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Surgical correction of aortic insufficiency

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SURGICAL CORRECTION OF AORTIC INSUFFICIENCY

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INTRODUCTION

The treatment of aortic insufficiency has always been a perplexing problem once signs of congestive heart failure have appeared. It is the purpose of this paper to bring out that once medical management of aortic insufficiency is failing, there is now a surgical approach to correct the mechanical defect which for the most part is responsible for the development of heart failure.

ANATOMY

Before going into the various causes of aortic insufficiency it might be best to describe briefly the normal anatomy of the aortic valve. There are three cusps surrounding the orifice of the aorta, two posterior and one anterior. They are attached to the wall of the aorta by their convex margins. In the middle of the free margins is a thickened nodule called corpus Arantii (5). From this nodule tendinous fibers radiate through the valve to its free and attached margin increasing its strength. Between the valves and the wall of the aorta are three dilatations called the sinuses of Valsalva. It is these structures which are pathologically involved when certain organism or conditions produce aortic regurgitation.

ETIOLOGY

Until recently it was generally accepted that syphilis with its vascular complications was the leading cause of this condition. The latest articles by Ellis (26) and Hufnagel (25) indicate that the etiologic factors in order of frequency are the

following: (1) rheumatic valvulitis, (2) syphilis, (3) subacute bacterial endocarditis, (4) trauma, and (5) anomalies of the aortic arch. Hufnagel (17) reports that as high as 80% of the patients he has seen with this condition have a history of rheumatic fever. Of his first forty-two cases of aortic insufficiency, thirty-two were rheumatic in origin, six were syphilitic, and one was traumatic. There was a thirty-six to six male to female ratio. The ages ran from 19 to 54, with the average age being 34.

With the etiology and anatomy in mind, before going into the diagnosis, it might be well to dwell a while on the pathology and pathologic physiology. Before there can be any degree of reflux of blood through the aortic valves there must first take place some pathologic change.

PATHOLOGY

In rheumatic valvulitis the basic pathologic change (6) is a swelling and hyalinization of the collagenous ground substance of the fibrous tissues. This occurs chiefly in the mitral valve when the valves are affected, but as reported by Hufnagel (17) aortic insufficiency as a pure lesion appears to be more common than as was once thought. Wartlike nodules called verrucae measuring 1-3 mm. in diameter form along the closing edges of the cusps. These are located on the ventricular surface of the aortic semilunar valves. These verrucae represent only the superficial reaction which involves the annulus and septa fibrosa as well.

In the subendothelial layers there is degeneration of

the connective tissue and proliferative activity. Usually the whole valve is involved in this inflammatory change which shows edema, and an exudate of macrophages, lymphocytes, plasma cells and formation of young capillaries. Eventually the endothelium is destroyed. Within this inflammed tissue areas of hyalinization and necrosis then develop. Resultant healing produces a thick, distorted, retracted, and finally insufficient valve.

In syphilitic heart disease the main lesion is that of aortitis (3). This granulomatous process affects the aortic valve by downward extension. The proliferating process tends to spread the commissures of the valves. It also spreads to the free portion of the cusps producing adhesions between the lateral portion of the leaflets and the aortic wall. Eventually hyaline placque formation results. Fibrous tissue replaces the destroyed elastic tissue. The valves then become incompetent as this fibrous tissue produces shortening and thickening of the cusps with rolling of their edges and widening of the commissures. Histologically there is enarteritis obliterans of the vasa vasorum and it is felt by Saphir (3) to affect the cornaries is a similar fashion.

The appearance of angina pectoris is felt to be the result of one of the following or a combination of both of them. In syphilitic heart disease the generalized process of replacement of elastic tissue in the ascending aorta by fibrous tissue is felt by Osler (2) and later confirmed by Saphir (3) and others to produce a narrowing or constriction of the coronary ostia.

This results in a decrease blood flow to the myocardium. In both syphilitic and rheumatic heart disease producing aortic insufficiency, there is a tremendous hypertrophy of the left ventricle. When this happens, a point is reached when there is a relative decrease blood flow and the oxygen supply to the myocardium is decreased. Along with this decrease in quantity of blood to the myocardium, there is a lowered diastolic pressure which lowers the coronary perfusion pressure. The result is anginal pain coming from a starved myocardium. Now that the two major forms of pathologic change in the aortic valve producing aortic insufficiency have been discussed, it is interesting to review the patho-physiologic mechanisms that result in certain signs and symptoms which we may observe and detect in making the diagnosis of aortic insufficiency.

