The Effects of Arterial and Pulmonary Shunts on the Dynamics of Aortic Coarctation

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This communication deals with the manner in which collateral arterial channels and a persistent ductus arteriosus affect the dynamics of an uncomplicated aortic coarctation which was studied experimentally by means of artificial shunts and registration of pressure pulses. The conclusion is reached that effective collaterals or a surgical subclavian-aortic shunt can be effective dynamically in reducing hypertension and left ventricular strain and in providing an adequate flow through tissues despite the fact that the femoral pressure pulse does not revert to a normal form. The presence of an aortic-pulmonary shunt affects aortic pressure both through reduction in aortic resistance and augmentation of systolic discharge until very severe degrees of coarctation exist. The right ventricle of the normal heart can function as a systemic ventricle only when the aorta is almost completely occluded.

The BASIC dynamics of aortic coarctation were analyzed in the preceding communication. In man, however, coarctation of the aorta is always complicated by the gradual development of numerous anastomoses which provide sufficient blood flow to the lower aortic branches to maintain life processes reasonably well. Nature's experiments, therefore, differ from acute experiments in that patients manifest hypertension above the constriction despite the development of such collaterals. The question, therefore, arises as to whether the resistance of the cephalic aorta and its branches is still high or whether additional constriction of the peripheral arteries is involved. The purpose of this investigation was to simulate naturally developing collaterals by making a functional shunt between a subclavian artery and the aorta, and to study the hemodynamic effects of opening and closing such a shunt. Incidentally, these studies also throw light on the dynamic adequacy of such surgical shunts in patients with coarctation of the aorta.

Human coarctation may also be complicated by a persistent ductus arteriosus. In the series collected by Maude Abbott, approximately 10 per cent of all patients with adult type of coarctation had an associated patent ductus arteriosus. The same proportion has been reported in cases of coarctation referred to the Mayo Clinic. In most of these cases blood from the aorta was obviously shunted into the pulmonary artery. However, several cases have been reported in which the flow of blood was apparently directed from the pulmonary artery to the aorta, the right ventricle acting as a "systemic ventricle." Douglas and associates described a case of patent ductus arteriosus with the unusual features of cyanosis, right ventricular hypertrophy, and obstructive pulmonary vascular lesions. An aortic intimal lesion, the so-called "jet lesion," was present opposite the entrance of the ductus, and its structure was consistent with that of a reaction to mechanical irritation. On that basis, they concluded that at least for part of the cardiac cycle the pressure within the right ventricle equalled or exceeded that within the aorta, thus causing a flow of blood from the pulmonary artery to the aorta.

In another case, described by Edwards and
co-workers, the systolic pressure in the left leg varied from 98 to 102 mm. Hg, and the diastolic pressure ranged from 50 to 60 mm. Hg, which compared very favorably with the diastolic pressure of 45 to 50 mm. Hg in the upper arm. Necropsy revealed that the right ventricle equaled the left in thickness and the systemic collateral circulation was not well developed. The lumina of the intrapulmonary arteries and arterioles, as seen on microscopic examination, were greatly narrowed. In still another case, described by Taylor and associates, the oxygen content of the femoral artery blood was low, although the femoral blood pressure was equivalent to that in the radial artery. The collateral circulation also was not developed in this case. This suggested a flow of blood from the pulmonary artery to the aorta. In order to determine whether dynamic conditions other than high pulmonary resistance and failure in the development of arterial collaterals might cause the right ventricle to propel blood through a patent ductus into the aorta below a constriction, an artificial shunt was made between the pulmonary and systemic systems and the effect of its opening during various degrees of aortic coarctation was studied.

Methods

Aortic pressure pulses above and below the constriction and in the femoral artery were recorded as described in the preceding paper. In experiments designed to study the effects of systemic shunts, the side tube of a glass cannula inserted into the aorta was connected with a subclavian artery. In experiments designed to study the effect of a patent ductus arteriosus, a pulmonary T-cannula, shown in figure 1A, was specially designed so that the blood might flow unobstructed to the left lung. The narrow side (M) was inserted into the distal side and the wider side (N) toward the pulmonary trunk side so that the lumen might correspond to the natural lumen of the vessel. An aortic-pulmonary shunt was made by connecting the aortic cannula by a short, wide plastic tube to the pulmonary T-cannula. In some of these experiments pressure pulses were also recorded immediately below the coarctation. To accomplish this an aortic cannula with two side tubes (Fig. 1B) was used, one side tube (M) for making the shunt and the other (N) for recording the pressure. Right ventricular pressures were secured in some experiments by means of a blunt needle (15 gage), introduced through the ventricular wall.

