［原著］The effects of compression of the bundle of nerves and vessels at the internal auditory meatus on cochlear blood flow and acoustically evoked responses

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	作成者: Tang, Anzhou, Ura, Masaharu
	メールアドレス:
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The effects of compression of the bundle of nerves and vessels at the internal auditory meatus on cochlear blood flow and acoustically evoked responses

Anzhou Tang and Masaharu Ura

*Department of Otolaryngology, Faculty of Medicine, University of the
Ryukyus, Okinawa, 903-01, Japan*

ABSTRACT

This study was performed to observe the changes in cochlear blood flow (CoBF), the compound action potentials (CAP) and auditory brainstem response (ABR) in the pressure-induced animal model of acoustic neuroma. By suboccipital approach, the bundle of nerves and vessels (BNV) at the entrance of the internal auditory meatus in the guinea pig was exposed, and two pressure points, one being anterior to and the other being posterior to the center of the bundle were selected to compress. After compression, the changes in CoBF, CAP and ABR were determined in a total of 31 animals. According to the responses of CoBF, we classified these changes into three types as follows: complete decrease (type I), incomplete decrease (type II) and increase in CoBF (type III). The proportion of change in each type revealed significant differences between the two pressure points. Seventy-four percent (14/19) of the compressions at the posterior point showed type I, whereas 67% (8/12) of the compressions at the anterior point showed type III. CAP and ABR in each type were closely related to CoBF. In type I and II, CAP and ABR deteriorated, and the deteriorations of both were attributed to cochlear ischemia. After transient occlusion of CoBF, N_1 of CAP was able to recover. The degrees of N_1 recovery were dependent not only on the ischemic duration but also on the degree of decrease in CoBF during ischemia. On the other hand, although there was no decrease in CoBF and wave I in type III, I-II interwave latency delay with decrease in the amplitudes of wave II-IV was observed. It is suggested that the changes in type III were mainly caused by the blockage of the cochlear nerve. The results presented in this study indicate that compression will induce damages to both the internal auditory artery and the cochlear nerve, depending mainly on the compression position. The mechanisms responsible for hearing loss caused by acoustic neuroma were discussed based on this pressure-induced model. *Ryukyu Med. J., 15(4)181~188, 1995*

Key words: internal auditory artery, cochlear nerve, cochlear blood flow, acoustically evoked responses, acoustic neuroma

INTRODUCTION

The characteristic signs of retrocochlear lesions including the poor speech discrimination score, abnormal adaptation in tone decay test, pathological stapedial reflex and the prolongation of I-V interwave latency in ABR are usually found in patients with acoustic neuroma. From these audiological test findings, acoustic neuromas are considered as one of representative retrocochlear disorder. Previous studies on the histopathology[1,2] have demonstrated that the destruction of cochlear nerve fibers is the main change responsible for hearing loss and provided the fundamental evidences for supporting the theory of retrocochlear lesions in acoustic neuroma. However, Ylikoski et al[2] analyzed the relationship between the audiological test results and the remaining population of cochlear nerve fibers and failed to establish the relationship between them. The damage of the cochlear nerve alone could not fully explain hearing loss in patients with acoustic neuroma.

In the cerebellopontine angle and within the internal auditory meatus, the internal auditory artery usually travels with the nerves[3-7]. From the anatomical features in the arrangements of the nerves and the vessels, it is suspected that mass lesions like acoustic neuroma could involve both the nerves and the arteries. Indeed, the typical signs of cochlear impairments such as the positive recruitment phenomenon were also found in these patients[8-14]. The disorder in CoBF due to compression of the internal auditory artery is considered as the main factor resulting in cochlear dysfunction in acoustic neuroma[1,2,8-14], but there is lack of direct evidence to show the changes in CoBF.

The direct measurement of CoBF will give a new

approach to elucidate the mechanism of hearing loss associated with the disorder in the circulation of the inner ear. Laser Doppler flowmeter is a noninvasive method that is particularly suitable for the assessment of dynamic changes in CoBF[15-17]. In this study, the pressure-induced model of acoustic neuroma was established by compression of the BNV at the entrance of the internal auditory meatus in guinea pigs, and the measurement of CoBF was used in combination with CAP and ABR to evaluate the pathophysiological changes in the auditory functions. The purpose of this study was to determine 1) the responses of CoBF, CAP and ABR to compression at different pressure points, and 2) the relationship between CoBF, CAP and ABR.

