CARDIAC ACTIVITY DISEASE BY EXPERIMENTAL INTERFERENCE IN BRAIN PROCESSES

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Summary : In 65 dogs by the method of motor alimentary condition reflexes and delayed reaction, an experimental model of the informational disease was created, which produced chronic increase of the heart rate and significant change in the segment of the ECG.

Key words : informational disease conditional stimuli heart rate ECG

INTRODUCTION

The term Experimental Informational Disease implies the disturbance of higher nervous functions which bring about impairments of the vital activity of the other systems of the organism. These appear during the prolonged exposure of the brain to the conditions of unfavourable combination of the three following factors : 1) a certain amount of information to be processed for decision making, 2) the factor of time allotted for such an activity of brain to occur and 3) the level of motivation determining the significance of information and necessity of its processing. The combination of these three factors (referred later as the informational triad) may be unfavourable for the brain if, firstly, it is necessary to process a great volume of information (including the stage of decision making) in the conditions of prolonged deficit of time allotted and the high level of motivation, or, secondly, if there is lack of information for a long time, while the motivation is high. Thus, in both cases the following triad of factors unfavourably combines and affects the brain : 1) information volume (in the first case it is excessive, in the second case it is scarce), 2) time (in the first case it is insufficient, in the second case it is excessive), 3) motivation (which in both cases is very high, while the hypodynamia appears to be an important factor favouring the decrease of the nervous system stability leading to the informational disease). According to our observations (7), such a decrease of stability besides the

hypodynamia is also due to the breeding of the animals in conditions of broken interspecies relationships, and to the inherited deviations of the lability in higher regions of the brain (according to I.P. Pavlov).

MATERIAL AND METHODS

Experiments were carried out on 65 adult dogs in which delayed reac tions were studied by motor-alimentary methods (Fig. 1). There were three feeders in the experimental chamber (45 mz) placed at a distance of 1.5 m from each other and 3 m from the

