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RESEARCH ARTICLE

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Saccular and utricular inputs to sternocleidomastoid motoneurons of decerebrate cats

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Abstract Connections from the otolithic organs to sternocleidomastoid (SCM) motoneurons were studied in 20 decerebrate cats. The electrical stimulation was selective for the saccular or the utricular nerves. Postsynaptic potentials were recorded from antidromically identified SCM motoneurons; these muscles participate mainly in neck rotation and flexion. Partial transections of the brainstem at the level of the obex were performed to identify the possible pathway from the otolithic organs to the SCM motoneurons. Saccular or utricular nerve stimulation mainly evoked inhibitory postsynaptic potentials (IPSPs) in the ipsilateral SCM motoneurons. Some of the sacculus-induced IPSPs were preceded by small-amplitude excitatory PSPs (EPSPs). The latencies of the PSPs ranged from 1.8 to 3.1 ms after saccular nerve stimulation and from 1.7 to 2.8 ms after utricular nerve stimulation, indicating that most of the ipsilateral connections were disynaptic. In the contralateral SCM motoneurons, saccular nerve stimulation had no or faint effects, whereas utricular nerve stimulation evoked EPSPs in about two-thirds of neurons, and no visible PSPs in about one-third of neurons. The latencies of the EPSPs ranged from 1.5 to 2.0 ms, indicating the disynaptic connection. Thus, the results suggest a difference between the two otolithic innervating patterns of SCM motoneurons. After transection of the medial vestibulospinal tract (MVST), saccular nerve stimulation did not evoke IPSPs at all in ipsilateral SCM motoneurons, but some (11/40) neurons showed small-amplitude EPSPs. Most (24/33) of the utricular-activated IPSPs disappeared after transection, whereas the other 9 neurons still indicated IPSPs. In the contralateral SCM motoneurons, no utricular-activated EPSPs were recorded after transection. These MVST transection results suggest that most of the otolith-SCM pathways are located in the MVST at the obex level. However, the results also suggest the possibility that other otolith-SCM pathways exist at the obex level.

Key words Vestibulocollic reflex · Saccular nerve · Utricular nerve · Sternocleidomastoid motoneuron · Cat

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Introduction

Saccular and utricular receptors sense linear accelerations. They detect the direction of the gravity or linear accelerations in each optimal plane and send this information to the central nervous system. The inputs from these receptors reach neck motoneurons in the upper cervical cord, forming the sacculo- or the utriculocollic reflex arc. The reflexes also contribute to the fixation of the image on the retina by activating the appropriate neck muscles (Wilson and Melvill Jones 1979).

Techniques for the selective stimulation of the saccular nerve (Uchino et al. 1997a) or the utricular nerve (Sasaki et al. 1991) have enabled the detection of input patterns to neck and ocular motoneurons innervating several muscles. Stimulation of the saccular nerve evoked disynaptic excitatory postsynaptic potentials (EPSPs) in ipsilateral neck extensor motoneurons, di- or trisynaptic EPSPs in contralateral neck extensor motoneurons, di- and trisynaptic inhibitory postsynaptic potentials (IPSPs) in ipsilateral neck flexor motoneurons and trisynaptic IPSPs in contralateral neck flexor motoneurons (Uchino et al. 1997a). In contrast, utricular nerve stimulation evoked disynaptic EPSPs in ipsilateral neck extensor motoneurons, trisynaptic IPSPs in contralateral neck extensor motoneurons (Bolton et al. 1992), disynaptic EPSPs in ipsilateral neck flexor motoneurons and trisynaptic IPSPs in contralateral neck flexor motoneurons (Ikegami et al. 1994). Thus, the sacculo-neck pathway has a bilateral symmetric organization, while the utriculo-neck pathway has an essentially left-right asymmetric organization (Uchino 1997). These previous

studies revealed the sacculo- and utriculo-neck connections focusing on extensor muscles and flexor muscles. Less information has been obtained about the muscles that participate in neck rotation, e.g., the sternocleidomastoid (SCM) muscle.

