THE EFFECT OF EXPERIMENTAL NEUROSIS ON AGE VARIATION OF HEART RATE IN NORMAL APES AND IN THOSE SHOWING A BRADYCARDIA WITH SINUS ARRHYTHMIA

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It has frequently been reported that apes are valuable as experimental animals for studying various pathological human conditions [1, 5, 6, 10]. Cases of coronary insufficiency and myocardial infarct resulting from experimentally induced neuroses have been described [6, 11].

Here we describe a further cardiac disturbance, bradycardia with sinus arrhythmia, also caused by experimentally induced neuroses. Since no reports are available of the normal range of heart rate in apes, we here report the results we have obtained for different age groups.

METHOD AND RESULTS

The heart rate was measured by an electrocardiogram (EKG), recorded by an ÉKP-4 apparatus or by an ink-writing electrocardiograph (Officine Galileo). The neurosis was induced by disturbance of the 24-hour rhythm [10], by altering the daily program at a frequency to which the animal was unable to adapt. We used four 2-year-old hamadryad baboons. (Abad, Omar, Barsuk, and Khisar).

In normal baboons, the heart rate is quite high. Observations on 79 specimens showed no difference between rhesus monkeys and hamadryad baboons, and the results may therefore be taken together. There was, however, a considerable difference according to age (see table and Fig. 1). The differences in heart rate were statistically significant.

It can be seen from the table that the highest rate in the very young animals is 59% above that of the adults; in the one-to-four age group it is lower, and in the adults, lower still. In the old animals, the few observations available indicate that the rate does not differ from that of younger adults.

The marked variation, caused especially by muscular activity and emotional factors, should be noted; it occurs within each group, and the rate for one animal also changes between successive measurements. In the experiments of N. I. Lagutina and A. A. Fufacheva, blocking of the vagi by subcutaneous injection of atropine, or (in an acute experiment) by a ligature, caused an increase in heart rate. However, in apes, the heart rate increases by 17-19% as a result of this procedure, whereas, in man, an atropine injection may increase the rate to 150 beats per minute, which represents a 100% tone is considerably less than in man. This low tone is also indicated by the fact that respiratory arrhythmia is very poorly shown. Figure 1f shows an EKG recorded over a period of 12 seconds when the respiration rate

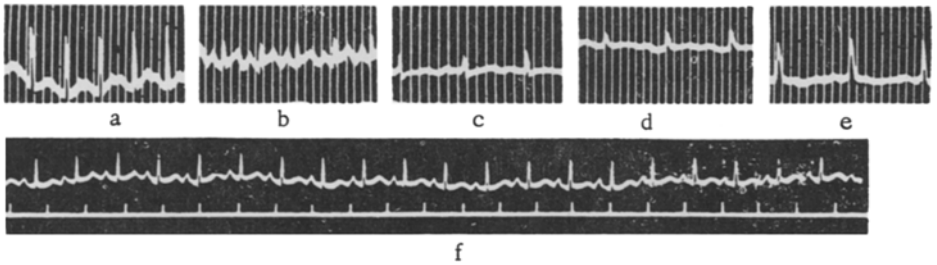

Fig. 1. EKG of apes of different age groups. a, b) 7 months (pulse 300 and 266 beats per minute); c) 2 years (pulse 184 beats per minute); d) 6 years (pulse 150 beats per minute); e) 16 years (pulse 133 beats per minute); f) EKG of adult ape, recorded over a period of 12 seconds (no respiratory arrhythmia).

Range of Variation and Mean Values of Heart Rate in Apes, According to Age Groups

Heart rate	Age			
	less than one year	from one to four years (young)	from four to fifteen years (adult)	from fifteen years (old)
Maximum	300	240	218	171
Minimum	218	133	120	120
Mean	258 ± 4.87 (from 23 measurements)	180 ± 2.89 (from 91 measurements)	162 ± 3.56 (from 35 measurements)	162 ± 2.7 (from 3 measurements)
P	P = 0.0000		P = 0.0007	

was 30-40 per minute. The R–R intervals remain strictly unchanged.

