SEGMENTAL CONCENTRACTILITY OF THE LEFT VENTRICLE DURING CARDIAC ARRHYTHMIAS

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UDC 616.12-008.318-07:616.124.2-008.3-07

KEY WORDS: arrhythmias; nonhomogeneity; intramural tension; left ventricle; apex

One of the distinguishing features of contractions of the left ventricle (LV) during arrhythmias is nonhomogeneity of the distribution of tension in its different parts [6], and which can be studied by echolocation [1], by measurement of deformation [7, 9], by methods involving the use of models [8], and by recording intramural tension [2, 4]. However, the particular features of the nonhomogeneities of tension arising under these circumstances have not yet been adequately studied.

The aim of this investigation was to study the redistribution of tensions in the wall of LV and in the apex of the heart with the onset of different kinds of arrhythmias, and to determine the effect of these redistributions on the homodynamic parameters of the heart.

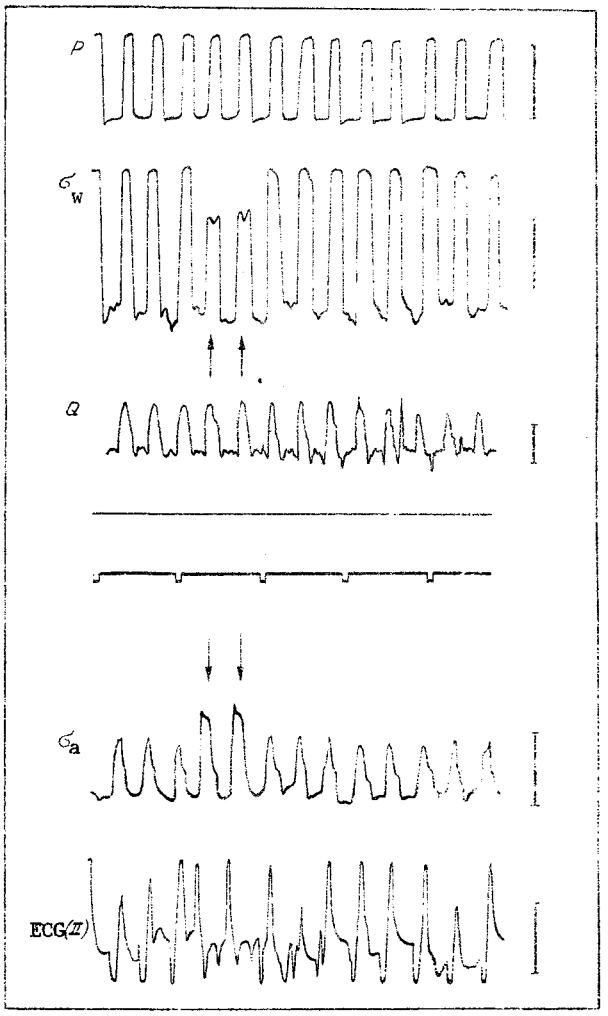

Fig. 1. Changes in functions P(t), $\sigma_w(t)$, $\sigma_a(t)$, Q(t), and ECG (II) with onset of ventricular parasystole. Calibration: P) $2 \cdot 10^4$ Pa, σ_w and σ_a) $0.6 \cdot 10^4$ Pa. ECG 1 mV, time 1 sec.

Department of Medical and Biological Physics, I. M. Sechenov First Moscow Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 110, No. 11, pp. 453-455, November, 1990. Original article submitted June 6, 1989.

 0007-4888/90/0011-1454$12.50

EXPERIMENTAL METHOD

Experiments were carried out on 9 anesthetized (trimeperidine 5 mg/kg, pentobarbital, 10-15 mg/kg) mongrel dogs, male and female, weighing 12-20 kg. Thoracotomy was performed and the pericardium opened during artificial ventilation of the lungs by the RO-2 apparatus (50 cm^3/kg, 16-17 inspirations/min). During the experiments the pressure (P) inside the left ventricle and aorta was recorded by means of catheters, the velocity of the blood flow (Q) was measured by means of a fluorometer (Nicotron, Norway), the ECG was recorded in standard leads II and III, and segmental contractility was estimated on the basis of local tension of concentrations in the wall of LV (σ_w) and in the region of the apex of the heart (σ_a). A latex rubber balloon, filled with liquid, was used as the transducer for σ. Full details of the method of measuring σ were described by the writer previously [4]. Output parameters were recorded by means of 6 NEK-4 and "Suney" monitors (Japan). After each experiment, the positions of the transducers in the structure of the myocardium was verified at autopsy. Arrhythmias were produced by intravenous injections of a 1:1000 solution of adrenalin in a dose of 0.2-0.8 ml or a 5% solution of ephedrine in a dose of 0.5 to 2 ml. The experiments were carried out at the Institute of General Resuscitation, Academy of Medical Sciences of the USSR.

