Basic Hemodynamic Changes Produced by Aortic Coarctation of Different Degrees

By Trilok Chand Gupta, M.D., Ph.D., and Carl J. Wiggers, M.D., Sc.D.

Basic hemodynamic studies on experimental coarctation of the aorta just beyond the left subclavian artery have revealed that hitherto unsuspected physical and physiologic factors are involved in creation of hypertension above a coarctation and in changes of pressure pulses below such a lesion. The effects are by no means explained by an increased resistance at the coarctation, as is generally believed. This communication analyzes the roles that changes in capacity and distensibility of the aortic compression chamber and increase in systolic discharge of the left ventricle play in the production of aortic hypertension, and discusses the physiologic compensations in blood flow by which an adequate return to the right heart is maintained despite extreme reduction in flow through the inferior cava. This communication also deals with the ways in which the pressure relations in the lower aorta and femoral artery are altered from the normal, emphasizing the relative shares that damping of the pulse wave and reduced input into the lower aorta play with different degrees of coarctation. The changing characteristics of the murmurs with progressive aortic constriction are also analyzed. The conclusion is reached that all the dynamic changes found in experimental and human coarctation are adequately explained without the assumption of accessory vasoconstriction through reflex or humoral agencies.

There is general agreement that virtually all clinical cases of aortic coarctation manifest systolic hypertension above and systolic hypotension below the constriction. The diastolic pressure in the cephalic vessels of the aorta is also generally elevated, though to a lesser extent, so that a large pulse pressure obtains. Differences of opinion exist as to whether the diastolic pressure in the femoral arteries is also raised. In 1928, Abbott,1 in her statistical review of 200 autopsied cases, stated that the femoral diastolic pressure is changed but little. In 1937, King2 reported cases in which diastolic pressures averaged more than those in normal subjects. Direct intra-arterial measurements of pressure in clinical cases of aortic coarctation have been reported more recently.4-7 Although comparisons with indirect methods have not generally shown close agreement, they have indicated that systolic hypertension exists in the upper portion of the vascular bed and reduced systolic pressure in the lower portion. Again, great variability was found regarding femoral diastolic pressure. Thus, Brown and his associates,4 using a hypodermic strain gage manometer, found that in 21 cases the femoral systolic pressure ranged from 87 to 133 (average 113) and the diastolic pressure from 63 to 99 mm. Hg (average 81). On the other hand, Bing and his associates6 found in a series of 22 patients that, with one exception, diastolic femoral pressure was below normal. Such variability in diastolic pressure readings is not surprising, since the degree of constriction that exists in different patients naturally varies, and cases of different severity might have been fortuitously included in different groups of patients studied. Furthermore, it has been observed in this laboratory that needling of an exposed femoral artery of a dog can induce a reduction in the plethysmographic volume of a hind limb suggestive of vasoconstriction. Hence, it remains to be demonstrated that the maintenance or elevation of femoral diastolic

From the Department of Physiology of Western Reserve University School of Medicine, Cleveland, Ohio.

This investigation was supported by a grant from the Life Insurance Medical Research Fund, and was carried out during the tenure of one of the authors (T. C. G.) of a Fellowship from the Government of Bihar, India. It constituted part of his thesis, submitted in partial fulfillment of work toward the degree of Ph.D. at Western Reserve University.

Circulation, Volume III, January, 1951
pressure is not due to physiologic alterations incurred by the procedure used for determination of femoral pressure.

In the pressure pulses recorded from patients, chief attention has been focused on the arterial pressure levels; little attempt has been made to discuss the reasons for the alterations in pulse contours. In 1939, Woodbury and his associates published simultaneous intra-arterial pressure curves of the radial and femoral pulses in a case of coarctation. These indicated that the femoral pulse rises and falls much more slowly than the radial and that it is diminished in volume. The upstroke is delayed and slow, the peak broad, low, and rounded, and the contour during diastole is almost a smooth declining curve. Landmarks indicating the beginning of diastole are absent. Femoral pressure pulses exhibiting similar characteristics were published by Brown and his associates and Bing and his group.7

These clinical observations have been supplemented by animal experiments, which, however, were designed chiefly to establish whether the hypertension above the coarctation can be explained as a physical result of the constriction. It has been amply demonstrated by means of acute experiments that the effects of complete aortic occlusion on mean pressure above the constriction depend on the level of compression. It is generally agreed that compression just above the renal arteries is without effect, whereas compression above the origin of the celiac axis or above the diaphragm causes an immediate rise of arterial pressure, as does compression of still more central regions. However, in chronic experiments it has been reported that constriction of the aorta just above the origin of the main renal arteries can also produce hypertension which is apparently not mechanical in origin.13–17

