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Effect of Partial Clamping of Aorta in Dogs upon Diastolic Pressure in Carotid and Femoral Arteries.

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It is well known that in the human congenital anomaly, coarctation of the aorta, both *systolic* and *diastolic* levels of pressure are frequently elevated in the arteries proximal to the constriction.[1, 2] It has recently been suggested that interference with the blood supply to the kidneys is the mechanism underlying the development of the arterial hypertension found in this condition.

The evidence that this is so is that arterial pressure rises in the upper half of the body only when the clamp upon the aorta is placed above the site of origin of the renal arteries. Rise in mean pressure in the carotid artery was studied by direct measurements in dogs[3] and increase in pressure in rats[4, 5] was inferred from the weights of the hearts at death being distinctly greater when the aortas were clamped above the origin of the renal arteries than when they were clamped below or not clamped at all.

If the mechanism of the development of hypertension occasioned by clamping the aorta resembles that which follows clamping the renal arteries there should occur an increase in diastolic arterial pressure throughout the body in parts distal, as well as proximal to, the constriction of the aorta. So far, diastolic pressure has not been measured in experimental work nor were pressures in the hind extremities measured. Yet in human cases of coarctation of the aorta collected by King[2] the data suggest and, more recently direct measurement of pressure in the femoral artery in a case[6] shows, that elevation of diastolic pressure may be present in the lower extremities.

In the present study observations were made of the changes in diastolic pressure in the legs of three dogs following partial clamping of the aorta above the origin of the renal arteries. The experiments were carried out as follows: Van Leersum loops[7] were made of one

1 Lewis, T., *Heart*, 1933, **16**, 205.

2 King, J. T., *Ann. Int. Med.*, 1937, **10**, 1802.

3 Goldblatt, H., and Kahn, J. R., Proc. Cent. Soc. Clin. Res., *J. Am. Med. Assn.*, 1938, **110**, 686.

4 Rytand, D. A., PROC. SOC. EXP. BIOL. AND MED., 1936, **38**, 10.

5 Rytand, D. A., *J. Clin. Inv.*, 1938, **17**, 391.

6 Steele, J. M., and Cohn, A. E., *J. Clin. Inv.*, 1938, **17**, 514.

7 Van Leersum, E. C., *Arch. ges. Physiol.*, 1911, **142**, 377.

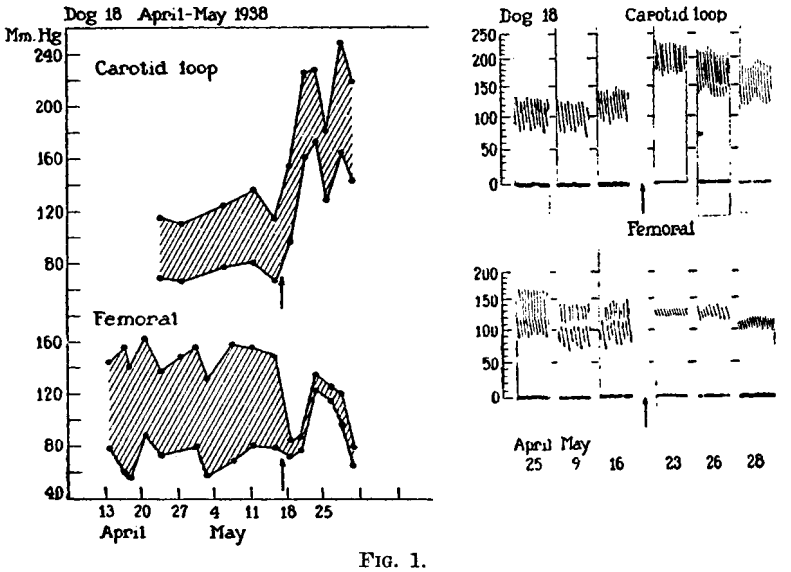

FIG. 1.

Chart of carotid and femoral arterial pressures from dog No. 18 read from records obtained by use of Hamilton's intra-arterial manometer. At the right (Fig. 1a) are reproduced a few of the original records. The black arrows indicate application of the aortic clamp on July 7.

carotid artery. Pressures were then recorded in both the carotid and femoral arteries by means of Hamilton's intraärterial manometer[8] about 4 times a week. The frequency and sensitivity of the manometer were, of course, such that both systolic and diastolic levels of pressure were accurately recorded. Relatively constant levels became established in 3 or 4 weeks. The animals were then anesthetized with pentobarbital and an adjustable metal clamp was placed upon the aorta above the renal arteries, but below the coeliac axis and adrenal arteries. Some interference with the adrenal blood supply occurred, in all probability, in most of the animals but it was obviously not sufficient to interfere perceptibly with the function of the glands. The aorta was gradually compressed by means of the clamp while pressure in the femoral artery was continuously recorded until pulsation had almost disappeared. The clamp was then fixed in this position. At this point little fall in diastolic level occurred, but if an attempt was made to obliterate the pulse altogether, the diastolic pressures fell abruptly. After securing the clamp the wound was closed. Records of pressure were obtained after operation almost daily for a period of about 2 weeks.