MATERIALS AND METHODS

Animals and preparation

Healthy guinea pigs (Hartley) weighing between 450 and 550 g with normal Preyer reflexes were used in this experiment. The animals were anesthetized with pentobarbital sodium (35 mg/kg, intraperitoneally) and artificially ventilated with a respirator (SHINANO, Model SN-480-7). The muscle relaxant pancuronium bromide (0.08/ kg, intramuscularly) was administered. Rectal temperature was regulated with a rectal probe sensor and maintained at 36-38°C by a heating pad. A cannula inserted in the left carotid artery was connected with pressure sensor (NIHON-KOHDEN, TP-400T) to monitor blood pressure (BP).

This experiment was performed in accordance with the National Research Council criteria for care and use of animal, and the guide for animal experiments of the University of the Ryukyus. The animal use protocol was approved by the Animal Care Committee of the University of the Ryukyus.

Surgical approach

Each animal was held in prone position with a head-holder. By postauricular approach, the right cochlea with a round window was exposed. The mucosa of the cochlea was carefully removed with a cotton pledge permitting placement of a measurement probe directly on the bony surface.

To access the cerebellopontine angle, a suboccipital approach was used. A small sharp burr was used to make a craniotomy into the right posterior cranial fossa. The cerebellum overlying the BNV entraining the internal auditory meatus was carefully suctioned. During this manipulation, care was taken not to touch or retract the BNV. Once the BNV was exposed, ABR was recorded, and the animals giving a normal waveform were used for the following experimental procedures.

Pressure probe and the BNV compression

A simple pressure probe was designed for compression (Fig. 1-a). This probe was composed of concentric needles. The outer one held by micromanipulator was exactly positioned to the point of the BNV but without contact. The sharp, beveled end of 18 gauge inner needle was replaced with 1 mm silver ball, and 3 g of clay was
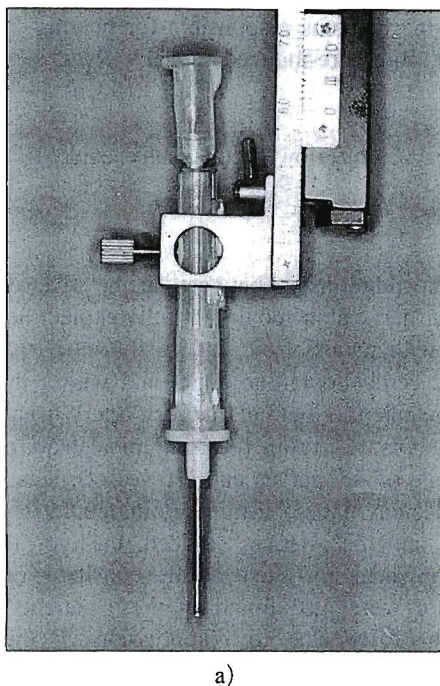
a)
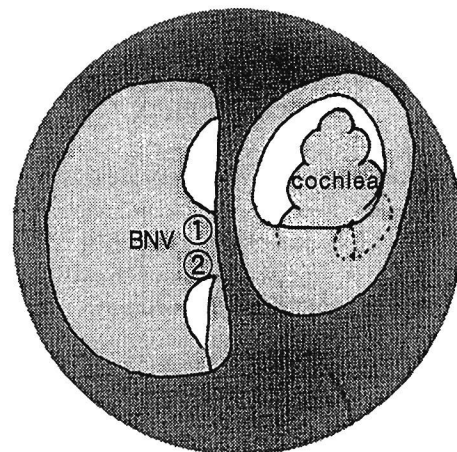
b)

Fig.1 a). Pressure probe used in compression.
    b). The diagram shows the positions of the bundle of nerves and vessels (BNV) at entrance of the internal auditory meatus and cochlea. ①
and ② show the anterior and the posterior pressure points respectively.

mounted on the head to compress the point of the BNV as desired. In order to observe the effects of compression at the different position, two pressure points, one being anterior to and the other being posterior to the center of the BNV were selected for compression (Fig. 1-b).