A clinical test of vestibular function using loud click stimulation has been reported (Colebatch and Halmagyi 1992). Loud clicks evoke typical myogenic potentials in the SCM muscle even among patients with hearing loss. Since this stimulation seems to affect the saccular nerve, these potentials may reflect the function of a sacculo-SCM pathway. However, the connectivities of such a pathway are not known. The purpose of the present study was to elucidate synaptic connectivities from the saccular and utricular receptors to the SCM motoneurons of the decerebrate cat.

Materials and methods

General procedures

Successful experiments were performed on 20 adult cats. The animal care in these experimental procedures followed the guidelines of the Physiological Society of Japan, 1988. All cats were initially anesthetized with ketamine hydrochloride (Ketalar, 30–50 mg/kg intramuscularly) followed by halothane and nitrous oxide mixture inhalation via artificial ventilation after a tracheotomy was performed. Blood pressure was monitored from the femoral artery. When necessary, 5–10% glucose was infused through the femoral vein and maintained at about 120 mmHg. The rectal temperature was kept at about 37°C with a servo-controlled heating pad. The end-tidal CO_2 was monitored and maintained at about 4–5%. The cat was suspended from hip pins and by a clamp on the spinal process of caudal cervices. Decerebration was done at the precollicular level in the last stage of the surgery. For stable recording, a bilateral pneumothorax operation was performed, and pancuronium bromide (Mioblock, 0.25–0.5 mg/kg per hour) was administered by the intravenous route. The animals were given artificial respiration using positive pressure.

Stimulating procedure

The left inner ear was opened through the ventrolateral aspect to expose the saccular nerve or the utricular nerve. Other branches of the vestibular nerve were resected. A bipolar silver electrode (insulated except for ~0.5 mm at the tip; interelectrode distance ~0.8 mm) was inserted into the saccular nerve or the utricular nerve as described (Sasaki et al. 1991). In four animals, we placed electrodes on the saccular and utricular nerves separately. Fluid from the inner ear was drained using twisted cotton, and all nerves and tips of electrodes were then covered with a semisolid paraffin-Vaseline mixture to prevent current spread during stimulation and also to avoid drying. Each electrode was fixed to the occipital bone with dental cement. The ipsi- and/or contralateral nerve branches innervating the SCM muscle were prepared for antidromic stimulation to identify motoneurons by using tunnel electrodes.

Recording and histological procedure

Access to the vestibular complex was gained by resecting the portion of the occipital bone which overlies the fourth ventricle and by removing the caudal part of the cerebellum until the floor of the fourth ventricle appeared. Vestibular field potentials were recorded from the vestibular nuclei with a glass micropipette containing 2 M NaCl saturated fast green dye with a resistance of 1–2 MΩ.

Dorsal laminectomy was performed at the C1–2 segments. A glass micropipette containing 2 M potassium citrate with a resistance of 5–10 MΩ was inserted into the SCM motoneuron located in the C1 and upper part of the C2 segments (Rapoport 1978; Holomanova et al. 1973), and then the PSPs elicited by the saccular or the utricular stimulation were recorded.

Transection of the brainstem near the midline was performed in six experiments in an attempt to elucidate the location of the descending axons. After recording the control PSPs in SCM motoneurons in the intact state, a fine blade (2.5 mm wide) was inserted from the surface into the medial vestibulospinal tract (MVST) at about the obex level, by using a manipulator under visual observation. The intracellular recording was then repeated to detect the input difference between before and after the transection. When the experiment was finished, an overdose of anesthetic was administered, and the brainstem was removed and fixed with 10% formalin. After 3–5 days of fixation, the brain was cut into 100-µm-thick serial transverse sections on a freezing microtome, and the sections were Nissl stained to confirm the transecting lesion.