PATHOLOGIC PHYSIOLOGY

The reflux of blood through the faulty aortic valve producing left ventricular hypertrophy and dilatation and a wide pulse pressure are the characteristic manifestations or aortic insufficiency (9). Because of this reflux of blood back into the left ventricle during diastole, a rumbling diastolic murmur is heard. As the blood travels from the third and fourth intercostal space downward over Erb's point and to the apex.

As the left ventricle is being filled from the left auricle and the reflux from the aorta, its muscle fibers are stretched. Consequently the force of ventricular contraction is increased. The greater portion of reflux mentioned above takes place during

early diastole when the intraventricular pressure is low and the intra-aortic pressure is relatively high (39). This back flow causes a sudden drop in the intra-aortic pressure early in diastole. As this continues into early systole the aortic walls soon develop a decreased resistance to ejection. Thus with the aorta's decrease resistance to ejection coupled with the increase stroke volume of the left ventricle the systemic systolic pressure is raised. As this process continues there is reached a point when the rise in intra-aortic pressure produces an increase resistance to ejection, so that during the latter part of systole when the volume is decreased little blood is expelled. This accounts for the sharp fall of pressure at the end of systole. This coupled with the decreased peripheral resistance results in a decrease in diastolic pressure. These variations of pressure and flow transmitted to the smaller vessels are responsible for the various peripheral signs mentioned below.

Corrigan (1) in 1832 gave the first detailed analysis of aortic insufficiency and its physical signs. His article "On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves" describes clearly the physical signs, one of which bears his name. For a while, following this excellent expose, the condition of aortic insufficiency was referred to as Corrigan's disease.

He felt that as the aortic valves became inadequate and allowed blood from the carotids, subclavians and ascending aorta

to return into the left ventricle, the aorta and these vessels of the upper extremity became partially flaccid. Then with the next ventricular contraction "the blood propelled into them is sent along as a rushing current, which throws the sides of these arteries into vibrations". (1) These vibrations give to the ear "bruit de soufflet," and to the finger "frennissement". (1) The latter is a peculiar rushing thrill felt by the finger over the carotid and subclavian arteries. The water-hammer character of the pulse was a later name attached to the peculiar pulse. The initial forcefulness is brought about by the increase stroke volume and systolic pressure, while the sudden drop is a result of the lower than normal diastolic pressure. These can be taken as specific signs produced by the general manifestations of the disease.

The decrease in diastolic pressure is caused by two pathophysiologic changes. The incompetent valves allow for a sudden drop of the intra-aortic pressure which helps to lower the peripheral resistance. The second change is that a large cardiac output sets up an exaggerated sino-aortic reflex at the height of systole with a resulting marked arteriolar dilatation. (9) This reflex leads to a rapid escape of blood from the arterioles into the capillaries with an overall decrease in peripheral resistance. It is the peripheral resistance which largely determines the diastolic pressure.

The capillary pulsation, or Quincke's pulse, is seen as a rhythmic expansion of the capillary loops with each heart beat.

This can be produced in young people at room temperatures after soaking their hands in water at 114 degrees Fahrenheit. These capillary pulsations should be regarded as dilated arterioles allowing the impulse of the heart beat to reach the skin vessels and may be normal or abnormal. The cause for ventricular hypertrophy as stated results from increase work load on the ventricle, but that of ventricular dilatation is another story.

The filling of the coronary arteries is impaired as there is a decrease in diastolic pressure which lowers the coronary perfusion pressure. This results in poor nutrition of the heart muscle. Because the heart fibers do not have adequate nourishment they loose their strength and ventricular dilatation results.

When the valves become so incompetent that the reflux of blood back into the left ventricle is greater than the relative increase in stroke volume, resulting in a decrease of peripheral blood flow, congestive heart failure is present. From the writings of Corrigan (1), Osler (2), Bailey (23), and Webster (16) it is seen that the prognosis for life expectancy over a five year period is markedly decreased when this occurs.