Measurement of CoBF, CAP and ABR

The needle-shape measurement probe (0.8 mm) held by another micromanipulator was placed on the bony wall of the basal turn or second turn of the cochlea. CoBF was then measured with a laser Doppler flowmeter (TSI Laserflo, 403A). CoBF as well as BP were recorded on a strip chart recorder (SANEI, 8K21) and preserved on the tape of cassette data recorder (TEAC, XR-7000). For recording CAP and ABR, a signal processor (SANEI, 7S 12) was used. The silver ball electrode (1 mm) placed on the round window membrane with reference electrode inserted into the neck muscles was used to record CAP. The electrodes placed on the vertex and the right mastoid process were used to record ABR. Clicks were presented to the right ear at a rate 10/s and at 80 dB (SPL) by a speaker placed 1 cm lateral to the right external auditory meatus. CAP and ABR were the average of 100 repetitive responses. The waveform of both was stored on the disk for later analysis. Block diagram of the experiment is shown in Fig. 2.

Experimental protocol

After CoBF, CAP and ABR became stable, the BNV was compressed. To determine the effects of compression at the different points on CoBF, CAP and ABR, a total of 31 guinea pigs were divided into two groups. In one group of 12 animals, compression was carried out at the anterior

point, while in the other group of 19 animals, at the posterior point. The duration of compression was 16 min in the former group. In the latter group, it was 5 (n=7), 16 (n=7) and 30 min (n=5) respectively. CoBF, CAP and ABR were simultaneously measured before, during and after compression.

Statistical analysis

The mean values for at least 3 min before compression were used as the original levels. Because CoBF measured by laser Doppler flowmeter has no absolute zero, we used the value after dissection of the BNV including the internal auditory artery as a reference zero to calibrate CoBF[17]. The values of CoBF, latencies and amplitudes of CAP and ABR were expressed as the percent change from its original level. Unless otherwise stated, these values are M ± SD. Data were analyzed using two sample Wilcoxon test and simple regression analysis with values at <0.05 significance level.

RESULTS

A: Classification following compression

With the manipulation on the BNV, no obvious change was observed in BP. As the BNV was compressed, CoBF, CAP and ABR showed changes. According to the responses of CoBF, we classified these changes into three types; complete decrease (Fig. 3), incomplete decrease
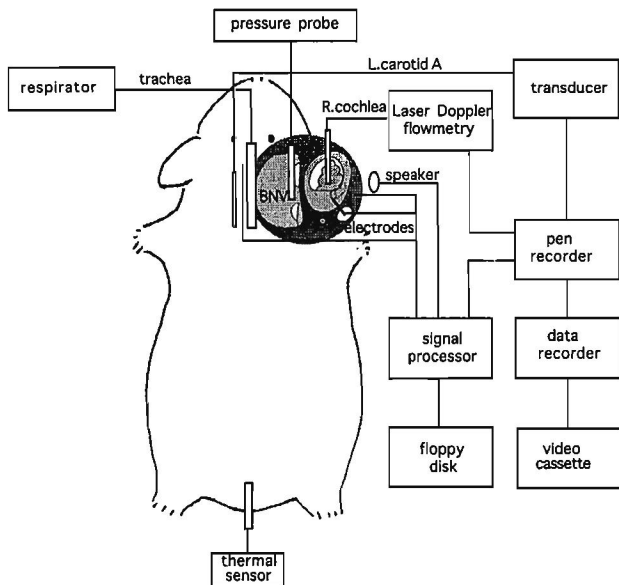
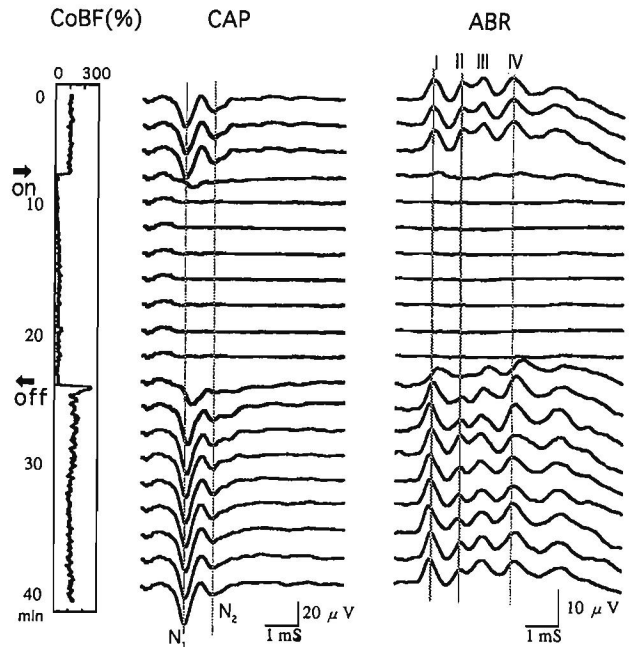