Results

Field potentials in the vestibular nuclei and SCM motoneurons

Before every experiment, to assess the validity of the selective stimulation, we recorded the first negative (N1) field potentials evoked by the saccular or utricular nerve stimulation in the ventral part of the vestibular nuclei, according to the method of Precht and Shimazu (1965). The threshold for N1 potentials (N1T) ranged from 8 to 40 µA (16.6±10.5 µA, mean±SD) with saccular nerve stimulation and from 3 to 30 µA (11.4±9.2 µA, mean ±SD) with utricular nerve stimulation. The latencies of the N1 potentials ranged from 0.7 to 1.3 ms (0.90±0.17 ms, mean±SD) with saccular nerve stimulation and from 0.8 to 1.4 ms (0.94±0.16 ms, mean±SD) with utricular nerve stimulation. These values are equivalent to previous results (Bolton et al. 1992; Ikegami et al. 1994; Uchino et al. 1997a). In the majority of experiments, the N1 field potentials increased as the stimulus intensity was increased, then reached a plateau at the stimulus intensity of approximately 5–7×N1T, as observed by Uchino et al. (1997a). The plateau presumably indicates the maximal activation of the saccular nerve or utricular nerve and no current spread to other vestibular branches. In the present study, intracellular recordings were usually made by stimulating the saccular or utricular nerve with a stimulus intensity of less than 5–7×N1T. If a response was not seen, supramaximal stimulation with double or triple shocks was applied to the saccular or utricular nerve to confirm the absence of response.

We recorded 142 SCM motoneurons intracellularly in the area of C1 and rostral half of C2 (81 ipsilaterally and 61 contralaterally). The resting membrane potentials of recorded cells were usually lower than –35 mV. We omitted the cells with unstable or unsatisfactory resting potentials.

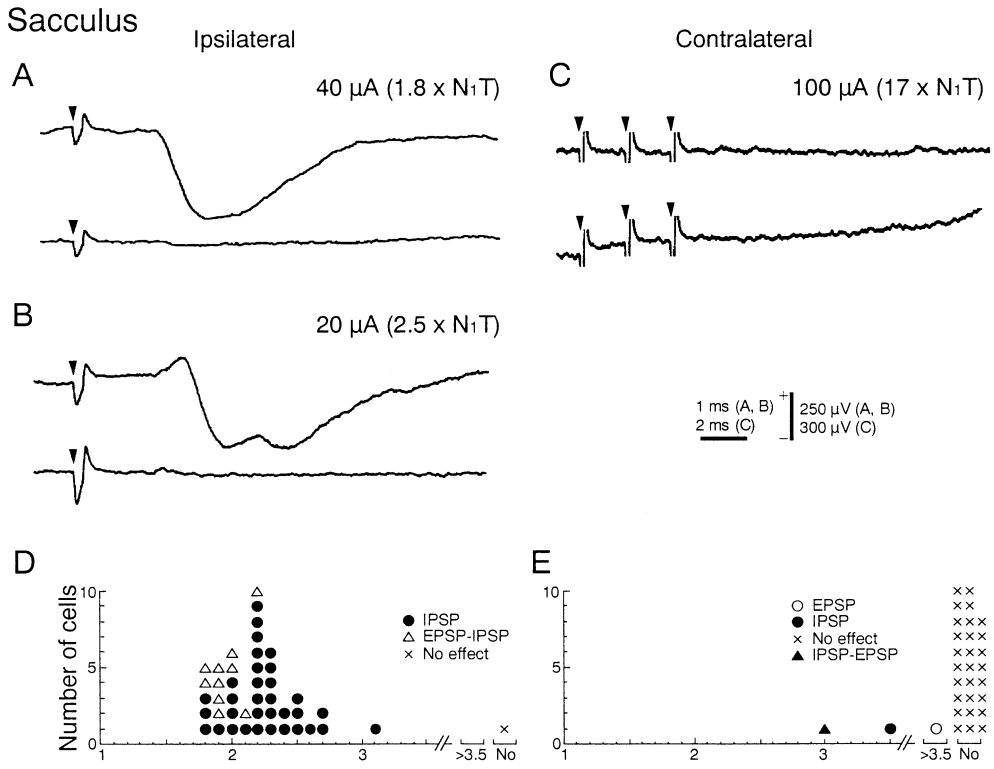

Fig. 1A–E Effects of saccular nerve stimulation on ipsi- and contralateral SCM motoneurons. One typical waveform of a sacculus-evoked IPSP **(A)** and an EPSP-IPSP sequence **(B)** in ipsilateral SCM motoneurons, and an example of no visible potential **(C)** recorded from a contralateral SCM motoneuron are shown. The input patterns of all records and the latency histogram of ipsi- **(D)** and contralateral **(E)** SCM motoneurons is shown. *The upper traces in* **A–C** are from intracellular recordings, and *the lower traces* are from outside adjacent to the neurons of *the upper traces. The filled triangle on each trace* shows the onset of stimulation

