Fall in Pressure in Radial Artery during Reactive Hyperemia

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Under conditions of rapid flow, the arterial bed between the aorta and radial artery offers an appreciable resistance to flow. During reactive hyperemia in normal subjects, approximately 30 per cent of the energy of pressure may be lost between the aorta and radial artery. Hypertension produced in normal subjects by infusion of levarterenol increases the gradient in pressure during reactive hyperemia between the aorta and radial artery. Hypertension in patients with hypertensive disease does not increase the gradient during reactive hyperemia beyond that which is produced in normal subjects.

IN A normal subject at rest, the mean arterial pressure is approximately the same in all arteries where it is commonly measured. This is not true for the systolic pressure which is higher in the dorsalis pedis than in the femoral artery and higher in the femoral than in the axillary artery. The digital systolic pressure is approximately equal to the brachial systolic pressure when finger blood flow is small, but the digital systolic pressure falls about 20 mm. Hg when the blood flow in the finger is increased by heat. The systolic pressure in a radial artery in which the blood flow has been increased by exercise of the corresponding arm is lower than that in the brachial artery of the opposite resting arm. The present observations were made to determine the effect of increasing the blood flow in one hand and forearm by means of reactive hyperemia on the pressure in the corresponding and opposite radial arteries.

METHODS

Eighteen or twenty gage needles were placed in each radial artery and the arterial pressure recorded continuously by means of strain gages. The mean pressure was determined by planimetry or by adding one third of the pulse pressure to the diastolic pressure. Data showing that the latter method is sufficiently accurate for our purposes have been reported previously. The circulation to one forearm and hand was occluded by a cuff placed above the elbow and inflated above systolic pressure. After a period of arterial occlusion, the cuff was released and the recording of pressure continued until preocclusion levels were reached. In some subjects the part was congested by a cuff pressure of 40 mm. Hg for 1 min. before the cuff pressure was raised to occlusion levels. This did not cause significant changes in the postocclusion pressures. The subjects were normal young males, normal young males receiving levarterenol, and patients with hypertension.

RESULTS

Normal Subjects with Arterial Occlusion. Fourteen observations were made in 11 subjects. The preocclusion pressures were equal in both arms and averaged 121/66 mm. Hg (mean pressure 84 mm. Hg). Arterial occlusion was maintained from 6 to 10 min. (average 9.5 min.). On release of the cuff, pressure in the unoccluded arm fell an average of 9 mm. Hg systolic and 8 mm. Hg diastolic, usually returning to normal in approximately 10 sec. In the occluded arm, the pressure fell to a low level during occlusion and rose immediately upon release of the cuff. With the first beat after release, the average pressure was 71/54 mm. Hg (mean pressure of 60 mm. Hg). It tended to fall slightly in the next two beats and slowly returned to normal, systolic pressure reaching the preocclusion value in an aver-
age time of 125 sec. and diastolic in 86 sec. (fig. 1). On release, the upstroke of the pulse wave in the previously occluded artery was slower and the dicrotic notch nearer the peak of the pulse wave (fig. 2).

**Normal Subjects with Levarterenol and Arterial Occlusion.** Seven observations were made in 6 subjects. The average arterial pressure before levarterenol was 128/71 mm. Hg (mean pressure 90 mm. Hg). During infusion of levarterenol the preocclusion arterial pressure averaged 188/100 mm. Hg (mean pressure 129 mm. Hg). Occlusion was maintained from 6 to 8.5 min. (average 7 min.). On release of the cuff, the pressure in the unoccluded arm fell as in the subjects without this amine. In the occluded arm, the pressure in the radial artery rose with the first beat to an average value of 104/72 mm. Hg (mean pressure 83 mm. Hg). In the next few beats, the fall was more constant than in the subjects without levarterenol.

**Patients with Hypertension.** Twelve observations were made in 6 subjects. The average preocclusion arterial pressure was 219/111 mm. Hg (mean pressure 147 mm. Hg). Upon cuff release, pressure in the unoccluded artery fell briefly as in the normal subjects. In the occluded artery, the average pressure on the first beat after release of the occlusion was 169/102 mm. Hg (mean pressure of 124 mm. Hg).