The method or methods of producing congestive heart failure are usually considered under the broad heading of forward and backward theories. Using as a starting point the left ventricle, the forward theory runs along the following lines. The decreased cardiac output results in decreased glomerular filtration rate, sodium and water retention, incfeased extracellular fluid and increased venous pressure; all of these are major factors in the

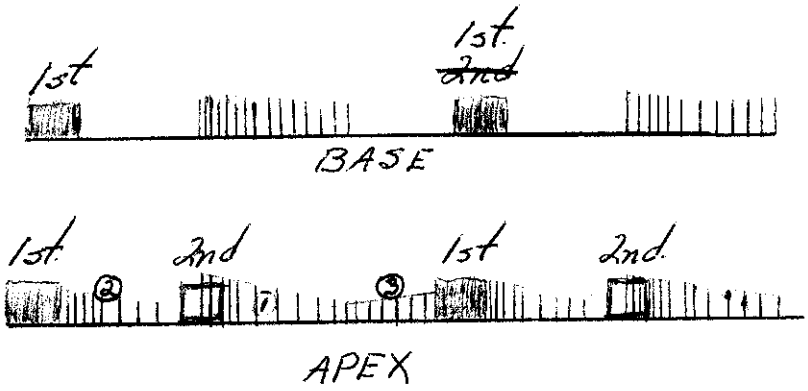
production of edema. The backward theory in essence is that a failing left ventricle produces increased pressure in the left auricle, increasing pulmonary pressure resulting in passive congestion of the lungs. This increases right ventricular pressure, increasing right auricular pressure, producing increase venous pressure which results in dilatation and pulsation of the neck veins, passive congestion of the liver and ankle edema.

SIGNS AND SYMPTOMS

With the pathologic and physiologic processes enumerated it might be well for clarification purposes to run over the significant signs and symptoms. Osler (2) felt that headache, dizziness, flashes of light and feeling faint on rising were some of the early symptoms of aortic insufficiency. The common symptom of fatigability is thought by Bailey (23) to be the result of anemia which is seen more commonly in this heart condition than other valvular lesions. Another outstanding symptom in comparison with other valvular lesions, is that of anginal attacks. It is felt by Hufnagel (25), and Bailey (23) to be present in well over half of all patients with aortic insufficiency. With the onset of palpitation and cardiac distress with exertion, congestive failure is just around the corner; dyspnea, orthopnea and peripheral edema are then seen. Osler (2) observed and now confirmed by Hufnagel (17) and Bailey (23) that the occurance of sudden death after the onset of congestive failure was and is more common in this condition than other valvular diseases.

Aortic insufficiency has been defined by Ellis (26) as

"a condition in which the aortic valves fail to close completely during diastole, with a resultant regurgitation of blood into the left ventricle during this period of the cardiac cycle." Pathognomonic of the lesion as stated by White (32) is a "diastolic murmur beginning early and best heard down the left border of the sternum, associated with the water-hammer pulse. The apex impulse is heaving and forceful in character. The point of maximum impulse is displaced downward more than outward so that it is palpated most commonly in the sixth intercostal space outside the midclavicular line. Auscultation reveals a rumbling diastolic murmur heard over the aortic area and over the third and fourth intercostal spaces to the left of the sternum which is transmitted downward. The murmur is diminuendo in character being heard loudest in the early part of diastole and then gradually diminish in intensity. (4) It is usually heard best over the third and fourth intercostal spaces, especially if the patient is placed in the upright, dorsal and left lateral positions with his trunk bent forward. Below is a diagram copied from Cabot and Adams' "Physical Diagnosis" showing the relationship of the murmurs to the heart beats.



At the base and along left sternal border a loud diastolic murmur is heard which replaces the second sound.

At the apex, there is the same diastolic murmur (1) as above which often partially masks the second sound. There may be either a systolic murmur (2) due to relative mitral insufficiency or if regurgitation is severe a diastolic murmur (3), (Austin Flint, Variety). The latter is caused by blood passing through the mitral orifice which is relatively narrowed as compared with the markedly dilated left ventricle.