EXPERIMENTAL RESULTS

In one series of experiments, segmental nonhomogeneity of contractility of the wall of LV and apex of the heart, due to different types of arrhythmias, was observed. In some cases, however, the hemodynamic parameters were compensated by tension nonhomogeneities, whereas in others compensation of this kind was absent.

The results in Fig. 1 show that after injection of the 1:1000 adrenalin solution (0.5 ml) centricular paroxysmal tachycardia developed at the second minute, with R−R = 0.36 sec. Under these circumstances P in LV reached $2.42 \cdot 10^4$ Pa, σ_w $1.38 \cdot 10^4$ Pa, and σ_a $0.4 \cdot 10^4$ Pa. The stroke ejection volume was 6.7 cm^3.

At one time ventricular parasystole appeared on the ECG (indicated by arrows in Fig. 1), the ectopic center for this pair probably arising in the right ventricle nearer to the apex. This leads to a sharp increase in σ_a for the two corresponding contractions, up to $0.78 \cdot 10^4$ Pa, i.e., twice the value for the previous contractions. A decrease of tension in the wall to $1.05 \cdot 10^4$ Pa, i.e., 1.3 times lower than the previous contraction, corresponded to these same contractions. With these sharp changes in tension, the values of the changes in the hemodynamic parameters of the heart remained virtually unchanged: P for these two contractions remained at $2.42 \cdot 10^4$ Pa and the stroke ejection volume at 6.8 cm^3 for the first and 6.7 cm^3 for the second contraction. In other words, the work done by the heart remained unchanged for each of the following contractions.

Closely similar results also were obtained in two other series, both for parasystoles, which occurred altogether 9 times, and for single extrasystoles, observed in 14 cases.

With the onset of arrhythmias cases were observed when, to compensate the stroke ejection, only one segment changes its operating schedule. This example is shown in Fig. 2, when 1 ml of 5% ephedrine solution was injected intravenously. As a result, an exit block from the atrial center appeared against the background of sinus rhythm. Moreover, R−R changes from 0.48 to 0.72 with a jump. Thus the duration of total diastole and the end-diastolic blood volume, as well as the preload, all increased. During the subsequent contraction, in accordance with Starling's law, the stroke ejection was increased by 1.54 times to reach 11 cm^3 (initially 7.2 cm^3).

The mechanism of realization of this increased ejection is very interesting. The most acceptable mechanism is a uniform increase in myocardial tension throughout the structure of LV. However, this did not happen. Initially, before the abnormal contraction (indicated by an arrow) the apex and wall of LV as usual worked on somewhat different schedules. (σ_w) had a characteristic plateau of the quasi-isotonic regime. Apical tension continued to rise after opening of the aortic valve up to a maximum, rising quite steeply in the rapid ejection phase, when it reached $0.75 \cdot 10^4$ Pa. At the time of the increase in ejection the work pattern of the apex remained virtually unchanged: σ_a increased only by 1.1 times, and σ_w changed back to its initial value, at the previous rate, up to $0.83 \cdot 10^4$ Pa. This was also the amplitude of the plateau. In an abnormal contraction with this value σ_w did not flatten out on a