Postsynaptic potentials in SCM motoneurons following saccular nerve stimulation

Saccular nerve stimulation evoked inhibitory postsynaptic potentials (IPSPs) in all but one (43/44) ipsilateral SCM motoneuron (the other neuron had no visible PSP). The nature of the IPSP was confirmed in three neurons with stable membrane potentials by the current injection test. A typical example of this IPSP, shown in Fig. 1A, was induced by the stimulus intensity of 1.8×N1T. Some (10/43) IPSPs were preceded by a small-amplitude EPSP (Fig. 1B). The amplitudes of these EPSPs never exceeded those of the IPSPs, and no IPSP-EPSP sequence was seen. The latencies of the IPSPs and the EPSP-IPSP sequence were mainly in the range 1.8–2.7 ms (Fig. 1D), indicating disynaptic connections (Uchino et al. 1997a). Only one IPSP had a latency of 3.1 ms. In the contralateral SCM motoneurons, the majority (28/31) did not show visible responses after saccular nerve stimulation. The wave shown in Fig. 1C is typical; the motoneuron did not show any response to saccular nerve stimulation even by repetitive and supramaximal (17×N1T) stimuli. Only three neurons showed late (≥3.0 ms) and faint PSPs (Fig. 1E).

Postsynaptic potentials in SCM motoneurons following utricular nerve stimulation

Utricular nerve stimulation evoked predominantly IPSPs (33/37) in ipsilateral SCM motoneurons; a typical example is shown in Fig. 2A. The nature of the IPSP was confirmed in three neurons with stable membrane potentials by the current injection test. The latencies of the IPSPs (1.7–2.8 ms) (Fig. 2C) were in the disynaptic range (Bolton et al. 1992; Ikegami et al. 1994). The other four neurons did not show any visible PSPs even at the supramaximal stimulus intensity. Of the contralateral SCM motoneurons, the majority (21/30) showed EPSPs (Fig. 2B). The latencies of EPSPs ranged from 1.5 to 2.0 ms (Fig. 2D), presumably indicating disynaptic connections from the utriculus to contralateral SCM motoneurons. The other nine neurons did not respond to the utricular nerve stimulation.

As for no-input neurons after the utricular nerve stimulation, all four ipsilateral and four of the nine contralateral no-input neurons were recorded from one cat (cat 10). In cat 10, even though vestibular field potentials were recorded as usual and SCM motoneurons were identified in the same manner, no PSPs were recorded from bilateral SCM motoneurons after utricular nerve stimulation.

Transecting effects on otolith-induced postsynaptic potentials

We tried to determine the pathways mediating sacculo- and utriculo-SCM motoneurons in six cats (five for the ipsilateral IPSPs induced by saccular and utricular nerve stimulation, one for the contralateral EPSPs induced by utricular nerve stimulation) by transecting at about the obex level, at the mid-portion in laterality. The lesions in transverse sections and PSPs before and after the tran-

