

# A STUDY OF MECHANICAL OSCILLATIONS IN THE PATHOLOGICALLY CHANGED MYOCARDIUM

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UDC 616.127-003.8-092.4

KEY WORDS: human myocardium; mechanical oscillations; frequency-strength curves.

The following signs of myocardial insufficiency were found previously in fragments of human atrial (auricular) myocardium removed during corrective operations for diseases of the heart valves: 1) a fall in amplitude of rhythmic contractions even during stimulation with frequencies of 1-3 Hz (the normal myocardium characteristically responds with an increase in the amplitude of contractions - a positive Bowditch's staircase); 2) a frequency - strength curve of monophasic type (in the normal myocardium it is triphasic); 3) absence of positive or appearance of a negative inotropic effect in response to a decrease in the external sodium ion concentration or after removal of potassium ions from the solution [2].

In the investigation described below a further sign of myocardial insufficiency was discovered, namely the presence of mechanical oscillations of tone arising after the principal contraction induced by an electrical stimulus. Oscillations usually developed in the myocardium of patients with acquired heart diseases and, as a rule, they were absent in the myocardium of patients with congenital heart diseases. Sometimes, however, they did occur in the myocardium of patients with congenital defects.

The object of this investigation was to study mechanical oscillations of tone in the pathologically changed myocardium in a group of patients.

## EXPERIMENTAL METHOD

Preparations from the auricles of the right atrium from 32 patients were studied. The auricle, removed before connection to the artificial circulation apparatus, was immersed in Tyrode solution, oxygenated with carbogen (95% O<sub>2</sub>+5% CO<sub>2</sub>) at room temperature. The tissue was sent from the operating theater to the laboratory in a container. The transportation time was 10-15 min. Trabeculae 3-5 mm long, 1-2 mm wide, and not more than 1 mm thick were removed from the auricle. Ligatures were attached to both ends of the preparation, which was immersed in a working chamber through which Tyrode solution flowed. The preparation was attached at one end to a stationary hook fixed to the floor of the chamber, and with the other end to the rod of a 6MKhIS mechanotron. The initial load on the preparation was 100-200 mg. For 1-1.5 h before the experiment began the trabeculae were stimulated with a frequency of 0.5 Hz. Square pulses, above threshold strength and 20-100 msec in duration, were used. Silver disk stimulating electrodes were arranged along the preparations. The trabeculae were perfused with solution circulating around a closed circuit with the aid of a peristaltic pump. The working chamber, made of Plexiglas, held 2 ml of solution. Throughout the experiment the solution was saturated with carbogen. Tyrode solution of the following composition (in mM) was used: NaCl 131, KCl 4.5, NaHCO<sub>3</sub> 11, NaH<sub>2</sub>PO<sub>4</sub> · 2H<sub>2</sub>O 0.6, MgCl<sub>2</sub> · 6H<sub>2</sub>O 0.25, CaCl<sub>2</sub> · 2H<sub>2</sub>O 2.16; glucose 11; pH 7.4. The temperature of the solution was 32-34°C.

## EXPERIMENTAL RESULTS

On the basis of rhythm and inotropic properties the myocardia of the patients could be divided into four main groups. In group 1 the frequency-strength curve was of the descending monophasic type, i.e., within the frequency range from 0.1 to 1.8 Hz the amplitude of contractions fell steadily to 50 ± 8% of its initial value at 0.1 Hz (100%) (Fig. 1; 12 experiments). In group 2 the curves were biphasic in type: Between 0.1 and 0.7 Hz the amplitude of contractions fell to 75 ± 7%, but later, within the frequency range from 0.9 to 1.2 Hz, the

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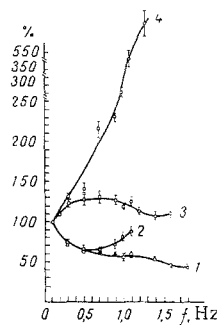


Fig. 1. The four types of frequency-strength curves for the patients' myocardium. 1, 2, 3) Acquired heart diseases; 4) congenital heart diseases. Abscissa, frequency of stimulation of preparations (in Hz); ordinate, amplitude of contractions (in %).

amplitude of contractions increased to  $90 \pm 9\%$  (six experiments). The frequency-strength curves in the myocardium of group 3 had the following appearance: Within the frequency range from 0.1 to 0.7 Hz the amplitude of contractions increased to  $130 \pm 8\%$ , but within the range from 0.7 to 1.4 Hz the amplitude of contractions then fell to  $100 \pm 6\%$  (eight experiments). In group 4 the curves in the frequency range from 0.1 to 1.4 Hz were of the rising monophasic type. The amplitude of contractions increased steadily to  $500 \pm 25\%$  at 1.4 Hz (six preparations). In a few experiments the amplitude of contractions began to fall only at frequencies of between 2.4 and 3 Hz.

