ARTERIAL PRESSURE RESPONSES TO IMPULSES IN SCIATIC NERVE A FIBERS IN DECEREBRATE AND SPINAL FROGS

G. I. Frolenkov, E. V. Lukoshkova,
T. B. Shuvalova, and V. M. Khayutin

UDC 612.143-06:612.819.94

KEY WORDS: amphibians; arterial pressure; reflex responses; decerebration; cordotomy.

Separation of the spinal cord from the medulla in mammals causes drastic inhibition both of tonic firing of sympathetic neurons and of reflex responses of the cardiovascular system to impulses in spinal afferents [2, 3]. These phenomena may be due either to prolonged spinal shock [2, 3] or, as is traditionally supposed [1], to the simple absence of appropriate mechanisms in the mammalian spinal cord. In amphibians spinal shock is of short duration [5] and, characteristically, in frogs the intensity of tonic discharges in the renal nerve, which contains vasoconstrictor fibers [8], is the same as initially only 10-20 min after high transection of the spinal cord [7], and the reflex rise of arterial pressure (BP) in response to sciatic nerve (SN) stimulation may not merely be restored to its previous value, but may actually exceed responses observed before cordotomy [3, 4]. Moreover, as the writers showed previously, the action of general anesthesia on responses of this kind in mammals (cats) [2, 3] and amphibians [6] is identical: conversion of pressor responses to impulses in SN A fibers into depressor. A detailed study of the contribution of impulses of different subgroups of SN A afferents to these responses in cats [2] and frogs [6] likewise revealed no differences in principle. The properties of responses evoked by volleys of SN A or A+C afferents in the renal nerve of cats and frogs also were found to be similar [2, 6]. It can accordingly be postulated that the organization of somatovasomotor reflexes in these representatives of two different classes of animals is similar in principle. However, in that case data on preservation both of tonic discharges of vasoconstrictor neurons and of re-

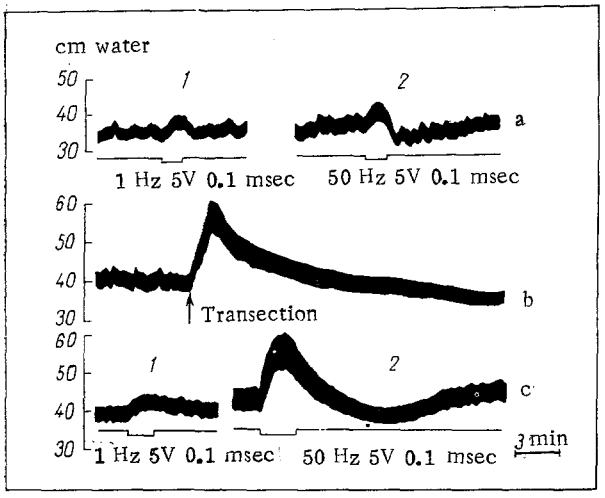

Fig. 1. Reflex responses of BP to stimulation of SN in a decerbrate frog before (a) and after (c) transection of spinal cord at first segment (1, 3 h 20 min after transection; 2, 1 h 30 min later), and also at time of transection (b) of spinal cord. Parameters of stimuli indicated below stimulation marker. Here and in Fig. 2, arrows indicate transection.

Laboratory of Biomechanics and Control of the Circulation, All-Union Cardiologic Scientific Center, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Sciences of the USSR E. I. Chazov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 11, pp. 536-538, November, 1985. Original article submitted December 4, 1984.

 0007-4888/85/0011-1486$09.50

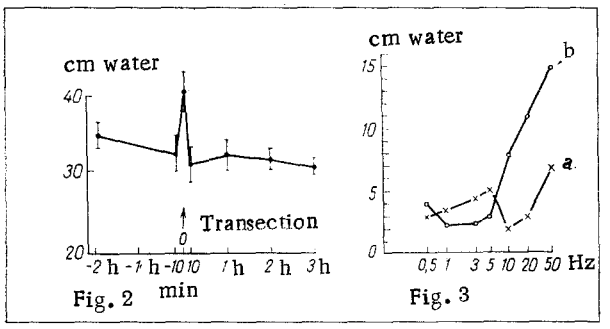

Fig. 2 Fig. 3

Fig. 2. Changes in average (11 experiments) value of BP as a result of transection of spinal cord with time.

Fig. 3. Dependence of magnitude of pressor reflexes on frequency of stimuli applied to SN (semilogarithmic scale) in decerebrate frog before (a) and after (b) cordotomy. Parameters of stimuli: 5 V, 0.1 msec.

flex responses of BP in spinal frogs can be regarded as confirmation of the correctness of views on the decisive role of spinal shock in the inhibition or even disappearance of these responses in cordotomized mammals. Admittedly, data on preservation of reflex responses of BP in spinal frogs [3, 4] have been obtained only for supermaximal stimulation of SN, i.e., for stimulation of A+C fibers, whereas in mammals responses of BP to impulses in A fibers of spinal afferents are inhibited to the greatest degree after cordotomy [2]. Data on reflex responses of BP to impulses in A fibers of spinal afferents in cordotomized frogs are not to be found in the literature.

In the present investigation an attempt was made to obtain such data and to show what changes in these BP responses are caused by cordotomy. In view of distortion of reflex responses of BP by general anesthesia [6], experiments were carried out on unanesthetized decerebrate and spinal frogs.