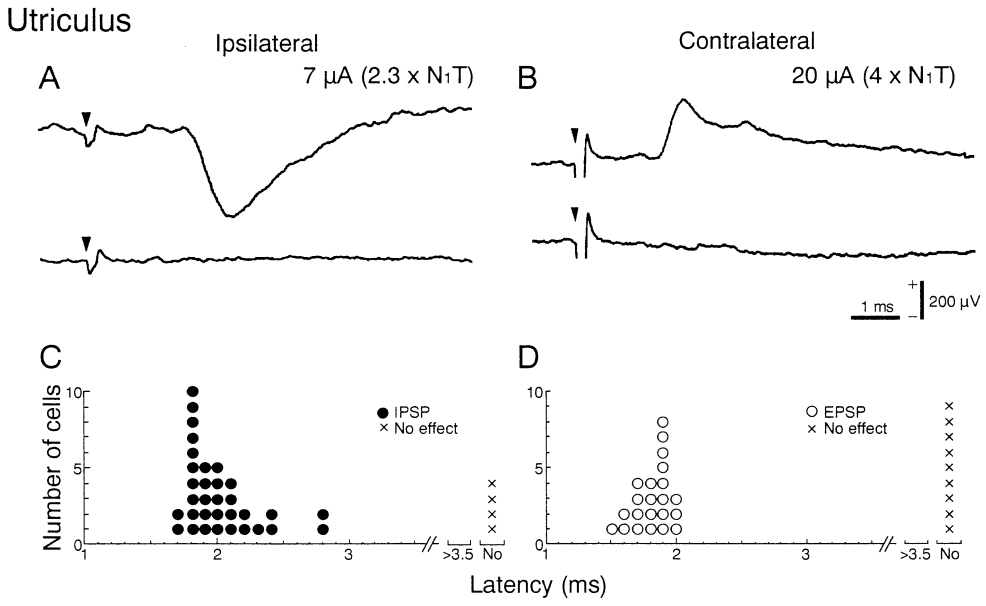

Fig. 2A–D Effects of utricular nerve stimulation on ipsi- and contralateral SCM motoneurons. One typical waveform of a utriculus-evoked IPSP **(A)** in an ipsilateral SCM motoneuron, and an EPSP **(B)** recorded from a contralateral SCM motoneuron are shown. The input patterns of all records and the latency histogram recorded in ipsi- **(C)** and contralateral **(D)** SCM motoneurons are shown. *The upper traces in* **A** *and* **B** are from intracellular recordings, where lower traces are from outside adjacent to the neurons of *the upper traces*. *The filled triangle on each trace* shows the onset of stimulation

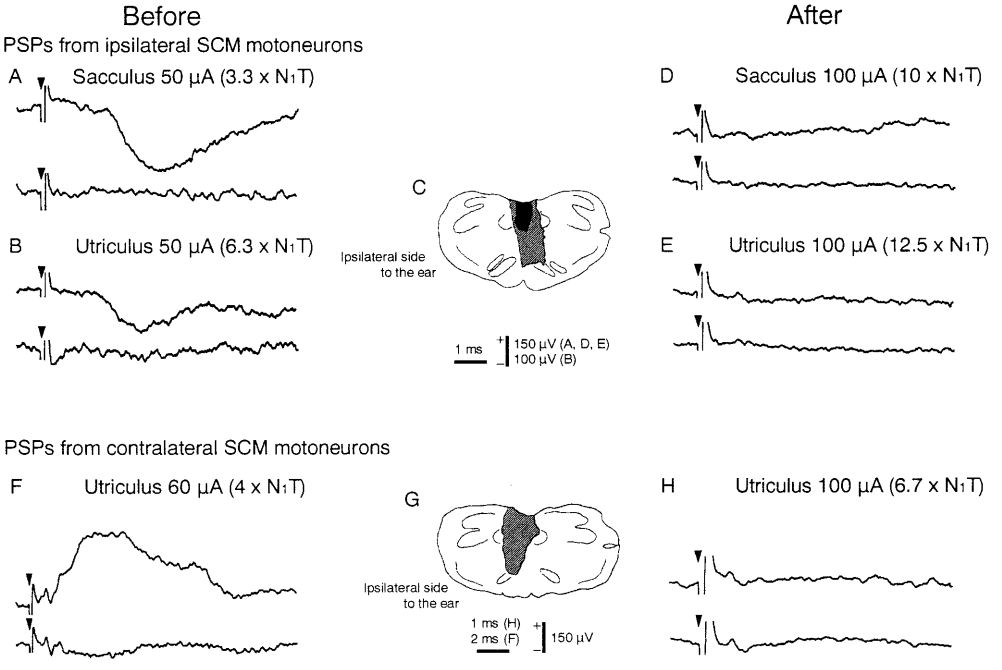

Fig. 3A–H Results from transecting experiments for the detection of otolith-SCM pathways. Postsynaptic potentials (*PSPs*) recorded in ipsilateral SCM motoneurons before **(A, B)** and after **(D, E)** the transection. These PSPs were obtained from one cat, and the transection was done as shown in **C** (*hatched area*). *The darker area in* **C** indicates the overlap portion of the lesion in five experiments for ipsilateral otolith-SCM pathways. The PSPs recorded in contralateral SCM motoneurons before **(F)** and after **(H)** the lesion **(G)** obtained from another cat are shown