In apes, there is a marked diurnal rhythm in the heart rate. Although the EKG was taken on waking, the conditioned reflex association with night-time causes a reduced rate by night. For adults, a rate below 120 per minute represents bradycardia, and above 240, tachycardia.

In the four animals in which neurosis was induced, the original rate was within the normal limits for the age group: In Barsuk it was 170 per minute, in Khisar 170-190, in Abad 155-170, and in Omar it reached the upper normal limit of 210-230 beats per minute.

For 82 days, the animals were subjected to a disturbance of the daily rhythm of their surroundings [10]. At the end of this time they had become neurotic. In addition to various physiological disturbances, in three of them there was a change in heart rate: In two it had fallen to 115 beats per minute, and in the third, Khisar, to 75 per minute. Figure 2 shows the reduction in the heart rate in Barsuk, Khisar, and Abad (as measured by the EKG). Thus, before the neurosis, the heart rate was normal for apes of that age, but when neurosis developed, there was a marked bradycardia. Subsequently, when the apes were returned to a normal daily rhythm, and the disturbing circumstances were removed, the heart rate gradually returned to normal (see Fig. 2). The EKG of Khisar, in whom the bradycardia was very marked, is shown in Fig. 3.

The EKG shows many abnormalities characteristic of vagal tone. Thus, together with the bradycardia, a respiratory arrhythmia develops. In Fig. 3 (7) it can be seen that the R–R intervals vary. Normally, as has already been pointed out, this is not the case in apes. In Fig. 3 (1 and 2), the EKG of the same ape is shown before neurosis was induced, and it can be seen that the R–R intervals are all the same. In addition, the amplitude of all the waves is reduced; the T wave is flattened, and the Q–T interval greatly increased, as is particularly marked in Fig. 3, (2 and 7), where the leads from the chest show the T wave particularly well.

Precisely the same EKG changes have been described by A. F. Tur [8], G. F. Lang [4], and others for the so-called "vagus neurosis" in man ("vagal heart"). Cardiac conditions involving a slow pulse (bradycardia) have been described previously by V. F. Zelenin [2] and L. I. Fogel'son [9].

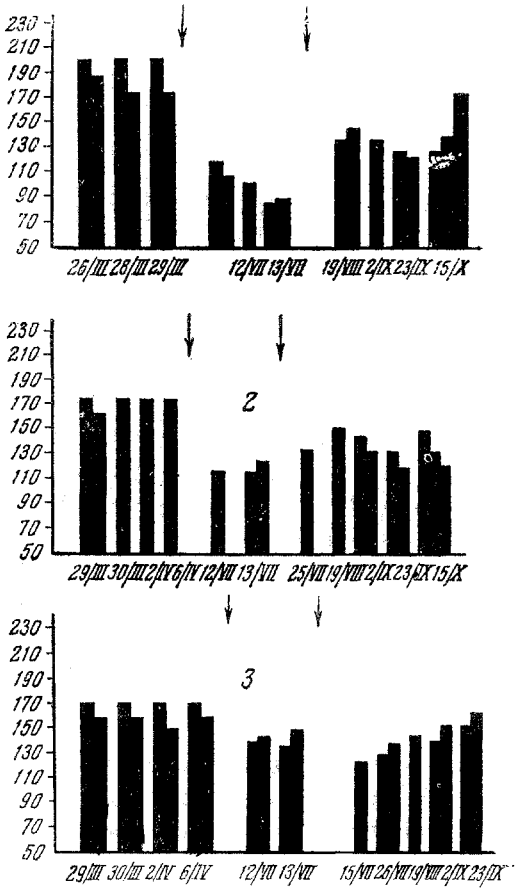

Fig. 2. Development of bradycardia during neurosis, and return to normal after the experiment. Above – in the ape Khisar; 2) in the ape Barsuk; 3) in the ape Abad. Heart rate is plotted upward; the arrows indicate the onset of the neurosis and the end of the experiment.

