MECHANISM OF THE DISTURBANCE OF CARDIAC ACTIVITY ON DECEREBRATION

V. S. Livshits

Laboratory of Experimental Pathology (Head of Laboratory: Professor S. I. Lebedinskaya) of the Department of General Pathology (Head of Department: Academician A. D. Speransky) of the Institute of Normal and Pathologic Physiology (Director: Full Member of the Acad. Med. Sci. USSR, Professor V. N. Chernigovsky)

(Received February 6, 1956. Submitted by Full Member of the Acad. Med. Sci. USSR, Prof. V. N. Chernigovsky)

The study of the role of various parts of the nervous system in compensating for disturbed cardiac functions has demonstrated [9] that occulsion of a branch of the coronary artery in decerebrate cats is accompanied by such disturbances of cardiac activity (prolonged extrasystole, paroxysmal tachycardia, irreversible fibrillation) as are not encountered with similar treatment of cats with intact nervous systems.

The present work is concerned with the study of mechanisms responsible for the appearance of disturbances of cardiac activity in decerebrate animals.

There are only a few references in the literature to the effect of decerebration on the cardiovascular system. Decerebration of dogs and cats gives rise to slowing of the cardiac rhythm, lowering of blood pressure, appearance of sinus arrhythmia, and more rarely of nodal and ventricular cardiac rhythm [16, 17, 19, 21]. In the experiments of Ya. M. Britvan and A. G. Kurdin [3] on dogs, decerebration was followed by the disappearance of the bradycardia evoked by the administration of morphine, and in three out of five experiments there was lowering of the RST segment in the electrocardiogram with a slight increase in the voltage of the R deflection in two of these. Unconditioned cardiovascular reflexes became considerably more pronounced after decerebration [1, 4, 5, 6, 7, 12, 14, 15, 16, 20].

EXPERIMENTAL

Experiments were performed on cats weighing from 2 to 3 kg; decerebration (intercollicular section) was carried out after preliminary disruption of the sympathetic regulation of the heart (excision of stellate ganglia), the parasympathetic regulation of the heart (section of the vagi in the neck) and all the extracardiac regulation (by combining the two operations mentioned above). In control experiments decerebration was performed on animals with intact cardiac innervation, and also partial or complete disruption of cardiac innervation in animals decerebrated prior to this. All the operative procedures carried out before decerebration were performed under (Barbamyl) or nembutal anesthesia (50-80 mg per 1 kg body weight intraperitoneally). Cardiac activity was recorded on an electrocardiogram (ECG) (Lead II) simultaneously with measurement of blood pressure; an ink-writing electrocardiograph and a mercury manometer were used.

RESULTS

In 18 of 20 cats with intact innervation of the heart, decerebration led to slowing of cardiac contractions (on an average by 40-50 contractions per 1 minute), appearance of mild sinus arrhythmia and fall of blood pressure (by 20-40 mm of mercury). In two of the animals there was nodal rhythm, in two— ventricular rhythm and in one— extrasystolic pulsus bigeminus. The latter disturbances of cardiac rhythm disappeared completely in the course of the first 5-10 minutes after decerebration. At the end of 40-60 minutes the slowed heart rate

in most animals (14) returned to the original, in the minority (4) it still remained 30-40 beats below normal after 2-3 hours. In a number of cases, decerebration was followed by lengthening of the PQ interval from 0.07 to 0.1-0.14 seconds, while the T wave underwent a change both in amplitude and direction. The most pronounced slowing of the cardiac rhythm was observed in animals with marked decerebrate rigidity.

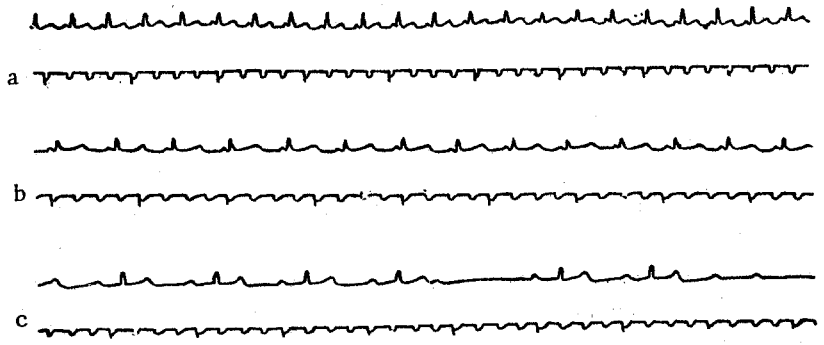

Fig. 1. Electrocardiogram of cat, Lead II.
a) original; b) after extirpation of stellate ganglia -slowing of cardiac contractions; c) 2 minutes after decerebration - still more marked slowing of cardiac contractions, heart block.
Time marker ($\frac{1}{4}$ second).

