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THE OBSCURE PHYSIOLOGY OF POSTSTENOTIC DILATATION: ITS RELATION TO THE DEVELOPMENT OF ANEURYSMS

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IN THE realm of rheology, the science of flow and elasticity, there exists a curious and puzzling phenomenon known as poststenotic dilatation. Many surgeons have noted it, few have provided an adequate reason for its presence or have ventured an explanation for the mechanism of its development. William S. Halsted was greatly intrigued by it, and as usual took the problem to the experimental laboratory, where a mild dilatation distal to partial ligation of the terminal aorta was observed in seven of thirty attempts in adult dogs.[1] He suggested that the "abnormal whirlpool-like play of the blood in the relatively dead pocket just below [distal to] the site of the constriction, and the lowered pulse pressure may be the chief factors concerned in the production of the dilation." But he also stated that "it is not denied that the paralysis of the vasomotor nerves, and the occlusion of the vasa vasorum may possibly play some part in the manifestation."

Although poststenotic dilatation was first observed and described in the subclavian artery beyond a congenital cervical rib, thoracic surgeons and radiologists have become increasingly familiar with its occurrence in the presence of other congenital abnormalities of the heart and large vessels. Striking is its occurrence beyond a subaortic stenosis, as illustrated by the following case:

CASE 1.—A. M., single, aged 43, was admitted to Stanford Hospital on Jan. 25, 1953, for study and possible operation upon an aneurysm of the ascending aorta.

His past medical history revealed that ever since infancy he had been told he had a bad heart, and that he had not been allowed to play with other children. He had led a fairly normal life, except that he had been turned down by the Army because of his heart. In October, 1952, he had been seized with a pain in the right chest which was thought to be pleurisy, but which promptly subsided on restricted activity. Recent to the 1953 admission, a pain in the left chest, together with some dyspnea, had again sent him to doctors for advice, and a diagnosis of aneurysm of the ascending aorta had been made. His past history revealed treatment for syphilis twenty years previously, which was said to have been con-

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genital, since his mother had died of neurosyphilis and his sister had been treated for interstitial keratitis.

Physical examination was negative except for a moderate increase in cardiac dullness, a harsh systolic murmur heard best over the right third intercostal space, and a loud aortic second sound with a soft, blowing early diastolic murmur over the left third intercostal

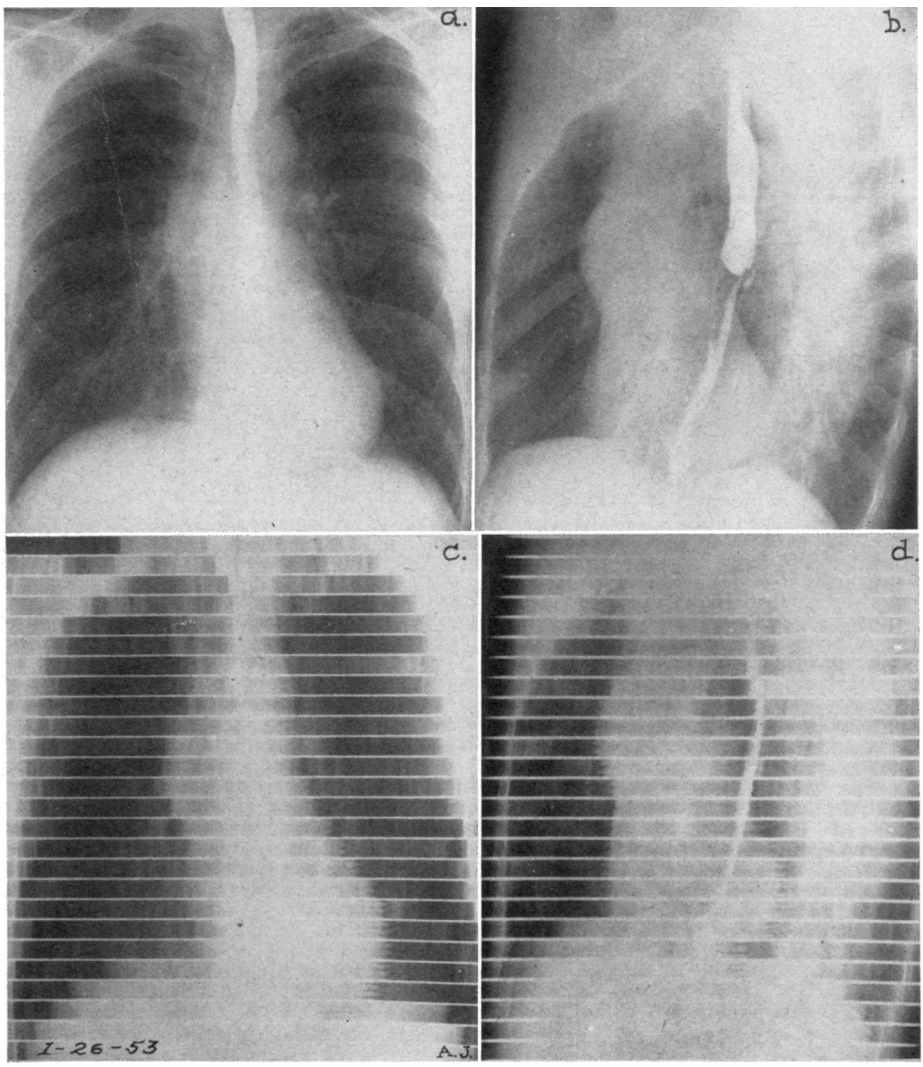


Fig. 1.—Case 1. Roentgenograms and kymograms show a markedly dilated ascending aorta originally considered a syphilitic aneurysm. However, the patient was 43 years old and was known to have had a faulty heart since infancy. A diagnosis of congenital subaortic stenosis with poststenotic dilatation was made.

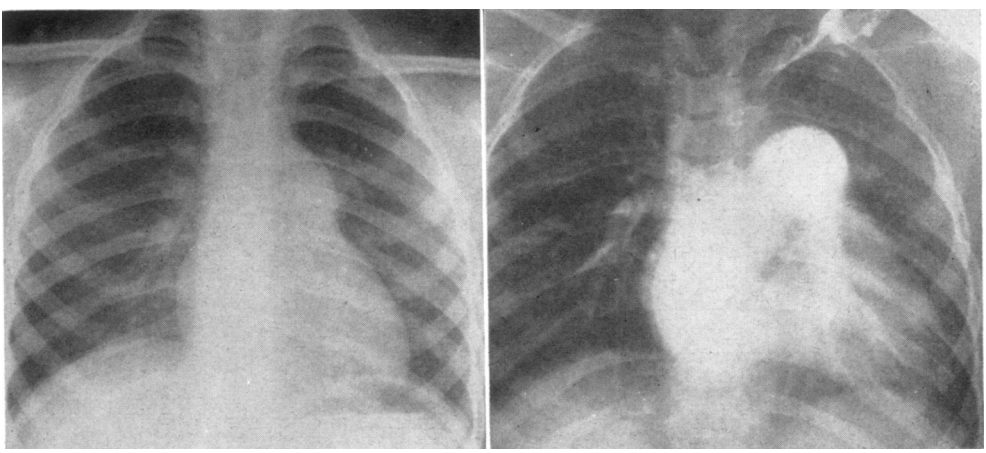
space. The blood pressure was 135/90, pulse rate 88. Roentgenograms and kymograms (Fig. 1) showed uniform dilatation of the ascending aorta with normal pulsation and with no evidence of regurgitation. Spinal fluid studies revealed a negative Hinton and a nor-

mal colloidal gold curve. The electrocardiogram was normal. A diagnosis of congenital subaortic stenosis with poststenotic dilatation of the ascending aorta was made. He was discharged without operation.

A conspicuous and more frequently seen form of poststenotic dilatation occurs distal to a congenital stenosis of the pulmonic valve, of which the following two cases are typical examples:

CASE 2.—J. B. was first seen in the Children's Cardiac Clinic on Aug. 1, 1951, when 4 years old, because of a cardiac murmur noted at birth.

Physical examination revealed a mild mental retardation, a loud harsh systolic murmur heard over the entire precordium, maximal over the second left intercostal space; pulse rate 88, blood pressure 112/70; hemoglobin 13 Gm. per cent; and 4,800,000 red cells. Roentgenograms and angiocardiograms in March, 1953, showed marked prominence and dilatation of the pulmonary artery (Fig. 2). Cardiac catheterization disclosed a right ventricular pressure of 98/4 mm. Hg, and a pulmonary artery pressure of 19/2 mm. Hg, with a femoral artery pressure of 96/60 mm. Hg. Oxygen analyses failed to disclose any evidence of an intracardiac shunt.



A. *B.*

Fig. 2.—Case 2. J. B. Roentgenograms and angiograms in a patient aged 6 showed marked prominence of the pulmonary artery. At operation a congenital stenosis of the pulmonary valve was found with marked poststenotic dilatation. *A*, Aug. 1, 1951; *B*, March 19, 1953.

Because of exercise intolerance (dyspnea and fatigue on running more than twenty yards) and the high right ventricular pressure, the patient was operated upon May 28, 1953. A valvular stenosis was incised and dilated with definite clinical improvement.

CASE 3.—R. W., aged 10, was first seen in June, 1949, at age 5, because of a cardiac murmur accidentally discovered two years previously. Although he seemed well at the present admission, he had begun to tire easily on exertion associated with the appearance of a circumoral blueness.

