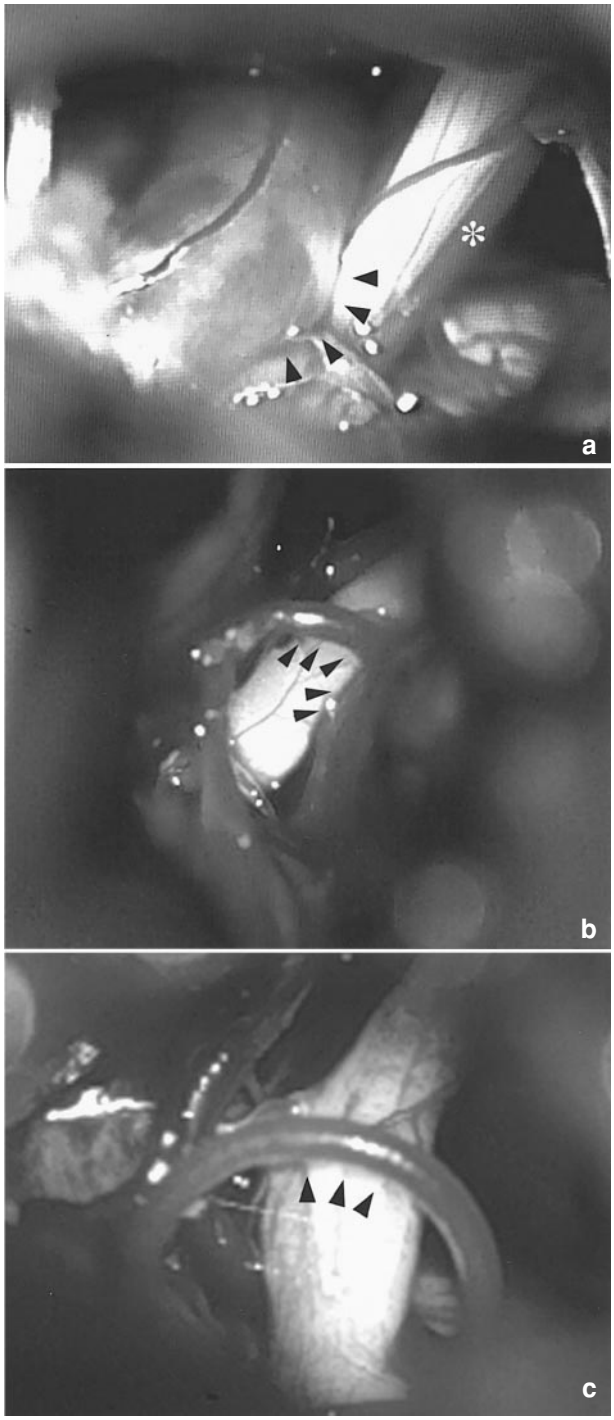

Fig. 5. Photographs of typical cases of NVC of the 8th N. (a) A case of vertigo only (Group V). The ventral (and probably the rostral) aspect of the 8th N is compressed by the vertebral artery (case 41 in Fig. 4b). An artery (*) is running parallel to the nerve, but is not compressing it. (b) A case of both vertigo and tinnitus (Group VT). The caudal and ventral aspects of the 8th N are compressed by an artery (case 15 in Fig. 4c). (c) A case of tinnitus alone (Group T). The caudal aspect of the 8th N is compressed by an arterial loop (case 40 in Fig. 4d). Arrowheads indicate the compression sites

Table 3. *Main Sites of Compression of the 8th N in Groups V, VT, and T*

		Main compression sites		
		CN	CN and VN	VN
Group V	(2 cases)	0	1	1
Group VT	(16 cases)	5	11	0
Group T	(11 cases)	11	0	0

There is a very close relationship between compression sites and symptoms which is confirmed statistically ($p < 0.001$, chi-squared test). *CN* Cochlear nerve; *VN* vestibular nerve.

vessels during surgery without intra-operative recording of CCAPs. Responses were easily identified even with only a single stimulation. The responses obtained usually had a triphasic shape, and sometimes exhibited a second negative peak, as others have reported [1, 3].

Colletti *et al.* [1] reported that the CN can be clearly identified using bipolar recording electrodes. However, bipolar recording could not be used in our cases since the craniectomy was quite small (2.5×2 cm) and it was impossible for both electrode tips to contact the surface of the nerve with equal pressure due to the limitations on the angles between the electrode and the nerve created by the small craniectomy. We therefore used a monopolar electrode to record through a small retrosigmoid craniectomy. As a result of current spread, we obtained CCAPs from the entire surface of the nerve with the monopolar electrode. The amplitude of the response, however, differed considerably according to the recording point, and the position of the largest amplitude could readily be identified by gross inspection during surgery, and as shown in Fig. 3, was subsequently confirmed statistically. The largest responses were consistently obtained from the central and adjacent points in each recording array on the surface of the nerve, and response amplitude markedly decreased outside these three points. This means that the CN is located along the midline on the surface of the nerve, and that when viewed through a retrosigmoid approach, the border of the CN is located very close to the line connecting the points 2, 7, and 12, and 4, 9, and 14, respectively, as shown in Fig. 4a. This border, however, is neither definite nor very accurate, but is simply a rough landmark. We were unable to identify the cochlear-vestibular cleavage even with this technique. Even so, we obtained sufficient data to study the relationship between the sites of compression and symptoms.