Many records were taken to determine the effects that opening of a shunt had on the aortic and femoral pressure pulses when varying degrees of coarctation existed.

Results

Dynamic Changes Resulting from the Opening of a Subclavian-Aortic Shunt. Figure 2 shows the effects of opening the subclavian-aortic shunt on the aortic (upper curve) and femoral (lower curve) pressure pulses with severe degrees of coarctation. Segment A shows the normal relationship, segment B the effect of 80 per cent stenosis with the subclavian-aortic shunt closed. Due to the resistance to the flow of blood through the stenosis, the femoral pressures have fallen from 186 to 91 mm. Hg systolic, and from 116 to 81 mm. Hg diastolic. The pulse pressure was greatly reduced. The aortic systolic pressure, on the other hand, rose from 171 to 194 mm. Hg, and the diastolic pressure remained unaffected. The heart rate decreased from 151 to 144 per minute. Segment C shows the effect of opening the subclavian-aortic shunt. The aortic pressures diminished substantially from 194 to 178 mm. Hg systolic, and from 124 to 117 mm. Hg diastolic. The femoral pressures increased from 91 to 142 mm. Hg systolic, and from 81 to 107 mm. Hg diastolic. The pulse pressure increased greatly. The heart rate came back to normal. The contour of the femoral pressure curve, however, did not return to normal. The ascending and descending limbs still have a gradual slope, though less gradual than in segment B, and there is still no appearance of the dicrotic wave. The
summit of the curve, however, has become more peaked. It greatly resembles curves after moderate coarctation (see fig. 3C of previous paper). From a series of 10 experiments it was apparent that the subclavian-aortic shunt has reduced the load in the upper part of the body by reducing the pressures substantially, and has increased the flow to the lower extremities.

**Dynamic Changes Resulting from Opening a Pulmonary-Aortic Shunt.** Figure 3 shows a series of paired tracings illustrating the effects of opening an aortic-pulmonary shunt when various degrees of aortic coarctation exist. The two curves in each pair represent the aortic (upper) and femoral (lower) pressure pulses.

![Fig. 2. Effects of opening the subclavian-aortic shunt on the aortic (upper curve) and femoral (lower curve) pressure pulse contours after severe degrees of coarctation. Segment A shows the normal relationships. Segment B, the effect of 80 per cent stenosis with the shunt closed, and segment C, after opening the shunt.](image-url)

Before and after opening the shunt. The pair of records A shows the pressure relationship with unconstricted aorta. The pair of records B, C, D, E, and F represent the effects of opening the shunt with 55, 65, 75, 85, and 90 per cent reduction of the aortic lumina. Normally, with the shunt closed, the aortic pressures were 105 mm. Hg systolic and 74 mm. Hg diastolic; the femoral pressures, 124 mm. Hg systolic and 68 mm. Hg diastolic. On opening the shunt, the aortic pressure pulse shows an increase in the gradient of the ascending limb. There is a late systolic collapse and the diastolic pressure becomes very low, resulting in a greatly increased pulse pressure. The femoral pulse shows an increased gradient of the ascending and descending limbs, resulting in a high systolic and low diastolic pressure with high pulse pressure. The dicrotic wave disappears on opening the shunt. With a moderate degree of coarctation in pair of records B, the general features are the same as in A, except that the femoral systolic pressure also falls to some extent. The aortic and femoral pulse pressures both become larger on opening the shunt.