In 1940, Page18 pointed out that occlusion of the aorta close to the site of origin of the renal vessels does not duplicate the dynamics of human coarctation, nor does it necessarily demonstrate that liberation of a renal factor is necessary for development of hypertension during aortic coarctation. Page discovered that, owing to development of collateral circuits, the aorta may be first partially and then completely constricted at the arch without development of hypertension proximal to the occlusion. On the other hand, he could produce hypertension of renal origin by constricting the aorta at the level of the diaphragm only when the aorta was also constricted a few centimeters below the origin of the renal vessels. In 1949, Harreveld restudied the subject in acute experiments. He found that the rise in arterial pressure produced by compressing the aorta above the celiac axis varies directly with the degree of stenosis and is roughly proportional to the diminution in blood flow through the constricted region. From this experimental evidence he concluded that the hypertension proximal to the coarctation is adequately explained by mechanical obstruction. Experimental compression of the aorta just beyond the aortic valves has also been studied to throw light on the dynamics of aortic stenosis.19,20 Coarctation just beyond the left subclavian artery, which alone simulates clinical coarctation, has apparently been studied experimentally only by Page,18 and surprisingly, the hemodynamic consequences have never been fully analyzed.

This investigation was, therefore, carried out to fill certain gaps in our knowledge by studying the arterial pressures and pulse contours in regions of the aorta above and below the stenosis. The effects that different degrees of constriction have on such pressure pulses, the reasons for changes in the femoral pulses that might be of prognostic value, and the determination of the extent to which physical factors alone suffice to explain the hypertension proximal to the constriction, all form part of this investigation.

METHODS

Mongrel dogs were adequately anesthetized with sodium barbital, administered intravenously. Their chests were opened by a midsternal splitting procedure. Artificial respiration was instituted by means of an alternating air pressure and regulated so that natural lung inflation was simulated. The unavoidable loss of circulating fluid was compensated by giving a venous infusion of 100 to 200 cc. of warm, filtered, directly cross-matched, heparinized blood immediately after the major operation and by maintaining a slow drip during the experimental procedure. Aortic pressure was recorded by intro-
ducing a sound through the left subclavian artery so that its tip lay near the aortic valves. Femoral pressure was taken just below Poupart’s ligament by means of a short cannula. In some experiments, abdominal aortic pressure was recorded by means of a small cannula via a renal artery. In other experiments, left ventricular pressure was registered by means of a blunt 15 gage needle introduced directly through the ventricular wall. In still other experiments, pressure pulses were also recorded immediately below the coarctation by inserting a glass T-cannula in the aorta. In these cases, in addition to the midsternal opening, the left side of the chest wall was separated sufficiently between the fourth and fifth ribs to give a satisfactory exposure of the posterior wall of the mediastinum. The upper 3 or 4 cm. of the descending aorta were freed from the posterior chest wall and the vertebral column, keeping close to the vessels so as to leave the thoracic duct undisturbed. The third and fourth intercostal arteries on both sides were then doubly ligated and divided. At this stage the animal was heparinized, and 0.5 cc. of heparin solution* was administered subsequently at 30 minute intervals. All cannulas were securely clamped to avoid vibrations.

Standardized Gregg-type optical manometers, with tensely stretched rubber membranes of adequate frequency and sensitivity, were used for the registration of all pressure pulses. Aortic constriction was produced by a special compressor, the construc-

* The heparin solution used in these experiments was supplied in part by The Upjohn Company, Kalamazoo, Mich.

One half to one hour was allowed, after the completion of the operative procedures, for stabilization of the circulation. The aortic compressor was then progressively tightened, records being obtained during successive steps of the compression. Following each record a short calibration of optical manometers was made with a fixed standard pressure. The zero pressure in all experiments was taken as the hydrostatic level of the animal board, hence pressures as measured were 15 mm. Hg higher than the actual pressures. A complete calibration of each manometer was carried out at the end of each experiment. After the experiment had been completed the loop of cord around the aorta was left in its original position, the aorta was cut without disturbing the cord and its internal diameter was measured, as previously described.4 The degree of stenosis in terms of the percentage of the normal was calculated from the reduction in the area of the aortic lumen with progressive degrees of coarctation.

