The Effect of Moderate Changes in Blood Volume on Left and Right Atrial Pressures

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The blood volume of 15 dogs was progressively increased an estimated 30 per cent by repeated blood infusions and decreased 30 per cent by stepwise hemorrhage. The pressures in the venous system, both atria and the pulmonary artery, rose and fell in unison with these moderate changes in blood volume and the new levels remained stable for at least 10 to 20 minutes.

Although the right ventricle anatomically separates the systemic venous bed from the pulmonary circulation, small hemorrhages and transfusions have so little effect on its activity that, from the point of view of pressure volume relationships, the systemic veins and the left atrium are parts of the same functional unit.

The preceding paper presented evidence that stretch receptors in the left atrium may be part of a mechanism linking changes in actively circulating blood volume with homeostatic responses of the kidney. This focuses attention on the effects of moderate changes in blood volume on intrathoracic vascular pressures and especially those in the left atrium. The present work describes simultaneous measurements of systemic venous, pulmonary arterial, left ventricular diastolic and systemic arterial pressures 10 to 20 minutes following moderate changes in blood volume.

Methods

Fifteen healthy mongrels ranging in weight from 15 to 30 pounds were employed. Light morphine-chloralose anesthesia was employed, as is described elsewhere. The animals were laid on their sides without restraint and pressure measurements were made through catheters in the femoral artery, the right atrium (via femoral vein), the pulmonary artery (via jugular vein) and the left ventricle (via carotid artery). Location of the catheters in the pulmonary artery and right atrium was confirmed by fluoroscopy. Pressures were measured by Statham Type P-23 strain gages placed at heart level. Photographic recording was used and mean pressures determined by planimetry of pressure curves during a full respiratory cycle. Pressures were measured 10 to 20 minutes after each infusion or hemorrhage to give time for redistribution of the blood volume and for possible readjustments of vascular tone.

Blood was withdrawn into syringes containing heparin and stored at 4°C until needed. To minimize acute fluctuations in vascular pressures the rate was 30 cc./minute. Bleeding was in stages from the femoral artery and the amounts were usually 10, 20 and 30, but occasionally, 40 per cent of the estimated blood volume. It was assumed that this is approximately 10 per cent of the body weight on the basis of the observations of Wang and Associates on the blood volume of mongrel dogs. The consistency of the data in the final mean curves justifies this rough approximation.

In some cases, reinfusion of blood withdrawn several days previously preceded the bleeding; in others, this extra blood was reinfused after returning that withdrawn during the hemorrhage phase of the experiment. Blood for reinfusion was usually obtained from the same animal two to four days prior to the experiment by bleeding into a sterile bottle containing heparin.

Results

The data for the pressures in the pulmonary artery, the two atria and a systemic artery are presented in figure 1, which represents the mean of 15 experiments. All pressures were taken 10 to 20 minutes after completion of the volume changes.

Right atrial pressure (CVP) showed a well marked increase or decrease as the blood volume was changed. The mean pressure attained a peak of 7.7 cm. water with a 30 per cent infusion falling to minus 3.5 cm. water with 30 per cent blood loss.
Mean pulmonary artery pressure (PAP) changed by approximately the same amount as the pressure in the right atrium; the highest value, 19.8 and the lowest, 10.8, differing by 9.0 cm. water. Apart from the high value for the 20 per cent infusion, the points fell close to a line paralleling the right atrial pressure changes.

Left atrial (LVDP) pressures were estimated from left ventricular end-diastolic pressures. There was in all cases a fall in the pressure following hemorrhage, resulting in a mean low value of minus 1.6 cm. water with an increase to a 13.2 cm. water peak following infusion. The total range was 14.8 cm. water for a change in blood volume from minus 30 to plus 30 per cent. There is an extra increase in the left atrial pressure in the higher blood volume ranges and the line of pressure change slopes upward more sharply for this atrium than the right.

Although the values in the low pressure system almost invariably decreased with hemorrhage and increased with infusion, in the systemic artery in seven cases out of the twelve submitted to hemorrhage, the pressure remained unchanged or actually increased. As a result, the mean systemic arterial pressure (AP) changed little with the first 20 per cent loss and fell by only 8 mm. Hg from the control value of 113 mm. Hg with 30 per cent hemorrhage. In 3 cases out of the 11 given transfusions the systemic arterial pressure fell, although there was no evidence of pyrogen or transfusion reaction. Hence the mean pressure rose by only 14 mm. Hg with the 30 per cent increase in volume.