With still greater degrees of stenosis in segments C and D, significant changes occur on opening the shunt. The systolic aortic pressures now increase considerably and the diastolic pressure first falls (pair of records C). But, as the degree of stenosis is increased, the diastolic pressure also increases (pair of records D). The femoral pressure pulses at this stage assume the typical appearance of the coarctation pulse. Opening of the shunt reduces both the systolic and diastolic pressures, but there are no significant changes in the contour of the curve and in pulse pressure. The pair of records (E) with 85 per cent stenosis shows the same pressure relationships in aortic curves as in D, but, though the femoral pressures are less on opening the shunt, the pulse pressure is definitely increased from 2 to 7 mm. Hg. The pair of records F shows the effect with 90 per cent stenosis. With the aortic-pulmonary shunt
Fig. 3. Effects of opening an aortic-pulmonary shunt after various degrees of coarctation. Segment A shows the pressure relationships with unconstricted aorta. Segments B, C, D, E, and F represent the effects of opening the shunt with 55 per cent, 65 per cent, 75 per cent, 85 per cent, and 90 per cent reduction of the aortic lumen. The two curves in each segment represent the pressures and pulse contours before and after opening the shunt.
closed, the femoral pulse pressure has become zero and the mean pressure has decreased to 11 mm. Hg, indicating that the flow through the stenosis had been reduced to a mere trickle. Upon opening the shunt, there is a fall in the aortic pressure, while the femoral pressure now increases from 11 to 23 mm. Hg systolic and shows a definite pulsation.

In order to study the effects of opening a shunt during various degrees of acute coarctation on the right heart, pressures were also recorded from the right ventricle. Figure 4 shows segments of such records, together with pressure pulses from the aorta above the stenosis, and immediately below the stenosis and shunt. Segment A shows the normal relationship with the aorta unconstricted. The pressures in the aortic arch and in the aorta below the shunt are essentially the same, as are also their pulse contours. The right ventricle shows initial and maximal pressures of 7 and 48 mm. Hg respectively. Segment B illustrates the effect of 70 per cent stenosis of the aortic lumen. The aortic systolic pressure above the stenosis has risen as before, the diastolic pressure remaining the same. The pulse contour of the thoracic aortic pulse below the coarctation (lower curves) has the murmur graphically recorded. The right ventricle shows only a slight increase of initial pressure. Segment C depicts the effect which follows opening the aortic-pulmonary shunt. The aortic systolic pressure is increased slightly from 137 to 141 mm. Hg, the diastolic pressure is reduced from 73 to 56 mm. Hg, resulting in a greatly increased pulse pressure. The gradient of the aortic pressure curve becomes steeper and the pressures below the shunt are further reduced, systolic from 92 to 52 mm. Hg, and diastolic from 74 to 38 mm. Hg. On the curve are superimposed marked marked vibrations due to the turbulence created by the large increase in the velocity of ejection from the left ventricle. The contour of the right ventricular pressure curve is not changed, but maximal pressure has increased from 43 to 50 mm. Hg systolic, and the initial tension from 8 to 9 mm. Hg. The dura-

Fig. 4. Effects of opening a shunt during various degrees of acute coarctation on the right ventricular pressures (upper curves in segments A, B, C, and middle curves in segments D and E), together with pressure pulses from the aorta above the stenosis (middle curves in segments A, B, C; upper curves in segments D and E) and below the stenosis and shunt (lower curves). Segment A shows the normal relationships with aorta unconstricted. Segment B, effect of 70 per cent stenosis of the aortic lumen. Segment C, after opening of the shunt, and segments D and E, before and after opening the shunt with 93 per cent constriction of the aorta.

The effect of ejection phase is slightly increased in segment C as compared with B.

Segment D shows the effect of 93 per cent stenosis of the aorta with the shunt closed. As seen before, the aortic pressure has risen to a very high level and the aortic pressure below the coarctation has zero pulse pressure and a mean pressure of only 16 mm. Hg. The right ventricular initial and maximal pressures are slightly higher than in the segment B with 70 per cent stenosis. Upon opening the shunt in segment E, the aortic pressures fall and the aortic pulse pressure becomes slightly reduced. On the other hand, the femoral systolic pressure is increased from 16 to 34 mm. Hg and the pulse pressure from 0 to 12 mm. Hg.

Since the femoral pressure wave is obviously due to right ventricular action, the changes in
the right ventricular pressure curves are especially important. It will be noted that the right ventricle accomplishes its ejection into the systemic circuit during nearly complete occlusion of the aorta with an actual slight reduction of initial and maximal pressures. The shunt was left open for 3 hours and 45 minutes to ascertain whether there are any further changes in behavior of the right ventricle and femoral pressures suggestive of other compensatory reactions, but none were found.

