Exp. Brain Res. 24, 75—87 (1975)

Experimental Brain Research

Functional Organization of Vestibulofastigial Projection in the Horizontal Semicircular Canal System in the Cat

N. Furuya, K. Kawano and H. Shimazu

Department of Neurophysiology, Institute of Brain Research, School of Medicine, University of Tokyo, Hongo, Tokyo (Japan)

Summary. Spike potentials of fastigial nucleus neurons were recorded extracellularly in decerebrate, unanesthetized cats. The neurons responding to head rotation in the horizontal plane with a type I fashion were located mainly in the middle and caudal regions of the fastigial nucleus. Three fourth of these fastigial type I neurons were antidromically activated by stimulation of the contralateral vestibular nuclei. These neurons were excited transsynaptically from the ipsilateral vestibular nerve or nuclei.

Intracellular recordings were made from those neurons which were located in the caudal half of the fastigial nucleus and were activated antidromically from the contralateral vestibular nuclei. Stimulation of the ipsilateral vestibular nerve produced EPSPs in these neurons with latencies of 1.0—6.6 msec. The shortest conduction time along primary vestibular afferents from the labyrinth to the ipsilateral fastigial nucleus was 0.7 msec. The EPSPs with the shortest latency of 1.0 msec were therefore postulated to be due to monosynaptic connections of primary vestibular afferents with fastigial neurons. Stimulation of ipsilateral vestibular nuclei also produced monosynaptic EPSPs in fastigial neurons. These EPSPs were facilitated by conditioning stimulation of the ipsilateral vestibular nerve, indicating the existence of polysynaptic activation of fastigial neurons from the ipsilateral vestibular nerve through the vestibular nuclei.

Key words: Fastigial nucleus neuron — Horizontal semicircular canal — Vestibulocerebellar input — Monosynaptic EPSP — Polysynaptic EPSP

Introduction

The fastigial nucleus has been reported to receive secondary vestibulocerebellar fibers (Brodal and Torvik, 1957; Carpenter *et al.*, 1959). With respect to primary vestibulocerebellar afferents, Carpenter (1960) described their termination in the fastigial nucleus, while Brodal and Høivik (1964) have found that a number of primary vestibulocerebellar fibers pass through the fastigial nucleus but have been unable to demonstrate termination of these fibers in the nucleus. Brodal (1972) mentioned, however, that negative evidence does not exclude the existence of primary vestibular fibers ending in the fastigial nucleus, since it is extremely difficult to verify the presence of terminal degeneration in an area where there are many degenerating fibers of passage. With Golgi methods Matsushita and Iwahori (1971) did not dare to conclude definitely regarding termination of primary

vestibular fibers in the fastigial nucleus. Thus, in anatomical studies, termination of primary vestibular fibers in the fastigial nucleus seems to be still an open question.

In physiological studies, Dow (1939) recorded action potentials in the fastigial nucleus in response to vestibular nerve stimulation. Arduini and Pompeiano (1957) found that cathodal polarization of the ipsilateral labyrinth resulted in an increased rate of firing of neurons in the rostral part of the fastigial nucleus. Using intracellular and extracellular recording from the fastigial nucleus, Precht and Llinás (1968) reported monosynaptic and polysynaptic activation of nuclear neurons from primary vestibular afferents.

As to the responses to natural stimulation of the labyrinth, it has been reported that neurons in the fastigial nucleus respond to tilt of the head in the cat (Ghelarducci, 1973; Ghelarducci *et al.*, 1974) or to angular acceleration of the head in the monkey (Gardner and Fuchs, 1975). In these studies neuronal organization between the labyrinth and the fastigial nucleus has not been analyzed.

In the present study we have attempted to locate the neurons in the fastigial nucleus which respond to activation of the ipsilateral horizontal semicircular canal by rotatory stimulation and to delineate the neuronal organization of the pathway from the labyrinth to the ipsilateral fastigial nucleus by intracellular and extracellular recording from fastigial neurons.

Methods

Preparation. Thirty-five adult cats were used. Under ether anesthesia a tracheal cannula was introduced and the animal was mounted on a stereotaxic frame placed on the turn table. Bipolar stimulating electrodes (fine Ag-AgCl wires insulated except for the spherical tips) were placed on the left vestibular nerve by a ventral approach as described by Shimazu and Precht (1965). On the right side bipolar Ag-AgCl electrodes were placed on the oval and round windows to stimulate the vestibular nerve with the receptors kept intact. Then, the animal was decerebrated at the intercollicular level. The occipital bone was removed to insert the stimulating and recording electrodes into the cerebellum and the vestibular nuclei. After ether was discontinued, the animal was immobilized by gallamine triethiodide (Flaxedil) under artificial respiration. Rectal temperature was kept at 37—38° C by a heating pad and the blood pressure at 120—140 mm Hg by intravenous injection of pressor agents (Carnigen, Hoechst and/or Effortil, C.H. Boehringer Sohn), when necessary.

