EFFECT OF INCREASED PRESSURE IN THE UPPER RESPIRATORY PASSAGES OF DOGS ON BLOOD PRESSURE IN THE RIGHT VENTRICLE

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When the pressure in the upper respiratory passages is raised to 20, 30, and 40 mm Hg, an equal increase was found in the systolic and diastolic pressure in the right ventricle, the difference between them being constant. It is concluded that the increase in pressure in the right ventricle is due to transmission of pressure by the dilated lungs and not by an increase in strength of the contractions of the right ventricle.

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An important role in the hemodynamic reaction of the body to raised intrapulmonary pressure is played by the right ventricle, which forces blood along the compressed pulmonary vessels [4, 7, 8]. The concept of functional overloading of the right ventricle under these conditions has been accepted without any special evidence for it. Electrocardiographic findings have been explained by overloading of the right side of the heart [1] or by changes in the position of the hear within the chest [2, 5]. Clinicians consider that one sign of increased work of the right ventricle is a high systolic pressure associated with low diastolic pressure [3].

We studied changes in pressure in the right ventricle when the pressure in the upper respiratory passages was raised.

EXPERIMENTAL METHOD

The main blood vessels in dogs were catheterized. A constant positive excess pressure (EP) of 20, 30, and 40 mm Hg in the upper respiratory passages was created by the oxygen pressure regulator while the animal breathed for 5 min in an airtight helmet. The blood pressure (BP), pulse rate (from the ECG), and respiration rate were recorded on an oscillograph with a jet-recording system.

EXPERIMENTAL RESULTS

An increase in pressure in the upper respiratory passages was accompanied by an increase in systolic and diastolic pressure in the right ventricle, $t > t_{001}$ (Table 1). The positive correlation was preserved between systolic and diastolic pressures. The coefficients of correlation (ρ) varied from +0.42 to +0.52 at inspiration and from +0.64 to +0.8 at expiration, with a level of significance $P < 0.05$.

The difference in BP during cardiac systole and diastole showed a tendency to increase slightly, but statistical analysis showed that its difference from the initial value (when EP was 0) was not significant ($t < t_{05}$). This important physiological index thus remained practically unchanged when the pressure in the lungs was increased.

In control model experiments the BP in the right ventricle and the pericardial pressure were measured simultaneously in living and dead dogs. The pericardial pressure in intact dogs depended on the phase of the cardiac cycle. The systolic pericardial pressure was 27-37 mm Hg higher than the EP in the lungs, and the diastolic pericardial pressure repeated the value of the raised intrapulmonary pressure (with an accuracy of within a few mm Hg). In dead dogs the pericardial pressure followed fairly closely after changes in the intrapulmonary pressure (remaining constantly 4-5 mm Hg below it). The intraventricular pressure was considerably below the EP in the lungs (6-8 mm Hg). This difference in level was probably due to the fact that part of the transmitted pressure was lost in overcoming the elasticity of the heart

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TABLE 1. Effect of Intrapulmonary Pressure on Pressure in Right Ventricle of Dogs.

EP in lungs (in mm Hg)	Phase of cardiac cycle	At inspiration	At expiration
0	Systole	26±2	36±2
20		44±3	64±6
30		49±4	66±4
40		53±4	71±5
0	Diastole	−6±1	4±1
20		3±1	21±3
30		12±2	28±3
40		18±2	32±3

and lung tissues. The increase in BP in the right ventricle on account of the pressure in the lungs evidently took place without any particular expenditure of muscular energy by the right heart. Other workers have shown experimentally that the tone of the muscle of the right ventricle was reduced during respiration under an EP in the lungs and rose sharply after the chest was opened [6].

The systolic pressure in the left ventricle rose by 5-10 mm Hg, and the diastolic by 20-30 mm Hg. The BP in the abdominal aorta and femoral artery, after a transient initial fall, remained near the original level. The increase in BP in the veins was 3-10 mm Hg less than the increase in intrapulmonary EP. A distorted reaction of the venous BP to respiration was observed. Whereas under normal conditions the BP in the abdominal veins increases at inspiration by 5-6 mm Hg, when the intrapulmonary pressure was raised it fell sharply, by 13-20 mm Hg. The increase in BP in the right ventricle was thus only one component of a general systemic circulatory reaction to elevation of the intrapulmonary pressure. It can be postulated that the principal changes in the systemic circulation take place on account of limitation of the venous return because of the raised intrathoracic pressure.

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