Oscillations of tone appearing after the end of the main contractions obtained in response to an electrical stimulus were recorded in the myocardium of group 1. The oscillations appeared at frequencies of stimulation of 0.1–0.5 Hz. It will be clear from Fig. 2 (three experiments) that the amplitude of the first oscillation was  $20 \pm 3\%$  of the amplitude of the main contraction during stimulation with a frequency of 0.1 Hz. With an increase in the frequency of stimulation to 1.6 Hz the amplitude of the oscillations gradually increased to  $42 \pm 6\%$ . In response to stimulation at frequencies higher than 0.7 Hz oscillations were recorded against the background of a marked increase in tone. Oscillations were observed in the myocardium of groups 2 and 3 in 12 of 14 experiments. In the myocardium of group 4 no oscillations were found in any of the six preparations. The period of the oscillations (the time from the maximum of the main contraction to the maximum of the first oscillation) did not change significantly with an increase in frequency (Fig. 2).

However, the myocardium of each group had its own period of oscillation: For group 1 it was  $0.33 \pm 0.05$  sec (at 1.8 Hz), for group 2  $1.02 \pm 0.05$  sec (at 0.7 Hz), and for group 3  $0.51 \pm 0.09$  sec (at 1.4 Hz).

The amplitude of the rhythmic contraction was found to decrease when the interval between contractions coincided in time with the period of the mechanical oscillations. This is shown in Fig. 3 for the myocardium of group 1. If an extra stimulus was applied against the background of a constant rhythm of stimulation of the preparation before oscillation developed, the amplitude of contraction rose sharply (see Fig. 3, I, frequency of stimulation 1.4 Hz). A similar increase in amplitude of contractions was observed when the frequency of stimulation increased to 3 Hz (Fig. 3, II).

A twofold increase in the external calcium concentration, a 40% decrease in the initial sodium ion concentration in the Tyrode solution, removal of potassium ions from the solution, and the addition of strophanthin in a dose of  $1 \times 10^{-6}$  g/ml all caused the development of a positive inotropic effect and increased the amplitude and number of mechanical oscillations.

The investigation showed that the myocardia of patients with congenital and acquired heart diseases could be divided into four main groups on the basis of rhythmic and inotropic properties. A myocardium of groups 1, 2, and 3 was present in patients with acquired heart diseases. The heart tissue of these patients was affected by rheumatic disease. Calcification was present in the heart valves. Cardiomegaly was observed.

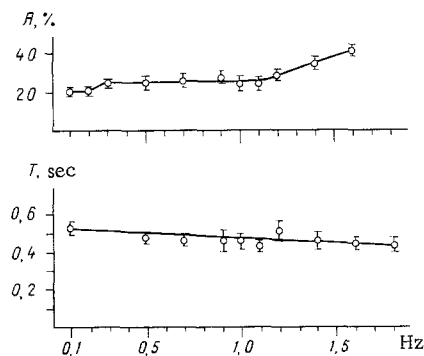


Fig. 2

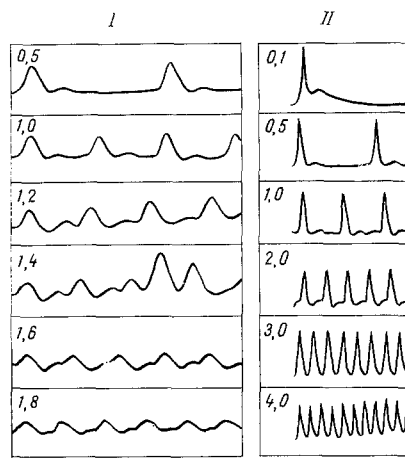


Fig. 3

Fig. 2. Amplitude and period of mechanical observations as functions of frequency of stimulation of myocardial preparations. Abscissa, frequency of stimulation (Hz); ordinate: above — amplitude of contractions (%), below — period of oscillations (in sec) — distance from peak of contraction evoked by electrical stimulus to peak of first oscillation.

Fig. 3. Amplitude of contractions as a function of phase of mechanical oscillations. Frequency of stimulation (in Hz) shown in top left hand corner; time marker below: I) 250 msec, II) 200 msec.

Atrial flutter was present. Patients with congenital heart diseases had a group 4 myocardium. In these patients the heart was normal in size and exhibited a sinus rhythm. Rhythmic and inotropic relations in the group 4 myocardium were similar to those which developed in the normal myocardium of warm-blooded animals, and for that reason the myocardium of these patients was considered to be normal. The investigation showed that mechanical oscillations develop in the pathologically changed myocardium of groups 1, 2, and 3 within the frequency range of 0.1–2 Hz, in Tyrode solution of normal ionic composition and at a temperature of 32–34°C. All procedures which caused the development of a positive inotropic effect in myocardial preparations increased the amplitude and number of mechanical oscillations. These effects were absolutely identical with those established previously in the myocardium of warm-blooded animals as a result of corresponding treatment. Coincidence between the period of oscillations and the interval of the rhythmic contractions led to a decrease in amplitude of the contractions. Analysis of the experimental data showed that the period of oscillations for myocardium of groups 1, 2, and 3 was equal to the period between contractions in the region of the minimum of the frequency–strength curves. The minimum of the frequency–strength curves was thus formed by superposition of rhythmic contractions on the peak of the oscillations. With a further increase in the frequency of stimulation the frequency–strength curves were again shifted upward along the amplitude axis, for the interval between contractions was reduced and they appeared before the development of mechanical oscillations, and as the investigation showed, the amplitude of the contractions increased in these cases. This increase in amplitude of the contractions was probably connected with an increase in activity of the slow sodium–calcium channels of the heart cells [1], which increased the combined contribution of Ca ions from the sarcolemma and the sarcoplasmic reticulum (SR) to the development of contraction.