Physical examination revealed a loud systolic murmur maximal in the second left intercostal space, obscuring pulmonic second sound; pulse rate 88, blood pressure 100/70; good femoral pulses, hematocrit 46; hemoglobin 14 Gm. per cent; capillary oxygen saturation 96 per cent. Roentgenograms showed a marked enlargement of the pulmonary artery, confirmed by angiograms (Fig. 3).

Cardiac catheterization in April, 1951, disclosed a right ventricular pressure of 103/14 mm. Hg and a pulmonary artery pressure of 19/13 mm. Hg, with a femoral artery pressure of 125/88 mm. Hg. The catheter passed easily into the left auricle through a patent interauricular septum, but oxygen studies gave no evidence at rest of an intracar-

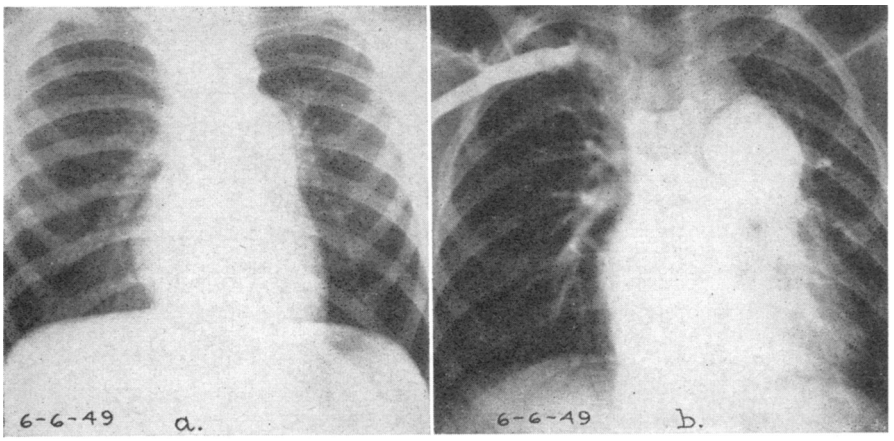


Fig. 3.—Case 3. Patient R. W. Marked prominence of pulmonary artery proved by angiograms and operation to be distal to a tight congenital stenosis of the pulmonic valve.

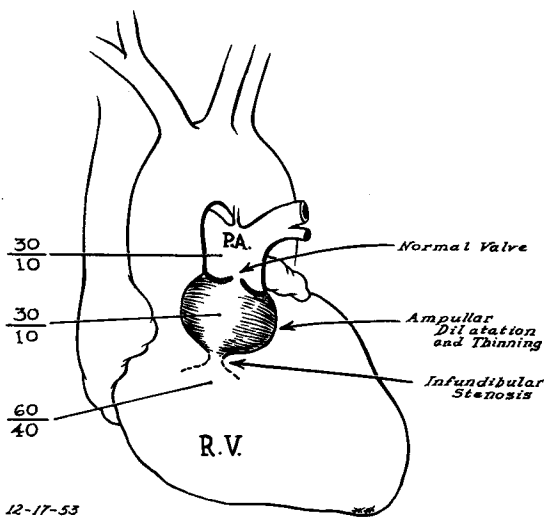


Fig. 4.—Case 4. Patient C. L. Low infundibular stenosis with normal pulmonary valve does not cause dilatation of pulmonary artery, but may cause dilatation of ampulla between the infundibulum and the valve.

diac shunt. Because of the high right ventricular pressure, operation was performed and confirmed the presence of a tight valvular stenosis with marked dilatation of the pulmonary artery distal to it.

In contrast to pulmonic valvular stenosis, the low infundibular stenosis of the pulmonary outflow tract with a normal pulmonary valve is rarely associated with dilatation of the pulmonary artery, but there may occasionally be seen in angiograms, or at operation, a small ampullar dilatation lying between the stenosis and the normal pulmonic valve (Fig. 4).

CASE 4.—C. L., a 12-year-old boy, was first seen at age 7 because of dyspnea, easy fatigability, a cardiac murmur known to have been present since birth, and a history of cyanosis at birth which soon disappeared, but which recurred slightly on exercise.

Physical examination disclosed a pale grayish cast to his skin, with questionable cyanosis of lips, pulse rate 84; blood pressure 118/82 mm. Hg in the arm, 124/77 mm. Hg in the leg; a systolic thrill in the second and third intercostal spaces with a loud systolic murmur heard over the whole precordium, but maximal in the left third intercostal space; 6,300,000 red cells, 16 Gm. of hemoglobin; and a right axis deviation in the electrocardiogram. On cardiac catheterization, a right ventricular pressure of 170/5 mm. Hg was found, with a pulmonary artery pressure of 19/2 mm. Hg and a femoral artery pressure of 110/64 mm. Hg. There was no evidence of an intracardiac shunt. Roentgenograms and angiograms (Fig. 5) showed a large right auricle and ventricle, and a small pulmonary artery, whose peripheral branches were also small.

At operation on Dec. 17, 1953, an infundibular stenosis was found, just distal to which lay a small round knob of poststenotic dilatation proximal to a normal pulmonary valve with normal pulmonary arteries. Dilatation and fragmentary removal of some cardiac muscle resulted in limited improvement.

Dilatation of the descending aorta beyond a coarctation occurs frequently, although not invariably, and may occasionally offer real difficulty in the reestablishment of continuity with the much smaller proximal aorta:

CASE 5.—C. M. was admitted to Stanford Hospital on April 4, 1952, when 3 months old because of coughing spells and difficult breathing.

At birth the baby had had a good color and weighed 7 pounds, 8 ounces. Within a few days of birth, difficult breathing accompanied by a slight but persistent cough was noted. When 23 days old, she was treated with penicillin because of a presumed bronchopneumonia. Temporary improvement was followed by gradual deterioration with increasing symptoms until readmission to the hospital.

Physical examination disclosed a poorly developed, chronically ill, pale baby, without cyanosis; pulse rate 190; respiration 74; a moderately enlarged heart, with a loud systolic murmur heard over the precordium and over the posterior lung fields; slightly palpable femoral pulses; an electrocardiogram that showed right ventricular hypertrophy; 11.7 Gm. hemoglobin; 3.75 million red cells; blood pressure 98/70 mm. Hg in the right arm, 92/60 mm. Hg in the left arm, and 58/40 mm. Hg in the left leg.

A retrograde aortogram through the left brachial artery revealed the presence of a coarctation at the usual site beyond the left subclavian artery (Fig. 6). A month after admission the child died with evidence of right upper lobe pneumonitis. Autopsy revealed a coarctation and a small interventricular septal defect.

Compression of the subclavian artery in the angle between the scalenus anticus muscle and the cervical rib to which it is abnormally attached may also produce moderate aneurysmal dilatation of the artery distal to the stenosis (Fig. 7),[2] although the astute W. W. Keen,[3] long dean of American surgeons, once expressed wonder that it did not occur proximal to the obstruction. William S. Halsted[1] abstracted 716 cases of cervical rib recorded in medical literature, and in twenty-seven cases an enlargement of the subclavian artery, fusiform, aneurysmal, or cylindrical, was described. He also reported[4] a unique example of acquired poststenotic dilatation following the application of a partially occluding metal band to the innominate artery for the cure of a subclavian aneurysm (Fig. 8). Twelve years later the common carotid artery distal to the stenosing band was strikingly dilated throughout its entire length.

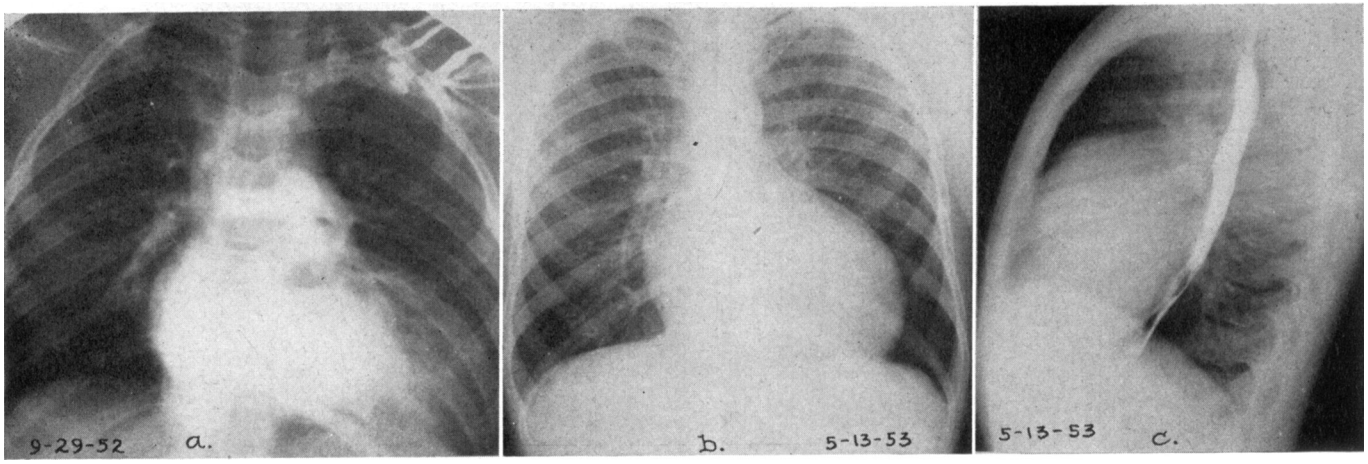


Fig. 5.—Case 4. Patient C. L. Roentgenograms and angiograms confirm absence of dilatation of pulmonary artery in presence of an isolated infundibular stenosis (see Fig. 4).