**Results**

The segments of figure 1 show the effects of progressive coarctation of the aorta on the central aortic, femoral, and left ventricular pressure curves. It will be seen that, as the degree of coarctation is increased, both systolic and diastolic pressures rise in the central aorta and that the amplitude of the curves, i.e., the pulse pressure, increases. Contrariwise, the
femoral pressure pulses show a progressive reduction in systolic, diastolic and pulse pressures until a stabilized mean pressure of 22 mm Hg is reached in segment D.

The average trend of aortic and femoral pressures in 22 dogs with different degrees of aortic constriction is shown graphically in figure 2. The following deductions are self-evident: (1) A reduction of more than 45 to 55 pressures above the stenosis, but the average diastolic pressure does not change until the aortic lumen is reduced to more than 65 per cent of its original size. It then shows a rise much smaller than systolic pressure. Thus, there is a progressive increase in the pulse pressure above the coarctation. The maximum increase in systolic and diastolic pressures does not supervene until the aortic lumen has been per cent in the aortic lumen is required before the arterial pressures in the aorta or femoral artery are affected. With a 55 to 65 per cent stenosis, the average aortic diastolic pressures are approximately the same and the average aortic systolic pressure has increased only slightly. At the same time, the average femoral systolic pressure declines to a greater degree. (2) With progressive increase in coarctation, there is an accelerating increase in the systolic reduced to about 90 per cent. With progressive reduction of the aortic lumen beyond 55 to 65 per cent, the fall in femoral systolic pressure is greatly accelerated. The diastolic pressure also falls, but not to the same extent. Thus, the pulse pressure keeps on decreasing, and at about 85 to 95 per cent stenosis it becomes so small that a femoral pulse is just discernible. This is quite understandable, for when the aorta has a larger lumen a certain percentile

---

**Fig. 2.** Chart summarizing average aortic and femoral systolic and diastolic pressure changes with progressive coarctation of the aorta.
decrease alters its area proportionately much less than the same percentile decrease does when the lumen is narrow. Neglecting any change in pressure, the volume of blood flowing through a stenosed lumen varies as a fourth power of the radius of the aorta at that level and, according to Poiseuille's law, the flow should be suddenly curtailed to a greater extent in extreme degrees of coarctation.

Effect of Progressive Aortic Compression on Aortic Pressure Contours

In order to interpret the effects of progressive narrowing of the aorta on the pulse contours beyond a constriction it is important to know how the parent pulses central to it are modified. This has so far not been accomplished by studies of human subjects.

An analysis of many records revealed that changes in the configuration of the central aortic pressure pulse depend not only on the degree of constriction but also, to a considerable extent, on the stroke volume and competence of the left ventricle. This is illustrated by aortic pressure pulses in figures 1, 3, 4 and 5. Figure 1 indicates that the systolic discharge previous to compression (segment A) was obviously below par. Consequently, progressive aortic compression merely serves to restore the aortic contours to forms that resemble those recorded from normal dogs and human subjects. This process was accompanied by changes in intraventricular pressure typical of increased aortic resistance. Figure 1A shows that the amplitude of the curves progressively increases, the isometric gradients become steeper, and the peaks are displaced progressively toward the end of systole; but in this case a measurable increase in initial tension and in duration of systole occurs only in segment D, i.e., after complete occlusion of the aorta. Apparently, the left ventricle is capable of compensating fully in such instances; indeed, judgments based on the increasing pulse pressure and forms of the aortic pulses strongly indicate that the systolic discharge is increased. This is supported by values obtained in several experiments through application of the Hamilton-Remington technic. The data from one of these analyses are concisely incorporated in table 1, which, incidentally, reveals that the calculated total peripheral resistance expressed in absolute units (A.U.) is not significantly raised until constriction equals 70 per cent of the natural aortic diameter.*

Figure 3 illustrates records from an experiment in which the control aortic pressure pulse has a contour which is essentially normal for dogs and man, although the diastolic and systolic pressures are a trifle high. With increasing aortic compression, as exhibited by successive segments, the top of the pressure curves assumes more and more an ascending plateau, terminating nearer and nearer the end of systole. These are characteristic effects, commonly described as due to increasing peripheral resistance.

* It has been our experience that calculations of systolic discharge and cardiac output by the pulse contour method are at times consistent and at other times inconsistent with expected or otherwise determined values during changing hemodynamic states. The augmentation of cardiac output as a result of aortic compression indicated in table 1 corresponds, at least directionally, with results in similar experiments in which cardiac output was determined from cardiomter records. It is interesting to note that this was true despite the fact that the basic premises upon which the procedure was founded were probably disturbed through high constriction of the aorta (C.J.W.).
HEMODYNAMIC CHANGES PRODUCED BY AORTIC COARCTATION

Indeed, an actual decline may take place. There is, furthermore, a considerable fall of diastolic pressure which is chiefly responsible for the larger pulse pressure. The pressure portion of systolic ejection. Such transformations are attributable to an altered pattern of

Fig. 3. Effects of progressive coarctation on the aortic (upper curves) and femoral pressure pulses (lower curves). Segment A, normal relationship between the central aortic and femoral pulses. Segments B, C, D, E, F and G show effects of 60, 65, 71, 76, 80 and 85 per cent reduction in aortic area at region of the constriction.