Electric Stimulation. The vestibular nerve was stimulated by rectangular pulses of 0.1 msec in duration. The stimulus intensity was less than 3 times the threshold for the N_1 field potential recorded in the ipsilateral vestibular nuclei (Shimazu and Precht, 1966). For stimulation of the vestibular nuclei, the electrodes consisted of four tungsten wires, 20 μ in tip diameter, with insulation except for a length of 500 μ from the tip, arrayed rostrocaudally with an inter-electrode distance of 1.5 mm. Each electrode had an AC (50 Hz) resistance of approximately 50 KΩ. The ensemble of electrodes was inserted into the vestibular nuclei on both sides under guidance of the stereotaxic atlas (Berman, 1968). The locations of the stimulating electrodes were adjusted by recording the N_1 potential through each electrode. The vestibular nuclei were stimulated rostrocaudally by pairing adjacent electrodes.

Natural Stimulation. The head of the animal was placed in the prone position at the center of the turn-table. The plane of the stereotaxic frame was inclined 30° upward in order to place the horizontal canal approximately on the horizontal plane. Stimulation was applied by manual rotation of the turn-table or electrically controlled sinusoidal rotation. In the text type I or type II neuron always means those recorded extracellularly and identified by horizontal rotation.

Recording. For recording extracellular spike potentials from the fastigial nucleus, glass micropipettes filled with Ringer solution saturated by Fast Green FCF (a resistance of

4—6 MΩ) were inserted in the dorsoventral direction through the cerebellum. For intracellular recording, glass micropipettes were filled with 3 M KCl saturated by Fast Green or 2 M K citrate solution, the resistance being 20—40 MΩ. A conventional input stage was used for recording and passing currents through the microelectrode.

Histological Investigation. After extracellular or intracellular recording from single neurons, dye was ejected electrophoretically through the recording microelectrode (Thomas and Wilson, 1965). In the intracellular study, one or two spots were stained in each experiment and the locations of unstained recording sites were estimated by reading the scale of the micromanipulator with reference to the stained spots. After each experiment DC currents were passed through each electrode in the vestibular nuclei against the indifferent electrode connected with the temporal muscle. The brain was perfused by intracarotid injection of 10% formalin. The stained spots and the location of the stimulating electrodes in the vestibular nuclei were examined histologically in Nissl-stained serial sections.

Results

Responses to Horizontal Rotation of Neurons in the Fastigial Nucleus

Spikes of 510 neurons were recorded extracellularly in the whole area of the right fastigial nucleus. Of these, 50 neurons exhibited responses similar to those of vestibular type I neurons when the turn-table was rotated in the horizontal plane (Gernandt, 1949; Duensing and Schaefer, 1958; Shimazu and Precht, 1965); i.e., an increase in their discharge frequencies with ipsilateral angular acceleration and a decrease in frequencies with contralateral acceleration. These neurons will be called fastigial type I neurons. Their amplitudes were usually 0.3—1 mV with negative polarity. Only negative spikes were postulated to be recorded from fastigial neurons and not from primary or secondary vestibular axons (see below).

Figure 1A exemplifies the response of a fastigial type I neuron to sinusoidal oscillation of the turn-table. The sequential changes of spike frequencies caused by sinusoidal rotation were more clearly seen by averaging the response by an electronic computer (ATAC 501, Nihon Kohden) (Fig. 1B). Angular acceleration to the contralateral side by manual turning of the table decreased the spike frequency and a sudden stop of the table increased it as seen in vestibular type I neurons (Fig. 1C). In a few fastigial type I neurons, their discharge frequencies were markedly increased with ipsilateral acceleration and only slightly decreased with contralateral acceleration. Although the threshold for frequency response of each neuron was not determined accurately, it appeared to be higher than that of vestibular type I neurons (Shimazu and Precht, 1965).

For 29 of the 50 fastigial type I neurons, the sites of recording were stained by electrophoretic ejection of the dye from the recording electrode. Figure 2 represents the location of fastigial type I neurons projected to each section of a 10% step from the rostral to the caudal end of the fastigial nucleus. Despite overall exploration in the fastigial nucleus, most of type I neurons were found in its caudal half and only 3 neurons were identified as type I in its rostral half. In the medio-lateral extension they tended to be located in the medial and central region of the fastigial nucleus. For the remaining 21 fastigial type I neurons, their precise locations were not examined by staining, but were estimated by reading the scale of the micromanipulator with reference to the stained spots. These neurons were also estimated to be located mainly in the caudal half of the fastigial nucleus.