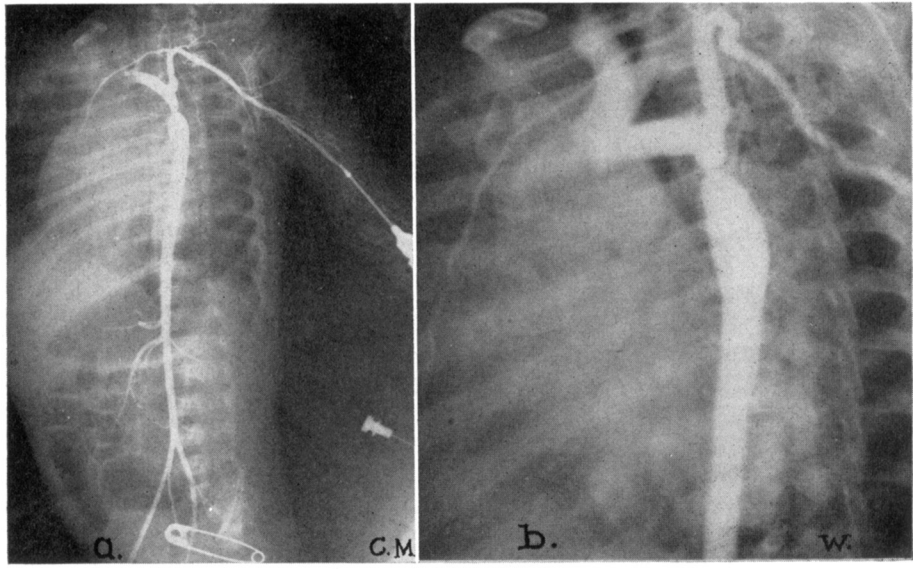


Fig. 6.—(*a*) Aortograms disclose marked dilatation of thoracic aorta beyond a coarctation in a 4-month-old infant; (*b*) dilatation in a second 4-month-old infant.

The most lucid description of the character of poststenotic dilatation as observed at necropsy is contained in a report, published in 1869, by Alfred Poland,[5] an English surgeon, who noted that "the lining membrane, as well as the middle and external coats of that portion of the artery affected by the swelling were perfectly healthy" and that "there was no atheromatous

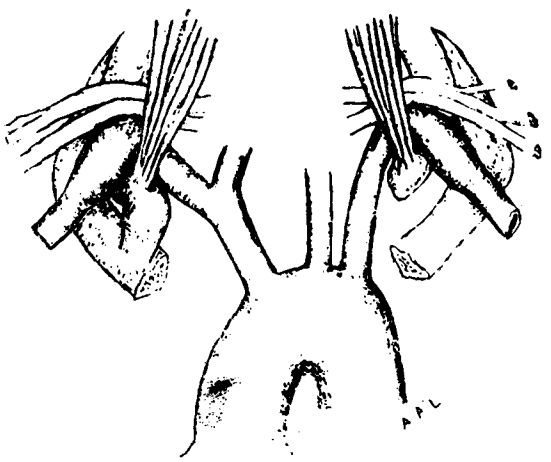


Fig. 7.—Mild bilateral aneurysmal dilatation of subclavian artery distal to compression of artery by scalenus anticus muscle at site of its attachment to congenitally abnormal cervical rib (Law: The Surgical Aspect of Cervical Ribs, Journal-Lancet 34: 330, 1914).

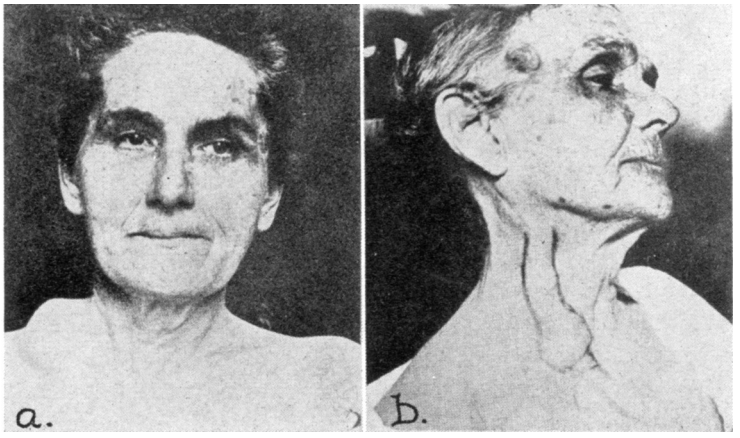


Fig. 8.—(*a*) Halsted's case of acquired poststenotic dilatation of right common carotid artery following partial stenosis of innominate artery by metal band for aneurysm of the subclavian artery. Aneurysm subsequently cured by ligation of subclavian artery proximal and distal to aneurysm. (*b*) During twelve years of observation, the dilatation of the carotid beyond the stenosing band gradually became more pronounced (Surg., Gynec. & Obst. 27: 547, 1918).

degeneration of the internal coat, nor any thickening of either the middle or external coats." He considered the peculiar lesion "a fusiform dilatation, unaccompanied with any special weakening of any of the three tunics, or with any rupture whatsoever. . . . "

This description of poststenotic dilatation as a fusiform enlargement without disease of the vessel wall conforms in every respect with the character of its occurrence beyond aortic and pulmonic stenoses, and beyond a coarctation of the aorta. That it can be reproduced experimentally almost at will has been demonstrated in our laboratory. Employing a technique which had previously been found effective in producing pulmonic and aortic stenosis in a study of hemicardiac hypertrophy,[6] Gerbode[7] produced coarctation of the

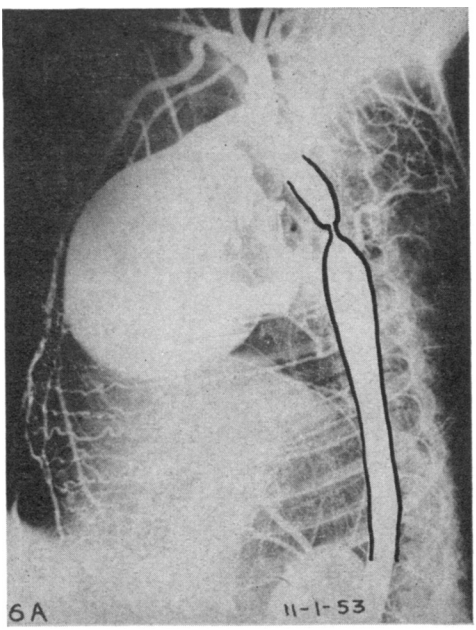


Fig. 9.—Post-mortem injection of vascular bed with 17 per cent bismuth oxychloride in a 20-month-old dog showed marked dilatation of aorta beyond an experimentally produced coarctation when pup was 8 days old.

thoracic aorta in 8-day-old puppies by a ligature so placed about the aorta that it did not constrict the lumen, but sharply limited the vessel to its primitive narrow diameter as the rest of the puppy grew to maturity. When these animals were killed one or two years later, dilatation beyond the stenosis was invariably present (Fig. 9). In our own studies, dilatation was observed eighty-four days following the application of a nonconstricting ligature to the thoracic aorta of three 21-day-old pups, all belonging to the same litter (Fig. 10).

In the course of studies to develop a method of uniting disparate cylinders,[11] the small left subclavian artery, with a diameter of 4 mm., in a 14-day-old puppy was joined to the distal end of the divided thoracic aorta, whose lumen measured 8 mm. in diameter, by the so-called V-plasty. In one instance, poststenotic dilatation immediately distal to the anastomosis appeared within twenty-four days, and in a second puppy, it was demonstrated by an aortogram at the end of ten weeks (Fig. 11). These two experiments demonstrated that

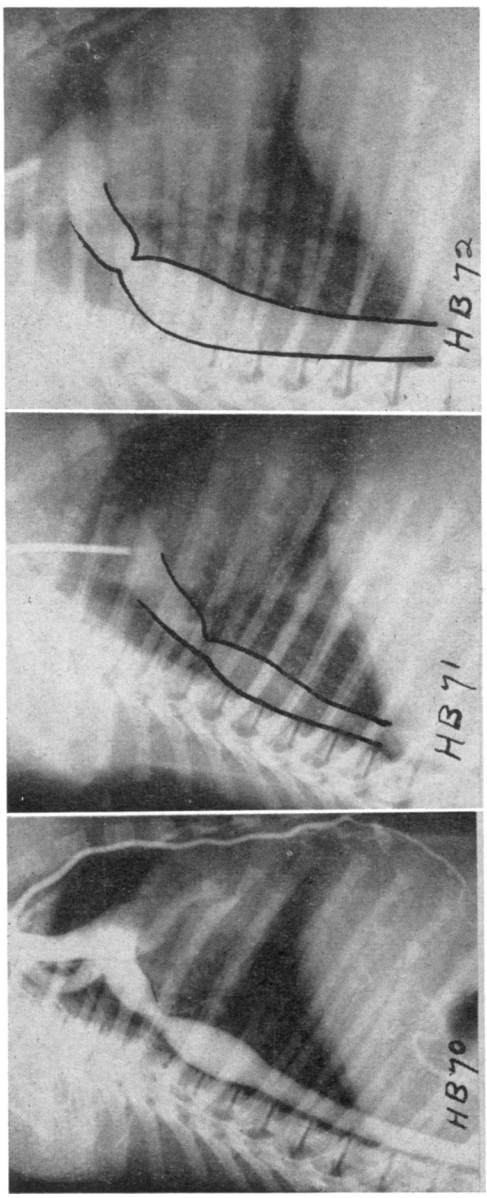


Fig. 10.—Dilatation beyond a coarcting ligature applied to the thoracic aorta was invariable, as illustrated in three pups from the same litter operated upon eighty-four days previously.

when a stream of small caliber suddenly flows into a vessel of larger diameter, the same hydraulic forces come into play as are found beyond an abrupt partial stenosis.