section are shown in Fig. 3. The data in Fig. 3A–E were obtained from one cat, and those shown in Fig. 3F–H were obtained from another cat.

In the intact state, saccular nerve stimulation evoked IPSPs (Fig. 3A) in all (12/12) recorded ipsilateral SCM motoneurons, some (4/12) of which were preceded by small-amplitude EPSPs. After blade insertion (Fig. 3C, hatched portion), the majority (29/40) of neurons showed no visible potentials (Fig. 3D), while the other 11 showed small-amplitude EPSPs (not shown). IPSPs were not recorded in any of the neurons tested. The darker portion in Fig. 3C indicates the commonly sectioned part in all five preparations. Since sacculus-induced IPSPs disappeared after blade insertion in all experiments, the commonly sectioned part (Fig. 3C, darker portion) may be the main pathway for the sacculus-induced IPSPs to ipsilateral SCM motoneurons. The result that small-amplitude EPSPs remained indicates that this pathway may be separate from the pathway of IPSPs at the obex level, but this was not confirmed.

Utricular nerve stimulation evoked IPSPs (Fig. 3B) in all (8/8) ipsilateral SCM motoneurons before transection. After transection (Fig. 3C, hatched area), most (24/33) of the recorded neurons showed no visible potentials (Fig. 3E), and the other 9 neurons showed IPSPs with disynaptic latencies (1.7–2.7 ms). This result suggests that most of the signals of utriculus-induced IPSPs pass through the portion around the midline

(hatched area in Fig. 3C) at least at the obex level. However, the pathways of the other nine neurons were not clarified.

In all (5/5) contralateral SCM motoneurons, utricular nerve stimulation induced EPSPs (Fig. 3F) in the intact state. After blade insertion (Fig. 3G), no visible PSPs (Fig. 3H) were recorded in any (10/10) of the contralateral SCM motoneurons. This result suggests that the excitatory pathway from the utriculus to contralateral SCM motoneurons is around the midline in the transverse plane at the obex level.

Discussion

This study demonstrated the presence of disynaptic pathways from the sacculus and the utriculus to SCM motoneurons. The main results are summarized in Fig. 4. Stimulation of the saccular nerve evoked mainly disynaptic IPSPs, and almost no effects contralaterally. Utricular nerve stimulation induced disynaptic IPSPs and EPSPs in ipsi- and contralateral SCM motoneurons, respectively. We surmise that these signals pass mainly through the middle portion of the brainstem at the obex level.

Current spread to other receptors

The problem of current spread to other nerve branches always accompanies the selective stimulation method. We confirmed that there was no current spread to other vestibular end organs or branches as described below. As to stimulus intensity, the stimulation we used was always below the intensity that exceeds the plateau (maximal activation of the vestibular nerve branch) in the stimulus response curve of N1 field potentials. The possibility of current spread to other receptors (especially semicircular canals) should be considered. The connections between the semicircular canal and SCM motoneurons have been investigated by previous studies (Fukushima et al. 1979; Shinoda et al. 1994a), which showed stimulation of all three semicircular canal nerves evoked disynaptic IPSPs ipsilaterally and disynaptic EPSPs contralaterally. In our experiments, saccular nerve stimulation evoked almost no responses in contralateral SCM motoneurons even at the supramaximal stimulus intensity, which was different from the EPSPs after stimulation of the semicircular canal nerves. This difference indicates that the current that was applied to the saccular nerve did not spread to the semicircular canal nerves. Regarding utricular nerve stimulation, the input patterns observed in the present study, i.e., IPSPs ipsilaterally and EPSPs contralaterally, are the same as semicircular canal-induced PSP patterns (Fukushima et al. 1979; Shinoda et al. 1994a). To clarify the input distinction between utriculus-induced PSPs and semicircular-canal-induced PSPs, we intentionally applied supramaximal stimulation to the utriculus in some SCM motoneurons in order to produce additional EPSPs or IPSPs of current spread effects that are presumably attributable to semicircular canals. In some cases, we confirmed additional components of EPSPs or IPSPs that may be attributable to semicircular canal inputs at the supramaximal intensity around 200 to ~500 μA. These components usually had shorter latencies (1.0 to ~1.4 ms), similar to previous results in neck motoneurons (Shinoda et al. 1994b; Isu et al. 1988), compared to those of sacculus- or utriculus-induced PSPs (≥1.5 ms).