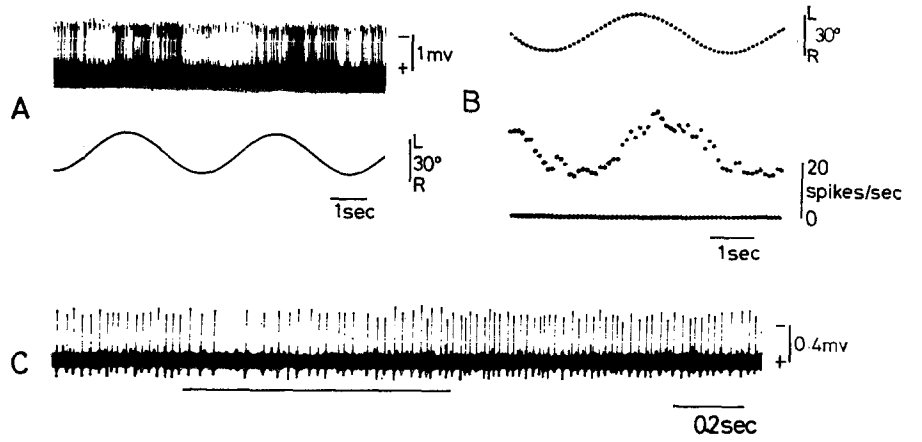

Fig. 1. Unit discharges of fastigial type I neurons in response to horizontal angular acceleration of the head. (A) Original record of unit spikes (top) and the signal indicating the position of the turn table (bottom) during sinusoidal oscillation. (B) Averaged response to sinusoidal oscillation of the same unit as in A (middle). The record was obtained with 100 sweeps. Top trace indicates the turn table position and the bottom trace the baseline for unit response. (C) Response of another fastigial type I neuron to contralateral acceleration. Horizontal bar below record indicates the period of manual rotation with rapid start and sudden stop

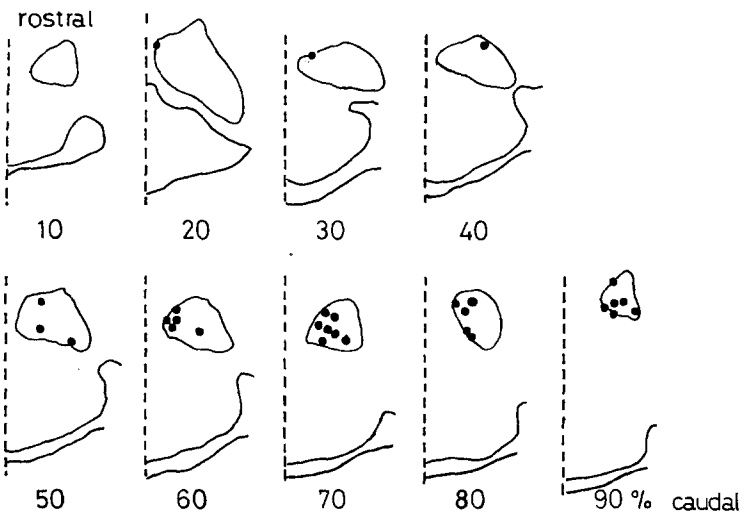

Fig. 2. Location of the fastigial type I neurons within the nucleus. Each drawing shows a section of a 10% step from the rostral to the caudal end of the nucleus

There was another type of fastigial neuron responding to horizontal rotation. The discharge frequencies of 12 neurons were increased with contralateral angular acceleration and were decreased with ipsilateral acceleration. These response characteristics are similar to those of vestibular type II neurons (Duensing and Schaefer, 1958; Shimazu and Precht, 1966). The locations of 7 fastigial type II