Fig. 4. Effects of progressive coarctation on the central aortic (upper curve), abdominal aortic (middle curve) and femoral arterial (lower curve) pulse contours. Segment A, normal relationships when the aorta is unconstricted; segments B, C, D and E, effects of 54, 64, 78 and 83 per cent reduction in the aortic lumen.
systolic ejection, the ventricles apparently emptying a larger fraction of their systolic stroke volume during the early moments of ejection.

Figure 5 shows the changes in central aortic pressures during an experiment in which a glass cannula had been inserted into the aorta. The curve is much more peaked, owing to a marked decline of pressure toward the incisura during late systole. Such a late systolic collapse indicates a disproportion between aortic uptake and efflux, due either to reduction in resistance or in ventricular discharge during the period under consideration. The possibility that the peak is deformed through reflected waves has been considered and discarded. That the altered form of central aortic pressure pulse is due to an abnormal pattern of ventricular ejection in this experiment is supported by the facts that (1) the collapsing characteristic is maintained even when pronounced constriction of the aorta has occurred (segments B, C and D), and that (2) in similar experiments the normal configuration was preserved.

The foregoing illustrations emphasize the importance of being familiar with the configuration of central aortic pressure pulses possible in experimental animals and in clinical cases, for they determine largely the effect of progressive coarctation on pressures above the lesion and on contours of pulses below it.

**Effect of Different Degrees of Aortic Compression on Contours of Femoral Pressure Pulses**

The progressive changes in femoral pulses are also shown in figures 1, 3, 5. The curves exhibit a progressive reduction in amplitude and a retardation of the anacrotic limb. The summit appears increasingly later in systole and, instead of being sharp and peaked, is broad and rounded. A more careful study of such records suggests that the transformation occurs in two stages: (1) With moderate constriction the femoral systolic pressure no longer exceeds aortic pressure, as is normally the case; it either equals it, as in figure 3B, or is less, as in figure 5B. Also, the contour of the femoral pulses is but slightly affected. (2) With further constriction, illustrated by subsequent segments of these figures, the dicrotic wave becomes less and less prominent and is finally abolished, the landmarks separating systole and diastole disappear, and the diastolic decline of pressure becomes essentially a smooth curve. At the same time, the pulse pressure diminishes progressively through a predominant fall in systolic pressure. The peak of the pulse is also conspicuously—and palpably—retarded, e.g., from 0.128 second in segment A to 0.2 second in segment G of figure 3, but an anacrotic halt characteristic of stenosis at the aortic valves is absent in the curves. The pulse wave is also delayed in its transmission, but
not conspicuously so. For example, the lag increased only from 0.056 second in segment A to 0.074 second in segment G of figure 3. Pulses were recorded in some experiments from the descending aorta at the level of the renal artery and immediately below the coarctation.

Fig. 6. Records showing the murmurs produced by coarctation of the aorta. Upper curves, aortic pressures above coarctation; middle curves, aortic pressures immediately below coarctation; lower curves, femoral pressures. Segment A, control with unconstricted aorta; segments B, C, D, E, and F, effects of 60, 68, 73, 78 and 86 per cent stenosis.

Effect of Progressive Compression on Pressure Pulses beyond a Constriction

In order to interpret the changes in femoral pulse contours more satisfactorily, pressure

In experiments in which the central aortic pressure pulses resembled those shown in figures 1, 3 and 5, the aortic pressure pulses recorded via a renal artery resembled those in
the femoral artery as regards main characteristics. This indicates that the transformation has taken place by the time the pulse wave reaches the lower aorta. The curves of figure 4 are introduced to illustrate that the basic transformation of the peripheral pulses is not altered by progressive coarctation when a state of hypertension exists before the compression. The middle record of this figure shows that the pressure pulses from the lower descending aorta resemble those taken from the femoral artery (lower curve). It may be noted that, as previously described by one of us in cases of human hypertension, the incisura of the central pulse is well transmitted to the periphery, even when the aorta has been constricted to 64 per cent of its original size (segment C).