**DISCUSSION**

The experiments on effects of opening a subclavian-aortic shunt were designed primarily to simulate and study the efficiency of natural shunts that develop in the body in individuals with aortic coarctation, and secondarily to determine the dynamic adequacy of artificial surgical shunts in alleviation of coarctation in human subjects. The results presented show clearly that a subclavian-aortic shunt is helpful in several respects, despite the fact that the femoral pressure pulses do not recover a normal form.

In the first place, the presence of such a shunt reduces the hypertension central to the coarctation. This is not wholly due to diminution in aortic resistance, for were this the case diastolic pressure should have declined more than systolic and the pulse pressure should have increased after opening of a shunt, not decreased, as in these experiments (fig. 2B, C). The predominant fall in systolic pressure and decrease in pulse pressure indicate a decrement in stroke volume of the left ventricle. The mechanism by which this is accomplished has been established by Wiggers and Katz (figs. 8 and 9) through registration of ventricular volume curves before and after decompression of the aorta. When aortic resistance is diminished the left ventricle empties itself more completely for a few beats, thereby reducing the residual blood and diastolic size. As soon as this eventuates the systolic discharge becomes less than during a state of elevated pressure. Such an automatic adjustment of the stroke volume of the left ventricle explains the reduction in pulse pressure and predominant decline of systolic pressure.

In the second place, the existence of a shunt increases the flow into the distal aorta to such an extent that diastolic femoral pressure is elevated considerably. This indicates that a good drainage reservoir exists for maintaining a continuous flow of blood through tissues below the coarctation, i.e., during diastole as well as systole.

In the third place, the persisting deviations of the femoral pulse from normal—slower rise and fall and absence of dicrotism—are not as serious dynamically as they seem. Excessive damping of the central pressure wave is responsible for the slower rise and to some extent for the more gradual fall. When the forceful impact of ventricular ejection is not transmitted to the lower aorta, as in coarctation, the blood column is not set into oscillation; no standing wave is created, the rising limb of the femoral pulse is not accelerated, systolic pressure does not overshoot, and the descending limb is not deformed by a dicrotic notch and wave. It more truly represents the gradual degradation of pressure energy to flow during late systole and diastole than does the normal femoral pulse. The deviations from a normal form of the femoral pulse are therefore less important than the elevation of diastolic pressure after opening a shunt.

The physical mechanism by which the rise of femoral pressure becomes steeper and greater after opening a systemic shunt still requires analysis. The rise is always smooth and steeper from the start; there is never an anacrotic halt or suggestion of summated waves on the rising pressure limb. This strongly suggests that the pressure waves entering the lower aorta through a stenotic aorta and the subclavian shunt have so little time difference that they combine to produce a smooth rapid elevation from the start. This is dynamically important in that turbulence is probably avoided.

On dynamic grounds, therefore, the surgical creation of subclavian-aortic shunts* would seem to constitute sound procedure for creating an adequate blood flow below the constriction.
and in alleviating the state of hypertension above it. Everyone is agreed that the ideal operation of clinical coarctation is one in which the stenotic area is excised and the proximal or distal ends of the aorta are united by suture as performed by Crafoord and Nylin,10 and Gross and Hufnagel.11 Unfortunately, there are some instances in which this does not appear to be feasible, either because the constricted area in the aorta may be too extensive or the left subclavian artery may arise too close to the localized area of constriction. The question has been raised as to whether one should employ the left subclavian to by-pass the point of stenosis if the ideal operation cannot be performed. These experimental results support the conclusion of Bing and associates,12 that the subclavian-aortic anastomosis does reduce the load to the upper part of the body and substantially increases the amount of blood to the lower part of the body.