Fig.2 Experimental block diagram.

Fig.3 CoBF (left column), CAP (center column) and ABR (right column) in type I. With compression (➡), complete decrease in CoBF and the disappearance of CAP and ABR were noted. When compression was released (⬅), CoBF, CAP and ABR returned to the original level. Compression was performed at the posterior point in this animal.
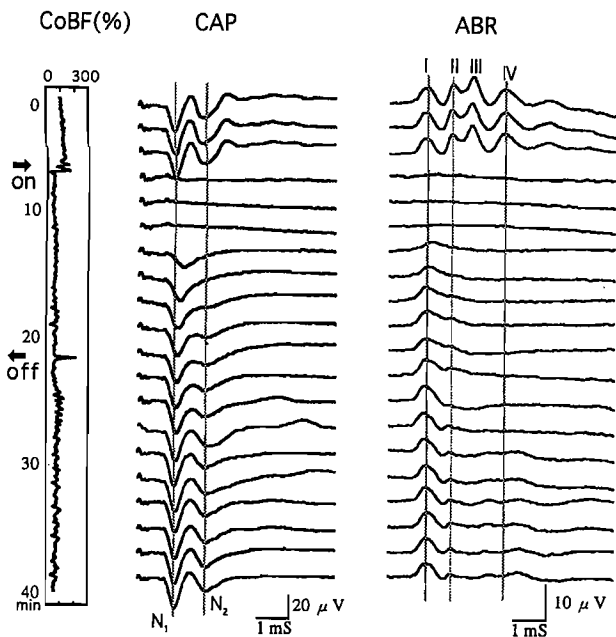
Fig.4 CoBF (left column), CAP (center column) and ABR (right column) in type II. With compression (➡), CoBF abruptly decreased and then gradually recovered. All waves of CAP and ABR transiently disappeared, and N_1 and wave I reappeared with recovery of CoBF. N_2 and waves II to IV also show recovery after compression was released (⬅). Compression was performed at the posterior point in this animal.
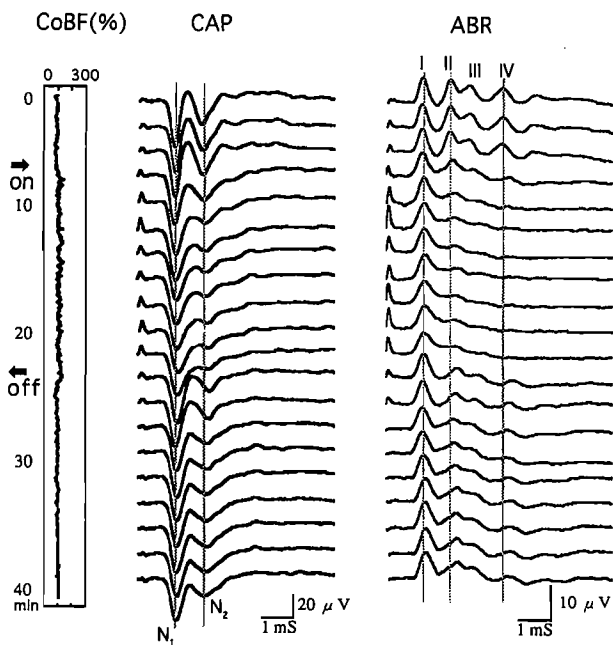

Fig.5 CoBF (left column), CAP (center column) and ABR (right column) in type III. With compression (➡), CoBF shows increase rather than decrease, while N_2 and waves II to IV become deteriorated. In this type, the prolongation of I - II interwave with the decrease in waves II to IV was obvious. Once compression was released (⬅), the changes in CoBF, N_2 and waves II to IV returned slowly to the original level. Compression was performed at the anterior point in this animal.