Returning to figure 5, the middle curve portrays pressure changes in the aorta just distal to the coarctation. Crosslines drawn in segment A show that at the time the peak of pressure is reached above the constriction (upper curve) the pressure is the same below the constriction (middle curve). However, the pressure continues to rise below the constriction while it declines above it. Since the summit practically coincides with the peak of the femoral pulse, it follows logically that a standing wave is added to the pressure curve in this region. This interpretation becomes more plausible by a glance at segment B of figure 5, in which a definite wave is superimposed after the aorta has been slightly constricted. With increasing degrees of compression, the initial steep rise becomes less and less and the curve continues as a protracted rise until the incisura. Fine vibrations, irregular in size and frequency, can also be observed on the rising pressure curve. These occasionally extend even beyond the incisura, which becomes progressively less conspicuous. The pressure oscillations are graphic representations of the systolic murmur that accompanies coarctation. It will be observed that these murmur vibrations do not start from the anacrotic halt of the pressure pulse but are separated from it by a short period of smoothly rising pressure. Since there is no similarity between these curves and those taken from the femoral artery, it is obvious that the transformations that occur in the femoral pulse with increasing degrees of constriction do not arise immediately below the coarctation.

Figure 6 is an original record of the murmurs in progressive coarctation of the aorta. Normally, in segment A the curve recorded from the glass cannula is free of any murmur. As the stenosis is increased to 60 per cent (segment B), a late systolic crescendo murmur of short duration appears. With 68 per cent reduction of the aortic lumen (segment C), the systolic murmur starts about the middle of the ascending limb, is still crescendo, and is continued as a diastolic decrescendo murmur. On increasing the coarctation to 73 per cent (segment D), a crescendo-decrescendo murmur reaching great intensity starts about one-third up the ascending limb. The decrescendo portion continues into early diastole. With still further increase in coarctation to 78 per cent (segment E), the murmur becomes less intense. In severe degrees of coarctation (segment F), the murmur is present throughout systole and diastole, but its amplitude becomes so small that it is no longer audible. The vibration frequency of the murmur varies between 120 and 138 per second.

Discussion

Similarities of Clinical and Experimental Coarctation. Femoral pressure pulses, recorded from patients with aortic coarctation by Woodbury, Brown, Bing, and their respective associates, are very similar to those that we produced by experimental coarctation. The results of our experiments are, therefore, applicable to clinical cases of coarctation. However, in patients, equivalent degrees of aortic constriction may not affect the femoral pressure pulses to the same extent, owing to established collateral channels. (See succeeding article.)

It is common clinical knowledge that a palpable delay exists between the radial and femoral pulses in patients with marked aortic coarctation. Measurement of our records, however, reveals that the increased delay in transmission of the pulse wave from the aorta to the femoral artery is too small to be detectable by palpation. Since the tactile stimulus is given by the primary rise of pulse waves,
The difference in their summits, which is significantly greater owing to the slow rise of the femoral pulse, probably accounts for the clinical impression of a delay. Somewhat similar interpretations were made by Lewis\textsuperscript{25} and Feil and Gilder\textsuperscript{26} in other disorders.

The slow rise of the femoral pulse, evident in experimental and clinical pressure pulses, may be appreciated by palpation. It is due in part to the damping of the parent wave at the constriction and in part to the failure of a less forceful impact to set up a standing wave in the aorta, thus preventing summation of pressure on the anatomic limb and a systolic overshooting, such as occurs in the normal femoral pulse.\textsuperscript{27} (For further discussion, see later part of paper.)

**Hemodynamic Alterations above the Coarctation.** The physiologic and physical effects cephalad to the region of coarctation are not limited to those produced by increased resistance alone, as is commonly inferred. They involve also changed characteristics of the aortic compression chamber, adaptations of the left ventricle, and adjustments of flow which assure an adequate venous return.

Our data reveal that the resistance effects exerted by progressive narrowing of the aorta are detected sooner through elevation of left ventricular and aortic systolic pressures than in calculations of total peripheral resistance (TPR). For example, as shown in table 1, a 60 to 65 per cent constriction is without significant effect on TPR, but causes a significant change in left ventricular pressures (fig. 1B). When central arterial pressures are greatly elevated, as in figure 4, the larger pulse pressure may be due, in part at least, to the rapidly diminishing distensibility of the aorta, but this does not explain the increase at lower pressure ranges, such as persist in the curves of figure 1C. The suspicion that systolic discharge is also increased is supported by calculations shown in table 1. This increase in systolic discharge is an important factor in the larger pulse pressure when the left ventricle compensates. When such compensation does not occur systolic pressure may even decline slightly, as in figure 4. In these cases the increase in pulse pressure is due entirely to reduction of diastolic pressure, the cause of which may now be discussed.