Experiments were then conducted with an artificial circulatory system (Fig. 12) which included a pump whose rate varied from 84 to 120 pulsations

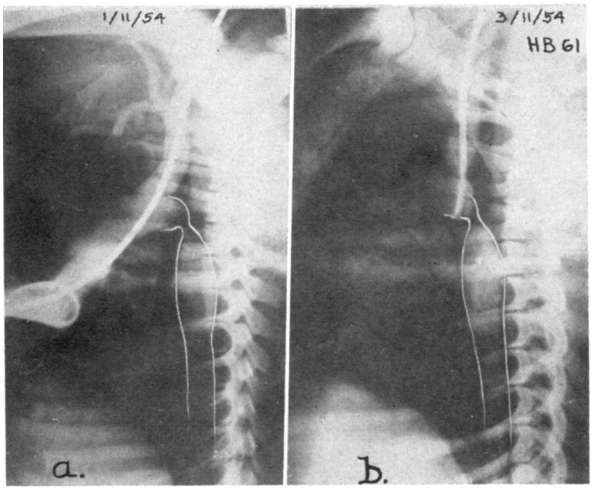


Fig. 11.—Aortograms disclosed progressive dilatation beyond end-to-end anastomosis of subclavian artery to distal end of divided thoracic aorta (*a*) ten weeks and (*b*) eighteen weeks after operation.

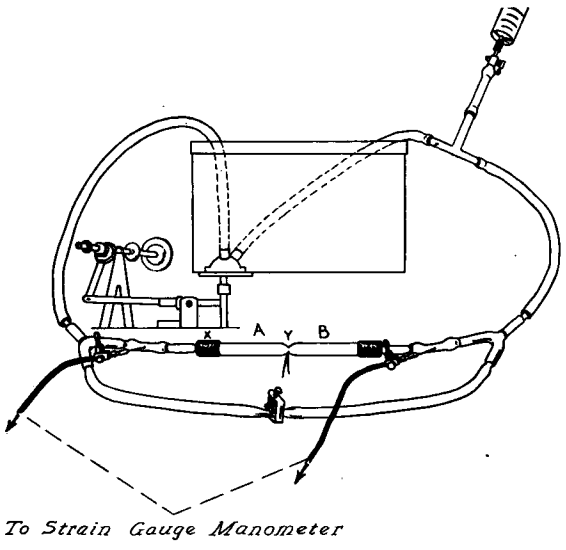


Fig. 12.—Schematic drawing of pump and conduits simulating the circulation with a segment of elastic rubber tubing introduced into system with constrictions at *X* and *Y*; capacity of system about 200 c.c. water.

per minute and whose stroke output varied from 1.7 c.c. to 9.7 c.c. The conduits consisted of rigid plastic tubing with an internal diameter of 0.6 cm., but included also a segment of elastic rubber tubing whose wall had an average

TABLE I

PRESSURE IN SYSTEM	STROKE		DIAMETER OF MAXIMUM POSTSTENOTIC DILATATION (CM.)	LENGTH OF POSTSTENOTIC DILATATION (CM.)
	RATE	OUTPUT (C.C.)		
120/70	96	5.7	2.0	1
	8 c.c. water added to system			
125/80	96	5.7	2.8	3
	Stroke output increased to 9.8 c.c.			
160/80	96	9.8	3.0	4
	Stroke output decreased to 5.7 c.c.			
125/80	96	5.7	2.5	2.5
	8 c.c. water added to system			
125/75	96	5.7	3.2	3.5
	Stroke output increased to 9.8 c.c.			
145/60	96	9.8	3.4	3.5

Capacity of circulatory system 200 c.c.

thickness of 0.033 cm. and whose internal diameter was 1.6 cm. The pressure in the system could be varied by increasing or decreasing the volume of circulating fluid, and the velocity of flow was varied by altering the stroke output. Table I records the effect upon the elastic segment of varying the pressure and the velocity of flow in the system.

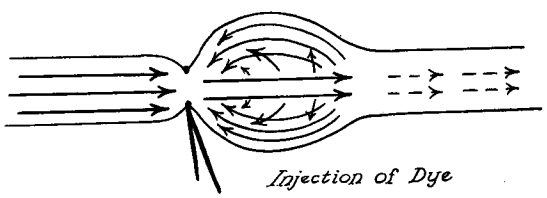


Fig. 13.—When dye is injected proximal to a constriction, remarkable eddies of turbulent and reversed flow are noted just distal to the stenosis where the rapidly ejected stream strikes the more slowly flowing and stagnant distal stream, and where high kinetic energy is transformed into high potential energy or lateral pressure.

In another experiment, the volume of circulating fluid was increased sufficiently to produce a mild poststenotic dilatation. The stroke output was then gradually reduced from 9.8 c.c. to 7.2 c.c., at which point the poststenotic dilatation disappeared. Restoring the stroke output to 9.8 c.c. caused the poststenotic dilatation to recur.

When a concentrated solution of methylene blue was injected into the lumen beyond the constriction, the dye promptly disappeared without delay into the fast-flowing distal stream. However, when the dye was injected proximal to the stenosis, there was noted just distal to the constriction a definite delay in its progress downstream (Fig. 13). It appeared to be involved in turbulent eddies before it hesitatingly resumed its forward flow. It was even seen to reverse its direction and to flow backward, momentarily, toward the stenosis. The site of delay and of turbulence of flow was identical with the site of dilatation, as well as with the location of the palpable thrill and audible bruit. Similar observations were made repeatedly.

The following experiment provided a completely convincing demonstration that the play of hydraulic forces against the elastic wall just beyond a stenosis will, in time, produce poststenotic dilatation:

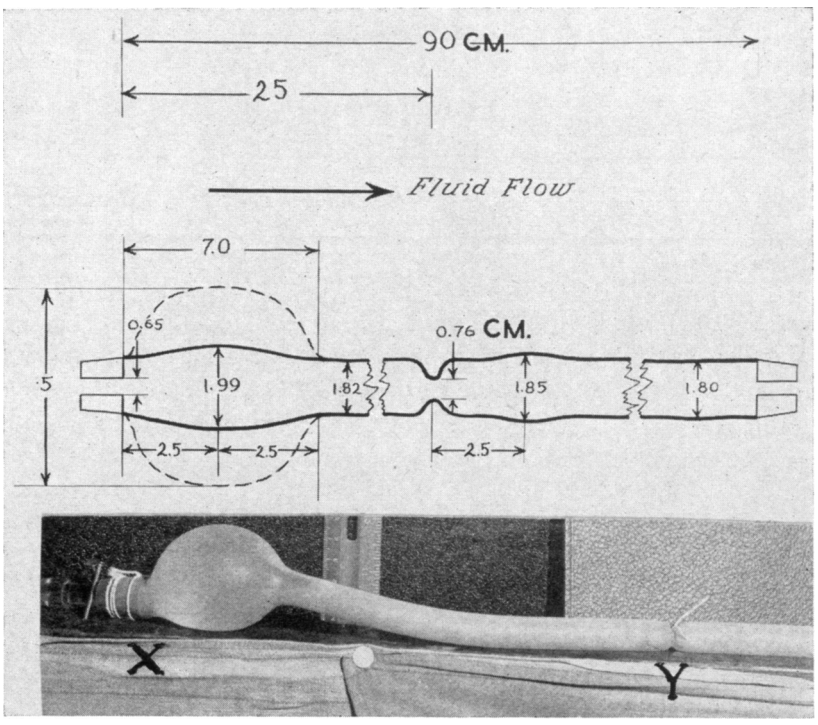


Fig. 14.—A 90 cm. long segment of rubber tubing, 1.8 cm. in diameter, with two points of constriction was subjected to twenty-one hours of continuous pumping, at which time mild dilatation appeared beyond both constrictions. After eleven more hours of continuous pumping, during which time there was no change in the mild dilatation, the tubing suddenly ballooned out beyond the first constriction to a diameter of 5 cm. over an area about 7 cm. long. This alteration was permanent and represents maximal structural fatigue at site of greatest lateral stress.

Into the artificial circulatory system was inserted a segment of elastic tubing 90 cm. long and 1.8 cm. in diameter (Fig. 14). Twenty-five centimeters beyond the junction of the rigid tubing with the elastic tubing, a second site of stenosis was produced by partial ligation, which reduced the diameter of the tubing to about 0.75 cm. Enough water was introduced into the system to distend the elastic tubing to a normal and uniform diameter throughout, without dilatation beyond either constriction. The pump was set at 84 pulsations per minute with an output per stroke of 9.8 c.c.