Figure 6 shows the topography of the three compo-

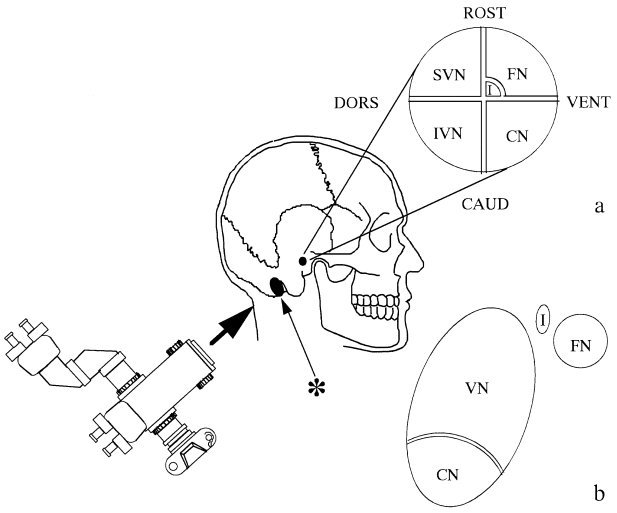

Fig. 6. Spatial arrangement of the three components of the right 8th N and the facial nerve at the fundus (a) and in the CP cistern (b). Because the IVN is located in front of the CN, the CN cannot be seen at the fundus (a) when viewed through the retrosigmoid approach, even if the posterior wall of the internal auditory canal is removed. We found that the CN is located in the caudal part of the 8th N in the CP cistern (b), and that the VN is rostral to the CN, because the 8th N rotates in the internal auditory canal. The boundary between the SVN and IVN could not be identified in this study. *I* Intermedius nerve; *FN* facial nerve; *VN* vestibular nerve; *IVN* inferior vestibular nerve; *SVN* superior vestibular nerve; * position of craniectomy

nents of the 8th N and the facial nerve at the fundus of the right internal auditory canal as viewed through the external auditory canal. The SVN is located rostral to the transverse crest, and the IVN caudal to the crest. The facial nerve is located ventral to the SVN, and the CN is ventral to the IVN. Therefore, the CN cannot be identified through the retrosigmoid approach because of the presence of the IVN at the fundus, even if the posterior wall of the canal is removed. Based on the results of the present study, however, in the CP cistern the CN is located along the midline of the caudal surface of the nerve. This indicates that the CN undergoes forward rotation in the internal auditory canal as it travels toward the porus. The SVN, IVN, and the facial nerve may rotate in the same direction, although the border between the two vestibular components could not be identified in the present study. This would result in the CN being located on the caudal side of the 8th N in the CP cistern, and the SVN and IVN on the rostral side. Silverstein *et al.* [8, 10] reported finding, in cadaver studies, that the CN, SVN and IVN rotate about 90 degrees, mainly in the internal auditory canal, and that only minimal rotation occurs in the CP cistern. Our results support their findings, although the rotation of the nerve was less than 90 degrees (unpublished data). The topography presented here is also supported by our recent clinical experience of using CCAPs and successfully and selectively dividing only the CN in the CP cistern, with no damage to the vestibular components, in a patient with intractable tinnitus and normal vestibular function [5].

The present study clarified the relationship between the sites of compression of the 8th N and the symptoms of NVC (Figs. 4b–d). In most patients with vertigo and tinnitus with or without sensorineural hearing disturbance, the site of compression mainly involved the caudal and ventral aspect of the 8th N. This indicates that the CN and the VN (SVN and IVN) had been compressed at the same time (Fig. 4c). In patients with tinnitus alone, the site of compression involved mainly the caudal aspect of the nerve, indicating that mainly the CN was compressed (Fig. 4d). In patients with vertigo alone, the site of compression was the ventral and rostral aspect of the nerve, indicating that the VN was mainly involved (Fig. 4b). These findings indicate that the sites of compression in patients accounted for their symptoms well, and they support the concept of NVC syndrome of the 8th N.

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Comment

This study is of great interest.

The intra-operative findings, i.e.: presence or not of a vascular conflict (Table 1); types of offending vessels (Table 2); sites of the conflicts (rostral, caudal, ventral, dorsal) are interesting, the more so as there are very few series of VIIIth nerve decompression reported, detailing intra-operatiove anatomical findings.

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