Lowering of resistance through opening of natural collateral channels can be excluded in these experiments, for the decline of diastolic pressure occurs too promptly and is not prevented by ligation of all accessible large collaterals. The temptation at once arises to invoke reduction of resistance in aortic branches above the coarctation through aortic and carotid sinus reflexes. In addition, it has been found in similar experiments performed in this laboratory that resistance to coronary flow decreases, partly for mechanical reasons\textsuperscript{28} and partly through local actions of metabolites.\textsuperscript{29} As a result, the coronary channels carry a large fraction of the flow that is prevented from leaving the aorta through a stenotic orifice. Incidentally, this insures a better blood supply for the hyperactive ventricles. However, the concept that the decline of diastolic pressure is due to reduced resistance in branches of the aortic arch is inconsistent with (1) the absence of changes in heart rate such as generally occur pari passu with reflex vasomotor changes when pressoreceptors in the aorta and carotid sinus are excited by a rise in arterial pressure; (2) the failure of calculations (table 1) to indicate that a reduction in TPR has occurred; and (3) the maintenance rather than decline of diastolic pressure below the stenosis. If reflex dilatation of arterioles had taken place, those in the splanchnic area, kidneys, and hind limbs would have been affected to an even greater extent and, hence, diastolic pressure below the stenosis would have declined. Obviously, some factor other than reduction in resistance in aortic branches must be responsible for the total changes in central pressure pulses, viz., the rise of systolic and decline of diastolic pressures, and the slight increases in systolic discharge and calculated total peripheral resistance while the heart rate remains constant (figs. 3, 5).

Dynamic studies on artificial circulation machines (Wiggers\textsuperscript{30}) show that such a com-
Combination of changes does result when the distensibility of the compression chamber is reduced. Upon reflection, this is the situation that develops during aortic coarctation: the compression chamber is greatly reduced in size and its walls are put under greater stretch. Hence, we attribute the effects on systolic pressure (in part) and on diastolic pressure (entirely) to an increase in the volume elasticity coefficient \( \frac{dp}{dv} \).

We have referred to the possibility that systolic discharge and cardiac output may be augmented as a reaction to aortic compression. Since this occurs with compression of 55 per cent or more, and despite the fact that aortic pressures below the constriction have declined to low levels, it would seem that the venous return via the inferior cava must be drastically reduced. Since augmented cardiac output can be maintained only when the total return flow is adequate, the inference follows that the reduced inferior cava flow is compensated by increased flow through the superior cava and coronary venous system. That this is true has been demonstrated by Katz and Wiggers, who found in similar experiments that right arterial and ventricular pressures may even be slightly elevated and that this is not due to "back pressure effects." As inferred by these authors, the greatly augmented flow through the coronary system contributes greatly to rebalancing the venous return.

Hemodynamic Alterations below the Coarctation. In order to interpret changes in pressures and pulse contours below aortic coarctation of various degrees, the dynamics of pressure transmission in the aorta under normal circumstances must be kept in mind. The transformation that the aortic pressure pulses undergo during their passage through the aorta has been discussed by several recent writers. The changes are chiefly caused by natural damping in a low frequency system and superposition of standing waves. Under normal conditions, natural damping results in (a) a retardation of the rise of the initial part of the pressure pulse, (b) some rounding of the summit, and (c) annulment of finer oscillations such as the pre-ejection vibrations and sharp incisura and its after-vibrations. The superposition of a standing wave causes the pressure wave in the lower aorta (a) to rise rapidly to a higher pressure peak than exists in the ascending aorta, (b) to decline rapidly during late systole and early diastole, and (c) to develop a dicrotic dip succeeded by a dicrotic wave of considerable size. These changes are transmitted to the femoral arteries (cf. figs. 3A; 5A.) These natural transformations can be modified decidedly by changes in (a) the elasticity coefficient of the aorta, (b) peripheral resistance, (c) other conditions that alter the natural frequency of the aortic reservoir and rate of travel of reflected waves and, as in these experiments, (d) damping of the parent pressure waves at the constriction. A great deal concerning this last factor can be learned by progressive damping of an optical manometer through gradual closing of a stopcock while aortic pressure pulses are being recorded. A large experience with such trials has helped us to interpret the effects of progressive degrees of coarctation on the femoral pressure pulses. But the effects are not quite identical, for a great deal depends upon the natural frequency of the system which picks up the damped vibrations. The inherent frequency of the descending aorta is very low, usually 3.6 to 5 per second. It increases when the walls are stretched, as in hypertension; it decreases when the walls become more lax during hypertension, as in these experiments.