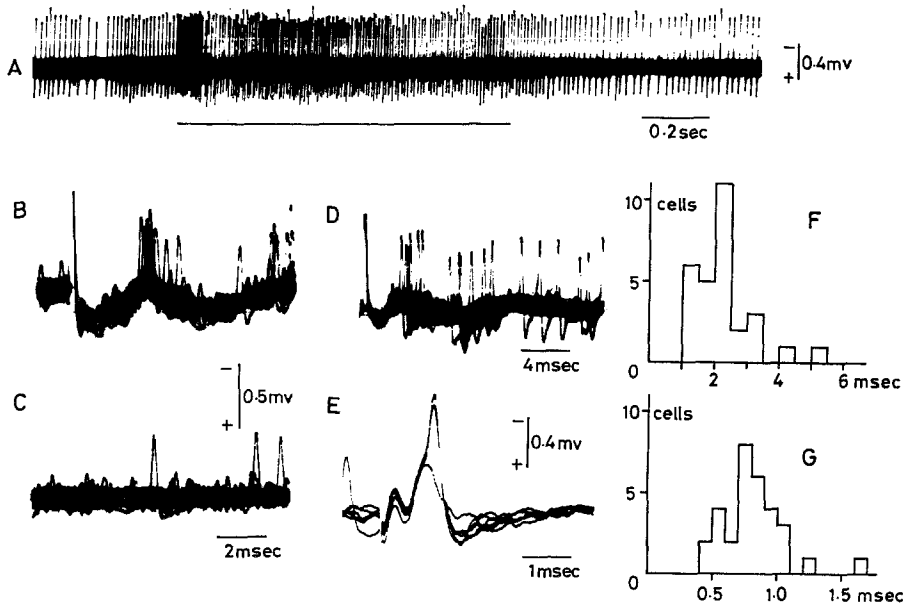

Fig. 3. Unit spikes of fastigial type I neurons in response to stimulation of the vestibular nerve and nuclei. Records A, D and E were obtained with the same neuron, and B and C were with another neuron. (A) Effect of ipsilateral vestibular nerve stimulation at 100/sec indicated by the horizontal line below the record. (B) Effects of single shocks to the ipsilateral vestibular nerve. (C) Spontaneous discharges without stimulation as a control for B. Records B and C were composed of 20 superimposed traces, respectively. (D) Effects of single shocks to the ipsilateral vestibular nuclei. (E) Antidromic responses to threshold-straddling stimulation of the contralateral vestibular nuclei. Records D and E were composed of 7 superimposed traces, respectively. (F) Latency histogram of spikes transsynaptically activated by stimulation of the ipsilateral vestibular nuclei. (G) Latency histogram of spikes antidromically activated from the contralateral vestibular nuclei

neurons were examined by marking the tip of the recording microelectrode. Six neurons were found in the rostral half of the fastigial nucleus and one was in its caudal region.

Orthodromic and Antidromic Activation of Fastigial Type I Neurons

Repetitive stimulation of the ipsilateral vestibular nerve at 50—100 Hz produced an increase in discharge frequency in all of 41 fastigial type I neurons examined (Fig. 3A). Although single shocks to the ipsilateral vestibular nerve were not consistently effective, spikes were clearly driven in 7 neurons with latencies of 1.7—6.0 msec (Fig. 3B).

In 29 out of the 41 fastigial type I neurons, spikes were evoked by single shocks to the ipsilateral vestibular nuclei with considerably fluctuating latencies (Fig. 3D), indicating that they were transsynaptically activated. In the remaining 12 neurons, effects of single shocks were not clearly detected because of their high frequency spontaneous discharges, though they were consistently excited by a repetitive pulse train applied to the ipsilateral vestibular nuclei. The shortest

latency of spikes evoked by single shocks to the ipsilateral vestibular nuclei was measured in each neuron and it ranged from 1.3 to 5.0 msec (Fig. 3F). Thresholds for orthodromic activation of individual fastigial type I neurons were determined with each stimulating electrode in the vestibular nuclei. They were as low as 20 μ A with single pulses of 0.1 msec duration. The locations of the electrodes having the lowest threshold were examined histologically after each experiment in five animals. They were located in the descending nucleus and the caudal half of the medial nucleus. Stimulation through electrodes located in the lateral or superior nucleus was found to be less effective.

Thirty-one out of the 41 fastigial type I neurons examined were antidromically excited from the contralateral vestibular nuclei. The spike potentials were evoked with fixed latencies, showed all-or-none responses to threshold-straddling stimulation (Fig. 3E) and followed double shocks with an interval of 1.6 msec. The latencies of antidromically evoked spikes ranged from 0.4 to 1.2 msec except for one neuron of which antidromic latency was 1.65 msec (Fig. 3G). The most effective site of stimulation in the vestibular nuclei for antidromic excitation of contralateral fastigial type I neurons depended on individual neurons and was found in each nucleus of the vestibular nuclear complex.

In summary, fastigial type I neurons receive excitatory inputs from the ipsilateral horizontal canal either directly or through the vestibular nuclei and most of them send their axons to the contralateral vestibular nuclei or further down to the deeper brain stem structures.