(Fig. 4) and increase in CoBF (Fig. 5), and these types were categorized as type I, type II and type III respectively. In type I, CoBF completely decreased and all waves of CAP and ABR disappeared during compression. CoBF in type II decreased, but not to zero, and then recovered gradually. With the decrease in CoBF, all waves of CAP and ABR transiently disappeared. When CoBF recovered, N_1 of CAP and wave I reappeared, but N_2 and waves II to IV remained deteriorated. In type III, although no decrease in CoBF occurred, the prolongation of I - II interwave latency with the decrease in the amplitudes of wave II to IV was observed. Similar to the changes in ABR, N_2 revealed the prolongation of latency and the decrease in the amplitude without the change in N_1.

The changes in CoBF, CAP and ABR following compression were reversible. Once compression was released, CoBF, CAP and ABR recovered in all types.

B: Types and the pressure points

Following the compressions at the anterior or posterior point, all of three types was found. Of the 12 animals that received compression at the anterior point, type I, II and III were observed in 1, 3 and 8 animals respectively. In the other 19 guinea pigs that received compression at the posterior point, type I, II and III were found in 14, 4 and 1 animals respectively. The proportion of type I was 74% of the compressions at the posterior point, and that of type III formed 67% of the compressions at the anterior point (Fig. 6). The results showed a significant difference between the two groups (two sample Wilcoxon test; p < 0.001), and indicated that the type of change was associated with the compression positions.
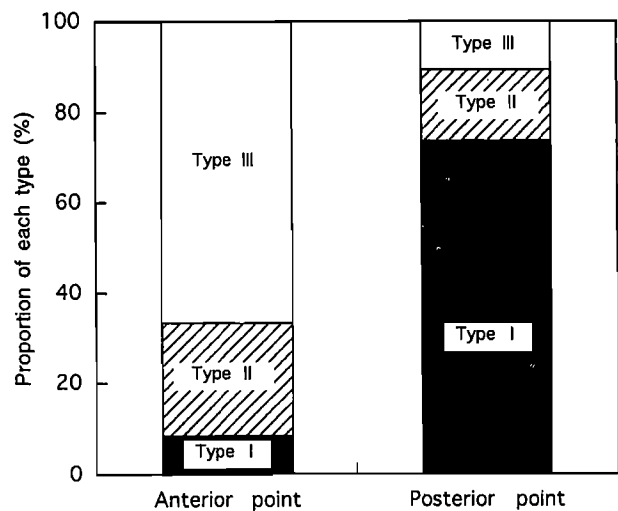

Fig.6 Pressure points and the proportion of each type. Type I, type II and type III formed 8%, 25% and 67% of the compressions at the anterior point, whereas type I, type II and type III formed 74%, 21% and 5% of the compressions at the posterior point. The proportion of each type shows significant difference between the two pressure points (two sample Wilcoxon test; p < 0.001).

C: Relationship between CoBF and N₁

From the changes in CoBF, CAP and ABR that occurred in type I, type II and type III, it is obvious that CAP and ABR were related to CoBF. We analyzed the relationship between CoBF and N₁ in the animals shown in Fig. 3, 4 and 5, and confirmed that there was a significant correlation between them (simple regression, $p < 0.001$) (Fig. 7). It is suggested that the change in N₁ depended upon CoBF. The results also indicated that when CoBF is reduced to as low as about 70% of the original level, N₁ deteriorated, and in turn, the disappearance of N₁ does not always represent zero level of CoBF.