**DISCUSSION**

The mean pressure in the aorta and in the vessels the size of the radial artery is approximately the same in a normal subject resting in the horizontal position. The velocity of flow in the radial artery is less than in the aorta. The conversion of energy of flow into energy of pressure as velocity decreases is balanced by the frictional loss of energy as blood flows from the ascending aorta peripherally, and the energy of pressure changes little. Under resting conditions the resistance is primarily at the arteriolar level. Dilating the arterioles in the skin and muscle of the arm by occluding the brachial artery increases the blood flow, on release of the occlusion, from an average level of 3 to approximately 30 ml./100 cc. of forearm/min. The increase in flow causes a rise in viscous resistance in the vessels between the aorta and the radial artery and energy of pressure is changed into heat by the frictional forces. A smaller amount of energy of pressure is converted into energy of flow. The net effect is a considerable decrease in the energy of pressure in the artery supplying the dilated part. Under these circumstances
Peterson pointed out the increased loss of energy of pressure as viscous resistance is increased by increasing flow. This phenomenon is well illustrated in the tracings published by Patterson although he did not comment upon this aspect of the problem. Hamilton has commented on the marked fall in systolic pressure in the femoral artery when a vasodilator drug is injected into the femoral artery. Eichna and Wilkins pointed out the importance of using low collecting cuff pressures to avoid obstructing the artery when measuring forearm blood flow during reactive hyperemia by the method of venous occlusion.

The clinician usually expects to find arterial disease when he encounters pressure gradients in the arterial tree as great as those described here during reactive hyperemia. He may not appreciate the fact that a fast flow may increase the viscous resistance sufficiently to cause a sharp gradient between the peripheral artery and the aorta. In occlusive disease of the arteries, the gradient of pressure across the partially obstructive vessel is markedly altered by changes in blood flow which cause only small changes in gradient in a normal subject. Vasodilation causes a sharp loss of energy of pressure from both viscous resistance and turbulence. These relations have been well described by Dornhorst and Sharpey-Schafer in a study of patients with arteriosclerotic disease of the main femoral arteries. With the patients at rest, the arterial pressure below the obstruction was decreased and the pulse pressure small. An occluding cuff placed below the recording needle in the femoral artery decreased the flow from the artery below the cuff and both mean arterial and pulse pressure above the cuff rose. Reactive hyperemia occurred when the cuff was released and the pressure fell below the preocclusive level.

The infusion of levarterenol in the normal subjects increased the arterial pressure and widened the gradient between the two radial arteries during the period of reactive hyperemia. The arterial bed between the aorta and radial artery assumed increased importance as an area of resistance to flow. These findings are compatible with either constriction of the arterial tree between the aorta and radial artery by levarterenol or increased frictional loss of energy of pressure because of the more rapid flow of blood caused by the rise in arterial pressure, or a combination of these two factors. Wilkins and Eichna observed that elevation of the arterial pressure by paresinol caused an increased flow during reactive hyperemia.

In the patients with hypertension, reactive hyperemia did not increase the gradient between two radial arteries beyond that present in the normotensive subjects. Plethysmographic studies during reactive hyperemia have shown that the increased arterial pressure in patients with hypertension does not cause a blood flow greater than that present.
under similar conditions in normal subjects. The data recorded here are compatible with an arterial tree which has not been changed in size by the high pressure and a persistent elevation in the resistance distal to the radial artery.

**Summary**

Pressures were measured in both radial arteries before and after the release of a cuff occluding one brachial artery above the elbow. When the blood flow is rapid the normal vessels between the aorta and radial artery offer an appreciable resistance to flow. The increase in blood flow during reactive hyperemia produced an average gradient in mean pressure of 24 mm. Hg between the two radial arteries. When hypertension was produced by the intravenous injection of levarterenol, the gradient in mean pressure, on release of the occluding cuff, reached an average value of 46 mm. Hg. In patients with hypertension the gradient in mean pressure on release of the cuff averaged 23 mm. Hg.

**Summario in Interlingua**

Esseva mesurate le tensiones in arterias radial tanto ante como etiam post le relaxa- tion de un manchette que oclu.deva un ar- teria brachial supra le cubito. Quando le fluxo de sanguine es rapide, vasos normal inter le aorta e le arteria radial interpone appreciabile resistentias al fluxo. Le aug- mento del fluxo de sanguine durante hyper- emia reactive produceva al media un gradi- ente de 24 mm. Hg inter le tensiones medie in le duo arterias radial. Quando hyperten- sion esseva produce per le injection intra- venose de norepinephrina, le gradiente de tension medie atingeva, post le relaxation del manchette de occlusion, un valor medie de 46 mm. Hg. In patientes con hypertension, le gradiente del tension medie post le relaxa- tion del manchette esesseva al media 23 mm Hg.

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