VERY LONG-LATENCY SOMATOSYMPATHETIC RESPONSE IN UNANESTHETIZED DECEREBRATE FROGS

G. I. Frolenkov, E. V. Lukoshkova, and V. M. Khayutin

UDC 612.143-06:612.899

KEY WORDS: amphibians; somatosympathetic responses; general anethesia

The writers showed previously that rhythmic volleys in sciatic nerve A-afferents evoke depressor blood pressure (BP) reflexes in frogs anesthetized with viadril, and pressor responses in unanesthetized (including decerebrate) frogs [3, 5]. It was suggested that the reason for this qualitative difference in reflex responses of BP in unanesthetized and anesthetized frogs could be discovered by studying somatosympathetic A-responses in decerebrate frogs before and after anesthesia. The aim of the present investigation was to test this hypothesis.

EXPERIMENTAL METHOD

In experiments on male frogs (*Rana temporaria*) anesthetized with ether the brain stem was divided at the rostral border of the thalamus by the ÉS-30 electrosurgical apparatus. The cranial bone was pared on the ventral aspect with a dental drill to expose the brain. At the end of the experiment the skull was opened widely and completeness of division of the brain stem verified visually. The animals were immobilized with flaxedil (40 μg/g, intraveneously). To record BP the catheter of an inductive electromanometer was inserted into the sciatic artery. BP was 20-55 cm water. The central end of the divided sciatic nerve was stimulated with square pulses with an ÉSL-2 stimulator, using anisolating transformer. Thresholds of excitation of the various subgroups of afferents of the stimulated nerve were determined previously [5] and their values for different durations (t) of stimulation were: for A_α-fibers 0.2-0.4 V (t = 1 msec), 0.5-0.8 V (t = 0.1 msec); for A_β-fibers 0.1-0.2 V higher; for A_δ-fibers 0.5-0.8 V (t = 1 msec), 1.2-1.4 (t = 0.1 msec); for C-fibers 3-5 V (t=1msec), 6-8 V (t=0.1 msec). Sympathetic discharges in the renal nerve, as representative of the vasoconstrictor system [8], were recorded. The renal nerve was dissected from surrounding tissues and divided near the kidney. Drying of the nerve was prevented by mineral oil. To detect regular reflex discharges evoked by volleys of sciatic nerve A-afferents (somatosympathetic responses) better, the 14G11 averager (Disa, Denmark) was used. After the end of investigation of the somatosympathetic responses in unanesthetized frogs the animals were anesthetized with viadril (0.13 mg/g body weight) and the responses again investigated. The results of eight experiments are described.

EXPERIMENTAL RESULTS

Changes in efferent signals in the renal nerve in response to a volley of pulses in A-fibers of the sciatic nerve, typical of decerebrate unanesthetized frogs are illustrated in Fig. 1. A volley of A_α + A_β-afferents of the sciatic nerve, just as in anesthetized frogs [5], led to inhibition of tonic discharges in the renal nerve (Fig. 1a). During excitation of A_δ-afferents as well, an intensive burst of spikes appeared (A1 response), after which inhibition of tonic discharges developed, and this was followed by a second intensive and incomparably longer burst of discharges (A2 response; Fig. 1b, c). The latent period of the A1 response was 90-150 msec and its duration 170-250 msec. Only in one experiment, illustrated in Fig. 1, was the inhibition following the A1 response complete, i.e., there was no spike discharge in the renal nerve. Usually after the A1 response the intensity of the tonic discharges was only slightly reduced (Fig. 2c). The latent period of the A2 response was 2.0-2.9 sec and its duration 1.2-2.8 sec. If the period between stimuli was shortened from 20 to 5 sec the A2 response was increased.

In unanesthetized frogs decerebrated along the rostal border of the thalamus, repetitive

Laboratory of Biomechanics and Regulation of the Circulation, All-Union Cardiologic Scientific Center, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR V. N. Smirnov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 101, No. 1, pp. 3-5, January, 1986. Original article submitted March 25, 1985.

0007-4888/86/0001-0001$12.50

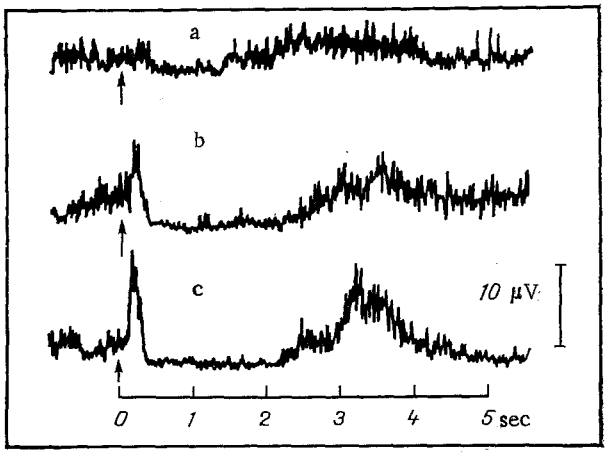

Fig. 1. Changes in spike discharge in renal nerve evoked by a single volley in Aβ-fibers (a) and A_β + Aδ-fibers (b, c) of sciatic nerve in unanesthetized decerebrate frogs. Averaging of 50 realizations. Duration of stimuli 0.1 msec, following frequency 0.1 Hz, amplitude: a) 1V, b) 2V, c) 5V. Times of stimulation indicated by arrows.