Within 24 hours both systolic and diastolic pressures in the caro-

8 Hamilton, W. F., Woodbury, R. A., and Harper, H. T., *Am. J. Physiol.*, 1934, **107**, 427.

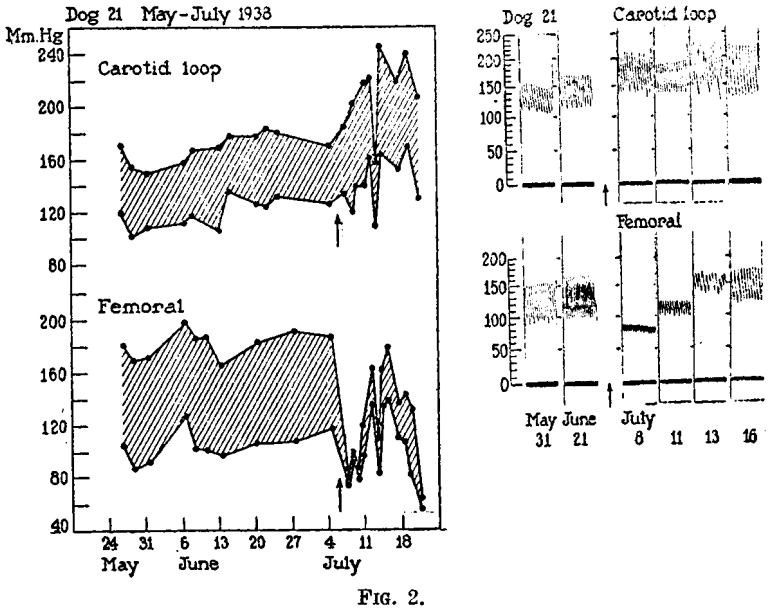

FIG. 2.
Same as Fig. 1, for dog No. 21. The clamp was applied on May 17.

tid artery rose (Figs. 1 and 2). During the next 72 hours both levels of pressure in the femoral artery began also to rise from the very low postoperative levels occasioned by constriction of the aorta, and the pulse pressure, though still small, increased. Within 5 days the *diastolic* pressure in the lower extremities rose to a level plainly higher than the preoperative one and remained elevated for about a week, or until slow hemorrhage into neighboring tissues took place through erosion of the aorta by the clamp.

The diastolic level in the hind legs does not, of course, always rise. Whether it does seems to depend upon the degree of occlusion of the aorta. If the aorta is completely occluded, and if sufficient collateral circulation fails to develop, as occurred in one instance, the pressure in the hind legs remains low though both pressures rise in the carotid artery; but if it is insufficiently clamped neither the pressures in the legs nor those in the carotid artery rise. The marked reduction in flow to the lower extremities seemed in the instance of complete occlusion responsible for the failure of the pressure to rise; the hind limbs were cold and failure of nutrition began before the animal was killed.

From these experiments the conclusion can be drawn that clamping the aorta in dogs above the orifices of the arteries to the kidneys may be followed by diastolic hypertension in the hind legs as well as in the neck. Increase in peripheral resistance is widespread. Elevation of

pressure does not depend therefore upon mechanical factors alone as in acute experiments when the aorta is occluded (Barcroft[9]). Nor can the hypertension in coarctation of the aorta be explained by local mechanical factors such as the increase in resistance offered by the narrowed aorta and the collateral paths around it.[10] It depends upon a reaction of the whole peripheral arteriolar system. Hypertension the result of clamping the aorta is in this respect similar to that following constriction of the renal vessels. In both cases the deciding factor is interference with the dynamics of the renal circulation.

One further remark seems pertinent. The observation of similar consequences to the arterial pressures of constriction of the aorta in man (coarctation of the aorta) and in dogs (clamping of the aorta) warrants the inference that interference with the hemodynamics of the renal blood supply in man may lead to arterial hypertension as Goldblatt has demonstrated that it does in dogs and monkeys.

Summary. Clamping the aorta above the orifices of the renal artery in dogs is followed by elevation of the diastolic level of arterial pressure in the hind legs as well as in the carotid arteries. Constriction of the peripheral arterioles is, therefore, a general phenomenon, just as when it follows partial clamping of the renal arteries. The hypertension which develops in coarctation of the aorta in man is on this evidence analogous to that which accompanies constriction of the renal arteries. The evidence suggests strongly that interference with the hemodynamics of the renal circulation leads to hypertension in man as well as in animals.

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Form of Ventricular Contraction in Cardiac Infarction; Fluoroscopic Studies.

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Infarction of the heart following coronary artery occlusion is attended by a profound disturbance in the circulation. While the circulatory dynamics and electrocardiographic changes have been

[9] Barcroft, H., *J. Physiol.*, 1931, **71**, 281.

[10] Blumgart, H. L., Lawrence, J. S., and Ernstene, A. C., *Arch. Int. Med.*, 1931, **47**, 806.