EXPERIMENTAL METHOD

Under ether anesthesia the brain stem of male frogs (*Rana temporaria*) was divided at the rostral boundary of the thalamus, or the spinal cord was divided at the caudal boundary of the first segment. Administration of ether was then stopped and the animals were immobilized with flaxedil (40 μg/g body weight intravenously). In the series of experiments in which BP changes as a result of cordotomy were studied the immobilized frogs were spinalized without ether anesthesia. The transection was carried out under an MBS-9 microscope, using the ES-30 electrosurgical apparatus with an active electrode consisting of a pointed blade 0.5-1 mm wide and a wire ring about 1 mm in diameter. The brain was exposed from the ventral aspect, through a midline incision in the palatal mucosa, and the spinal cord from the dorsal aspect, after removal of part of the suprascapula. The bony tissue covering the brain was ground away with a dental drill. Completeness of transection was verified visually at the end of the experiment, after opening the skull and vertebral canal widely. Responses of BP in the decerebrate frogs and in the cordotomized frogs under ether anesthesia began to be studied not earlier than 2 h after transection of the spinal cord, and in the unanesthetized cordotomized frogs, not earlier than after 15-20 min. To record BP the catheter of an inductive electromanometer was introduced into the sciatic artery. BP was 20-55 cm water (disregarding reflex changes). The central end of SN was stimulated by square pulses 0.1 msec in duration and 5 V in amplitude. This was sufficient to stimulate all subgroups of A fibers, but was below the threshold of excitation of the C fibers of SN [6]. Drying of SN was prevented by the use of petrolatum. The results of experiments on 11 decerebrate and 18 spinal frogs are presented (four animals underwent decerebration first, followed by cordotomy). The significance of differences between values of the reflexes and changes in BP level as a result of cordotomy were evaluated by Student's t test. In the latter case, the degree of correlation between the values compared was calculated.

EXPERIMENTAL RESULTS

Cordotomy caused a transient (3-5 min) increase in BP, followed by a fall (Fig. 1b). In a series of 11 experiments the value of BP 10 min after spinalization was higher than initially (at most by 20%) in four frogs, lower than initially (at most by 39%) in four frogs, and indistinguishable from BP before cordotomy in three experiments. The mean values of BP

for all 11 animals 10 min and 1, 2, and 3 h after cordotomy differed from values of BP 10 min before cordotomy, but not statistically significantly ($P > 0.4$; Fig. 2).

Repetitive stimulation of A fibers of SN in both decerebrate and spinal frogs usually evoked only pressor reflexes, whatever the frequency of stimulation used (0.2-50 Hz). Three experiments on decerebrate frogs were exceptions, during which pressor reflexes occurred to a frequency of 1 Hz or more, but less frequent stimulation (under 1 Hz) evoked depressor reflexes. In four of eight decerebrate frogs and in two of 11 spinal frogs, a depressor "rebound" was observed at the end of stimulation with a frequency of over 10 Hz: a temporary fall of BP below its initial level (Fig. 1).

Spinalization did not lead to inhibition of pressor reflexes in the frogs: At a low frequency of stimulation responses of BP to volleys of A afferents of SN before and after cordotomy were comparable in value, but at a high frequency, responses after cordotomy were actually much larger (Fig. 1). Typical relationships between amplitudes of pressor reflexes and frequency of volleys in A afferents of SN for decerebrate and spinal animals respectively are shown in Fig. 3. The result obtained by averaging these curves, obtained in nine decerebrate and nine spinal frogs (three of these experiments were on the same animals before and after cordotomy) shows that at frequencies up to 5 Hz the averaged curves coincided (differences not statistically significant: $P > 0.4$), whereas at frequencies above 5 Hz a steep drop of the curve was observed, but a steep rise in the curve for spinal frogs. Under these circumstances the pressor reflexes in the spinal frogs were greater ($P < 0.001$) than in decerebrate frogs.

High transection of the spinal cord in frogs thus does not lead to any statistically significant changes in BP and, in addition, unlike in mammals, inhibition of pressor reflexes to impulses in A afferents of SN does not take place in frogs. This confirms the view that a decisive role in the inhibition of such reflexes in cordotomized mammals is played by spinal shock [2]. In decerebrate frogs, during stimulation of A fibers of SN at high frequencies (5 Hz or higher) limitation of the pressor reflexes takes place, but disappears as a result of spinalization.

LITERATURE CITED

1. B. Folkow and E. Neil, Circulation, Oxford Univ. Press, New York (1971).
2. V. M. Khayutin, R. S. Sonina, and E. V. Lukoshkova, Central Organization of Vasomotor Control [in Russian], Moscow (1977).
3. V. M. Khayutin, Fiziol. Zh. SSSR, No. 8, 1032 (1982).
4. O. V. Shablonshkaya and V. A. Tsybenko, in: Central Regulation of the Circulation [in Russian], Volgograd (1977), pp. 225-227.
5. C. S. Sherrington, Integrative Action of the Nervous System, Yal Univ. Press, New Haven (1947).
6. T. B. Shuvalova and E. V. Lukoshkova, Byull. Éksp. Biol. Med., No. 6, 12 (1982).
7. E. Dieter, Pflügers Arch., 271, 64 (1960).
8. S. Yamagishi and T. Azuma, Jap. J. Physiol., 13, 399 (1963).