Under circulatory conditions that develop with progressive aortic compression, damping reduces the force of the initial impact as blood is ejected. Consequently, as soon as this is not sufficiently violent to set the aortic blood column into oscillation no standing wave is induced. The effect of critical damping appears early during compression, so that with approximately 55 per cent compression systolic pressure in the femoral artery becomes equal to or slightly less than that in the aorta, but, owing to the low frequency of the arterial system, the pulse contour is deformed. After
greater degrees of stenosis, the systolic and pulse pressures progressively decline and the ascent becomes more gradual. However, the delay in transmission is not conspicuously affected.

The effects simulate progressive damping of mercury manometers used for arterial pressure recording. As in these, so in the aorta, progressive damping decreases the amplitude of oscillations until only a mean pressure is transmitted. Since the configuration of pressure waves places the mean nearer to the diastolic pressure, it follows that diastolic pressure is sustained relatively better than systolic. In this way, the relatively high diastolic pressure is explainable without invoking the operation of arteriolar vasoconstriction and liberation of a humoral agent to activate such a process. Parenthetically, it is our considered opinion that, while it is quite possible, in chronic coarctation, that a renal factor may contribute to the hypertension above a stenosis and to the sustenance of diastolic pressure below, the latter cannot be used as evidence for the existence of increased resistance. Likewise, studies of blood flow in the lower extremities of patients with coarctation are incapable of determining the existence of increased arteriolar constriction, for it is not known what the pressure-flow relations would be at equivalent pressures under normal conditions.

Concerning Turbulence. It has long been suspected, from a priori physical considerations and the presence of a murmur, that a state of turbulence is created beyond an aortic constriction. Our records reveal that they resemble vibrations previously reported in aortic valvular stenosis.19,20 In some cases (fig. 6) the murmur is preceded by a sharp incisura, but this is not followed by an anacrotic femoral pulse, indicating, as Dow20 found, that the two are not related as cause and effect phenomena.

We have seen no evidence that such a local turbulence beyond the stenosis has any effect on the pulse contours in the lower aorta and femoral artery. However, a few interesting features were noted: (1) The murmur appears before pressure relations are significantly affected (fig. 6B); hence, greater importance should perhaps be placed on auscultatory phenomena in suspicious diagnosis of incipient states of coarctation. (2) The murmur increases in intensity up to a degree of stenosis critical for pressures cephalad to the constriction, and thereafter diminishes (fig. 6D, E). The magnitude of constriction can, therefore, not be judged from the intensity of murmurs heard on auscultation. (3) The murmur starts after ejection and extends into the early phase of succeeding diastole, i.e., as long as a considerable pressure gradient for maintaining the turbulence continues.

The question has, naturally, been considered whether the development of turbulence, like the erosive action of a whirlpool on stream banks, may not have some deteriorating effect on the vascular walls, causing them to weaken or lose elasticity, thus creating a hemodynamic factor which is responsible for the subsequent dilatation below the stenosis. Such a suggestive explanation must be held sub judice until we have more basic information regarding the effects of turbulence on stream boundaries of fluids contained in tubes.

Summary

1. The basic hemodynamic factors that determine pressure relations above and below an aortic coarctation were studied experimentally in dogs by recording pressure pulses from the aortic arch and femoral artery, and also, in certain experiments, from the left ventricle and different levels of the descending aorta, by means of calibrated optical manometers.

2. Various degrees of circular constriction up to complete occlusion were produced in the aorta just beyond its left subclavian branch by means of a special compressor that could be calibrated at the end of an experiment.

3. Measurements of calibrated pressure curves revealed that a reduction in aortic lumen greater than 45 to 55 per cent is required to produce any changes in arterial pressures. The effects of successively greater reductions on systolic, diastolic, and pulse pressures above
and below the constriction are conveniently summarized in figure 2.

4. In order to evaluate the pressure changes beyond an aortic coarctation one must be familiar with the configuration of the parent aortic pressure pulses above the stenosis. Since these alterations have apparently not been taken into account in previous hemodynamic analyses of aortic coarctation, modifications of the aortic pressure pulses, induced by varying degrees of constriction, were first carefully analyzed.