The peripheral signs accompanying this disease are enumerated below. Osler (2) felt the visible pulsations were more commonly seen in the peripheral vessels in this condition than in any other disease. One of the most striking signs when present is the hyperactive pulsation of the carotid artery. When this is severe it causes the whole head to throb. Ophthalmoscopic examination may reveal pulsating arteriolar vessels which may even have the jerking quality so often associated with the Corrigan pulse. The capillary pulse is observed in the fingernails. It can also be elicited by seeing a flush come and go after rubbing the forehead, or by pressing on the ear lobe with a glass slide. A to-from murmur heard over the femoral artery is called Duroziez's sign and is often heard in this condition. These signs are by no means pathognomonic as they may be seen in association with hyperthyroidism, anemia (especially pernicious anemia), fevers, nervous hearts and certain instances of hypertension, as stated by Levine (11).

In the uncomplicated case the electrocardiogram shows left ventricular hypertrophy with or without strain. There is inversion of the T waves in Lead I and in precordial leads usually.

The roentgenograms as related by Shanks and Kerly (14) concerning aortic insufficiency are non-specific as aortic stenosis will produce like shadows. In some cases there may be prominence of the ascending aorta and an aortic knuckle. The apex beat is seen below the left diaphragm. Most characteristic of all is the increase in the size of the left ventricle. Its transverse diameter is increased to the left, and the left border is rounded and very dense.

In an oblique view of a patient with syphilitic heart disease, there is an exaggerated forcible pulsation of the left border of the aorta, dilatation of the ascending aorta, and prominent ascending aorta. In a patient with congestive failure as a result of aortic insufficiency, there are certain signs which corroborate the diagnosis. These are a left ventricle which has increased transversely more to the left than any other direction. The pulsations are less marked, and the pulmonary vessels are engorged. The left auricle is increased in size. As failure progresses, the shadows of the right ventricle and pulmonary artery fill in the concavity between the aorta and the left ventricle.

The tracing of the ballistocardiogram shows characteristic changes for aortic insufficiency. The tracing itself represents the longitudinal movements of the body resulting from heart motion. (12)

Men trained in this technical work calculate the cardiac output from the tracing which in essence records the stroke volume of the heart. Smith (21) ran ballistocardiograms on 50 people taken at random in an attempt to establish normals. They felt they corresponded closely with the findings of others writing on this subject. They then recorded the results of 10 patients known to have aortic insufficiency. They felt a diagnostic pattern was presented which included the following: (1) higher amplitude in all measurements, (2) deeper velocity K waves, (3) higher amplitude acceleration K waves when measured from a base line, and (4) notching of accelerated J wave with a ratio of J to K over 1.3. They felt these abnormal deviations were so constant that the ballistocardiogram could provide a sensitive measure for demonstrating small aortic valve leaks and thus help in the differential diagnosis of aortic stenosis.

Gorlan (37) ran a series of cardiac catheterizations on 24 patients with aortic valvular disease. Correlating his findings, he was able to determine enough significant difference to differentiate with a fair degree of accuracy the amount of aortic stenosis and/or insufficiency.

There have been two main methods employed to obtain these results. Bjork (34) prefers the transthoracic approach. He introduces a 20 cm. long needle paravertebly and directs it towards the left atrium. He then threads a catheter through the needle into the left ventricle, and then into the aorta. As he withdraws the catheter certain pressures are obtained in the different

chambers. Zimmerman (8) was among the first to carry out left heart catheterization by the retrograde method, from the brachial, ulnar or femoral artery.

Correlating the various pressures obtained plus the clinical information, the above authors felt that they are now able to tell with some accuracy if the aortic valvular lesion is pure stenosis, insufficiency, or a combination of the two. The only findings which may require some explanation is the left auricular pressure curves. The "a" wave is caused by auricular systole; the "c" wave by the rising pressure in the ventricle with the onset of ventricle systole; the "x" wave or "descent of the base" following the "c" wave by negative intra-auricular pressure and by drawing caudad of the A-V septum as the ventricle contracts; the "v" wave is caused by inflow of blood into the auricle from pulmonary veins.

Bjork felt that a withdrawal curve similar to the following was indicative of aortic stenosis; aortic systolic pressure of 105 mm Hg., left ventricular systolic pressure of 220 mm Hg., and left strial systolic pressure of 10 mm Hg. Below is a graft which summarizes the major differences.