Intracellular Recording from Fastigial Neurons

In the first step of each experiment, an extracellular microelectrode was inserted into the right fastigial nucleus to find the most prevalent location of type I neurons. Then, with the aid of the micromanipulator which had been used for extracellular recording, an intracellular electrode was inserted into the left fastigial nucleus at the same rostrocaudal level. After the experiment the location of intracellular recording was marked by Fast Green and examined histologically.

In the present study intracellular recordings were made from those neurons which were located in the caudal half of the fastigial nucleus and activated antidromically from the contralateral vestibular nuclei. Thirty-one fastigial neurons impaled satisfied the above criteria which were in line with the characteristics of fastigial type I neurons as described above. Figure 4A exemplifies an antidromic activation after stimulation of the contralateral vestibular nuclei. The spikes had a fixed latency of 0.5 msec and followed double shocks at an interval of 1.8 msec with a more marked inflexion in the rising phase of the second spike. The latencies were measured in 24 neurons and they ranged from 0.45 to 1.3 msec (Fig. 4D). These values resembled well the latencies of antidromically excited fastigial type I neurons recorded extracellularly (Fig. 3G).

Stimulation of the ipsilateral vestibular nerve produced excitatory postsynaptic potentials (EPSPs) and, in some cases, spike potentials in these neurons (Fig. 4B). The amplitudes of the EPSPs attained 2—3 mV with maximal stimulation. The thresholds of vestibular nerve stimulation for production of the EPSPs were as low as 1.4 times the threshold for N_1 field potential in the ipsilateral vestibular nuclei. The latencies of the EPSPs were determined in 27 neu-

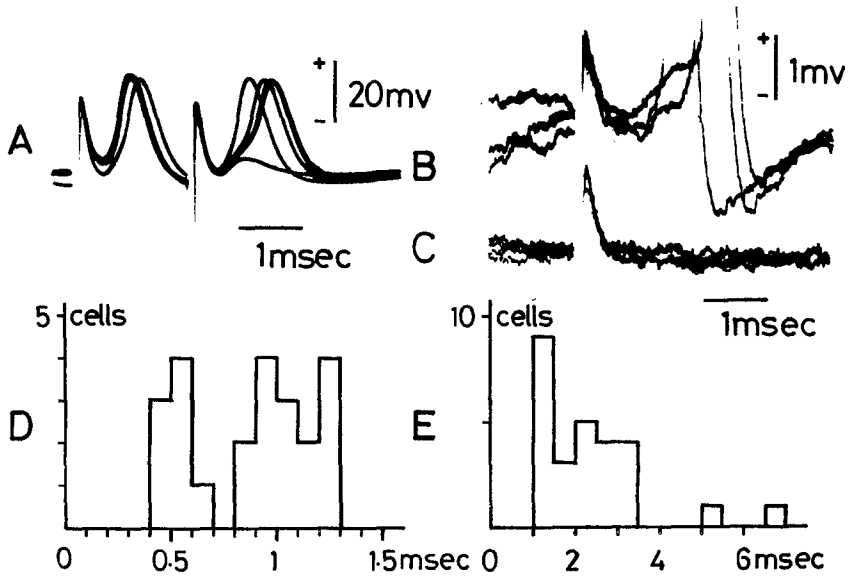

Fig. 4. Intracellular recording from neurons in the caudal part of the fastigial nucleus. (A) Antidromic activation from the contralateral vestibular nuclei. (B) Monosynaptic EPSPs and action potentials induced by stimulation of the ipsilateral vestibular nerve. Stimulation intensity was 1.8 times the threshold for N_1 potential. (C) Extracellular field potentials recorded outside the cell as a controll for B. (D) Latency histogram of intracellular spike potentials antidromically evoked by stimulation of the contralateral vestibular nuclei. (E) Latency histogram of EPSPs evoked by stimulation of the ipsilateral vestibular nerve

rons by subtracting the extracellular field potentials (Fig. 4C) from the intracellular records. They ranged from 1.0 to 6.6 msec after stimulation of the ipsilateral vestibular nerve (Fig. 4E).

When the microelectrode was advanced to the ventral part of the fastigial nucleus, a clear positive-negative field potential was recorded following stimulation of the ipsilateral vestibular nerve, the latency of the positive peak being 0.85 msec (Fig. 5E). This value may approximate the time of arrival of presynaptic impulses conducting along the primary vestibular fibers. The above explanation was supported by intraaxonal recording within the caudal half of the fastigial nucleus. Figure 5A—D exemplifies the axon spikes directly evoked by vestibular nerve stimulation. The positive spikes rose abruptly from the baseline without any prepotential, showed all-or-none response to threshold-straddling stimulation with fixed latencies (Fig. 5C and D) and followed double shocks with an interval of 1.5—2.0 msec (Fig. 5A and B). The latencies of the spikes ranged from 0.7 to 2.0 msec (Fig. 5F). Thus, it is postulated that the EPSPs with the shortest latency of 1.0 msec are produced monosynaptically from the vestibular nerve, if 0.3 msec are allowed for a single synaptic delay (Eccles, 1964).