The method whereby the bradycardia develops may be represented as follows: Disturbance of the daily rhythm in apes induces a neurosis which is associated with severe disturbances in the functions of the internal organs [11]. The normal relationships between the processes of excitation and inhibition in the cerebral cortex are then upset, with the result that there is also a disturbance of the mutual inductive effects between cortex and subcortex. Normally, vagal tone is under cortical control [7]. Consequently, any change in cortical activity must react on the heart. The cortex exerts an inhibitory effect on the vagal center. Therefore, removal of cortical control leads to an increase in vagal tone. A. K. Smirnov has observed this phenomenon when removing the cerebral cortex in dogs [7]. K'ang K'ong-yeh [3] has shown that, when the cerebral cortex of pigeons is removed and vagal tone increased, there is a marked bradycardia and sinus arrhythmia. The reduction of the cortical control over the bulbar centers of the vagus nerve may also develop without operative interference, and may be caused by a neurosis which disturbs the normal cortical activity and its relationship with the subcortex. Consequently, vagal tone may be raised, as is shown by a marked bradycardia and sinus arrhythmia, and probably also by many disturbances of other systems.

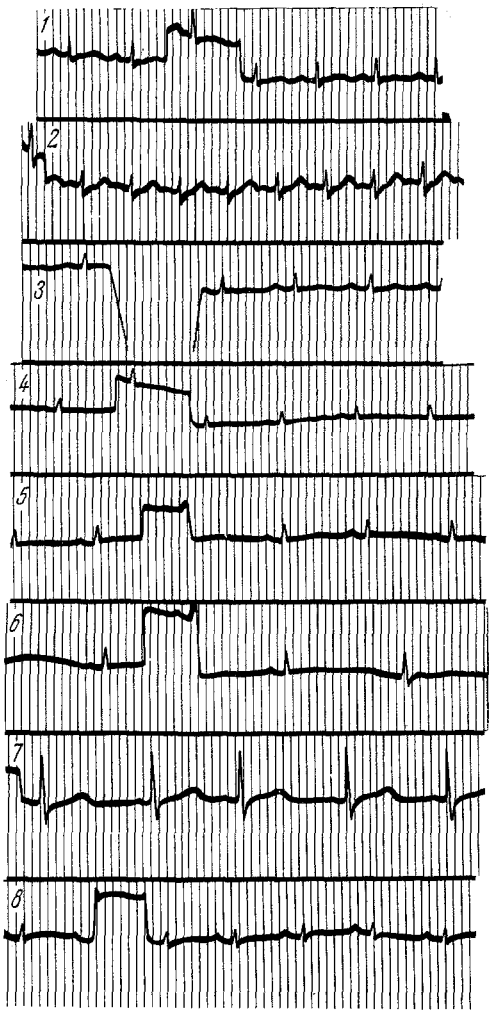

Fig. 3. Development of bradycardia and return of pulse to normal, as measured by EKG, in the ape Khisar. 1, 2) Initial EKG (lead II and CR_4; pulse – 200-184 beats per minute); 3) beginning of bradycardia (pulse 150 beats per minute); 4, 5, 6, 7) subsequent development of bradycardia (pulse – 120-100-86-75 beats per minute); 8) beginning of recovery (pulse – 150 beats per minute). Time-marker – 0.05 second.

SUMMARY

Studies have been made of changes in the heart rate in 79 apes, and it was found to decrease with age. The data were statistically significant. In adult apes a heart rate below 120 per minute indicates bradycardia and above 240, tachycardia.

Neurosis was induced in 4 hamadryad baboons, aged two years, by disturbing their 24-hour rhythm; three developed bradycardia, and in one of them, the heart rate fell to 75 beats per minute, and a respiratory arrhythmia developed. The EKG changes were similar to those occurring in "vagal neurosis" in man. After the experiment, the heart rate returned to normal.

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