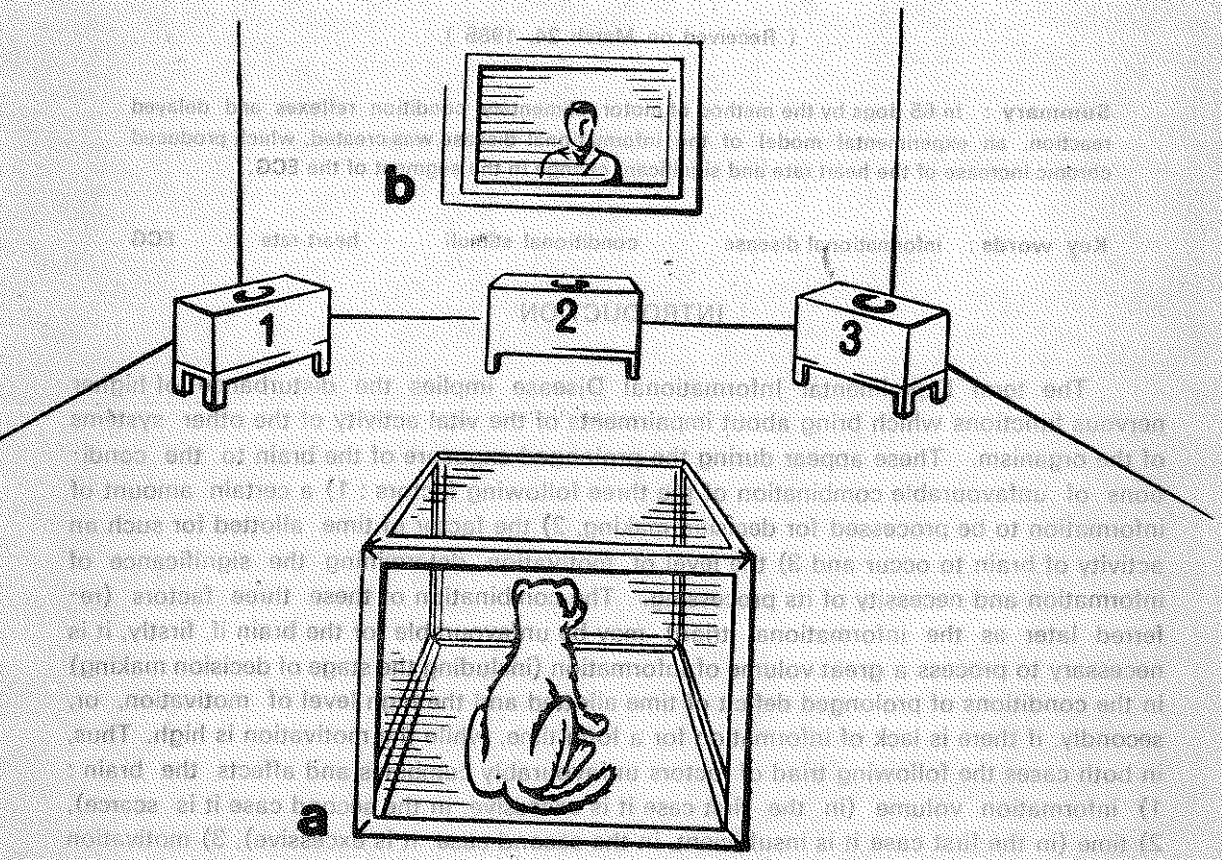

Fig. 1 : Experimental room for the study of delayed reactions and elicitation of the disease :
(a) Starting cage. The front door of the cage is operated by the experimenter.
(b) 1, 2, 3 — Feeders.

starting cage (0.5 mz), the front door of which was opened by the experimenter. The foud-speakers providing conditioned stimuli (tone 500 Hz) were located close to the feeders. Conditioned signal was delivered during 5 sec., the door of the cage was immediately opened after the cessation of sound, and if the animal approached the correct feeder the food reinforcement was provided. During the intersignal intervals the animal was in the cage where it returned after the food reinforcement.

Delayed reactions were studied according to Khanter, referred in literature as the indirect method (2). In order to test the short-term memory function the animal was released from the cage after a lapse of certain time (delay time) from the cessation of the conditioned stimulus. Stimuli trace was considered to be retained in the short-term memory if the animal went to the correct feeder. Each experiment consisted of 8-10 trials with 2-3 minutes intertrial interval.

Experimental informational disease was produced by sharp shortening of intertrial intervals (upto 10-15 sec) between separate tests, It was noted that this procedure of the pathology development was based on the earlier established work deprivation of complex analytical-synthetical activity due to the conditions of constant deficit of time and a high level of motivation leads to the development of morbid state which in the animal model was labelled as experimental informational disease (8).

ECG was registered by lead plate electrodes in chest leads according to Heb modification and was recorded throughout the experiment on ink-writing electroencephalograph, before and after the experiments. Heart rate was registered every second before (background) and after the experiments, in the intersignal intervals during conditioned stimulus and during the delayed reactions. Pattern of the ECG was systematically monitored. The obtained data was processed statistically.

RESULTS

As for the alterations in separate organs of autonomic system in animals with the informational disease, they differ in the various functional systems. However, among them the alterations in cardiovascular system are the most constant and typical.

In normal experimental dogs, i.e., when there was no locomotor activity and no signal stimuli were presented, heart rate was 70-85 per minute. Isolated presentation of positive conditioned stimuli caused a statistically significant increase ($P < 0.001$) of heart rate at 15-25%. While at the presentation of positive stimuli, signaling the delay,

the heart rate increased at 4-7%. This frequency increase was observed during the first two seconds of stimulus presentation, afterwards, during the following 3-4 seconds the heart rate returned to the initial level. During the delay the heart rate remained unchanged. When comparing the rate before and after the experimental session the statistically significant ($P < 0.01$) deceleration was observed. In normal dogs, ECG remained unaltered throughout the experiments.