Stimulation of the ipsilateral vestibular nuclei near the border between the medial and the descending nuclei produced EPSPs in these fastigial neurons (Fig. 6C). The latencies of the EPSPs ranged from 0.8 to 6.5 msec (Fig. 6G). When allowance was made for the conduction time between the vestibular nucle

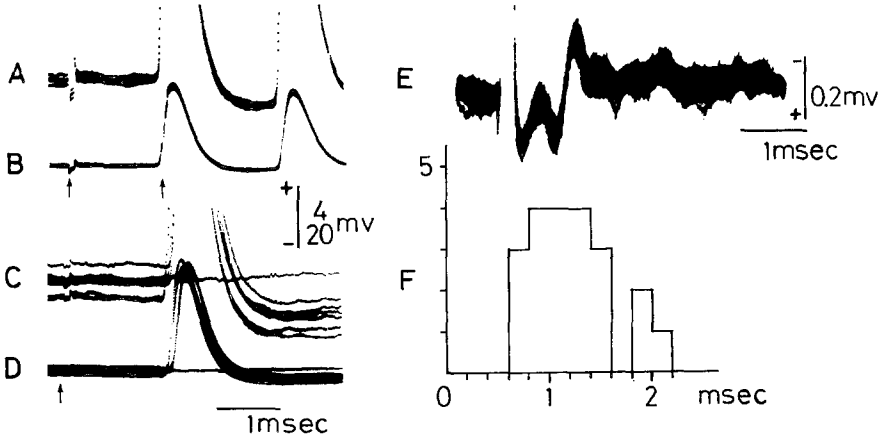

Fig. 5. Intraaxonal recording from primary vestibular fibers (A—D) and extracellular field potentials (E) in the fastigial nucleus in response to stimulation of the ipsilateral vestibular nerve. (A—B) Responses to double shocks. (C—D) Responses to threshold-straddling stimulation. Arrows indicate the time of stimulation. Calibration 4 mV applies to A and C (high gain records) and 20 mV to B and D (low gain records). (E) Field potentials recorded in the ventral part of the fastigial nucleus in response to stimulation of the ipsilateral vestibular nerve. (F) Latency histogram of axonal spikes recorded in the fastigial nucleus and evoked directly from the ipsilateral vestibular nerve

and the fastigial nucleus being around 0.5 msec (Shimazu and Smith, 1971), the value of 0.8 msec for the shortest latency of the EPSP indicates the existence of monosynaptic excitatory connection with the fastigial neurons from the ipsilateral vestibular nuclei.

Attempts were made to find whether the long latency EPSPs (2.0—6.6 msec in Fig. 4E) from the primary vestibular nerve are produced polysynaptically and, if so, where the interneurons are located. In Fig. 6A, a single shock was applied to the ipsilateral vestibular nerve at the stimulus intensity near the threshold for the EPSP. When the shocks were paired at the same intensity as in Fig. 6A, clear EPSPs were produced with the latency of 4.2 msec from the effective shock (Fig. 6B). This indicates that at least some of the long latency EPSPs are mediated through polysynaptic pathways rather than a monosynaptic, slowly conducting path.

Figure 6D—F exemplifies the interaction between the effects of stimulation of the vestibular nerve and the nuclei. The stimulus intensities were so adjusted near the threshold as to produce only small EPSPs in the fastigial neuron. The nuclear stimulation (Fig. 6E), when preceded by the conditioning nerve shock (Fig. 6D), induced the EPSP which was larger in amplitude and steeper in rising slope than the algebraical summation of both control EPSPs (Fig. 6F). The latency of the EPSP was 2.0 msec from the nerve and was 1.1 msec from the nuclei. In the histological study the nuclear stimulating electrode was located in the ventrolateral part of the medial vestibular nucleus. These results indicate that the fastigial neurons receive monosynaptic excitation from secondary neurons in the ipsilateral vestibular nuclei which are in turn excited from the vestibular nerve. Similar results were obtained in 2 other fastigial neurons.