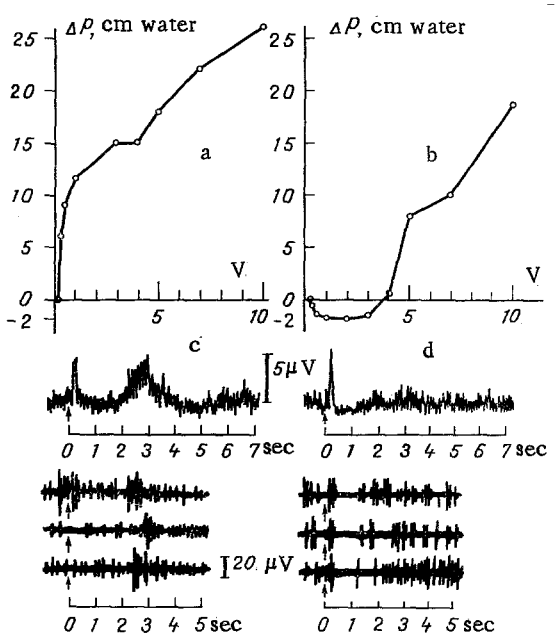

Fig. 2. Dependence of magnitude of reflex responses of BP on amplitude of stimuli applied to sciatic nerve (a, b), and changes in renal nerve activity in response to volley in Aβ+Aδ-afferents of sciatic nerve (c, d) in decerebrate frogs before (a, c) and after (b, d) injection of viadril. a, b) Results of one experiment; c, d) results of another experiment: top trace — averaging of 50 realizations, bottom three traces — single realizations. Parameters of stimuli: a, b) duration 1 msec, frequency 10 Hz; c, d) duration 0.1 msec, amplitude 5 V, following frequency 0.1 Hz. Arrows indicate times of stimulation.

stimulation of the sciatic nerve evoked pressor reflexes, which increased with an increase in stimulus strength [5] (Fig. 2a). After injection of viadril into such a frog the reflex responses of BP to A-afferent volleys changed into depressor (Fig. 2b). Volleys in A+C-afferents continued to evoke pressor reflexes (Fig. 2b). Comparison of somatosympathetic A responses in decerebrate animals before and after injection of viadril (Fig. 2c, d) shows that, first, general anesthesia inhibited the A2 response, and second, deepened the inhibition which followed the A1 response (which became complete). It can be tentatively suggested that the appearance of depressor reflexes in response to repetitive stimulation of sciatic nerve A-afferents in the anesthetized animal is associated either with depression of the A2 responses or with deepening of inhibition, or with both these factors. As regards the A1 response, before injection of viadril it was not found in every realization, but after viadril it was present in all (Fig. 2c, d, single realizations). The amplitude of the averaged A1 response thus increased under general anesthesia. This increase was considerable (by 2.5-4 times) if the A1 response in the unanesthetized animals was weak.

The somatosympathetic response to volleys of myelinated afferents with a latent period as long as that of the A2 response (about 2 sec) has not been described in the literature. However, this duration of its latent period does not seem surprising if it is recalled that sympathetic delay in amphibians may be from 2.5 to 7 times longer than in mammals [6]. The great sensitivity of the A2 response to general anesthesia, and also correlation of its depression with the appearance of depressor reflexes (instead of pressor), which is a feature of the very late A response in mammals [2, 4], suggests that the A2 response is the analog of the very late A response in mammals (the latent period of which is about 300 msec)[1, 7]. However the problem of whether the very late response in mammals and the A2 response in frogs are generated by systems with similar organization and have the same physiological function, namely, they form pressor reflexes to volleys in myelinated afferents, requires further investigation.

LITERATURE CITED

1. Yu. B. Gailans, E. V. Lukoshkova, and V. M. Khayutin, Byull. Éksp. Biol. Med., No. 11, 519 (1978).
2. Yu. B. Gailans, E. V. Lukoshkova, and V. M. Khayutin, Byull. Éksp. Biol. Med., No. 10, 390 (1979).
3. G. I. Frolenkov, E. V. Lukoshkova, T. B. Shuvalova, and V. M. Khayutin, Byull. Éksp. Biol. Med., No. 11, 536 (1985).
4. V. M. Khayutin, R. S. Sonina, and E. V. Lukoshkova, Central Organization of Vasomotor Control [in Russian], Moscow (1977).
5. T. B. Shuvalova and E. V. Lukoshkova, Byull. Éksp. Biol. Med., No. 6, 12 (1982).
6. H. S. Meij, K. S. Holemans, and B. J. Meyer, J. Exp. Neurol., 14, 496 (1966).
7. A. Sato, Pflügers Arch., 332, 117 (1972).
8. S. Yamagishi and T. Azuma, Jpn. J. Physiol., 13, 399 (1963).