	Aortic Stenosis	A S and A I	Aortic Insufficiency
Left ventricular mean pressure	230-250 mm Hg. maintain with work.	200-230	? - rose 35-80 mm Hg with work.
Diastole (left ventricle)	6-15	60 mm Hg.	15-20 mm Hg.
Arterial B.P. (Avg.)	109/72	134/65	174/55
Left ventricle work/min.	2 x norm	3 x norm	3-4 x norm
Left atrial pressure pulse	large A waves negative mid-systole X waves U wave = to or larger than V waves.	same as A S	no prominent "a" waves. Absent mid-systole dip in X wave.
Diastolic pressure	70 or more		60 or less
Pulse pressure	45 or less		60 or more
Mean aortic diastolic gradient	100-140 mm Hg.		50-70 mm Hg.
Effect work	Arterial pressure ↑→stroke. Volume ↓		Arterial pressure ↑→ stroke Volume ↑
Systole upstroke/sec.	.15 or more		.10 or less
Systole period/sec.	.30 or more		.30 or less

DIFFERENTIAL DIAGNOSIS

Other conditions producing a diastolic murmur heard along the left sternal border which must be differentiated from aortic insufficiency are (1) mitral stenosis, (2) Graham Steell murmur, (3) congenital heart disease, and (4) organic tricuspid stenosis. The presence of auricular fibrillation points toward mitral stenosis. The absence of the above mentioned peripheral findings would mitigate against the presence of a Graham Steell murmur. In the latter there is no increase in the systemic arterial pulse pressure. On roentgenograms the pulmonary artery is usually quite prominent, and upon fluoroscopy pulmonary regurgitation results in marked pulsation of the hilar shadows of both lungs producing the "hilar dance". The electrocardiogram will show a tendency toward right ventricular hypertrophy which is not tenable with aortic insufficiency. The roentgenogram finding of a dilated pulmonary artery along with the finding of a "machinery-like murmur" in a child would strongly indicate the possibility of a patent ductus arteriosus. The findings of an enlarged right ventricle with a strain pattern on electrocardiogram, transmission of the murmur downward and to the right, and pulsating liver would indicate organic tricuspid stenosis.

TREATMENT IN THE PAST

When contemplating any surgical procedure, the question should always be asked: is there a more conservative form of treatment which will provide the patient with as good a prognosis? Although there has been no well conducted series with controls,

it is the general opinion of most authors as stated before that once the patient shows symptoms of severe left ventricular failure, his prognosis is guarded.

Webster (16) et al have analysed 1020 patients with a clinical diagnosis of syphilitic aortic insufficiency followed at John Hopkins Hospital and New York Hospital for 20 years. Of interest here is Webster's group classified as (1) having failure at diagnosis, and (2) having failure at diagnosis or later. He presents a graph representing survivors out of 100. Class (1) has a 31% - 2½ year survival, and class (2) a 26% - 2½ years survival rate. This is with medical treatment.

Hufnagel (19) is currently reporting 59% survival rate 2½ years postoperatively. However, of his patients only 20% were due to syphilis. All of his patients had symptoms of failure before operation which would place them in a class similar to Webster's class (1).

Because of the different percentages of disease states causing the aortic insufficiency, no definite conclusions should be drawn. It is interesting to note that Webster shows a further steady decline so that class (1) has an 18% and class (2) a 11% five year survival. Hufnagel (31) maintains that once the patient has survived the first postoperative year his prognosis is much improved and that the cause of his death is likely be due to an unrelated condition.

Before going into the various surgical procedures, the

present day medical treatment will be summarized. According to White (32) and Stead (33) the object of treatment is to restore the balance of supply and demand, and to remove and prevent the accumulation of fluid in the body tissues. Methods by which an attempt is made to accomplish these objectives are the following. Reduce the body's requirement for blood by placing the patient on complete bed rest until the acute episode is over, or until you are sure there is some myocardial reserve. A low calorie diet for the obese patient will help. Increase heart output by digitalization. White (32) has found that as a base line "digitoxin .15 mg. three times a day for a week and then once daily thereafter" is quite satisfactory. This can be regulated on any patient until the therapeutic level is reached or the patient shows toxic manifestations. These include for the most part nausea, vomiting, decrease appetite, diarrhea, disturbed vision and/or mental confusion. The last is the prevention and elimination of edema. First the sodium intake should be regulated to 150-200 mg. per day. The low salt syndrome is seen in congestive heart failure more commonly in a patient with renal disease and/or who has taken mercurial diuretics. If reduction of sodium intake is not sufficient to produce the above effect, 1-2 cc. of neohydrin or mercuhydrin is commonly employed. Often ammonium chloride is used to potentiate the effect of the mercurial diuretics in the refractory case.