After twenty-one hours of continuous pumping without the slightest evidence of dilatation, there began to appear definite but mild dilatation beyond the first constriction, with an even milder dilatation beyond the second constriction. At this time the pulsations were increased to 120

per minute. Ten more hours of continuous pumping produced no significant change in the dilatation beyond either site of constriction. Suddenly, however, after thirty-one hours of pumping and after eleven hours of mild dilatation, there occurred a remarkable aneurysmal dilatation just beyond the first constriction. An outside diameter of 2 cm. was suddenly increased to 5 cm., and the poststenotic dilatation was now 7 cm. long (Fig. 14, *B*). Moreover, the dilatation was now permanent, whether the pump worked or not. The elastic tubing had yielded suddenly and irrevocably to the play of hydraulic pressures just beyond the stenosis. Previously the mild dilatation had promptly subsided when the pumping ceased. With the appearance of the balloonlike dilatation beyond the first constriction, the dilatation beyond the second constriction disappeared completely because of the dissipation of pressure and pooling of fluid from the rest of the small system in this excessively dilated area.

In another experiment, a second 90 cm. length of rubber tubing with two points of stenosis was subjected to the continuous operation of the pump (Fig. 15, *a*). After about ninety hours of pumping, mild dilatation appeared just beyond the first constriction (Fig. 15, *b*), which subsided when the pumping ceased (Fig. 15, *c*). By applying the abrasive action of sandpaper, the wall of the tubing 2 cm. beyond the constriction was slightly thinned out, resulting in a small pouchlike dilatation at this weakened area when the pumping was resumed. The pressure in the system was then increased by the addition of only 5 c.c. of water. Instantly there occurred an accentuation of the pouchlike protrusion, followed gradually by a balloonlike dilatation of the tube just beyond the stenosis. When the pump was stopped, the aneurysmal dilatation disappeared. The effectiveness of the play of hydraulic forces in producing an aneurysmal dilatation immediately beyond a site of constriction was vividly portrayed before our almost incredulous eyes.

In another illuminating experiment, a segment of the small intestine of a cat was introduced into the circulatory system in place of the rubber tubing. After about eighteen hours of pumping, first the circular and longitudinal musculature split and then the fibrous submucosa burst 3 cm. beyond the junction of the small rigid tubing and the larger distensible intestinal lumen, the rent extending for about 2 cm.

These various observations of the effects of a stenosis in an artificial circulatory system demonstrated conclusively that poststenotic dilatation was the direct result of the play of hydraulic forces set in motion by the stenosis, and that it varied in degree with the pressure and velocity with which fluid was ejected from the stenotic channel. Moreover, there appeared to be a critical level of pressure and of velocity below which poststenotic dilatation did not occur. The element of time was most important, dilatation appearing in one experiment only after the play of hydraulic forces had persisted for a prolonged period, thirty-one hours in one experiment and ninety hours in a second experiment. It is quite evident that other important factors may determine the duration of the play of hydraulic stresses and strains necessary before dilatation occurs. These are the inherent resistance of the vessel wall to

dilatation, depending upon its structural qualities, the relationship between the elasticity of the wall and the pressure and velocity of flow to which it is subjected, the degree of stenosis present, and perhaps the viscosity of the circulating fluid. It appears that unless the stenosis reduces the lumen to one-third or one-fourth its original diameter, turbulence of flow will be minimal and only minor dilatation will occur, and may be long delayed.

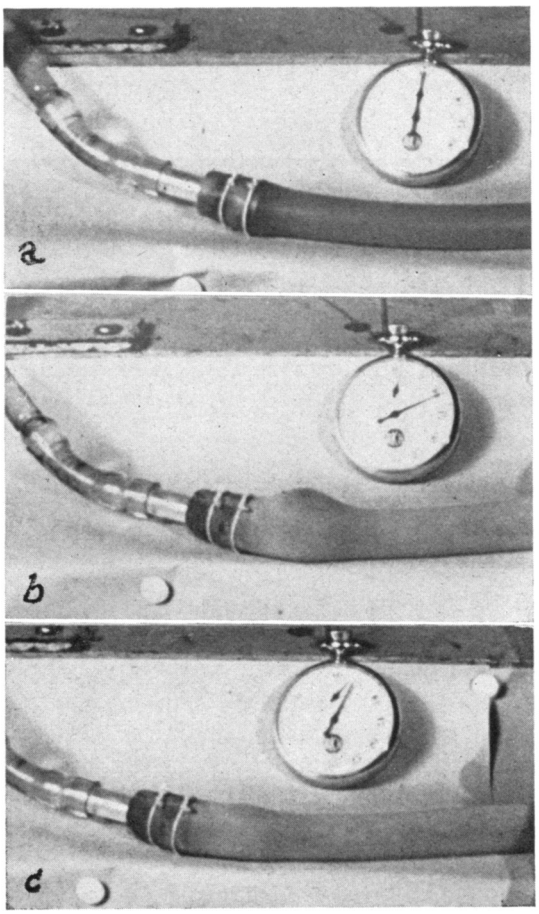


Fig. 15.—(*a*) Appearance of rubber tubing at beginning of experiment showing complete absence of dilatation beyond constriction despite pumping action.

(*b*) Postconstriction dilatation appeared after ninety hours of continuous pumping.

(*c*) Dilatation disappears promptly but not completely when pumping ceases. Incomplete disappearance due to permanent alteration as the result of structural fatigue.

In reviewing these experimental observations with due regard to certain principles of hydraulics, as emphasized by Professor John K. Vennard[8] of the Department of Civil Engineering at Stanford University, certain causes and effects were postulated as operating beyond a narrow stenosis in a large vessel transporting blood under relatively high pressure and high velocity:

During diastole in a pulsating system, there occurs normally a momentary retardation in flow which is markedly accentuated just beyond a stenosis. During systole, there emerges from the narrow stenotic channel a stream of blood at a greatly increased velocity, as compared with the velocity in the broader channel either proximal or distal to the stenosis. Mathematically, the velocity of flow, other factors remaining the same, is inversely proportional to the square of the radius. Obviously, if a tube 18 mm. in diameter is suddenly reduced to a diameter of 6 mm., the velocity through the constriction is increased nine times. Conversely, if a tube 6 mm. in diameter is suddenly increased to a diameter of 18 mm., the velocity will be reduced to one-ninth its previous rate. The rapidly ejected stream runs head on, as it were, into the more slowly flowing, and relatively stagnant, poststenotic stream, resulting in a sudden retardation in its forward flow. High kinetic energy is suddenly converted into high potential energy or lateral pressure. Moreover, the sudden arrest of the rapid stream produces eddies of turbulent and reversed flow, which strike the elastic vessel wall with alternating high- and low-pressure waves, causing the wall to vibrate, these vibrations being recognizable as a palpable thrill and an audible bruit. The location of maximal clash of streams and of the site of conversion of high kinetic energy into high lateral pressure, and therefore also the zone of greatest turbulence, lies about 1 to 3 cm. beyond the constriction, depending upon the caliber of the conduit, and upon the pressure and velocity of flow. At this point, also, the maximal dilatation occurs.

A factor supremely important to the hydraulic expert concerns the phenomenon of structural fatigue. Unevenly distributed in the zone of turbulent flow are impacts or shocks of increased pressure alternating with decreased pressure, which produce points or minute areas of high local stress against the limiting vessel wall. Depending upon the pressure in the system, the velocity of flow, and the structural qualities of the vessel wall, this oscillation of high and low pressures against the individual fibers of the vessel wall results in the prompt, or very gradual, onset of structural fatigue and in a potential thinning or distention of the wall at these points of impact. As these points of shock or stress vary in position from moment to moment and are diffusely distributed in diminishing vigor throughout the zone of increased lateral pressure, the sites of structural fatigue are correspondingly diffusely distributed in diminishing severity, resulting in a tapering dilatation of the vessel wall.

The absence of dilatation proximal to the constriction is dependent upon the concept that the pressures are evenly and uniformly distributed against the limiting vessel wall, and since the forward flow here is not subjected to sudden retardation or arrest, there is no conversion of kinetic energy into lateral pressure. General pressure proximal to the stenosis may be higher than the pressure distal to it, but it is a relatively uniform level of pulsatile pressure, evenly distributed throughout and not subject to the rapidly oscillating variations in localized pressure such as occur in the turbulent flow produced by the

clash of streams distal to the stenosis. As a result of this uniform distribution of pressure proximal to the stenosis, uniform expansion of the vessel occurs with each pulsation.

In analyzing further the play of hydraulic forces distal to a stenosis, it appears that there are three factors operating here to produce greater dilatation of the vessel: The first factor is the sudden conversion of high kinetic energy into high potential energy or lateral stress. The second factor is the turbulence of flow due to a clash of streams of differing velocities, which causes eddies of alternating high and low pressures to strike against the elastic wall. The third factor is the lowered velocity due to the widening of the stream diameter produced by the first two factors. According to the Bernoulli principle, this lowered velocity will of itself increase lateral pressure. In a pulsating stream, the first two factors recur repeatedly with each systole and set the stage, as it were, for the third factor to act, also in repetitive manner. The result of these three inexorably recurring influences in a pulsating stream, operating in a localized segment of an elastic distensible vessel, is a vicious circle: greater lateral pressure → greater widening of the stream-diameter → lessened velocity of flow → greater lateral pressure → greater widening of the stream diameter → lessened velocity of flow, and so forth. Insidiously progressive dilatation is an inevitable result unless the resistance to further dilatation inherent in the intact vessel wall itself or the incompressibility of the surrounding structures prevent it. Even these conditions may not prevent it, so effective and insistent are these hydraulic forces, given sufficient time.