Opening of an aortic-pulmonary shunt re-duplicates the dynamic condition of humans in which the ductus arteriosus persists without a complicating coarctation. The primary effect on the arterial pressure pulses illustrated in figure 3A is due to lowering of peripheral resistance; diastolic pressure declines predominantly and the pulse pressure increases in the aorta. To this is added the almost immediate increase in systolic discharge of the left ventricle; the blood which leaves the aorta by way of the pulmonary shunt quickly augments the return of blood to the left heart and is disposed of by augmentation of stroke volume. The magnitude of this flow is indicated by observations of Eppinger and co-workers13 that 40 to 75 per cent of the left ventricular output may pass through a patent ductus. This brusque increase in systolic discharge accentuates femoral pressure by causing a great overshoot of systolic pressure, but the reduced aortic resistance occasioned by the aortic-pulmonary shunt results in a marked drop in diastolic pressure, much as in cases of A-V fistula and aortic insufficiency. As in these cases also, the distinctive feature is the loss of the dicrotic wave.14 The picture does not change significantly when an aortic-pulmonary shunt is complicated by a degree of aortic stenosis up to 55 or 60 per cent of the aortic lumen (fig. 3B).

As aortic coarctation becomes more severe the resultant changes of systolic discharge and aortic resistance caused by opening of a shunt alter continually. In this connection it is important to remember that as far as effects on aortic pressure are concerned, change in resistance caused by opening a shunt is not determined alone by the caliber and length of the shunt and the pressure in the aorta below the coarctation; resistance is a physical ratio of pressure: flow. While changes in resistance are not actually determinable, their relative importance in accounting for aortic pressure changes can be inferred from alterations that occur in aortic diastolic pressure on opening the shunt. Since diastolic pressure is reduced when a shunt is opened in cases of mild aortic constriction (fig. 3B, C), but increased when constriction is severe (fig. 3D, E), it is obvious that reduction in resistance plays a recognizable role only when the degree of constriction is moderate (about 65 per cent reduction). Judging from the increase in aortic systolic and pulse pressures (fig. 3D) after opening of a shunt, augmentation of systolic discharge continues to be the dominant factor until an extreme state of coarctation exists. Finally, however, the flow of blood through the shunt is so far reduced—or even reversed—that systolic discharge becomes less and consequently aortic pressures fall on opening the shunt (fig. 3F).

Even in fairly marked aortic coarctation, opening of the shunt has comparatively little effect on the femoral pulse except to reduce systolic and diastolic pressures and to smooth the dicrotic wave (fig. 3C, D). However, when femoral pressure has declined to very low levels (fig. 3E, F), an augmentation of the pressure pulse is produced by a reversal of flow. Attention may be called to the fact, apparent from the curves C, that 65 per cent coarctation with complicated patent ductus is much more serious than a pure case of coarctation. The femoral pressures and pulse contours with 65 per cent stenosis and with the shunt open, resemble a
stenosis of about 75 per cent with the shunt closed. The pair of curves $E$ with 85 per cent stenosis still show reduced femoral pressures on opening the shunt, but the femoral pulse pressure is increased. The reduced femoral pressure is explained by the fact that blood is still flowing from the aorta to the pulmonary artery on opening the shunt, thus lowering the femoral pressures. The fact that pulse pressure is increased strongly supports the inference that the right ventricle is pumping blood into the aorta during some part of the cardiac cycle. This would have been more certain if the femoral pressure curve had shown a second hump on the ascending limb. In $F$, however, with 90 per cent coarctation the aortic pressures fall and the femoral pressures rise on opening the shunt. In this case, the right ventricle was certainly pumping blood into the aorta below the coarctation.

The manner in which the right ventricle responds when blood is suddenly shunted into the pulmonary circuit can be deduced from changes in the right ventricular pressure curves. The pulmonary arterial pressure against which the right ventricle is required to eject its blood is probably raised to nearly the same level as in the aorta below the constriction. In figure 4 the pressure varies from 38 mm. Hg diastolic to 52 mm. Hg systolic. The right ventricular pressure must obviously be raised above this level in order to expel its contents. In figure 4C this is the case during the early phase of ejection but during the later portion of ejection the aortic pressure tends to exceed right ventricular pressure slightly. This probably augments the residual volume of the right ventricle and is responsible for the slight elevation of initial tension seen in this record. However, the right ventricle at no time appears to be under increased strain such as follows pulmonary stenosis, for example.