SURGERY, GENERAL

As can be seen from the above, once the patient is in

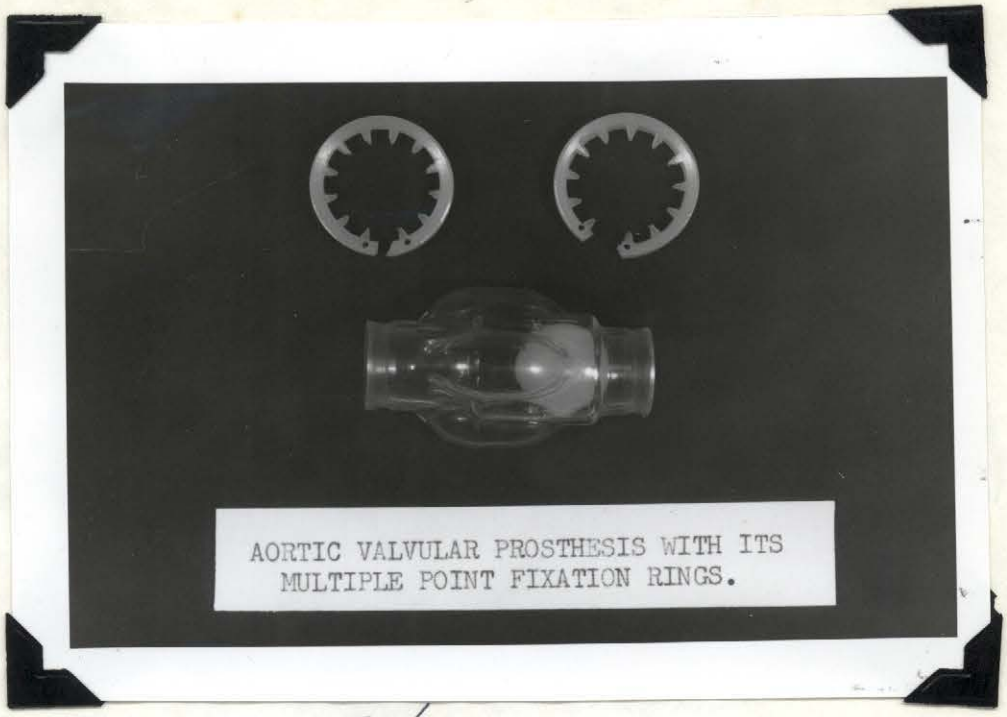
class three and especially class four of the American Heart Association classification, not only is his life expectancy markedly reduced, but he is forced to very limited activity. As the mechanical elements plays such an important part in this, Hufnagel (25) and Bailey (23) have both felt that if the defect could be corrected the prognosis of the patient would be greatly improved.

The first surgical attempt at the correction of aortic insufficiency was by Bailey (23) in 1951. This was along the line of reconstruction of the defective valve. This consisted chiefly of transplantation of tissue in an effort to occlude the leaking portion of the aortic orifice. At first, Bailey (23) used plastic materials and rubber like substitutes in an effort to form a good valve. He made fifteen attempts to correct aortic insufficiency by the placement of a pedunculated ball or a hammock of pericardial tissue within the aortic lumen just above the level of the valve. It was believed that the free movement of these grafts could produce a considerable tamponade of the valve opening. This thought was at first confirmed by the resulting elevation in diastolic blood pressure postoperatively. However, with the passage of time these attempts did not live up to expectation for what appears to be the following reasons. The inability to palpate the valve made it difficult to place the pericardial tissue accurately above the regurgitant orifice. The grafts swelled initially and then became fibrotic and finally retracted from their tamponading position. This technique could not cope anatomically with the type of valvular dysfunction which results from a dilatation of the annulus.