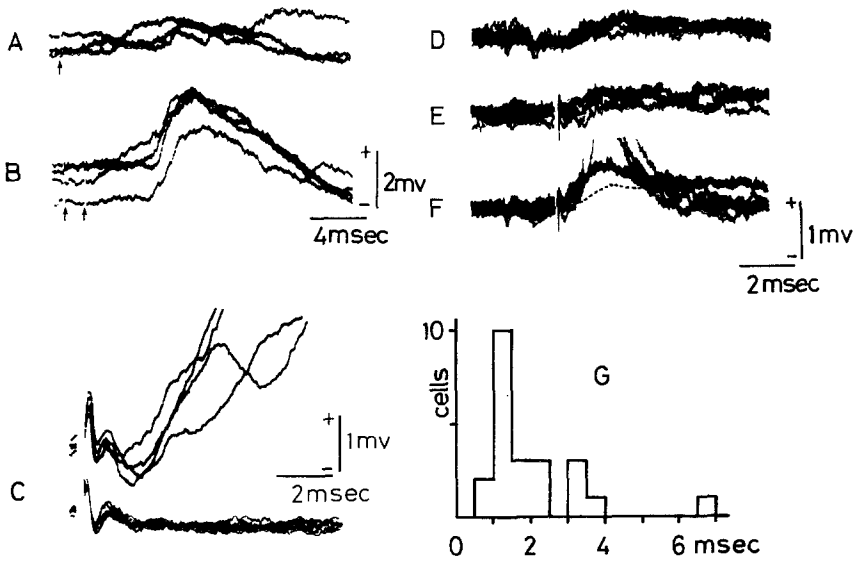

Fig. 6. Facilitation of EPSP in fastigial neurons. (A) Effects of weak single shocks to the ipsilateral vestibular nerve. Stimulus intensity was 1.6 times the threshold for N_1 potential and was near the threshold for the EPSP in this cell. (B) Facilitation of the EPSP by double shocks at the same intensity as in A. (C) EPSPs evoked by stimulation of the ipsilateral vestibular nuclei. Bottom trace indicates extracellular record. (D—F) Facilitation of EPSP in another neuron. (D) Disynaptic EPSP evoked by conditioning shock to the ipsilateral vestibular nerve at the intensity of 1.4 times the threshold for N_1 potential. (E) Monosynaptic EPSP induced by test shock to the ipsilateral vestibular nuclei. (F) EPSP (and spikes in some traces) evoked by conditioning plus test shocks. Dotted line indicates an algebraical summation of records D and E. (G) Latency histogram of EPSPs induced from the ipsilateral vestibular nuclei

Discussion

The present study has shown that type I neurons in the fastigial nucleus are located mainly in its middle and caudal region and are scarcely found in its rostral area. These neurons exhibited responses similar to those of ipsilateral primary vestibular afferents or secondary vestibular neurons in the horizontal canal system. Thus, the horizontal canal input projects to the caudal half of the ipsilateral fastigial nucleus either directly or through the vestibular nuclei. More than 75% of fastigial type I neurons were antidromically activated from the contralateral vestibular nuclei. This is in accord with the anatomical study that the majority of crossed fastigiovestibular fibers are derived from the caudal half of the fastigial nucleus (Jansen and Jansen, 1955; Walberg *et al.*, 1962). Fastigial type II neurons, which exhibit responses to horizontal rotation reverse to type I, were located mainly in the rostral part of the nucleus. Gardner and Fuchs (1975) also found in the monkey that type II neurons were located in the rostral part of the fastigial nucleus and that type I neurons were scattered in more caudal parts of the nucleus.

The shortest latency of the EPSPs produced in the caudal fastigial neurons was 1.0 msec following stimulation of the ipsilateral vestibular nerve. The shortest latency of axon spikes directly evoked from the vestibular nerve was 0.7 msec

when recorded in the ipsilateral fastigial nucleus. It is thus postulated that some neurons in the caudal half of the fastigial nucleus receive monosynaptic activation from ipsilateral vestibular afferents. Since the vestibular nerve contains efferent fibers to the receptors, the possibility may not be excluded that the monosynaptically induced EPSPs from the vestibular nerve are mediated through axon collaterals of these efferent fibers. However, this possibility is unlikely, because the data so far obtained suggest that efferent fibers to the labyrinth are inhibitory in nature (ref. Precht, 1974). It should be noted that spikes of fastigial type I neurons were driven by a single shock to the ipsilateral vestibular nerve in only 7 out of 41 neurons examined. This is a remarkable contrast to the fact that all vestibular type I neurons clearly exhibited spike potentials in response to single shocks to the ipsilateral vestibular nerve (Shimazu and Precht, 1965). This difference may partly be attributed to a small number of direct connections of vestibulocerebellar fibers with fastigial neurons (Carpenter, 1960) as compared to the dense projection of primary afferents to the vestibular nuclei (Walberg *et al.*, 1958).