The neurotized dogs, when placed in the starting cage, were unable of locomotor activity and in the absence of signal stimuli had tachycardia — increase of heart rate at 25-40%, frequency being 110-130 beats per minute. When the conditioned stimuli, signaling the delay were applied, the heart rhythm increased at 15-20%. After the cessation of conditioned signal during the delay (for 20-30 sec) the heart rate was at the same level, but in some cases the increased tachycardia was observed (Fig. 2). Later on

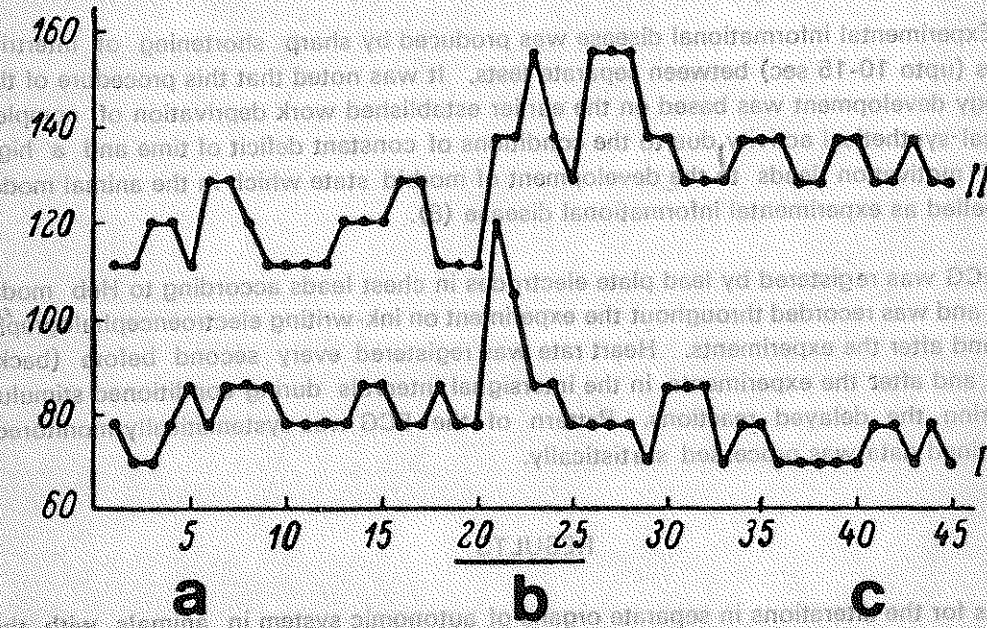

Fig. 2 : Dynamics of heart rate changes during the delayed reactions in normal dogs (I) and in the dogs with informational disease (II) :

On the abscissae — time in sec.

On the ordinate — frequency of heart beats per 1 min.

