EVOKED POTENTIALS OF VESTIBULAR NUCLEI DURING STIMULATION OF VISCERAL AND SOMATIC NERVES

A. A. Shlyakhovenko

UDC 612.828.014.423

Evoked potentials in the bulbar complex of the vestibular nuclei in response to stimulation of somatic and visceral afferent fibers were investigated in actue experiments on cats anesthetized with chloralose and nembutal. Evoked potentials were recorded in the medial and lateral vestibular nuclei to stimulation of the vagus, splanchnic, pelvic, brachial, and sciatic nerves. Interaction between somatic and visceral stimuli at the level of the vestibular nuclei was studied. During application of a conditioning pulse to the visceral nerve, the second response evoked by test stimulation of the somatic nerve was blocked if the time intervals between the two pulses were 10-75 msec. A less marked occlusion phenomenon was observed during interaction in the reverse direction.

By contrast with efferent connections of the vestibular nuclei, which have been well-studied, their afferent organization has been inadequately described, and principally from the results of morphological investigations [3, 5, 7, 8]. No information is available on the projection of visceral afferents in the vestibular nuclei.

This paper gives data on the representation of certain visceral and somatic afferent systems in the bulbar complex of the vestibular nuclei and on their functional interaction at the level of those structures.

EXPERIMENTAL METHOD

Experiments were carried out on 30 cats anesthetized with chlorase and nembutal (40 and 15 mg/kg, respectively, intraperitoneally), and immobilized with listhenon. The representation of the brachial, sciatic, vagus, splanchnic, and pelvic nerves in the bulbar complex of the vestibular nuclei was studied by the evoked potentials (EP) method. Stimulation was by the application of single square pulses (0.2 msec, 2-30 V). The animal was fixed in a stereotaxic apparatus. Stainless steel recording electrodes with a tip 15-50 μ in diameter were inserted in accordance with the coordinates of Snyder's stereotaxic atlas. The indifferent electrode was fixed to the bones of the frontal sinus. Potentials were recorded by means of the four-beam oscilloscope of the ÍÉM electrophysiological apparatus, using an ac amplifier with symmetrical input, transmission band 0.2-3500 Hz, and time constant about 2 sec.

EXPERIMENTAL RESULTS

During stimulation of the central end of the brachial nerve, EPs consisting of positive-negative waves were recorded in the lateral and medial vestibular nuclei on the ipsilateral side after a latent period of 5-16 msec. The duration of the positive phase averaged 19 msec, and the amplitude was 247 μV. The duration of the negative wave of the response averaged 36 msec and its amplitude was 160 μV. EPs in response to sciatic nerve stimulation were similar in shape to potentials evoked by stimulation of the brachial nerve, the only differences being quantitative: their latent period was 8-21 msec, the duration of the positive and negative phases of the response was 22 and 35 msec, respectively, and their amplitude was 242 and 151 μF, respectively (Fig. 1).

Department of Physiology, Ivano-Frankovsk Medical Institute. (Presented by Academician V. V. Parin.)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 72, No. 9. pp. 6-9, September, 1971. Original article submitted January 22, 1971.

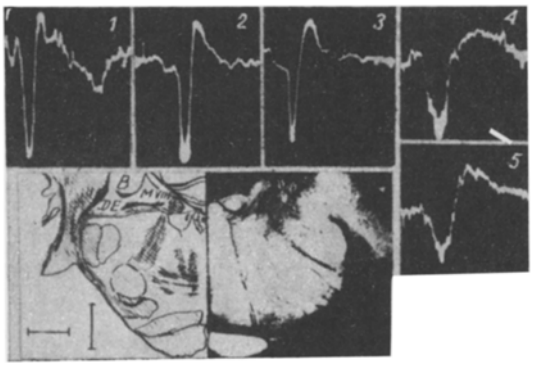

Fig. 1

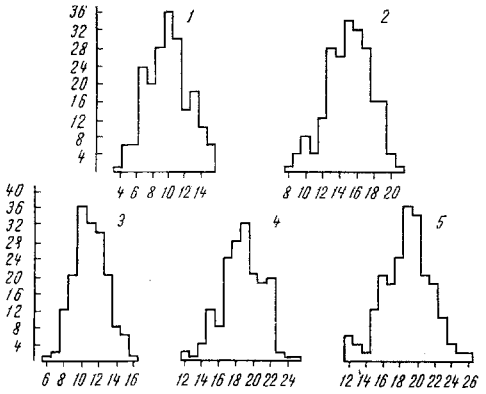

Fig. 2

Fig. 1. Evoked potentials recorded in lateral and medial vestibular nuclei in response to stimulation of brachial (1), sciatic (2), pelvic (3), splanchnic (4), and vagus nerves (5). Time marker 10 msec; calibration 50 μV.

Fig. 2. Histograms of latent periods of evoked potentials in vestibular nuclei during stimulation of the brachial (1), sciatic (2), vagus (3), splanchnic (4), and pelvic nerves (5). Abscissa latent periods (in msec); ordinate, number of cases.