Certain clinical observations confirm the great importance of the height of pressure and velocity of flow in the development of poststenotic dilatation (Fig. 16). It occurs most prominently in the ascending aorta beyond a subaortic stenosis where the left ventricle ejects blood under maximal pressure, at maximal velocity, in maximal mass, against a maximal peripheral resistance. It is almost equally prominent beyond a stenosis of the pulmonic valve where the hypertrophied right ventricle ejects an equally large mass of blood under high pressure and at high velocity. It is less prominent beyond a coarctation of the aorta since the pulsatile end pressure here is less than in the ascending aorta, the distal peripheral resistance is less, and the mass of blood forced through the coarctation and the resulting velocity of its flow have been reduced significantly by the escape of blood through the large branches of the aortic arch. Its moderate development in the subclavian artery beyond a cervical rib is also related to the lower pressure in this smaller vessel with lessened peripheral resistance beyond it. Its apparent absence beyond constrictions in vessels of still smaller caliber is related to the lower pressure and lower velocity as one approaches the periphery, where the disparity between systolic and diastolic pressures is gradually being reduced and where pulsatile flow gradually changes to more uniform and even flow. Such observations indicate that the occurrence and degree of dilatation depend upon the height of systolic pressure with which blood is thrust against the constriction, which in turn determines the velocity of flow with which blood is ejected from the short stenotic channel. The greater this velocity, the more distantly distributed

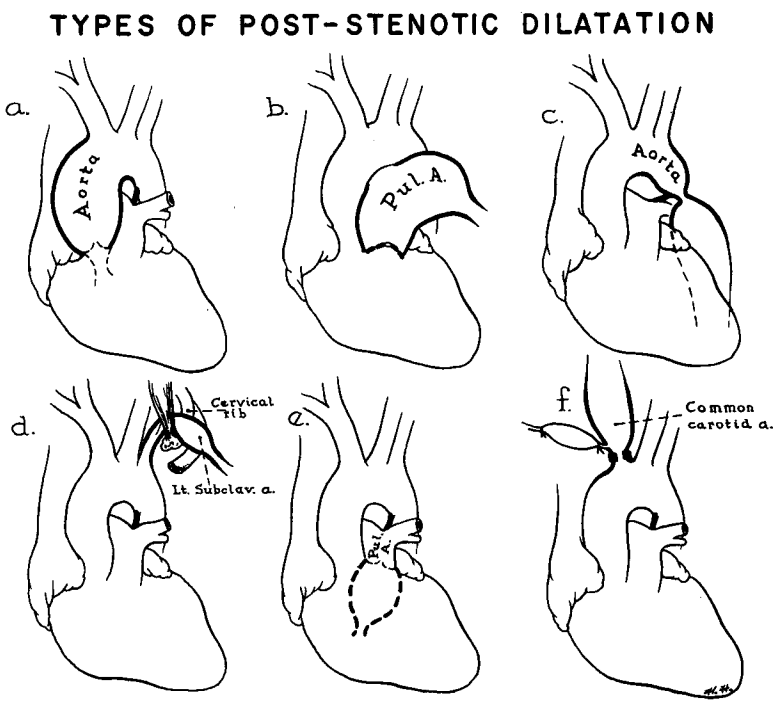


Fig. 16.—Poststenotic dilatation is most pronounced beyond a subaortic stenosis, equally prominent beyond a pulmonic stenosis, less developed beyond a coarctation of the aorta, mildly present distal to compression of the subclavian artery between the scalenus anticus muscle and its attachment to a cervical rib, occasionally present in the small ampulla between an infundibular stenosis and the pulmonic valve, and in a unique instance it occurred distal to a partially occluding band applied to the innominate artery. The degree of poststenotic dilatation is intimately dependent upon height of pressure and upon velocity of flow.

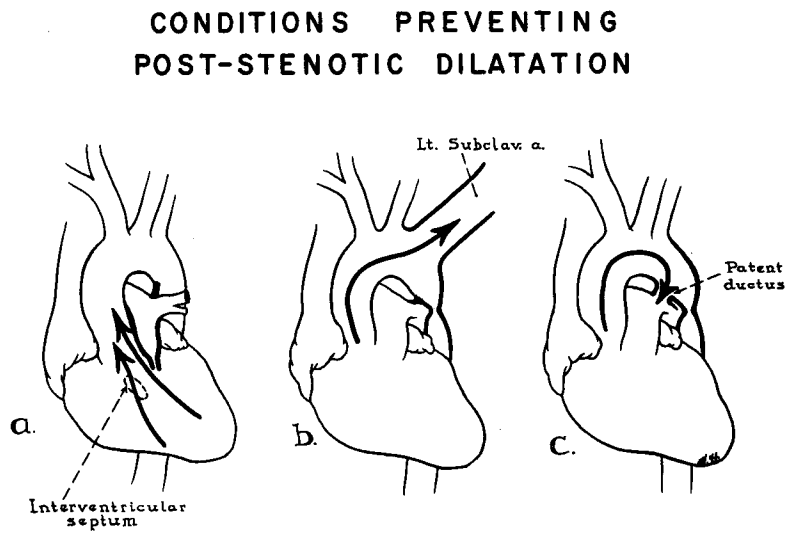


Fig. 17.—Certain conditions prevent dilatation beyond a stenosis: (*a*) an interventricular septal defect and an overriding aorta provide diversion of blood away from the pulmonary artery, thus diminishing pressure and velocity of flow through the accompanying infundibular stenosis (tetralogy of Fallot); (*b*) a large subclavian artery may divert a large volume of blood away from a coarctation, thus preventing dilatation beyond the coarctation; (*c*) a large open ductus proximal to a coarctation effectively diverts a volume of blood away from the coarctation sufficient to prevent dilatation of the aorta beyond the coarctation.

are the impacts of the swiftly flowing systolic stream against the momentarily retarded distal diastolic stream and, therefore, the more distantly effective are the shocks of increased lateral pressure against the vessel wall.

Other clinical conditions have been observed which prevent the development of poststenotic dilatation or determine its degree (Fig. 17). Most importantly, the volume of blood forced through the constricted channel and, therefore, the velocity of flow with which the blood is ejected from the stenosis may be greatly altered by conditions just proximal to the stenosis: a large collateral channel such as a greatly dilated left subclavian artery may reduce the volume flow through a coarctation; an open ductus located just proximal to a coarctation may drain a large volume of blood into the pulmonary artery away from the coarctation; an interventricular septal defect and an overriding aorta as in the tetralogy of Fallot may provide a large "runoff," or diversion, of blood away from the infundibular stenosis, thus reducing markedly the mass and the velocity of blood flow through the stenosis.

The difficulty encountered by Halsted and Reid in their experimental attempts to produce postconstriction dilatation by partial ligation of the terminal abdominal aorta may be due to the diversion of blood away from the site of partial ligation through the many large branches just proximal to it, such as the celiac axis, the two large renal arteries, the inferior mesenteric artery, and the several paired lumbar vessels. These can effectively reduce both the velocity and pressure with which blood is ejected through a stenotic channel produced in the terminal aorta and thus prevent or greatly curtail poststenotic dilatation. In our experiments, when the coarctation was produced in the thoracic aorta, where pulsatile pressure, velocity of flow, and mass of blood thrust against the constriction are higher than in the terminal aorta, dilatation beyond the coarctation invariably occurred.

That a runoff or diversion of flow away from the stenosis may effectively prevent poststenotic dilatation was convincingly demonstrated by the following case of a large patent ductus proximal to a coarctation of the aorta:

CASE 6.—K. B., aged 10,* was admitted to Stanford Hospital in January, 1954, for correction of a coarctation of the aorta accompanied by a patent ductus, both of which had been recognized soon after birth. There was no history of cyanosis.

Physical examination disclosed a very pale but well-developed normal child except for a loud continuous murmur at the left cardiac base with a loud pulmonic second sound; heart definitely enlarged and hyperactive (Fig. 18); pulse rate 98; hemoglobin 13 Gm. per cent; hematocrit 39; 4,250 white cells, urine normal; blood pressure: right arm 142/90-58 mm. Hg, left arm 140/92-60; no pressure obtainable in either leg; after the patient spent two days in bed, blood pressure in the arm dropped to 128/70-56.

At operation, a widely open ductus arteriosus, 8 mm. in diameter, was found, transmitting blood from the aorta to the pulmonary artery. Just distal to this open ductus lay a typical coarctation, but the caliber of the aorta distal to it was diminished rather than increased. The aorta proximal to the constriction had a diameter of 1.7 cm., and the distal aorta a diameter of 1.3 cm. (Fig. 19). The coarctation itself had an inner lumen of about 3 mm. in diameter. To re-establish continuity of the two disparate ends, the small distal aorta was incised longitudinally for about 7 mm. in its anterior half, thus providing

*A patient of Dr. Ann Purdy.

the necessary extra circumference for satisfactory suture to the larger proximal aorta. As a result of this V-plasty, the lumen of the anastomosis exceeded the lumen of the smaller vessel, and there was no constriction at the site of union.