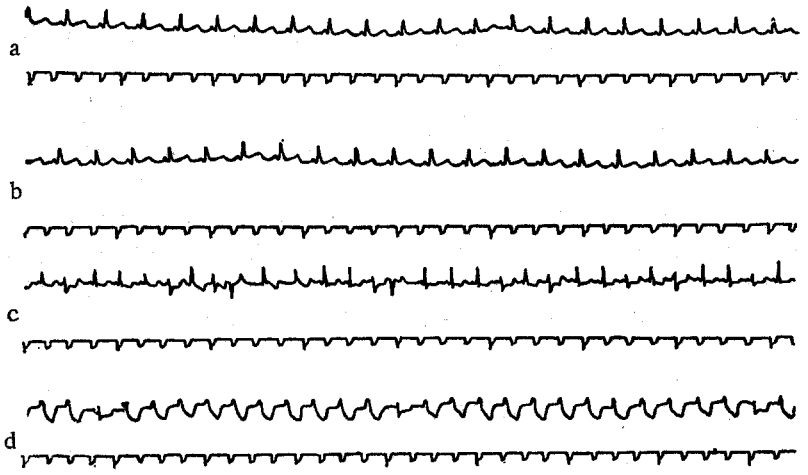

Fig. 2. Electrocardiogram of cat, Lead II.
a) original; b) after section of vagi - rate of cardiac contractions unchanged; c) 2 minutes after decerebration - acceleration of cardiac contractions, polymorphic extrasystole; d) 4 minutes after decerebration - ventricular rhythm, interrupted by single sinus contractions. Time Marker ($\frac{1}{4}$ second).

In 2 of the 20 cats with intact innervation of the heart, decerebration led to acceleration of cardiac contractions (by 10-15 per 1 minute) and a slight rise of blood pressure (by 10-15 mm of mercury).

Extirpation of the stellate ganglia produced slowing of the heart rate (by 20-40 beats per minute, Fig. 1, b) and fall of blood pressure (by 10-20 mm of mercury) in all cats.

Section of the vagi produced mild acceleration of the heart rate (by 10-20 beats per minute) or, more frequently, left it unchanged (Fig. 2, b), although there was a rise of blood pressure by 30-80 mm of mercury in half the animals. With complete disruption of extracardiac gulation (section of the vagi and extirpation of the stellate ganglia) the heart rate slowed down to 90-140 instead of 120-200 contractions per minute and blood pressure fell to 90-130 mm instead of 100-170 mm of mercury as in the normal.

Decerebration in all 8 cats with removed stellate ganglia produced considerable slowing of the heart rate (on an average by 50-70 contractions per 1 minute) (Fig. 1, c) and marked fall of blood pressure (by 30-40 mm of mercury). In 4 of the cats greater slowing of the heart rate was noted than in decerebrate control animals, while in 2 there was complete transverse heart block for 10-15 minutes (with the ratio of atrial and ventricular contractions 2:1 and 3:1) during which maximal blood pressure fell to 50-70 mm of mercury.

Decerebration in all 8 animals with sectioned vagi in the neck produced considerable acceleration of cardiac contractions (by 40-70 per 1 minute) (Fig. 2, c), and a rise of blood pressure (by 30-80 mm of mercury);