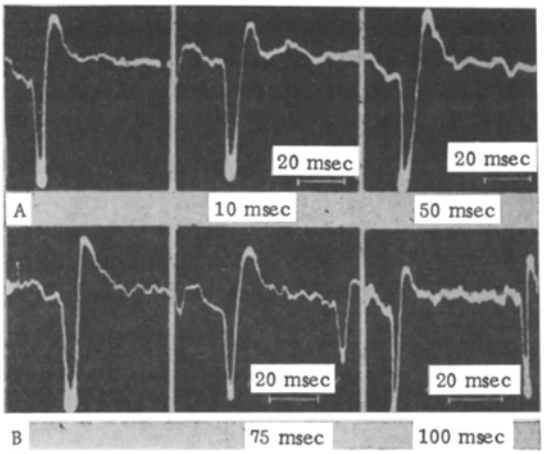

Fig. 3. Interaction between visceral and somatic stimuli in lateral vestibular nucleus. Conditioning stimulus applied to pelvic nerve (A), testing stimulus to sciatic nerve (B), with intervals of 10, 50, 75, and 100 msec between them. Time marker 20 msec; calibration 50 μV.

When potentials were recorded from the same nuclei but on the contralateral side, the EPs were similar but their latent periods were 1.5-2 msec longer and the amplitude of the positive phase was reduced.

No focus of maximal activity could be found for these nerves. EPs in response to stimulation of the brachial and sciatic nerves were recorded in both the rostral and the caudal parts of the nuclei. However, the potentials evoked by stimulation of the brachial nerve were slightly larger in amplitude in the rostral part of the lateral vestibular nucleus, while those in response to stimulation of the sciatic nerve were greater in the caudal part. In some cases EPs to stimulation of the somatic nerves were complex in configuration. Sometimes a fast positive or positive-negative wave, with a latent period of about 6-7 msec, appeared before the positive phase of the response.

During stimulation of the vagus nerve, EPs with a latent period of 7-16 msec, with positive phase of duration 24 msec, and with amplitude 130 μV were recorded in the lateral and medial vestibular nuclei. During stimulation of the splanchnic and pelvic nerves no differences were found in the configuration of the EPs, although their latent period was longer (12-24 msec). No selective localization of representation of the visceral nerves could be found in particular areas of the nuclei. However, responses to stimulation of the pelvic nerve were found in the majority of experiments and they were more constant than responses to stimulation of the splanchnic and, in particular, the vagus nerves (Fig. 2).

These experiments showed that the magnitude of the response is largely dependent on the depth of anesthesia. For instance, nembutal in a dose of 20 mg/kg reduced, and in a dose of 40 mg/kg or more it completely suppressed the EPs of the vestibular nuclei, in agreement with data in the literature [1].

With the facts described above, indicating extensive overlapping of the representation of the somatic and visceral afferent systems in the nuclei of Deiters and Schwalbe, it was decided to study the functional

interaction between the somatic and visceral afferents at the level of these nuclei. A single conditioning stimulus applied to a somatic nerve (brachial, sciatic) as a rule caused suppression of the response to test stimulation of a visceral nerve (vagus, pelvic) provided that the time intervals between them were 10–75 msec. If pairs of stimuli separated by intervals of 100–150 msec were applied, an EP of lower amplitude than in response to a single stimulus was recorded in response to the test stimulus. In the case of interaction in the reverse direction, i.e., when the visceral nerve was stimulated first and the somatic nerve second, the occlusion phenomenon was less marked. A somatic response was observed if the time interval between the two successive stimuli was 100–150 msec (Fig. 3).

The results thus indicate that visceral and somatic afferent systems are represented in the bulbar complex of vestibular nuclei, and that complex interaction between visceral and somatic stimuli takes place at the level of these structures.

It can be concluded from the results of morphological and functional investigations so far carried out that somatic and visceral stimuli reach the vestibular nuclei via polysynaptic nonspecific systems [1], through reverberation arising between the cerebellum and the vestibular nuclei [6], via connections formed by collaterals of the dorsal spino-cerebellar tracts, and also by direct spino-vestibular fibers [2, 5]. The possibility likewise is not ruled out that impulses may also reach the vestibular nuclei secondarily from the cerebral cortex [4]. The study of the mechanisms of viscero-somatic influences on the vestibular nuclei will be the subject of further investigation.

LITERATURE CITED

1. S. Feldman, J. H. Wagman, and M. B. Bender, J. Neurophysiol., 24, 350 (1961).
2. J. M. Frederickson, D. Schwarz, and H. H. Kornhuber, Acta Oto-Laryng. (Stockholm), 61, 168 (1966).
3. R. Lorente de No, Trab. Lab. Invest. Biol. Univ. Madrid, 22, 51 (1934).
4. L. S. Massopust and H. J. Daigle, Exp. Neurol., 2, 179 (1960).
5. O. Pompeiano and A. Brodal, J. Comp. Neurol., 108, 352 (1957).
6. O. Pompeiano and E. Cotti, Arch. Sci. Biol. (Bologna), 43, 57 (1959).
7. F. H. Thiale and V. Horsley, Brain, 24, 519 (1901).
8. V. Wilson, M. Kato, B. Peterson, et al., J. Neurophysiol., 30, 603 (1967).