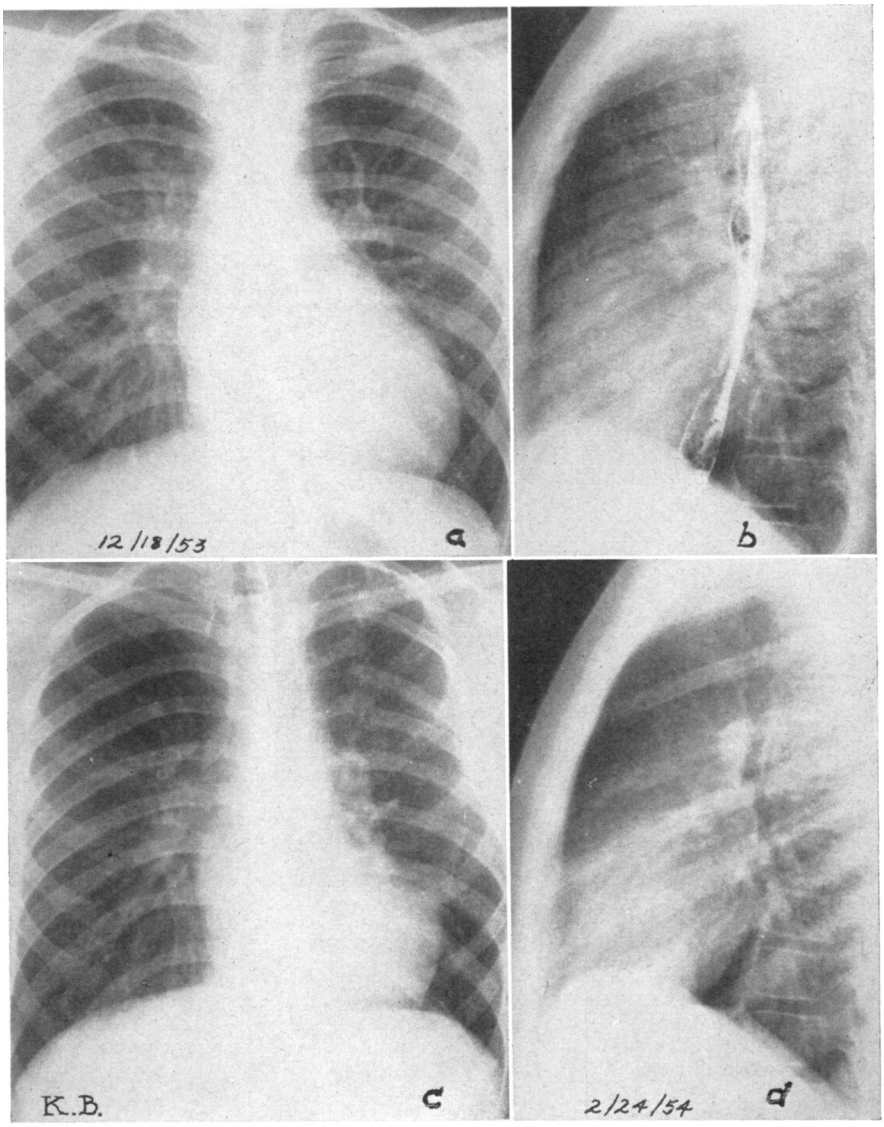


Fig. 18.—Roentgenograms (*a* and *b*) of a 10-year-old child before excision of a coarctation and ligation of a patent ductus proximal to the coarctation showed a marked dilatation of the heart which subsided promptly following operation (*c* and *d*) (see Fig. 19).

When the flow through the aorta was re-established by removing the occluding clamps, pressure in the right arm dropped from 170/110 to 120/90, and the conditions in the aorta proximal and distal to the anastomosis were promptly reversed: the proximal aorta was now smaller than the aorta distal to the anastomosis! Good pulses could now be felt in both feet, and on her discharge from the hospital ten days later, the pressure in the right arm was 128/90 and in the right leg 132/96 mm. Hg.

The absence of dilatation beyond the coarctation in this instance was the direct result of the diversion of a large volume of blood through the patent ductus into the pulmonary artery which reduced markedly the pressure and velocity of blood flow through the coarctation.

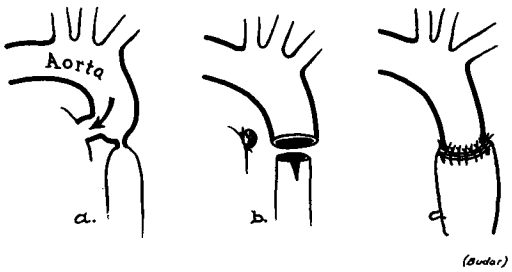


Fig. 19.—(*a*) Dilatation beyond a coarctation does not occur in the presence of a large open ductus proximal to the coarctation which diverts blood flow away from the stenosis.
(*b*) Anastomosis of disparate ends effected by use of V-plasty to avoid stenosis at site of union.
(*c*) Note larger diameter of distal aorta as compared with proximal aorta after elimination of ductus and excision of coarctation.

In the tetralogy of Fallot, in which the interventricular septal defect and the overriding aorta provide channels through which blood from the right ventricle is diverted away from the pulmonary outflow tract, no dilatation of the pulmonary artery is observed. On the contrary, in a tetralogy the caliber of the pulmonary artery and its two main branches is usually greatly diminished, commensurate with the reduced volume flow of blood through these channels. When the pulmonary artery and its branches are found to be dilated, one would suspect that the interventricular septal defect is small and that the stenosis involves the pulmonary valve rather than the infundibulum.

In the presence of pure pulmonic stenosis, without intracardiac shunts, the absence of dilatation of the pulmonary outflow tract as disclosed by roentgenograms of the chest strongly favors an infundibular stenosis. On the other hand, a dilated pulmonary artery as disclosed by roentgenograms in the presence of a presumed pure pulmonic stenosis is suggestive evidence that a valvular and not an infundibular stenosis is present (Fig. 20):

CASE 7.—W. M., aged 33, entered Stanford University Hospital in January, 1954, for possible operation upon a presumed patent ductus. He had a murmur over the heart which he believed had been present from birth, and certainly from the age of 12. He had been barred from high school football because of his heart, and had been turned down for military duty by the draft board on a cardiac disability. Recent to admission he had become more easily fatigued, associated, on excessive activity, with a dry nonproductive cough and discomfort in the chest. He disclaimed exertional dyspnea, ankle edema, cyanosis, or orthopnea.

Physical examination revealed no cyanosis, nor clubbing of the fingers, pulse rate 82, loud systolic murmur maximal in left first and second intercostal spaces, pulmonic second sound split with loud second component, blood pressure 130/74, good femoral pulses, hemoglobin 15.8 Gm. per cent, hematocrit 48, 7,100 white cells; right axis deviation, with conduction defect. Plain roentgenograms showed a marked prominence in the left hilar area which was identified by angiocardiography as dilatation of the pulmonary artery

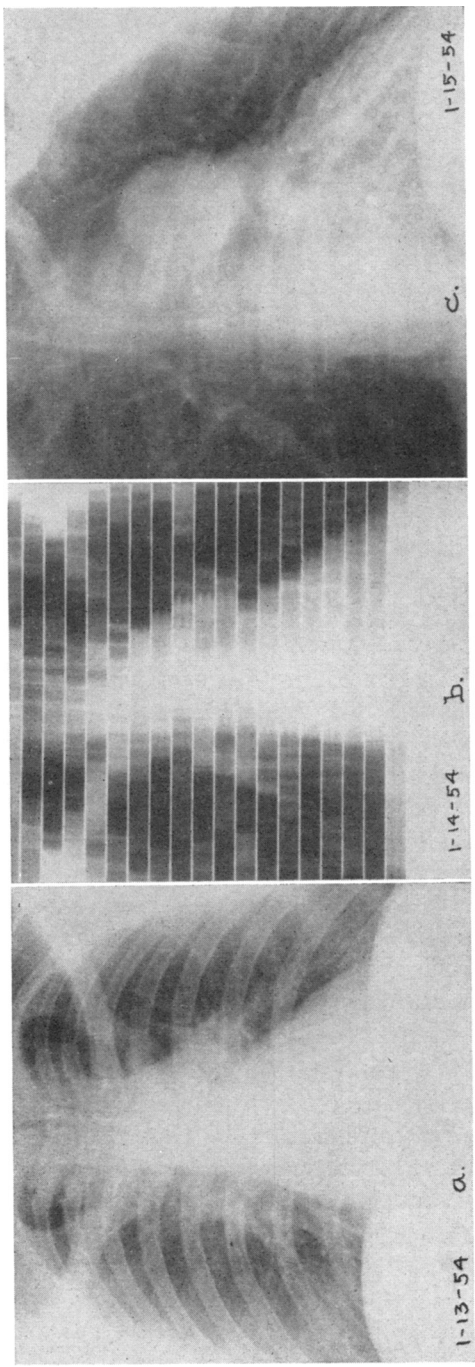


Fig. 20.—Case 7. Patient W. M. When roentgenograms in the presence of a presumed pure pulmonic stenosis without intracardiac shunts reveal marked dilatation of the pulmonary artery, it is presumptive evidence of a stenosis of the pulmonic valve rather than of the infundibulum.