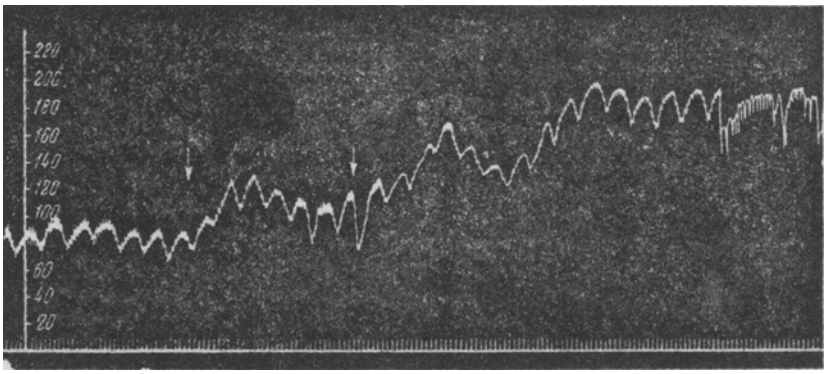

Fig. 3. Blood pressure of cat. First arrow – section of vagi – rise of blood pressure. Second arrow – decerebration – further rise of blood pressure, arrhythmia. Zero line – also time marker (1 second).

in some of these cases the heart rate reached 240-275 per 1 minute, and the blood pressure 180-210 mm of mercury (Fig. 3). Acceleration of heart rate and rise of blood pressure did not occur at once but developed gradually after a latent period of 30-60 seconds and reached a maximum after 2.5-3 minutes following decerebration . In half the cases as the heart rate rose the regular sinus rhythm became replaced by pulsus bigeminus, which was succeeded by right- and left-ventricular polymorphus extrasystole and then by ventricular paroxysmal tachycardia (Fig. 2, c and d); there were appreciable changes in the ventricular complex of the ECG, with appearance of a pronounced Q deflection, change of the T wave from positive to diphasic, and descent of the S-T interval below the isoelectric line. Such changes in the electrocardiogram, indicating diffuse disturbance of bioelectric processes in the myocardium, are usually considered to be due to anoxia of the heart muscle.

10-20 minutes after decerebration, the disorders of cardiac rhythm (excepting tachycardia) disappeared, while the blood pressure dropped although remaining 15-30 mm of mercury higher than the original level.

Decerebration in 4 cats with removed stellate ganglia and sectioned vagi did not alter the character of cardiac activity, although it produced a fall of blood pressure (by 15-30 mm of mercury).

It should be pointed out that decerebration in an overwhelming majority of control cats without lesions of extracardiac nerves was followed by slowing of the heart rate and fall of blood pressure, i.e., reactions indicating predominant excitation of centers concerned with parasympathetic regulation of the heart. However, in two cases, decerebration was accompanied by a definite acceleration of the heart rate and rise of blood pressure – reactions indicating some degree of predominance of excitation of sympathetic innervation centers.

Decerebration in cats with removed stellate ganglia, i.e., with considerable if not complete disruption of the connection between the heart and centers of sympathetic innervation, produced in some cases greater slowing of the heart rate than in the control animals, i.e., it was associated with greater vagal influence on the heart than in the controls. Conversely, decerebration in cats with sectioned vagi produced not slowing but marked acceleration of the heart rate and rise of blood pressure, i.e., it was accompanied by reactions indicating excitation of centers concerned with sympathetic regulation.

Consequently, the present experiments, as distinct from those of earlier workers, permit the conclusion that elimination of all the forebrain and part of the midbrain by section at the intercollicular level (decerebration) is accompanied by simultaneous pronounced and persistent excitation of centers concerned in the

parasympathetic and sympathetic regulation of the heart, and, in contradistinction to physiologic conditions, when the influence of the vagi and sympathetic nerves on the heart is to a considerable extent synergistic, their antagonistic action becomes clearly apparent following decerebration.

It is reasonable to infer that the heightened excitability (or tonus) of the centers of extracardiac nerves results from the elimination of the higher cerebral divisions and also trauma to the midbrain as well as the increased excitability of the bulbar centers (associated with this trauma) to reflex influences from the various vascular reflex zones.

The concept of simultaneous excitation of sympathetic and parasympathetic centers under the influence of decerebration is confirmed by the results of the following group of experiments in which section of the vagi was performed after decerebration. In these cases section of the vagi produced acceleration of the heart rate which was much greater than in control, nondecerebrate, cats. In these experiments the heart rate accelerated in controls to a maximum of 200-220 per 1 minute. In decerebrate cats, however, section of the vagi accelerated the heart rate to 260-280 per 1 minute.

This excessive acceleration of the heart rate in decerebrate cats following section of the vagi evidently results from excitation of smypathetic nerve centers previously masked by the accompanying excitation of vagal centers.

The experiments have also demonstrated the significance of cardiac innervation for the state of cardiac activity in cerebral lesions.