**DISCUSSION**

The data in this work with animals, under morphine and chloralose narcosis, accords well with those obtained with conscious human beings. The decrease in pressure differential between the pulmonary artery and the left atrium as the blood volume increased has also been observed in humans following rapid saline infusions. Like Doyle, and co-workers, we would attribute the decreased differential to a decreased flow resistance in the distended pulmonary vascular bed.

The most striking impression given by the curves in figure 1 is their parallelism. Since the central venous pressure and the pulmonary arterial pressure lines remain equidistant throughout, the pattern of activity of the right ventricle has not changed significantly with the changes in filling pressure. Although the right ventricle anatomically separates the systemic venous bed from the pulmonary circulation, small hemorrhages and transfusions have so little effect on its activity that it does not disturb pressure volume relationships.

In the +15 to −15 per cent range of changes in blood volume the response of the entire low pressure portion of the cardiovascular system was as though the blood was contained in a single functional unit. Hence in this volume
range right and left atrial pressures reliably reflect changes in blood volume. However, during more severe changes in excess of 15 per cent, the pressure curves flatten out, suggesting that the venous system has actively altered its tone. It is well known that after severe blood volume losses the venous pressure may even return to control values.8

The overall changes in pressure of 10 to 15 cm. water are so large that minor inaccuracies involved in the use of end diastolic left ventricular pressure instead of effective left atrial pressures do not affect the conclusions. Indeed in experiments in men involving a moderate loss of effective blood volume, Warren, Brannon, Stead and Merrill6 could find no significant difference between central venous pressure and true effective atrial pressure as determined by simultaneous atrial and intrapleural pressure measurements.

An increase in systemic arterial pressure following hemorrhage has been noted in studies of moderate blood loss in unanaesthetized men18 and animals.11 Bazett12 and Armin and Grant11 have attributed this rise to responses initiated by the pressure fall in the great veins. However, the sensitive region could be more extensive than this and the left atrium should also be considered. Our results suggest that for small blood volume changes, the whole low pressure system normally acts in unison as though it were an elastic reservoir. The atria are the most distensible portions of this system, hence stretch receptors located in them, which may lead to the release of vasoconstrictor substances11 and influence urine flow10 or systemic arterial pressure,12 would be effectively stimulated by changes of total blood volume or its distribution.

**Summary**

The blood volume of 15 dogs was increased an estimated 30 per cent by repeated blood infusions and decreased 30 per cent by stepwise hemorrhage. Systemic venous, pulmonary arterial, left ventricular diastolic and systemic arterial pressures were measured 10 to 20 minutes after each blood volume change.

As a result of the 60 per cent total change in blood volume, systemic venous pressure was altered by 11.2 cm. water, pulmonary arterial pressure by 9.0 cm. water, left ventricular diastolic pressure by 14.8 cm. water and systemic arterial pressure by 29 cm. water or 22 mm. Hg. These changes persisted for at least 15 to 20 minutes.

In spite of the interposition of the right ventricle, the systemic veins and lung bed act as though they were one continuous system. Hence stretch receptors in the left as well as the right atrium are directly affected by moderate changes in blood volume.

**Summario in Interlingua**

Le volumine sanguinee de 15 canes esseva augmentate per estimatemente 30 pro cento per medio de repetite transfusiones de sanguine e reducete per 30 pro cento per medio de hemorragias serial. Dece a 20 minutas post omne alteration del volumine de sanguine nos mesurava le pressiones venose systemic, pulmono-arterial, sinistro-ventricular diastolic, e arterial systemic.

Como resultato del alteration total de 60 pro cento in le volumine de sanguine, le pression venose systemic esseva alterate per 11,2 cm H2O, le pression pulmono-arterial per 9,0 cm H2O, le pression sinistro-ventricular diastolic per 14,8 cm H2O, e le pression arterial systemic per 29 cm H2O o 22 mm Hg. Iste alterationes persisteva durante al minus 15 a 20 minutas.

In despecto del interjacent ventriculo dextere, le venas systemic e le lecto pulmonar age como si illos eseva un sol systema continue. Per consequente, tensioneceptores in le atrio tanto dextere como etiam sinistre es directamente afficite per moderate alterationes del volumine de sanguine.

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Effect of Norepinephrine on Cardiac Output

While the influence of norepinephrine on cardiac output has been repeatedly investigated, the results have been rather discordant. In view of the frequently stated view that the elevation of arterial pressure is accompanied by no change or a slight reduction of cardiac output, it is of interest to note that Belgian investigators have recently reported the l-norepinephrine in doses of 1 to 30 γ/Kg. increased cardiac output fairly proportionally to the injected doses in dogs, anesthetized with morphine and chloralose. Injection of 5 to 10 γ/Kg. seemed to produce optimal effects.

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