(Fig. 20). A significant observation in the angiograms was the pronounced delay in the emptying of the poststenotic dilatation: dye which normally should leave this area in 3 seconds provided opacification of the dilated vessel for 4½ to 5½ seconds, a phenomenon also observed in our experimental studies of the flow distal to a constriction. Cardiac catheterization disclosed a pressure of 39/5 mm. Hg in the right ventricle, and a pressure of 19/7 mm. Hg in the pulmonary artery, and no evidence at rest of an intracardiac shunt.

A diagnosis was made of an isolated pulmonic valve stenosis without intracardiac shunt and only moderate right ventricular pressure. No immediate surgical treatment was advised, but the patient was requested to return for cardiac catheterization in one year's time to determine whether any increase in right ventricular pressure had occurred. If so, surgery would be indicated.

These experimental and clinical observations remove all doubts as to the cause of the poststenotic dilatation—it is not biologic, nor supernatural; it is not due to disruption of sympathetic nerves, nor to vasomotor paralysis; it is not the result of occlusion of the vasa vasorum nor of localized impairment of circulation; it is not the result of external trauma nor of an unusually well-developed collateral circulation entering the vessel distal to the coarctation; it is not the result of a dead pocket, nor of stagnation of fluid in this pocket. It is the simple, mechanical effect of the operation of natural hydraulic laws that govern the flow of fluid through a conduit—in this instance not a rigid-walled conduit, but an elastic vessel, capable of distention in response to increased lateral stresses repeatedly applied over a prolonged period of time.

These observations also carry other wide implications. They are strongly suggestive that the initiating factor in the development of the arteriosclerotic aneurysm may be occasionally a stenosis of the arterial lumen imposed by a segmental atherosclerosis, through which a narrowed stream flows with increased velocity into a still normally distensible distal segment, thus setting the stage for the play of hydraulic forces capable of producing a localized aneurysmal dilatation of the vessel (Fig. 21, *a*). If there is a limited area of pronounced localized weakening of the wall in the segment just beyond the stenosis, where the play of hydraulic forces is greatest, a linear rupture may occur, resulting in the initiation of a dissecting aneurysm (Fig. 20, *c*). Or a localized weakened area in the vessel wall may yield gradually to the play of hydraulic pressures, resulting in a sacculated aneurysm distal to the stenosis (Fig. 20, *b*).

Once a dilatation has been initiated, the process is perpetuated and enhanced through the application of the principle that a widened lumen slows the stream, a slowing of the stream increases lateral pressure (the Bernoulli principle), and increasing lateral pressure further dilates the vessel, thus setting up a vicious circle which is limited only by the inelasticity and rigidity of the vessel wall or by the incompressibility of surrounding structures. In reality, the play of these hydraulic forces may prove at times irresistibly destructive, as evidenced by the inexorable dilatation of an aneurysm against such seemingly incompressible or indestructible substances as bone. Through the operation of the phenomenon of structural fatigue and by the processes of compression, interference with blood supply, and consequent absorption, even the bodies of vertebrae may gradually be worn away. The progressive thin-

ning of the elastic vessel wall itself, once the dilatation has been initiated, is an additional important factor in its progressive dilatation by reducing progressively its resistance to the play of these hydraulic forces. The progressive nature of an aneurysmal dilatation once initiated is thus more easily understood.

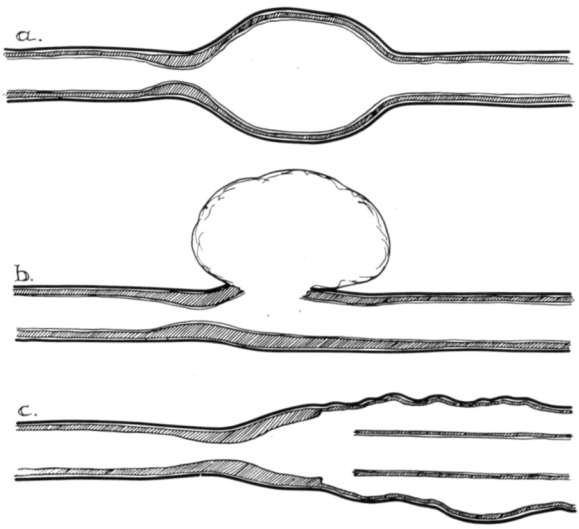


Fig. 21.—The play of hydraulic stresses accompanying the flow of blood through a stenosis due to segmental atherosclerosis in an arteriosclerotic aorta may be an important factor in the development of (*a*) a fusiform aneurysm, (*b*) a sacculated aneurysm, or (*c*) a dissecting aneurysm (see text).

Occasionally, a vessel may show segmentary thickening and rigidity of its wall due to degenerative changes without narrowing of its lumen but with complete destruction of its elasticity and with complete absence of dilatation in systole, whereas immediately distal to this rigid segment, the vessel retains its elasticity and distensibility. The emergence of a rapidly flowing stream from such a rigid segment into the more elastic and distensible distal segment may initiate a play of hydraulic forces quite comparable to those unleashed distal to a true stenosis, thus setting the stage for the development of an aneurysmal dilatation, either fusiform, saccular, or dissecting, depending upon local circumstances.

Moreover, the sudden appearance after many hours of pulsatile flow of a pouchlike dilatation or of a marked irreversible ballooning out of the rubber tubing just beyond a stenosis, as observed in several of our experiments, may well have its counterpart in the development of aortic aneurysms. A weakened area imposed by atherosclerosis or by degenerative processes in the vessel wall just beyond a stenosis may be subjected suddenly to a great increase in the play of these hydraulic forces due to the sudden increase in pulse rate and in general blood pressure accompanying unusual physical activity or great emotional strain. The suddenly heightened pressure and the increased velocity of flow through the stenosis increase greatly the hydraulic stresses localized just beyond

the stenosis, and a ballooning out of the aorta into a fusiform or a sacculated aneurysm, or even in a tear of the media with the production of a dissecting aneurysm, is the inevitable result.

SUMMARY

A mass of fluid ejected through a narrow and limited constriction under high velocity strikes against a more slowly moving mass of fluid distal to the stenosis resulting, first, in the conversion of high kinetic energy into high potential energy or lateral pressure and, second, in the lateral deflection of the rapid stream and even in a complete reversal in its direction of flow, thus resulting in a clash of opposing streams that produces eddies of alternating high and low pressures whose repeated impacts over a prolonged period against an elastic wall are capable of inducing structural fatigue and distention of that wall. Accentuating the play of these forces is a third factor: the hydraulic principle that a widening stream causes decreasing velocity which in turn produces increasing lateral pressure. The resulting interplay of these three factors recurring repetitively with each systole in a pulsating stream, and operating in a limited segment of an elastic vessel, produces eventually and inevitably the phenomenon of poststenotic dilatation. It is a highly intriguing thought that this sequence of events may be the initiating and possibly the most important factor in the development of the fusiform dilatations, the saccular aneurysms, or even of the dissecting aneurysms commonly observed in the arteriosclerotic aorta in which segmental atherosclerosis or fibrosis may produce a localized or relative stenosis.

The recognition of the potential power of the hydraulic forces liberated beyond a real or relative stenosis may assist in the explanation of the destruction of bone and surrounding structures that occasionally accompanies the development of a thoracic aneurysm. They explain also the aneurysmal dilatation of the vein usually seen at the site of an arteriovenous fistula where the arterial stream under high pressure and high velocity is ejected through an abnormal communication into the widened bed of the accompanying vein.

The occasional aneurysmal dilatation of the pulmonary artery in the presence of a patent ductus may also be ascribed to the hydraulic forces liberated when the arterial stream of high pressure and high velocity is ejected from the narrow ductus into the wider and less rapidly flowing stream of the pulmonary artery with its lower pressure.

It is highly probable, also, that the sudden cerebral hemorrhage attributed to the rupture of a small aneurysmal dilatation has its inception in the precipitate unleashing of hydraulic forces suddenly augmented by the heightened pressure and increased velocity of flow incident to emotional stress or physical exertion.

Similarly, sudden death from rupture of a larger thoracic or abdominal aneurysm may follow the sudden increase in blood pressure and pulse rate that accompanies emotional stress or physical exertion and produce an increase in neocity of flow which greatly augments the play of hydraulic forces, with result-

ing sudden increase in lateral pressure just distal to the junction of the narrow arterial lumen with the greatly widened bed of the aneurysmal dilatation.

These and other similar vascular phenomena may find their proper explanation in the application of the hydraulic laws that govern the pulsatile flow of fluid through a system of elastic vessels subjected to real or relative narrowing by congenital abnormalities, by degenerative processes, or by accidental injuries.

They give stern warning also of the need to employ, in vascular surgery, methods of end-to-end anastomosis that permit growth at the site of union as the child grows, thus avoiding future stenosis, and methods that prevent or minimize the development of constriction at the site of anastomosis, either by puckering at the time of operation, or by cicatricial contracture as healing progresses.

Grateful acknowledgment is hereby accorded to Professor J. K. Vennard of the Department of Civil Engineering at Stanford University for his helpful advice and criticism, and to Drs. Ernest Schnoor, Eldon Ellis, and Iseu da Costa, fellows in surgery, for their invaluable help in executing the experimental studies presented in this paper.

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