Mechanical oscillations were observed previously in the myocardium of warm-blooded animals in response to an increase in the Ca ion concentration in the medium [13, 14], lowering of the temperature of the myocardium [1, 3, 5, 10, 11], in the presence of cardiac glycosides [8, 15], and in response to other procedures [12, 13]. However, in Tyrode solution of normal ionic composition, and at a temperature of 32–34°C, no oscillations were found in the myocardium of warm-blooded animals. In the present experiments no oscillations likewise were recorded in the normal myocardium of patients with congenital heart diseases. In accordance with modern views mechanical oscillations in the myocardium of warm-blooded animals developed because the SR of the heart cells, when overloaded with Ca ions, cannot restore the original Ca ion concentration in the myoplasm in the course of a single contraction. As a result of this the Ca ions remaining in the myoplasm after the end of a contraction evoked by an electrical stimulus again potentiate the liberation of Ca from SR and cause the development of mechanical oscillations, unaccompanied by changes in membrane potential [1, 3, 13]. In the pathologically changed myocardium of the patients, in which the sequestering ability of SR to take

up Ca ions is disturbed [9, 14], even that part of the Ca which participates in the formation of contraction under the original conditions is unable to be evacuated from the myoplasm down to its initial level after completion of the evoked contraction. This leads to the development of mechanical oscillations in accordance with mechanisms described for the myocardium of warm-blooded animals [6, 7].

#### LITERATURE CITED

1. K. Yu. Bogdanov, S. I. Zakharov, and L. V. Rozenshtaukh, *Fiziol. Zh. SSSR*, No. 6, 859 (1980).
2. E. G. Vornovitskii, A. A. Galfayan, A. N. Kaidash, et al., *Byull. Éksp. Biol. Med.*, No. 7, 8 (1978).
3. E. G. Vornovitskii and E. Yu. Bychkova, *Byull. Éksp. Biol. Med.*, No. 11, 7 (1980).
4. E. Bozler, *Am. J. Physiol.*, 139, 477 (1943).
5. P. Braveny, J. Sumbera, and V. Kruta, *Arch. Int. Physiol.*, 74, 169 (1966).
6. A. Fabiato and F. Fabiato, *J. Physiol. (London)*, 249, 469 (1975).
7. A. Fabiato and F. Fabiato, *Circulat. Res.*, 40, 119 (1977).
8. G. R. Ferrier, *Circulat. Res.*, 41, 622 (1977).
9. S. Harigaya and A. Schwartz, *Circulat. Res.*, 25, 781 (1969).
10. R. Jensen and B. Katzung, *Nature*, 217, 961 (1968).
11. R. Kaufman, A. Fleckenstein, and H. Antoni, *Pflüg. Arch. Ges. Physiol.*, 278, 435 (1963).
12. J. Kedem, R. Jarom, J. Mahler, et al., *Cardiovasc. Res.*, 6, 353 (1972).
13. D. Masher, *Pflüg. Arch. Ges. Physiol.*, 323, 284 (1971).
14. Z. I. Penefsky et al., in: *Myocardial Biology*, Baltimore (1974), pp. 31-39.
15. M. Reiter, *Arch. Pharmacol. Exp. Pathol.*, 242, 497 (1962).

#### INTRACEREBRAL PRESSURE AND EEG DURING MEASURED CHANGE OF THE CSF VOLUME

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UDC 616.831-008.918+616.831-073.  
097]-02:616.832,9-008.811

**KEY WORDS:** changes in CSF volume; CSF pressure; intracerebral pressure; EEG; power spectrum of EEG.

As a rule the clinical state of neurosurgical patients and the effectiveness of their treatment are assessed on the basis of the results of a neurological examination, investigation of the state of the brain function by electroencephalography, and determination of the level of intracranial hypertension, assessed as changes in the CSF pressure (CSFP). Evidence has now been obtained that the CSFP is not identical with the pressure inside the brain tissue and does not always accurately reflect mechanical pressures developing in it [2, 3, 7, 8].

To study the biophysical characteristics of brain tissue methods based on a measured change in the CSF volume have been developed [4-6].

This paper describes the results of an investigation of the effect of a measured change in the CSF volume on the clinical state of the patient, the CSFP and the intracerebral interstitial fluid pressure (ICIFP), and on the EEG for the purpose of comparing the clinical and physiological data and of determining the advisability of an artificial change in the volume and pressure of the CSF in the acute postoperative period.

#### EXPERIMENTAL METHOD

Altogether 31 patients were studied after removal of supra- and subtentorial brain tumors (basal meningiomas in 14 patients, intracerebral tumors in 6, tumors in the region of the posterior cranial fossa in 11).

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