PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

CARDIAC REFLEXES DURING CHRONIC NOCICEPTIVE STIMULATION

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UDC 612.178.6:612.884

The action of transient nociceptive stimuli on the cardiovascular activity has been studied reasonably fully [5], but few investigations have been made of the influence of prolonged (chronic) stimuli on the cardiovascular activity and the state of the reflex centers regulating it.

The object of the present investigation was to study changes in the cardiac reflexes during chronic nociceptive stimulation.

EXPERIMENTAL METHOD

Chronic experiments were carried out on 4 dogs with the carotid arteries exteriorized in separate skin cuffs. The oculo-cardiac reflex was elicited by applying pressure (140 mm Hg) to the eye with a small rubber balloon connected to a tonometer for measuring the pressure. The reflex from the carotid sinus to the heart was elicited by the simultaneous compression of both carotid arteries. The pressure on the eye and the compression of the carotid arteries lasted for 20 sec, and the pulse rate was counted for intervals of 15 sec before eliciting the reflexes (no fewer than 4 times) and thereafter until recovery was complete.

A focus of chronic nociceptive stimulation was created by applying two ligatures, on which small, sharp-pointed beads were strung, to the sciatic nerve. The ligatures were tied to the muscles in opposite directions.

EXPERIMENTAL RESULTS

The heart rate in the experimental dogs was 86-120 beats/min; in the same animal variations from experiment to experiment were very small – within limits of 4-6 beats/min. After creation of a focus of chronic nociceptive stimulation the pulse rate fell by 12-30 beats, and remained at that level for 2.5-3 months, after which it became quicker, so that the changes in the pulse rate were periodic in character (Fig. 1). The oculo-cardiac reflex was well marked in all the animals, and the pulse rate fell by 20-30 beats/min, but after creation of the focus of chronic nociceptive stimulation this reflex also developed periodic changes (Fig. 2). The first phase, lasting 15-20 days, was marked by depression of the reflex (instead of falling by 20-30 beats/min, the pulse rate fell by only 2-10 beats/min or remained unchanged). In the second phase the oculo-cardiac reflex was opposite in form, i.e., instead of slowing when pressure was exerted on the eye, the pulse rate quickened by 6-8 beats/min.

The reflex was restored 2.5-3 months after creation of the focus of nociceptive stimulation.

The reflex from the carotid sinus to the heart in response to compression of both carotid arteries took the form of a quickening of the pulse by 20-30 beats/min and was well marked in all dogs. After creation of the focus of nociceptive stimulation, the changes in this reflex also were periodic in character (see Fig. 2).

During the first 15-20 days the reflex was sharply depressed (first phase), and in response to compression of the carotid arteries the pulse rate either remained unchanged or changed by only 5-6 beats/min. This was followed by a reversal of the reflex reaction (second phase), i.e., instead of a quickening of the pulse, it fell by 10-20 beats/min. These changes continued for about 2 months. The reflex was restored 2.5-3 months after the creation of the focus of nociceptive stimulation.

Comparatively weak, transient nociceptive stimuli, increasing the tone of the sympathetic nervous system, are known to cause quickening of the heart rate and an increase in vascular tone, as a result of which the blood pressure rises [1, 2]. Strong or prolonged nociceptive stimuli cause an increase in the tone of the parasympathetic nervous system and give the opposite effect [7, 10]. During chronic nociceptive stimulation the blood pressure rises [6]; possibly because of this the heart rate falls by a reflex mechanism. In the first phase the reflex centers regulating cardiac activity are depressed, while in the second phase the reflexes are distorted.

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Fig. 1. Slowing of the pulse rate and its periodic changes during chronic nociceptive stimulation. Along the axis of abscissas—time of experiment; along the axis of ordinates—pulse rate (per minute).

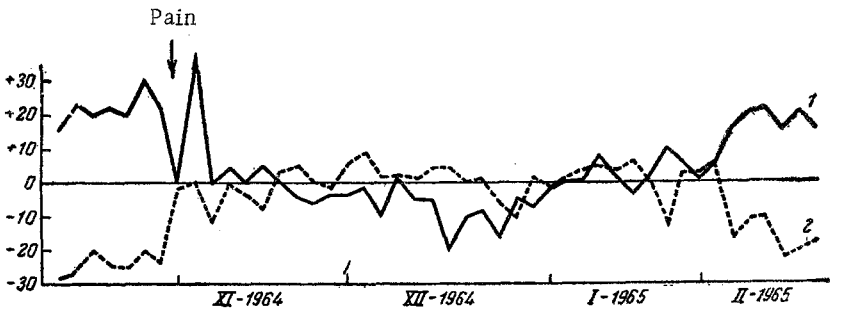

Fig. 2. Changes in the oculo-cardiac reflex and the reflex from the carotid sinus on the heart during chronic nociceptive stimulation. Along the axis of abscissas—time of experiments; along the axis of ordinates—difference in pulse rate during reflex action (+ increase; — decrease). 1) Compression of carotid arteries; 2) oculo-cardiac reflex.

The distortion of the reflexes evidently takes place because of a decrease in the lability of the reflex centers, and any additional stimulus addressed to this center causes its inhibition, while the other center, possessing higher functional lability at this time, produces its effect, so that the opposite reaction arises: in response to pressure on the eye the pulse rate quickens, and in response to compression of the carotid arteries, the pulse rate slows. There is a difference in principle between the action of transient and of chronic nociceptive stimulation, because in the latter case the functional state of the organism is established on a different level for a longer time, with changes in many of its indices [3, 4, 8, 9].

SUMMARY

Repeated experiments were carried out on four dogs to study the influence of chronic stimulations with pain on the oculo-cardiac reflex and the reflex from the carotid sinus to the heart during compression of both common carotid arteries.

A focus of chronic stimulation with pain was produced by placement on the sciatic nerve of two ligatures with tiny beads strung on them. The ligatures were drawn up and sutured to muscles in opposite directions. After a focus of irritation with pain had been produced, investigations were carried out during three to three and a half months. The changes in reflexes occurred by phases: during 15-20 days there was a distortion of reflexes, i.e., quickening of the pulse-rate upon pressure applied to the eyeball and a slowdown of the pulse upon compression of the carotid arteries. The reflexes returned to normal two and a half to three months after a focus of irritation with pain had been produced. Through all the period of investigations one could observe bradycardia (by 12-30 beats per min) as compared to the initial, painless, experiments.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of the first issue of this year.
