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Hypertension from Obstruction of the Aorta.

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Goldblatt[1] and others have found in chronic experiments that by stenosing the renal arteries and thereby decreasing the blood supply to the kidneys a persistent elevation in blood pressure can be obtained. This phenomenon Goldblatt explains by the hypothesis of a "pressor substance" which he believes is formed as a result of the ischemia of the kidneys. He found also that stenosis of the aorta above the origin of both renal arteries resulted in a persistent elevation of blood pressure while stenosis below the origin of these vessels did not cause hypertension.

Rytand,[2] using heart weight increase in rats as evidence of hypertension, obtained similar results. Stenosis of the aorta below the right but above the left renal artery, he found, also produced hypertension; however, if the distal (left) kidney be removed hypertension does not result.

Goldblatt's[3] conclusion was that hypertension from experimental aortic stenosis is of the same nature as that due to stenosis of the renal arteries directly, that is, that the stenosis of the aorta caused an elevation of blood pressure by producing renal ischemia.

This idea was further expanded by Rytand to the supposition that renal ischemia with its resulting "pressor substance" is the important factor in the elevation of blood pressure so regularly found in the upper extremities in coarctation of the aorta.

Inasmuch as these results were obtained in chronic experiments it was felt that it would be interesting to investigate the aortic phenomenon with acute stenosis at similar levels.

Using dogs, intraperitoneal nembutal (pentabarbital) as anesthetic and carotid cannula recordings with the kymograph, the following experiments were performed:

1. A screw clamp was placed about the aorta just above the origin of the celiac axis, 2 or 3 cm above the origin of the right (upper) renal artery. The clamp was then closed at first partially

1 Goldblatt, Harry, M.D., Experimental hypertension induced by renal ischemia, Harvey Lectures, 1937-1938.

2 Rytand, David A., *J. Clin. Invest.*, 1938, **17**, 391.

3 Goldblatt, Harry, M.D., *Transactions and Studies, College of Physicians of Philadelphia*, 1938, 4 Ser., Vol. 6, No. 1.

and then completely. Immediately after partial narrowing of the aorta the pressure began to rise and reached its maximum within several heartbeats. Further stenosis raised the pressure still more, the highest values being found with complete closure of the aorta and amounting to an average increase of 52 mm Hg in 4 experiments. This rise was maintained until the clamp was released. Upon release of the clamp there was an immediate drop in pressure, following which it gradually rose to about the original level. Moreover, partial release of the clamp caused a sudden but partial drop and the pressure readings could be decreased in steps by releasing the clamp a little at a time.

Next the renal arteries were ligated. No change in pressure was observed.

With the renal arteries ligated the aorta was again stenosed as above. Exactly the same type of rise and fall of blood pressure occurred. In one instance the rise was 76 mm Hg. This was maintained for over 40 minutes. Upon release of the clamp the same type of sudden drop occurred. The average rise in 2 experiments with the renal arteries ligatured was 65 mm Hg.

2. Stenosis and then complete closure of the aorta between the origins of the renal arteries was performed. The rise was rapid but small, averaging 13.5 mm Hg in 2 experiments. It was greatest with complete closure of the aorta. Release of the clamp gave a sudden drop with return to approximately the original level. With both renal arteries tied off a similar rise and fall were obtained.

3. Stenosis and complete closure of the aorta immediately below both kidneys caused a rapid but small rise of blood pressure with a sudden fall upon release of the clamp. With the renal arteries ligatured a similar result was obtained. The average rise in 7 experiments was 8.8 mm Hg.

TABLE I.

Percentage of Blood Flow Obstructed at the Different Levels and Average Rises of Blood Pressure Produced.

(A, liver and portal bed; B, kidneys; C, lower trunk and lower extremities.)

Level of stenosis	% of flow obstructed (Levy, Blalock)[4]	Avg rise mm Hg	No. of exp.
1. Above celiac	A + B + C = 60%	56	6
2. Between kidneys	½B + C = 22%	16	3
3. Below kidneys	C = 13%	8.8	7

[4] Levy, S. E., and Blalock, A., *Am. J. Physiol.*, 1937, **118**, 368.