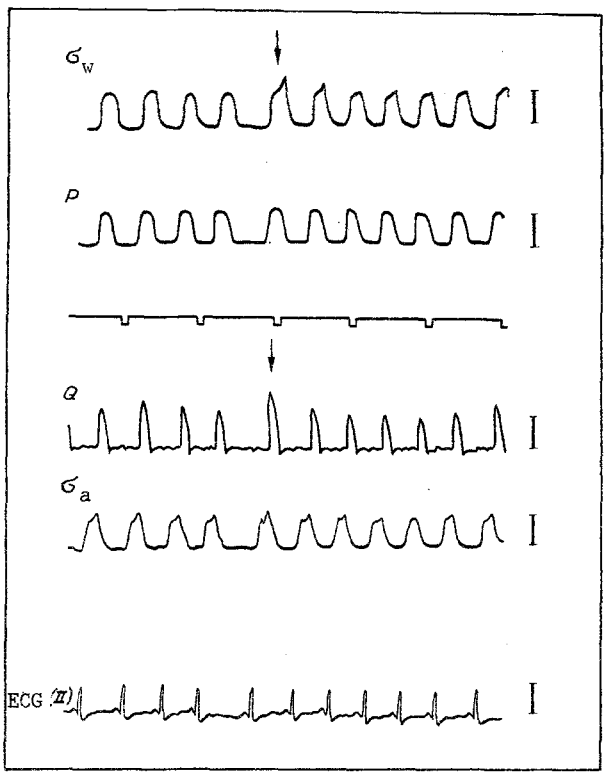

Fig. 2

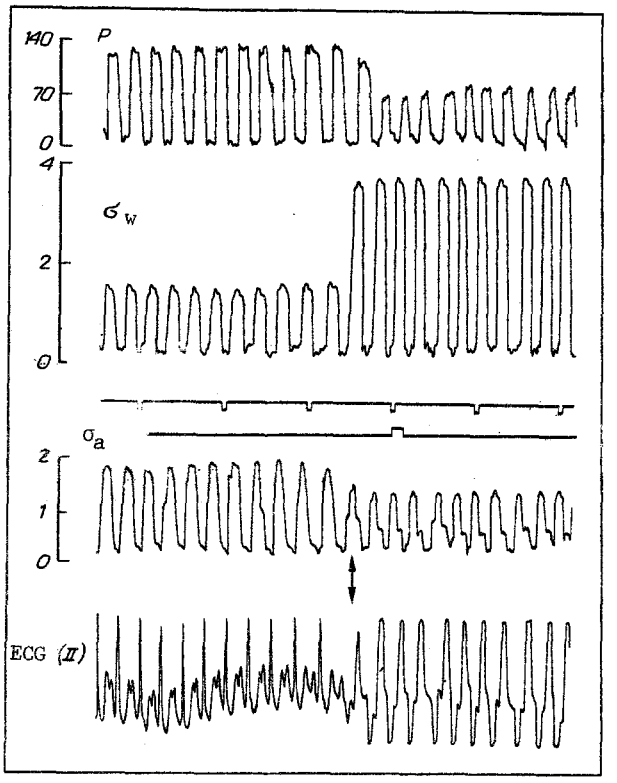

Fig. 3

Fig. 2. Changes in functions P(t), $\sigma_w(t)$, $\sigma_a(t)$, Q(t), and ECG (II) with an exit block from the atrial center. Calibration: P) $1.5 \cdot 10^4$ Pa, σ_w and σ_a $0.75 \cdot 10^4$ Pa, ECG 1 mV, time 1 sec.

Fig. 3. Changes in functions P(t), $\sigma_w(t)$, $\sigma_a(t)$, and ECG (II) on the appearance of an ectopic focus of excitation. P given in mm Hg, σ_w and $\sigma_a \cdot 10^4$ Pa; calibration of ECG: 1 mV, time 1 sec.

plateau but continued to rise virtually in a straight line to $1.3 \cdot 10^4$ Pa, i.e., it was increased by 1.6 times. The rate of growth of σ_w in the ejection phase was $2.65 \cdot 10^4$ Pa/sec. The next contraction also was a little longer: 0.56 sec, and σ_w increased its amplitude by a lesser degree (by 1.3 times) and, as a result, ejection was increased by 1.2 times. Later the process became stabilized and σ_w adopted a quasi-isotonic regime. In this case σ_a preserved its magnitude and shape throughout all the changes described. Thus compensation of the hemodynamic change (increased ejection) took place not due to an equal increase in tension of the whole structure of LV, but due to the appearance of segmental nonhomogeneity with respect to tension. Moreover, virtually the whole increase in stroke volume took place on account of the increase in σ_w, and the apex preserved its constant pattern.

Compensation of the hemodynamic parameters due to segmental nonhomogeneity is a statistically special case. In our series it was found in 70% of cases, when this particular type of arrhythmia occurred. This type of compensation takes place more frequently through the appearance of segmental nonhomogeneity than of a uniform increase of tension of the whole LV.

Segmental nonhomogeneity in an ectopic focus of excitation arising as a result of injection of 1:1000 adrenalin solution in a dose of 0.5 ml was recorded in Fig. 3. After 12 sec a positive inotropic effect began to appear at the apex, and after 21 sec σ_a reached its peak value (about 1.5 times its initial level). At the same time of 21 sec, the inotropic action of adrenalin in the wall of LV was weak – only 1.1 times its initial value. Under these circumstances P rose slightly to 1.2. Normal complexes of sinus origin were recorded on the ECG. After 21 sec an ectopic focus of excitation appeared in LV (Fig. 3, arrow), and this was recorded on the ECG [3], and localized on the basis of the extracellular potentials (EP). The technique of recording EP was described in [5]. EP appeared sooner at the site of the σ_w transducer than at the apex. Contraction of LV caused by the ectopic focus

had the following characteristics: σ_w rose in a jump by 2.3 times its value for the previous contraction, whereas σ_a fell in a jump to 0.7 of the previous contraction, P increased in a jump to 0.8 of the previous contraction, phase relations between the functions $\sigma_w(t)$, $\sigma_a(t)$, and P(t) were altered, and P increased to 1.8-2 times its initial value after 5-10 sec. The same effects were recorded in experiments on another three hearts. A sharp local increase in σ_w led to redistribution of tensions throughout the structure of the left ventricular myocardium. A synchronized fall of σ_a was observed. At the same time P in LV fell and, consequently, the pressure in the coronary arteries decresed. At the site of the focus of excitation, the local muscle tension could therefore exceed the blood pressure in the coronary vessels in this area, and this may contribute to the development of the destructive changes in the myocardium.

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