The normal right ventricle acts as a systemic ventricle only when an intense degree of aortic constriction exists and the pressure in the distal aorta falls to a level below that in the pulmonary artery. Despite repeated trials, this proved successful only after arterial pressure had been reduced to a low constant level, as in figure 4D. Since opening the shunt decreases pulmonary resistance, the transfer of right ventricular blood is effected with an actual slight decline of maximal right ventricular pressure. As a result also, its residual volume decreases and initial tension tends to decline slightly, as in figure 4E. In short, the right ventricle is relieved of strain and the commonly accepted determinant for provoking its hypertrophy is absent. It is obvious that other factors may enter into clinical cases. The suggestion has been made on deductions from necropsy studies that in clinical cases the excess flow of blood through the pulmonary system occasioned by a patent ductus may gradually cause sclerosis of pulmonary vessels. It is by no means clear that such a "cause and effect" relation exists; the sclerotic process may be wholly unrelated to the dynamic disturbances. However, regardless of the way in which pulmonary arterial sclerosis is induced, the increased resistance thus occasioned may react functionally on the right ventricle to cause its hypertrophy. Proof of this attractive hypothesis awaits evidence by catheterization studies that initial tension is unquestionably elevated in the right ventricle. It is quite possible that ejection of blood by such a hypertrophied right ventricle against a higher pulmonary resistance will be able to transfer blood to the aorta when pressures are at higher levels than in acute experiments.

**SUMMARY**

1. To elucidate the purely dynamic effects of collateral arterial circuits in aortic coarctation, an artificial shunt was made between the subclavian artery and the aorta distal to the constriction and pressure pulses were recorded by optical manometers from the aortic arch and the femoral artery.

2. Analysis of pressure pulses revealed that with moderate degrees of aortic constriction, opening of a subclavian-aortic shunt causes the following changes: (a) Above the constriction systolic pressure is reduced more than diastolic, hence the diminution in hypertension is not
wholly determined by decrease in aortic resistance, but significantly also by an automatic reduction in systolic discharge. (b) Below the constriction, as exemplified by the femoral pressure pulse, the systolic and diastolic pressures are elevated and the pulse pressure increases, but the form of the pulse does not return to normal; it maintains a more gradual ascending and descending limb.

3. The persisting deviations of the femoral pulse from normal are not as serious as they seem. The fact that diastolic pressure is markedly elevated indicates that enough blood reaches the lower aorta to make it a good diastolic drainage reservoir from which a continued blood flow through tissues can be maintained. These favorable effects combined with the reduction in hypertension above a stenosis and the relief of the left ventricle from strain make the creation of an artificial subclavian-aortic shunt a procedure which is dynamically sound.

4. In order to study the dynamic alterations that result from the coexistence of a patent ductus arteriosus and aortic coarctation, an artificial shunt was made between the aorta and pulmonary artery just below the coarctation, and the effect of opening such a shunt on aortic, femoral, and right ventricular pressure pulses was determined when the aorta was constricted to different degrees.

5. Analysis of such records revealed that with moderate degrees of coarctation, i.e., up to 55 to 60 per cent of the aortic lumen, opening of the shunt causes a predominant fall of diastolic pressure and a marked increase in pulse pressure above the coarctation. This is primarily due to the reduction in aortic resistance. However, the increased return of blood to the left heart very quickly augments the systolic discharge of the left heart so that systolic pressure remains essentially unchanged or actually rises. The increased systolic discharge also accentuates systolic femoral pressure, but the reduced aortic resistance causes a rapid decline of pressure to a low level, much as in aortic insufficiency or an arteriovenous fistula.

6. With more severe degrees of coarctation, the presence of a shunt alters the relative roles that reduction in aortic resistance and increase in systolic discharge play in determining aortic systolic and diastolic pressures. Only in cases of extreme degrees of coarctation does the presence of a shunt cause the stroke volume to diminish and aortic pressures to fall. This occurs when the blood flow through the shunt is reversed from the pulmonary to the systemic vessels.

7. The presence of an aortic-pulmonary shunt has no dynamic effects on the right ventricle of a normal dog by virtue of which pulmonary pressures can be raised sufficiently to exceed aortic pressures below the coarctation until occlusion of the aorta is nearly complete. The possibility that the right ventricle acts as a systemic ventricle in clinical cases through coincident increase in pulmonary resistance is not excluded by these experiments.

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