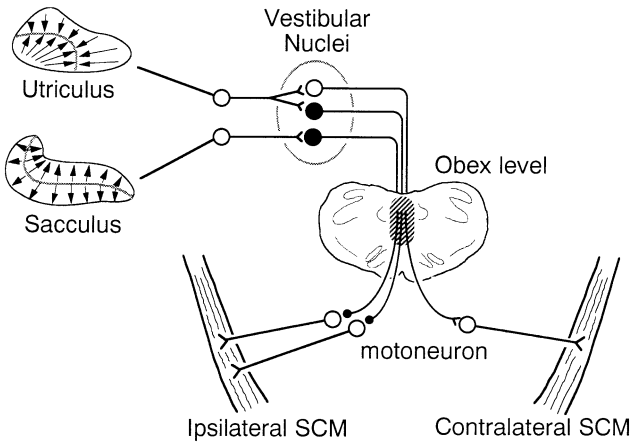

Fig. 4 Schematic diagram of the sacculo- and utriculosternocleidomastoid pathways. *Filled neurons* are inhibitory, and *the open ones* are excitatory. *The hatched portion* indicates the main pathway of the otolith-SCM connection

The possibility of current spread between the sacculus and the utriculus remains. However, previous results obtained using our method showed different inputs to the neck motoneurons after selective saccular or utricular nerve stimulation (Bolton et al. 1992; Ikegami et al. 1994; Uchino et al. 1997a). Thus, the problem of current spread between two otolith receptors can be excluded.

Vestibular evoked myogenic potentials

It is known that vestibular (especially saccular) function is tested by using the loud click stimulation (Colebatch and Halmagyi 1992). In normal subjects, the loud click stimulation evokes a typical waveform (p13–n23 response) of the averaged unrectified electromyogram (EMG) in the voluntarily activated ipsilateral SCM muscle. Halmagyi et al. (1994) showed the inhibition of unit activities of the SCM muscle in the period 8–16 ms after the stimulation, and proposed that the p13–n23 response in the EMG was due to this inhibition. This p13–n23 response was only seen ipsilaterally, and is believed to be produced by direct mechanical activation of the vestibular end organs, mainly the sacculus. In agreement with their studies, we found the saccular-SCM pathway was mainly composed of ipsilateral disynaptic inhibition.

Results of electrical stimulation

The selective stimulation technique has been applied to the saccular and utricular nerves, and their short-latency reflex arcs have been revealed in neck extensor and neck flexor muscle motoneurons of the cat (Bolton et al. 1992; Ikegami et al. 1994; Uchino et al. 1997a). Saccular nerve stimulation facilitates bilateral neck extensor motoneurons and inhibits bilateral neck flexor motoneurons. Utricular nerve stimulation facilitates ipsilateral neck extensor and flexor motoneurons, and inhibits contralateral neck extensor and flexor motoneurons. These connections are characterized by a bilateral symmetric organization in the saccular-originating and a left-right asymmetric organization in the utricular-originating reflex pathway. In the present study, the saccular nerve stimulation evoked IPSPs in ipsilateral SCM motoneurons. This exclusive unilateral connection is unique compared with previous results of sacculoextensor and sacculoflexor connections (Uchino et al. 1997a). In contrast, the utricular nerve stimulation induced IPSPs ipsilaterally and EPSPs contralaterally. A left-right asymmetric organization is also applicable in this case; however, as for input patterns, the ipsilateral IPSP and the contralateral EPSP are the opposite of previous results observed in neck extensor (Bolton et al 1992) and neck flexor (Ikegami et al 1994) motoneurons.