SURGERY, HUFNAGEL

In 1944 Hufnagel (12) began his investigative work which has lead him to the use of a plastic valvular prosthesis in the surgical correction of aortic insufficiency. In his early experiments with dogs, he was stymied because of two major complications. The first was that of peripheral embolization. Autopsy revealed consistently the presence of a thrombosis at the prosthetic-aortic junction. He felt that the clotting tendency at this point was the result of angulation of the prosthesis and a discrepancy between the size of the prosthesis and the diameter of the aorta. He them decreased the risk of peripheral embolization by (1) excising a 1-2 cm. segment of aorta which prevented angulation, (2) by placing a band of orlon mesh around the ends of the aorta and inside the prosthesis so that this portion of aorta was snug with the valve, and (3) by producing the plastic valves in four sizes pertaining to diameters.

The second complication was the production of necrosis of the aorta distal to the valve and the appearance of sudden death due to the formation of an artificial or false aneurysm at this point. This was solved by using "multiple point fixation" rings which allow secure fixation of the aorta to the prosthesis. This was brought about by using split nylon rings which exert pressure at numerous points, leaving spaces between the points of pressure so that the blood supply to both segments of the aorta was not disturbed. See Picture (1). The nylon multiple point fixation rings are so constructed that the teeth are longer than



AORTIC VALVULAR PROSTHESIS WITH ITS MULTIPLE POINT FIXATION RINGS.

Picture -1-

the thickness of the aortic wall which guards against slippage of the prosthesis. This type of fixation was then used experimentally several hundred times with no fatality caused by erosion or slippage of the fixation device. To date, Hufnagel (15) feels this the only method to be entirely satisfactory for the fixation of the plastic valve to aorta.

The valve itself is made of a plastic material called methyl methacrylate. The surface properties of this plastic material are such that they inhibit coagulation of blood. The best results are obtained when the surface is highly polished. As can be made out from the picture above, the valve is composed of an inlet, a chamber containing the polyethylene ball, and an outlet. The valve is made out of a single piece so that the inner surface is without seams and is very smooth. The polyethylene

ball is so light that it requires only 5 mm Hg. of pressure differential to open and close the valve. For clinical use the valve is made in four sizes, 3/4", 7/8", 1", and 1 1/8", which correspond to their diameters at either end. The more recently made valves are such that the proximal end is approximately 1/32" larger than the distal end.

Before going into the actual description of the operation, it might be of interest to relate a case story. Hufnagel (15) inserted his first prosthetic valve in a human September 1952. This was a 37 year old white female who had symptoms of severe aortic insufficiency and had rheumatic fever as a girl. She was treated on a conservative basis until signs of congestive heart failure developed. With the development of an Austin-Flint murmur, a diastolic gallop rhythm, angina pectoris, cardiomegaly, grade IV aortic diastolic murmur, her prognosis was deemed poor and an operation was advised. Before the operation the blood pressure in her arm was 160/0-180/0, her cardiac output was very low, and her circulation time was 32 seconds arm to tongue.

During the operation, it was necessary to insert the valve lower than the usual 4-6 cm. beyond the origin of the left subclavian artery because of a constriction of the aorta three inches below the artery. Actually this placed the valve in the thoracic aorta.

Three weeks postoperatively, the venous pressure was 90 mm of water and her circulation time arm to tongue was 15 seconds.

The Austin Flint murmur decreased in intensity, and the gallop rhythm disappeared. The above mentioned aortic diastolic murmur was reduced to grade II. This is believed to be the first application of a prosthetic valve in ahuman for aortic insufficiency. According to the latest report, August 1955 (25), the patient is very much alive and working daily.

Hufnagel (17) has chosen as the site for the insertion of the plastic valve, a point, some 4-6 cm. beyond the origin of the left subclavian artery. His reasons being: (1) The blood supply to the brain is not interrupted, (2) The danger of air embolism is minimized, (3) Manipulation of the aortic valve is at a minimum as manipulation of the valve produces disturbing cardiac arrhythmias. Most patients have a left sympathectomy of the second, third, fourth and fifth thoracic ganglia, and a ligation of the left internal mammary artery distal to the origin of the pericardiophrenic branch. This procedure is thought to increase the collateral blood supply of the left ventricle and to lessen coronary insufficiency (29).

