cell probably mainly initiates mechanisms preventing a marked rise in the RE level under these conditions. Meanwhile, under the influence of PGE_2 a broader spectrum of changes in the state of the redox systems of the neurons developed. In this case the pattern of unit activity was characteristically altered.

The variability of the cell responses is evidently associated both with differences in the type of the neurons identified and also with their original functional state. Further investigations will be devoted to the solution of this problem.

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FACTORS INFLUENCING THE ELASTIC RESISTANCE OF THE AORTIC COMPRESSION CHAMBER

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Elastic resistance plays an important role in optimization of the cardiac contraction during ejection of blood from the left ventricle into the aorta. This value has a significant effect on the level of the input impedance of the arterial system [4] which, as we know [12], determines the afterload on the left ventricle.

According to Frank's definition [9] the elastic resistance (E) of the aortic compression chamber (ACC) is obtained by the equation

$$E = \frac{\Delta P}{\Delta V},$$

where ΔP is the pulse pressure (the difference between the systolic P_s and diastolic P_d arterial pressure); ΔV the increase in volume of the ACC during the period (s) of ejection of blood from the ventricle. However, no easy method has yet been devised for determining the value of ΔV and, consequently, the value of E. Some workers [8, 10, 14] have attempted to use for this purpose analysis of the wave phenomena arising in ACC during cardiac ejection. This approach to the problem proved ultimately to be not completely satisfactory: The values of E calculated by equations deduced by the authors cited above differed, other conditions being the same, by as much as 50-100%. Accordingly attempts to determine E by using only the principal hemodynamic indices appear to be more promising. This problem is solved in the present communication from this standpoint.

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Let us examine a classical single-chamber model of ACC [9] in which the pressure P(t) changes in the course of the cardiac cycle from P_d to P_s. The pressure P(t) in ACC can be found by solving the following known [5] equation:

$$\frac{dP}{dt} = E(Q(t) - \frac{P(t)}{R}, \tag{1}$$

where Q(t) is the inflow of blood into the aorta and R the peripheral resistance.

The pressure P(t) in the aorta reaches its maximum P_s at the beginning of the period of ejection of blood from the left ventricle [2], after which it does not change significantly until the end of this period. This character of the change in pressure during the ejection period can be derived from (1), when the blood flow Q(t) has the following form:

$$Q(t) = Q_s\left[\frac{\alpha}{\tau}(\theta(t) - \theta(t-\tau) + \frac{1-\alpha}{s-\tau}(\theta(t-\tau) - \theta(t-s)\right], \tag{2}$$

where Q_s is the stroke volume; τ the time from the beginning of the ejection period to the time of reaching maximal pressure; $\theta(t)$ the Heaviside function [1]; the coefficient α is determined by the equation

$$\alpha = 1 - \frac{P_s(s-\tau)}{RQ_s}. \tag{3}$$

The solution to Eq. (1) in the region of the initial rise in pressure ($0 < t < \tau$) is accordingly expressed as follows:

$$P(t) = P_d e^{-mt} = \frac{\alpha RQ_s}{\tau}(1 - e^{-mt}), \tag{4}$$

where

$$m = E/R. \tag{5}$$

In diastole, when there is no cardiac ejection, i.e., Q(t) = 0, the solution to equation (1) has the form

$$P(t) = P_s e^{-m(t-s)}.$$

Substituting in this solution t = C (C is the duration of the cardiac cycle) and considering that at the end of diastole the pressure is P_d, by analogy with [5] we obtain:

$$m = \ln(P_s/P_d)\cdot D^{-1}, \tag{6}$$

where D = C − s.

By means of Eqs. (5) and (6), E can be represented in the following general form:

$$E = R\ln(P_s/P_d)\cdot D^{-1}. \tag{7}$$

Equation (7) is not perfectly convenient for undertaking practical calculations of E. This is mainly because it includes the term R. To find this term, by substituting $t = \tau$ in (4) and considering (3), the following equation is obtained:

$$R = \frac{P_s\cdot s - e^{-m\tau}(P_s(s-\tau) + \tau P_d)}{Q_s(1 - e^{-m\tau})}. \tag{8}$$

This in turn can be simplified, because τ is sufficiently small [2]:

$$R = \frac{P_s\cdot s + \Delta P/m}{Q_s}. \tag{9}$$

Further, if the function $\ln\chi$ is represented by continued fractions [7], Eq. (6) can be represented in the form

$$m = \frac{\Delta P}{P_m\cdot D}, \tag{10}$$

where P_m is the mean arterial pressure.

It will be noted that Eq. (10) is analogous to that deduced by Savitskii [6] by a different method.

Substituting (10) in (9) we obtain the final equation for R:

$$R = \frac{P_m D + P_s \cdot s}{Q_s}. \quad (11)$$

Next, by substituting (10) and (11) in equation (5), we obtain an equation for E:

$$E = \Delta P \left(1 + \frac{P_s \cdot s}{P_m D}\right) \cdot Q_s^{-1}. \quad (12)$$

The value of the elastic resistance of the ACC is thus determined both by the ratio $\Delta P/Q_S$ and the correction coefficient $qR = P_S S/P_m D$. The coefficient qR, in accordance with [13], characterizes the ratio of the systolic and diastolic outflow of blood from ACC into the capillaries. It is very individual and has a substantial influence on the value of E. For instance during changes in heart rate from 50 to 100 beats/min under normal conditions s/D varies from 0.3 to 0.6. The value of P_S/P_m at normal arterial pressure varies from 1.22 to 1.28, falling in hypotension to 1.15 and rising during hypertension to 1.4. All this indicates that there is no justification for representing qR as a constant value [3, 10, 14].

In conclusion it can be stated that during an investigation of 37 healthy persons the value of E, calculated by Eq. (12) varied from 806 to 1642 dynes/cm^5, with a mean value of 1296 ± 182 dynes/cm^5. This value is close to that given in the literature [11, 13], where E was determined in man by a direct method.

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