5. Such studies revealed that the hypertension in the aorta above a coarctation is not due solely to increased resistance, as has been commonly inferred. Equally important are (a) the reduced capacity and distensibility of the aortic compression chamber into which the left ventricle empties its blood during each systole and (b) the physiologic reactions of the left ventricle whereby its systolic discharge is increased. The latter were interpreted by analyses of left ventricular pressure curves.

6. Since the venous return to the heart by way of the inferior vena cava must decrease progressively as the degree of aortic constriction increases, it would obviously be impossible for the left ventricle to maintain a larger cardiac output unless compensatory increases in flow took place through various branches of the aortic arch, including the coronary system. Previous evidence from this department that such compensatory flow takes place was substantiated. It is evident that any mechanism which reduces the flow through branches of the aortic arch, such as the reflex or humoral constriction of arterioles, would not be a propitious event, and we have discovered no evidence that such reactions supervene.

7. The corollary follows that augmented resistance in small terminals of the aorta proximal to a coarctation can probably contribute little to the hypertension of aortic coarctation. By reducing blood flow through these circuits, the venous return to the heart would be so seriously impaired that cardiac output of the left ventricle would be reduced and aortic pressures would automatically decline. In short, the hypothesis that the hypertension of aortic coarctation is due to a renal factor is difficult to square with dynamic laws.

8. Studies of pressure pulses in the descending aorta at various distances beyond the stenosis indicate clearly that the changes in configuration of the femoral pulse start above the level of the renal arteries. The slow gradients of the anacrotic and catacrotic limbs and the abrogation of the dicrotic wave and notch, which characterize femoral pulses in severe coarctation, are the direct result of damping of the aortic pressure wave in its passage through the constriction. Since the impact imparted to the blood column by the force of ventricular systole is reduced, the aortic blood column is not thrown into oscillation and the normal standing wave is not created. In consequence of the latter, the gradient of pressure rise decreases, the normal systolic overshoot is prevented, and the dicrotic wave is abrogated. The damping factor is also the important factor that causes a smaller pulse amplitude during moderate degrees of aortic constriction, but the diminishing pulse volume that reaches the descending aorta is obviously of dominant importance in more severe grades of stenosis.

9. To a considerable extent the relatively good maintenance of femoral diastolic pressure as systolic pressure falls is also an indirect effect of damping. Damping tends to reduce pressure variations toward a mean level, such as is actually realized in very severe stenosis. With lesser degrees of stenosis and damping, pulse pressures merely diminish, but since the mean pressure lies nearer to the diastolic level the latter pressure is better maintained than is systolic pressure. The corollary follows that a comparatively high femoral diastolic pressure is not dynamic evidence that additional vasoconstriction exists in branches of the lower aorta.

10. With progressive aortic constriction, a murmur indicating a state of turbulence just beyond the constriction begins to appear before pressure relations are significantly altered above and below a constriction. The intensity of the murmur first increases and then de-
creases with progressively greater constriction. It begins shortly after the pressure rise and extends slightly into diastole, i.e., it continues as long as a pressure gradient exists across the constricted region. The question whether the turbulence created below the coarctation develops forces which may weaken the arterial wall and subsequently lead to dilatation of the aorta cannot be answered until we have more information regarding the effects of turbulence on stream boundaries of fluids in elastic tubes.

REFERENCES

1 Abbott, M. E.: Coarctation of the aorta of the adult type; a statistical study and historical retrospect of 200 recorded cases with autopsy of stenosis or obliteration of the descending arch in subjects above the age of 2 years. Am. Heart J. 3: 574, 1928.


12 —, and Formline, P.: The relation of the central nervous system to the increase in systemic flow produced by the occlusion of the thoracic aorta. J. Physiol. 82: 377, 1934.


26 Feil, H. S., and Gilder, M. D.: The pulse in aortic disease as felt and graphically inscribed. Heart 8: 4, 1921.


28 Gregg, D. E., and Green, H. D.: Effects of viscosity, ischemia, cardiac output and aortic pressure on coronary blood flow measured under
a constant perfusion pressure. Am. J. Physiol. 130: 108, 1940.

29 —, AND SHIPLEY, R. E.: Augmentation of left coronary inflow with elevation of left ventricular pressure and observations on the mechanism for increasing coronary inflow with increased cardiac load. Am. J. Physiol. 142: 44, 1944.


Basic Hemodynamic Changes Produced by Aortic Coarctation of Different Degrees
TRILOK CHAND GUPTA and CARL J. WIGGERS

Circulation. 1951;3:17-31
doi: 10.1161/01.CIR.3.1.17

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1951 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/3/1/17

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not
the Editorial Office. Once the online version of the published article for which permission is being requested
is located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/