(a) between the test sessions

(b) during the conditioned signal

(c) during the delay

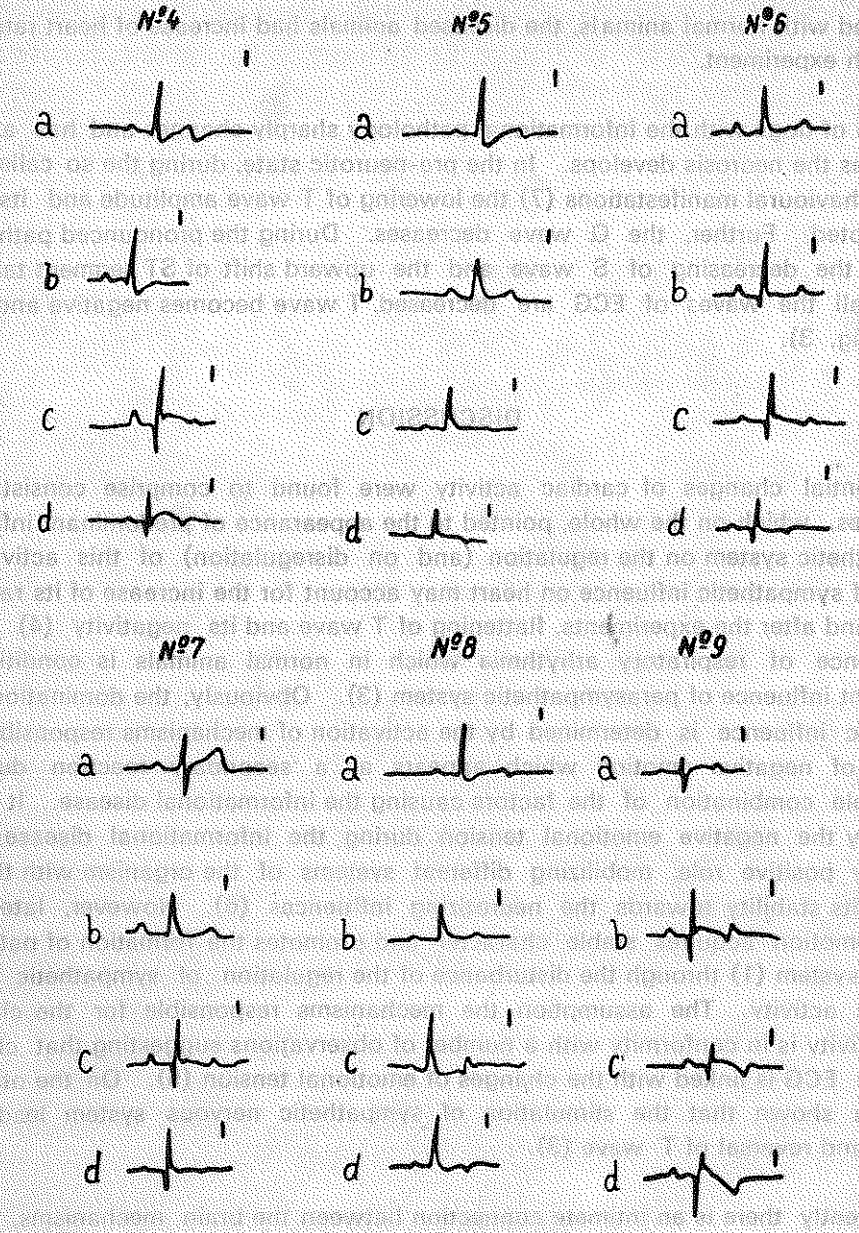

Fig. 3 : Dynamics of ECG changes in :
(a) normal dogs (NN 4, 5, 6, 7, 8, 9)
(b) at the initial stages of informational disease
(c), (d) during the deep pathology
Calibration — 500/aV
Sweep time — 60 mm per sec

as compared with normal animals, the diseased animals had increase of heart rate at the end of each experiment.

ECG of dogs with the informational pathology sharply changes and has a definite dynamics as the neurosis develops. In the pre-neurotic state, during the so called circularity in behavioural manifestations (7) the lowering of T wave amplitude and its flattening are noted. Further, the Q wave decreases. During the pronounced pathology of behaviour the decreasing of S wave and the upward shift of ST segment take place. Later on all the waves of ECG are decreased, T wave becomes negative and Q wave deepens (Fig. 3).

DISCUSSION

Essential changes of cardiac activity were found to comprise consistant ECG disturbances, which on the whole, pointed to the appearance of predominant influence of the sympathetic system on the regulation (and on disregulation) of this activity. The increase of sympathetic influence on heart may account for the increase of its rate during the delay and after the experiments, flattening of T wave and its negativity (4) and the disappearance of respiratory arhythmia which in normal animals is conditioned by predominant influence of parasympathetic system (3). Obviously, the domination of the sympathetic influence is determined by the activation of mechanisms responsible for the formation of negative emotion which appears as a secondary reaction during the unfavourable combination of the factors causing the informational disease. It is known that initially the negative emotional tension during the informational disease plays a biologically positive role, mobilizing different systems of the organism with the aim of increasing its stability towards the neurotizing influences (6). However, later on the negative emotion acquires stable character and promotes the formation of pathological functional system (1) through the disturbance of the regulation of sympathetic influence on cardiac activity. The assumption the mechanisms responsible for the changes of cardiac activity is in conformity with a number of observations suggesting that change of T-wave in ECG is linked with the changes of emotional tension (5). On the other hand, it has been shown that the stimulation of sympathetic nervous system leads to the flattening and reversal of T. wave (3).

Evidently there is an intimate connection between the brain mechanisms, responding to the components of the informational triad and the mechanisms regulating the activity of cardio-vascular system. That is why the disturbances in the higher nervous activity, conditioned by the unfavourable combination of the components of informational triad are first of all manifested in the pathological activity of cardio-vascular system.

As the experimental informational disease in animals is considered as a model of the same disease in humans, the data obtained probably casts light on the growth of different heart diseases during the last two decades, some of them being characterized by excessive loading on the higher functions of the brain.

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