Decerebration in 5 of 20 cats with intact innervation of the heart, as already mentioned, produced brief (5-10 minutes) disturbances of cardiac rhythm. The character of these disturbances (mainly nodal or ventricular rhythm) suggests their dependence on increased vagal tonus.

Decerebration in cats with sectioned vagi produced not only excessive acceleration of the heart rate, but also the appearance of polytopic extrasystoles, brief bursts of ventricular paroxysmal tachycardia and irreversible cardiac fibrillation; these effects were particularly prominent in cases of vagal section following decerebration. The incidence of fibrillation should be especially emphasized.

It is known that experimentally induced arrhythmias, produced by various myocardial lesions, disappear on section of the vagi [18, 8, 9, 13], andthat myocardialfibrillation in cats appears with difficulty, and when it does develop it is reversible [2, 11, 10]. In decerebrate cats, as seen from the present experiments, these usual inter-relations have been disrupted.

It follows from this that decerebration, which excites the centers of sympathetic and parasympathetic regulation of the heart, also causes changes in the properties of the heart muscle (appearance of the ability to show irreversible fibrillation and to respond with inverted reactions to section of the vagi).

In the presence of cerebral lesions, preservation of intact vagi protects the heart from the appearance of irreversible fibrillation not only because it entails slowing of the heart rate and prevents the appearance of ventricular paroxysmal tachycardia (which is particularly favorable for the development of fibrillation) but also because it exerts a positive trophic influence on the heart. This is indicated by the character of the ECG changes seen after vagal section in decerebrate cats as well as by the fact that in half the cases, fibrillation following section of the vagi occurred against the background of relatively slow, for cats, sinus rhythm (about 200 contractions per 1 minute).

In connection with the data obtained it becomes apparent that disturbances of cardiac activity arising on ligation of the coronary artery only in decerebrate cats and not observed in intact animals, result from the simultaneous extreme excitation of centers of sympathetic and parasympathetic regulation, evoked by decerebration, leading to changes in the functional properties of the heart muscle.

LITERATURE CITED

[1] V. A. Alekseev, cited in Transactions of the Navy Medical Academy* (1944), Vol. IV, Part 1, p. 24.

[2] L. A. Arshavsky, Nervous Regulation of the Cardiovascular System in Ontogenesis* (Moscow, 1936).

* In Russian.

[3] Ya. M. Britvan and A. G. Kudrin, Byull. Eksptl. Biol. i Med. 40, 9, 18 (1955).

[4] A. I. Ivanov, Esophageal and Gastric Interoceptors* (Navy Medical Academy Press), 1945).

[5] N. V. Klykov, cited in Kuban State Medical Institute Tenth Scientific Conference* (Krasnodar, 1955), p. 16.

[6] G. A. Kovaleva, Byull. Eksptl. Biol. i Med. 25, 4, 302 (1948).

[7] V. A. Lebedeva, Thesis* (Leningrad, 1947).

[8] V. S. Livshits, Voprosy Eksptl. Biol. i med. 1951, 127.

[9] V. S. Livshits, Problem of Reactivity in Pathology* (Moscow, 1954), p. 179.

[10] Ibid., p. 188.

[11] V. B. Malkin, Thesis* (1950).

[12] S. S. Musyashchikova, Byull. Eksptl. Biol. i Med. 37, 1, 13-18 (1954).

[13] A. I. Smirnova and N. I. Shumilina, Klin. Med. 32, 2, 62 (1955).

[14] V. N. Chernigovsky, Afferent Systems of Internal Organs* (Kirov, 1943).

[15] V. N. Chernigovsky, Transactions of the Navy Medical Academy* (1940), Vol. XVII, p. 395.

[16] I. C. Gupta, Z. Kreislaufforsch. 1936, No. 28, 492.

[17] H. C. Hoff, C. G. Breckenridge and W. A. Spencer, J. Physiol. 1925, 171-178.

[18] Jamada, Mitt. med. fac. Tokyo (1920).

[19] A. Schweitzer, Pflugers Arch. ges. Physiol. 1934, 235, 110.

[20] A. Schweitzer, Die Irradiation Autonomer Reflexe (Basel, 1937).

[21] C. S. Sherrington, The Integrative Action of the Nervous System (London, 1906).

* In Russian