Functional considerations

The SCM muscle activities induced by a sudden abrupt fall were recorded in canal-plugged upright postured cats (Watt 1976) and in supine human subjects (Ito et al. 1995). These studies showed that the SCM muscle activities commenced as early as 15 ms (canal-plugged cats) or 22–25 ms (human subjects) after the onset of the sudden release, and they concluded that the muscle excitation was probably due to the activation of a direct vestibulocollic pathway originating in otolithic receptors.

The electrical stimulation results in the present study indicate that saccular nerve stimulation mainly inhibits ipsilateral SCM muscle activity, which implies that the shortest sacculo-SCM pathway may contribute less to the muscle activation. However, our findings do not conflict with previous results, for Ito et al. (1995) suggested that SCM muscle activation is attributed to a second effect, namely the n23 effect (Colebatch and Halmagyi 1992; Halmagyi et al. 1994) after the initial effect, which was named p13. Thus, our finding of the sacculus-induced ipsilateral IPSP may prove to be the shortest linkage that may induce the p13 effect.

Utricular nerve stimulation evoked disynaptic IPSPs ipsilaterally and disynaptic EPSPs contralaterally. With the supine or upright posture, bilateral utriculi would be activated in the same manner. In such conditions, both ipsilateral disynaptic inhibitory and contralateral disynaptic excitatory inputs would create a conflict. To produce prominent SCM activation like that seen in the free-fall experiments, there may be an as yet unidentified neural connection that enhances the effect of contralateral facilitation.

One of the major differences between the natural and electrical stimulation of otolith receptors is the activated portion of the saccular or utricular macula. Natural stimulation activates one side of the macula divided by the striola and inhibits the other side, where our method applied the stimulation to both sides of the striola simultaneously. Thus, our method may make it difficult to interpret the functional meaning directly. However, the recent study by Uchino et al. (1997b) used a focal stimulating method in the saccular macula that applied the stimulation to the dorsal and the ventral part of the region across the striola separately. In that report, Uchino et al. found that single neurons in the vestibular nuclei are excited monosynaptically from one side of the striola, and simultaneously inhibited disynaptically from the other side of the striola. This means that such neurons emphasize the linear acceleration information from one side of the striola. If such neurons connect to neck motoneurons and contribute to the vestibulocollic reflex, the functional interpretation will be much clearer.

Otolith-SCM pathway

In light of the location of the lesion and the PSP differences between before and after the transection, the main pathway of sacculus- and utriculus-induced IPSPs to ipsilateral SCM motoneurons is thought to be via the MVST at the obex level. This is in agreement with pathways from the three semicircular canals to SCM motoneurons (Fukushima et al. 1979), in which all semicircular canal-induced PSPs pass through the medial longitudinal fasciculus (MLF), which includes the MVST. Although we performed the blade insertion in the same manner in all experiments, a few sacculus-induced EPSPs (the initial part of the EPSP-IPSP sequence in the intact state) or utriculus-induced IPSPs remained in some experiments. It is known that vestibulospinal axons project to the upper cervical spinal cord via multiple funicular paths (Donevan et al. 1990). Thus, there is no doubt that a small population of otolith-SCM pathways are scattered widely in the transverse plane.

Utriculus-induced EPSPs recorded in contralateral SCM motoneurons pass mainly around the midline and are thought to belong to the MVST. This is similar to IPSPs recorded ipsilaterally. This is also similar to the pathways of three semicircular canal-induced EPSPs to contralateral SCM motoneurons. This pathway probably crosses the midline around the obex and connects the SCM motoneurons located at the medial and dorsal portions of the ventral horn of C1 and C2 segments (Rapoport 1978; Holomanova et al. 1973).

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