17122. Arterial Hypertension Produced by Experimental Stenosis of the Thoracic Aorta.

W. C. SEALY. (Introduced by J. W. Beard.)

From the Department of Surgery, Division of Thoracic Surgery, Duke University School of Medicine, Durham, N. C.

Arterial hypertension has been produced in dogs by stenosis of the thoracic aorta. The hypertension is sustained and has many of the characteristics of that observed in coarctation of the aorta in man.

Materials and methods. The method of producing permanent and prolonged stenosis of the thoracic aorta in dogs, described in detail elsewhere,[1] consists in the substitution of a section of lucite tubing for a segment of the aorta. This tube is designed to reduce turbulence by making the bore hourglass in shape. In the dogs used in this experiment, the tube was 3.7 cm long with 3 mm as the narrowest diameter. The lumen of the aorta was reduced in the dogs in this group approximately 75 to 85%.

Blood pressure records were obtained by direct puncture of the femoral or carotid arteries and measurement of the pressure with either a mercury or Hamilton manometer. From the records obtained with the Hamilton manometer, the systolic and diastolic pressures were calculated. Mean pressures were obtained either from estimations with the planimeter of the area under the pulse waves, or read directly from the mercury manometer. All blood pressure determinations were made with the animals under nembutal anesthesia, 27 mg per kg of body weight.

Results. There were 11 of 16 dogs that survived the operative procedure. Six of the 11 died at intervals of 7 days to 3 weeks after operation, 4 dying because of spastic paralysis of the lower part of the body. Of the 5 animals that survived for longer than one month, 4 are still living. One dog died at the end of 4 months from unknown cause. The remainder are still living at intervals of 4 to 14 months after stenosis of the aorta.

The changes in the blood pressures in these animals has followed, in all instances, a definite pattern as demonstrated in Table I. The femoral mean pressure has shown a drop after operation that persisted in some animals for as long as 7 days; but by the 14th day the pressure returned to normal. After 2 weeks the femoral mean pressure increased above the normal. The carotid mean pressure increased by the end of the first week to levels well above normal, and by 14 days reached hypertensive levels. The mean pressures in both the femoral and carotid increased during the next 3 months. After this time there was in the 2 dogs observed for one year, a decrease in the mean pressure both above and below the constriction, but the pressure still remained in the hypertensive range. The results with 5 animals are tabulated in Table II.

Recordings of the blood pressure with the Hamilton manometer have shown a large increase in the pulse pressure above the constriction and a decrease below. The diastolic pressures both above and below the constriction have increased along with the rise in the mean pressure.

Discussion. The experiments described here show that severe arterial hypertension can be produced in dogs by stenosis of the thoracic aorta. The various mechanisms that contribute to the elevation of the blood pressure are not evident from these results. It has been suggested that the hypertension in coarctation of the aorta is renal in origin.[2] Though the blood flow to the kidneys is not recorded in these experiments, it is interesting to note that for only about 14 days was the mean pressure below the constriction lower than the normal. After this interval there was a progressive rise in the mean pressure. On the other hand, there was a diminution in the

[1] Sealy, W. C., and McSwain, George H., *Surgery*, in press.

[2] Rytand, D. A., *J. Clin. Invest.*, 1938, **17**, 391

TABLE I.
Experimental Hypertension. Blood pressure changes associated with stenosis of the thoracic aorta in the dog.

Days after constriction	Blood pressure in mm mercury Femoral Mean	Femoral S/D	Blood pressure in mm mercury Carotid Mean	Carotid S/D
Normal	127	165/108	126	149/110
6	45	45/45	138	161/116
16	133	151/121	158	200/137
30	157	181/135	190	233/149
40	169	184/154	197	236/162
124	176	199/167	238	292/198

S = Systolic. D = Diastolic.

TABLE II.
The Blood Pressure Changes Associated with Experimental Coarctation of the Aorta in Dogs.

Animal No.	Normal blood pressure Femoral M	Femoral S/D	Carotid M	Carotid S/D	Duration of aortic constriction, mo.	Last recorded blood pressure Femoral M	Femoral S/D	Carotid M	Carotid S/D
14	118		124		14	133	144/123	158	185/130
16	128		130		5	196		222	
22	134		140		13	154	170/137	193	241/160
29	127	165/108	126	149/110	4	176	199/167	238	292/198
31	125	157/107	161	184/146	3	162	170/157	239	299/199

M = Mean pressure. S = Systolic. D = Diastolic.

pulse pressures below the constriction that persisted. This might be interpreted as evidence for renal origin of the hypertension as suggested by Kohlstaedt and Page.[3]

The substitution of a rigid narrow tube for a section of the aorta would alter the characteristics of the pulse waves above and below the constriction; but this would not explain the marked elevation of the blood pressure that occurs in experimental stenosis of the aorta.

Summary. 1. By stenosis of the thoracic aorta in dogs, arterial hypertension can be produced above and below the stenosis.

2. This type of experimental hypertension has many of the characteristics observed in the hypertension occurring in coarctation of the aorta in man.

[3] Kohlstaedt, K. G., and Page, I. H., *J. Exp. Med.*, 1940, **72**, 201.

Received March 18, 1949. P.S.E.B.M., 1949, **71**.