D: Recovery of N₁ after transient occlusion of CoBF

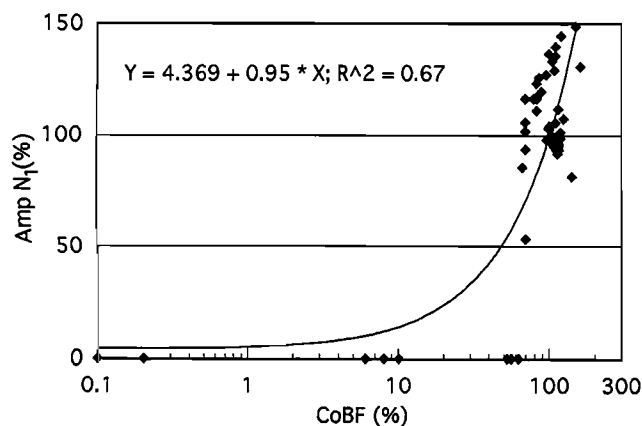Fig. 8 demonstrates the changes in CoBF and N₁ in the type I animals. With compression, CoBF decreased com-

Fig.7 Relationship between CoBF and the amplitude of N₁. Data from animals shown in Fig.3, 4 and 5. Significant correlation between CoBF and the amplitude of N₁ was found (simple regression, p <0.001).

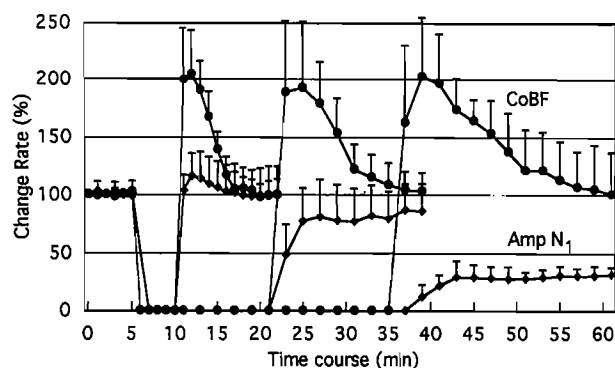pletely, and was maintained at zero level until release of compression. Once compression was released, CoBF recovered and transiently increased above the original level. The patterns of change in CoBF showed no obvious difference in 5, 16 and 30 min occlusion groups, while the recovery of N₁ after transient occlusion revealed wide variation in the different groups. N₁ recovered to about 105% of the original level in 5 min group, to 80% in 16 min group and to 30% in 30 min group. In the group with 30 min compression, however, we found an animal with incomplete decrease during compression which showed better recovery of N₁ than those with complete decrease in CoBF (Fig. 9). The different recovery of N₁ between them may be attributed to the different degrees of decrease in CoBF during compression.

E: No CoBF decrease and the change in ABR

In type III as shown in Fig. 5, no decrease in CoBF was observed, while an obvious prolongation of I - II interwave latency with the decrease of waves II to IV in amplitudes was observed in ABR. Because the changes in wave I and wave IV were easily identifiable than the unstable waves II and III, we used the amplitudes of waves I and IV as the parameters to reflect the changes in ABR. Fig. 10 shows the time courses of CoBF and the amplitudes of waves I and IV from the type III animals (n = 9). About 10% increase in CoBF occurred during compression. In ABR, remarkable decrease in the amplitude of wave IV was detected compared to the unchanged wave I. Comparison of the average changes in CoBF, wave I and wave IV of each animal during compression shows that the decrease of the amplitudes of wave IV had the widest individual variations (Fig. 11). No decrease in CoBF was found in these animals. In addition, there was no significant correlation between the increase in CoBF and the decrease in wave IV.
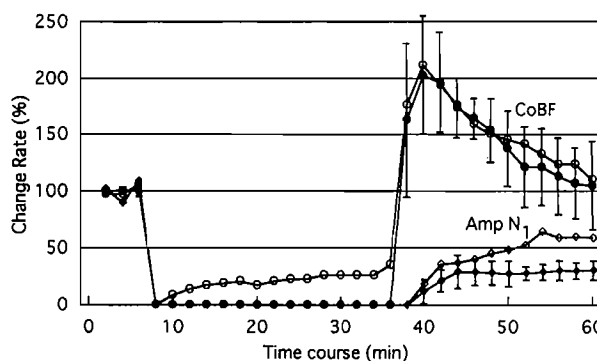

Fig.8 Recovery of N₁ after transient occlusion of CoBF. Occlusion duration was 5 min (n=5), 16 min (n=6) and 30 min (n=4). CoBF (filled circle) and the amplitudes of N₁ (filled square) in each group are expressed as M ± SD. Recovery degrees of N₁ after occlusion duration of 5, 16 and 30 min were about 105%, 80% and 30% of the original level respectively. Recovery degrees of N₁ after occlusion show a significant difference between the groups.