Stimulation of the vestibular nuclei also induced monosynaptic EPSPs in the ipsilateral fastigial neurons with the shortest latency of 0.8 msec. Regarding the neural origin that produces these EPSPs, the following possibilities should be considered; 1) fibers originating in the vestibular nuclei, 2) fibers passing through the vestibular nuclei and 3) axon collaterals of fibers projecting to the vestibular nuclei. The present experiments have shown that the monosynaptic EPSPs (latency: 1.1 msec) in the fastigial neurons produced by stimulation of the vestibular nuclei are facilitated by conditioning, weak stimulation of the ipsilateral vestibular nerve and that the vestibular nerve stimulation itself produces disynaptic EPSPs (latency: 2.0 msec) in the same neuron. From these results it can be deduced that the primary vestibular afferents activate the vestibular nucleus neurons which project to the ipsilateral fastigial nucleus and produce monosynaptic EPSPs. The latency difference between the EPSPs produced from the nerve and the nuclei, 0.9 msec, may be attributed to the time required for monosynaptic initiation of spikes of secondary neurons in the vestibular nuclei (Precht and Shimazu, 1965; Wilson *et al.*, 1968; Ito *et al.*, 1969; Kawai *et al.*, 1969).

Stimulation of the middle and caudal regions of the vestibular nuclei was more effective than stimulation of their rostral part to excite transsynaptically ipsilateral fastigial type I neurons. This is in agreement with the anatomical study that the major sources of the fastigial projection from the vestibular nuclei come from the caudal part of the medial nucleus and the ventrolateral part of the descending nucleus (Brodal and Torvik, 1957; Carpenter, 1960). These regions, however, do not receive an appreciable number of primary vestibular fibers innervating the cristae of the semicircular canals: canal afferents terminate chiefly in the superior nucleus and the rostral part of the medial nucleus (Stein and Carpenter, 1967), though some are found in the caudal region of the medial and descending nuclei (Gacek, 1969). Thus, the long-latency EPSPs in the caudal fastigial neurons induced from ipsilateral primary vestibular afferents may be mediated via multisynaptic pathways within the vestibular nuclei (Precht and Shimazu, 1965) or indirect pathways from the vestibular nuclei through the reti-

cular formation (Brodal, 1972). According to Gardner and Fuchs (1975), the mean phase lag of fastigial type I neuron responses to sinusoidal oscillation of the head of the animal was $108 \pm 30°$ at 0.9 Hz with respect to the applied angular acceleration, while the phase lag reported by Fernández and Goldberg (1971) for vestibular nerve fibers measured $68 \pm 10°$ at 1.0 Hz. The multisynaptic mechanisms mentioned above may be a possible substrate for the greater phase lag of fastigial type I neurons than those of primary vestibular afferents or even secondary vestibular neurons (Melvill Jones and Milsum, 1971; Shinoda and Yoshida, 1974).

The characteristics of fastigial type I neurons resembled those of the impaled fastigial neurons in every respect examined in the present study. Both were located mainly in the caudal half of the fastigial nucleus. Both were antidromically excited from the contralateral vestibular nuclei with an almost identical range of latencies. Stimulation of the ipsilateral vestibular nerve or nuclei produced EPSPs in the impaled neurons and spikes in fastigial type I neurons. The shortest latency EPSPs appeared earlier than the shortest latency spikes of fastigial type I neurons by 0.5—0.6 msec which may correspond to the time required for spike initiation from the EPSP. The above correspondence suggests that the neuronal organization studied by intracellular recording may apply to that of the vestibulofastigial pathway in the horizontal canal system.

The neurons in the fastigial nucleus that were activated from the cutaneous mechanoreceptors of the forefoot and hind foot were concentrated in the extreme lateral zone of the fastigial nucleus (Eccles, 1973). The neurons in the fastigial nucleus that responded to tilting of the head were mainly located in its rostral half (Ghelarducci, 1973; Ghelarducci *et al.*, 1974). In the present study the neurons in the fastigial nucleus that receive ipsilateral horizontal canal inputs were found chiefly in its medial and caudal area. Thus, it seems likely that in the fastigial nucleus there is a functional localization of neurons receiving different modality of sensory inputs which play an important role in the control of movement.

Acknowledgements. We express our thanks to Miss Y. Oguchi and Mrs. J. Kohase for their histological preparations and to Mrs. K. Katagiri for her secretarial assistance. This study was supported by a grant from Japan Ministry of Education.

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Received June 27, 1975

Prof. H. Shimazu
Department of Neurophysiology
Institute of Brain Research
School of Medicine
University of Tokyo
7-3-1 Hongo, Bunkyo-ku
Tokyo
Japan