Increase in mean circulatory pressure in Goldblatt hypertension

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RICHARDSON, TRAVIS Q., JOSE D. FERMOSO, AND ARTHUR C. GUYTON. Increase in mean circulatory pressure in Goldblatt hypertension. Am. J. Physiol. 207(4): 751-752. 1964.—Ten dogs were made hypertensive by the Goldblatt technique. The mean arterial pressure increased from an average control value of 117 mm Hg up to 161 mm Hg. After the animals had become hypertensive, the mean circulatory pressures were measured. These averaged 9.03 (±0.31 SEM) mm Hg, compared with measurements of mean circulatory pressure in ten normal dogs which averaged 7.0 (±0.17 SEM) mm Hg. The increase in mean circulatory pressure was highly significant (t = 5.75).

Since it is not possible to measure all the different pressures in the circulatory system to determine the mean pressure under dynamic conditions, another method has been devised to measure the mean pressure of the circulatory system (7). Briefly, this method necessitates stopping the heart either by fibrillating it or by injecting acetylcholine and quickly pumping blood from the arterial to the venous system, until an equilibrium of pressures is effected. The equilibrated pressure is termed "mean circulatory pressure" and is a reasonable measure of the mean pressure present in the circulatory system under normal, dynamic conditions.

Previous studies in this laboratory have demonstrated the importance of the mean circulatory pressure as a major factor influencing venous return and cardiac output (5, 6, 8, 9). Also, the effects of changing various circulatory parameters on mean circulatory pressure have been reported (11-14). The present study was designed to determine whether or not the increase in mean arterial pressure during Goldblatt hypertension is associated with increased mean circulatory pressure.

METHODS

Twenty mongrel dogs of various sizes and divided into two groups containing ten each were used in the present experiments. Ten were used as controls and ten were made hypertensive.

Goldblatt hypertension was produced in ten of the dogs by removing the right kidney and placing a Goldblatt clamp on the left renal artery. Following surgery the animals were allowed approximately 3 weeks to recover completely from the trauma and to develop sustained hypertension.

Arterial and venous pressures were measured by mercury manometers through catheters inserted into a femoral artery and an external jugular vein. The "physiologic reference point" (4) for making these measurements was 0.61 times the thickness of the chest going anteriorly from the back. The mercury manometers recorded the two pressures continuously on a kymograph. Also, the manometers were equipped with a vibrator which eliminated friction in the system and permitted the manometers to react quickly and accurately to changes in the arterial and venous pressures.

Mean circulatory pressures were measured on the hypertensive animals and the ten control dogs in the following manner. Anesthesia was induced by injecting 30 mg/kg sodium pentobarbital into the dogs. Heparin, 5 mg/kg, was injected to prevent coagulation during the procedure. Then the heart was stopped by injecting 8 mg acetylcholine through a catheter directly into the right atrium. Immediately after the heart had stopped, blood was pumped rapidly from the left femoral artery into the right femoral vein, which brought the arterial and venous pressures to equilibrium within 2-5 sec. The equilibrated pressures were recorded, and the heart recovered from the effect of the acetylcholine and began beating again within 20-30 sec. Within 15 min mean arterial pressure and heart rate returned to control values and another measurement of mean circulatory pressure could be made.

RESULTS

Figure 1 shows the mean circulatory pressures measured in the 20 dogs of this study. As can be seen in the figure, the mean circulatory pressure in the hypertensive, 9.03 (±0.31 SEM) mm Hg averaged considerably higher than that of the control dogs, 7.0 (±0.17 SEM) mm Hg.
This increase in mean circulatory pressure in the hypertensive dogs was highly significant ($t = 5.75$).

Figure 1 also shows a very high degree of correlation between the arterial pressure and the mean circulatory pressure in the entire spectrum of hypertensive and normotensive dogs. The correlation coefficient was 0.89.

**DISCUSSION**

An increase in mean arterial pressure by the Goldblatt technique is associated with an increase in mean circulatory pressure. The mean circulatory pressure is normally almost exactly 7.0 mm Hg, and the three most common factors known to increase this above normal are increased blood volume, increased interstitial fluid volume, and increased sympathetic tone. Ledingham (10), Grollman et al. (3) and Douglas et al. (2) have demonstrated that renal hypertension causes increased blood volume, and Douglas has shown marked increases in interstitial fluid volume. On the other hand, Wilkins (15) has shown that unilateral splanchic and lumbar sympathectomy does not elevate blood flow to the calf, thus indicating that increased sympathetic tone is probably not a factor in hypertension. Therefore we suspect that the increased mean circulatory pressure in these experiments was probably caused by increased fluid volumes.

The mean circulatory pressure in the hypertensive dogs averaged 2.03 mm Hg greater than in the controls. This much increase, under acute conditions, can increase the cardiac output as much as 30%. For this reason, the rise in mean circulatory pressure in the hypertensive dogs played a major role in the development of the hypertension, possibly through its tendency to increase cardiac output. In support of this reasoning is the work of Conway (1) which showed that a chronic increase in blood volume in a dog causes a rise in cardiac output for the first few hours followed by an increase in total peripheral resistance and a return of cardiac output back to the normal level. Although all of these effects may result from changes in volume alone, it is not possible to rule out the many other factors which are known to be altered in hypertension.

**REFERENCES**