Fig.9 Example of recovery process of N₁ after incomplete decrease in CoBF. Occlusion duration was 30 min. Filled circle and square indicate CoBF and the amplitude of N₁ in the group with complete decrease in CoBF (M ± SD) (see Fig. 8); open circle and square indicate CoBF and the amplitude of N₁ in an animal with incomplete decrease in CoBF. In the former, N₁ recovered to only 30% of the original level, but in the latter, N₁ recovered to about 60% of the original level.
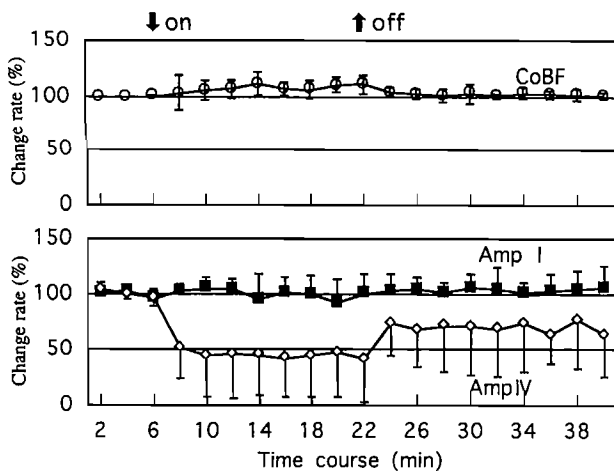
Fig.10 Time courses of CoBF and the amplitudes of wave I and wave IV in type III animals (n=9). CoBF shows the increase rather than the decrease. Wave I had no obvious decrease, while wave IV show the remarkable decrease.
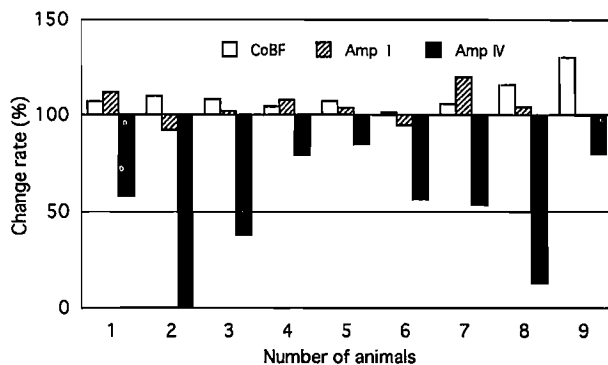

Fig.11 The average changes in CoBF, the amplitudes of wave I and IV during compression in type III animals (n=9). All animals show no obvious decrease in CoBF and the amplitude of wave I. The decrease in wave IV shows wide individual variations.

The results show that the change in CoBF is independent of wave IV.

DISCUSSION

It is well known that each wave of ABR corresponds to a specific brain stem nucleus. Wave I originates from the auditory nerve and wave II originates from the cochlear nucleus[18-20]. Similarly, the origin of N_1 and N_2 in CAP is located at the region of the habenulae perforata, and the medial cochlear nerve and the cochlear nucleus respectively[21-23]. Due to the changes in CAP and ABR, the location of the damage in the auditory pathway could be distinguished. For example, the prolongation of I - II interwave latency reflects conductive blockage of the cochlear nerve[24-27], whereas the disappearance of N_1 or wave I may be attributed to the cochlear dysfunction. Several authors[28-32] have described the changes in CAP and ABR following compression or retraction of the BNV at the cerebellopont-

ine angle. These studies[29-31] show that the more the pressure masses were used, the more the deterioration of CAP, ABR was found. In the sequential changes in CAP and ABR, N_1-N_2 interwave delay or I - II interwave delay was identified earlier. With progression, N_1 and wave I became obliterated. Finally, all waves of CAP and ABR disappeared. Although CoBF was not measured in these studies, the authors[29-31] speculated that the disappearance of CAP and ABR was attributed to the obstruction of the internal auditory artery. From these sequential changes, the cochlear nerve seems more susceptible to damage than the artery following compression or retraction. By observing the changes in CoBF combining with CAP and ABR, we found that both changes in CoBF and acoustically evoked responses coexist in compression of the BNV, and documented that the disappearance of CAP and ABR is associated with the decrease in CoBF. It is obvious that the changes in type I, type II and type III observed in this study represented the damage to the internal auditory artery, both the artery and the cochlear nerve, and the cochlear nerve respectively. Moreover, we found that the damage to the cochlear nerve and the artery were closely related to the compression positions rather than the pressure masses. Compression at the anterior pressure point easily injured the cochlear nerve, whereas compression at the posterior pressure point of the BNV induce the occlusion of the internal auditory artery resulting in cochlear ischemia.

Brown et al[33] demonstrated that N_1 is more sensitive to hypoxia among the cochlear potentials. In the present study, we found that the alteration in N_1 is due to the decrease in CoBF, and that the pressure on the cochlear nerve (expressed as I - II interwave delay or N_2 latency delay) could not affect N_1. The results were consistent with previous investigations which showed no obvious change in N_1 even after dissection of the cochlear nerve[21-23]. For this reason, we used N_1 not N_2 as the parameter to evaluate the effect of cochlear ischemia on the auditory function. As shown in Fig. 7, a significant correlation between CoBF and N_1 was found. With the reduction of CoBF, the amplitude of N_1 decreases. As CoBF reduced to as low as 70% of the original level, N_1 deteriorated and disappeared. In other words, the loss of N_1 did not always represent complete occlusion of CoBF.

There is some controversy on the recovery of the cochlear potentials after transient occlusion of CoBF[34-37]. We found that complete recovery of N_1 is difficult as the ischemic duration lasted 15 min, while Fuse[34] reported that ABR could completely recover even after the loss of ABR over 60 min caused by vertebrobasilar ischemia. The differences in recovery may reflect the degree of decrease in CoBF during ischemia because the N_1 recovery between complete occlusion and incomplete occlusion of CoBF show the obvious difference. The results presented here suggest that the changes in CAP and ABR are only an indirect parameter reflecting cochlear ischemia. As CAP and ABR were used to monitor auditory function during operation of

hearing preservation in acoustic neuroma[38-40] and vertebrobasilar occlusion[34,41], one must keep in mind that the loss of all waves of CAP and ABR is not always related to complete occlusion of CoBF. It is proposed that if the absence of N_1 were caused by partial decrease in CoBF, the recovery of auditory function will be better than those of complete decrease in CoBF.

Based on the arrangements of the cochlear nerve and the internal auditory artery at the entrance of the internal acoustic meatus, we could select the pressure point to block the cochlear nerve without cochlear ischemia. The changes in ABR as shown in type III are usually found in acoustic neuroma and mainly attributed to the conductive damage of the cochlear nerve[24-26]. However, the enlargement of the tumor could invade not only the cochlear nerve but also the arteries[1,2,42]. In the present study, the changes in CoBF were detected in all animals following compression of the BNV. It proved that the disorder in circulation of the inner ear is an important factor resulting in cochlear dysfunction. The different patterns of changes in CoBF associated with different pressure points may reflect the degrees of obstruction of the internal auditory artery. Clearly, the decrease in CoBF abolish cochlear potentials and results probably in histopathological changes[36,43]. The increase in CoBF is considered as a response to the disturbance in circulation of the inner ear induced by partial obstruction of the internal auditory artery. Through autoregulation, the blood supply to the inner ear may be maintained, but this situation is thought to be easily damaged by any stress such as vasospasm. Once the balance of circulation in the inner ear were destroyed, the hearing loss will be brought about. This mechanism may be contributed to acoustic neuroma presenting as sudden deafness.

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