Hufnagel (25) has set up the following criteria for an ideal patient before the prosthetic valve operation is performed. The patient should (1) be under 50 years of age, (2) have pure aortic insufficiency, or have a correctible second lesion, (3) not have active rheumatic fever, (4) show signs of progression on medical management, (5) have only moderate cardiac enlargement, (6) not present symptoms of intractable angina pectoris, (7) have no serious changes in ventricular conduction, and (8) not show

signs of serious renal or hepatic disease. He thus concludes that in the relatively ideal patient who has five or more of the above, the operative mortality should approximate 10%; while the very advanced case approaches 40%.

The following concerns the details of the actual operation and operative technique. The patient is usually prepared with a barbiturate, morphine and scopolamine. Demerol is contraindicated due to its belladonna like action. As in all cardiac surgery the patient is carried in a very light plane of anesthesia, usually by a mixture of nitrous oxide and ether. Should a drop in blood pressure occur, O'Donnel (24) prefers the use of neosynephrin to combat this complication. Because Rogers (27), Longan (28) and Lord (29) have encountered some difficulty in reduplicating Hufnagel's results, the following is a direct quotation of his technique for the insertion of the plastic valve.

"After the patient is prepared and draped for a left thoracotomy, the chest is entered through a posterolateral incision through the bed of the fifth rib. Segments of the fifth and sixth ribs are then usually removed. The aorta distal to the origin of the left subclavian is mobilized for 6-8 cm., and 4-5 pairs of intercostal vessels are ligated. The nylon rings along with their attached ligatures are passed around the intact aorta. The closing instrument is then inserted into each of the nylon rings. The valve is then washed with fresh saline solution. Prior to placing the valves on the operating table, they are sterilized by soaking

them for eighteen hours in 1:500 Zephiran solution, and triply rinsed in sterile saline solution. The valve is prepared for insertion by applying the valve holder. The aorta is then cross-clamped with Pott's ductus clamps proximally and distally. Following this, a one-half to one inch segment of aorta is excised. The distal end of the aorta is then grasped at equidistant points in Judd-Allis clamps and held open widely. The valve is inserted into the opened distal aortic end, and the ring closed into the groove with the holding clamp. The ligature is then tied, consisting of 5 braided silk ligature and No. 30 wire ligature through the appropriate holes in the rings. Great care is taken to see that the ring fits well and that the edges of the ring are well approximated when the ring is closed. The ring-holding clamp is removed. The Judd-Allis clamps are then applied to the proximal aorta. The proximal end of the valve is inserted, and the ring similarly closed and tied. The ring clamp and valve holder are then removed. The valve is filled with saline solution containing 1 mg. of heparin per 10 cc. As much air as possible is removed from the valve. The distal aortic clamp is then removed, and following this, the proximal clamp is removed. These clamps are taken off slowly, and blood flow to the aorta is now resumed. A twisted Surgaloy No. 000 suture is placed through the holes in the ring and tied. This gives further security to the valve and to the ring, insuring against any slippage, giving additional support in case the ligatures should break. A pleural flap is then turned down from the

lateral chest wall and sutured over the valve after a small square of Gelfoam is placed between the valve and vertebral bodies. After the pleural flap has been sutured in place, the valve is covered completely. The chest is then closed in the usual fashion." (17)

PHYSIOLOGY OF HUFNAGEL'S SURGERY

Before looking into the results of this operation let us first ask ourselves or see if the operation in any way corrects the pathophysiologic mechanism causing the trouble as enumerated earlier in this paper. Sarnoff and Case (35) feel that as a basis to understand the physiologic effects of the operation the relationship between "oxygen requirements of the myocardium and the work it is called upon to perform" is fundamental. In an attempt to more clearly understand the above relationship Rose (18) had nine patients undergo preoperative and postoperative clinical trials to determine their (1) brachial and femoral artery pressure pulse contours, (2) cardiac output and stroke volume, (3) T-1824 dye concentration curves, and (5) blood volumes.

The following were their results. The femoral artery pulse tracing showed in all 8 cases a decrease in systolic pressure and an increase in diastolic pressure. However, the brachial artery pulse tracings showed in all 8 cases except one an increase in systolic pressure and in all a decrease in diastolic pressure. See Picture (2). In six of the eight cases the cardiac output showed a varied increase from 19-77% from that of preoperative levels. In these six this was associated, with one exception,

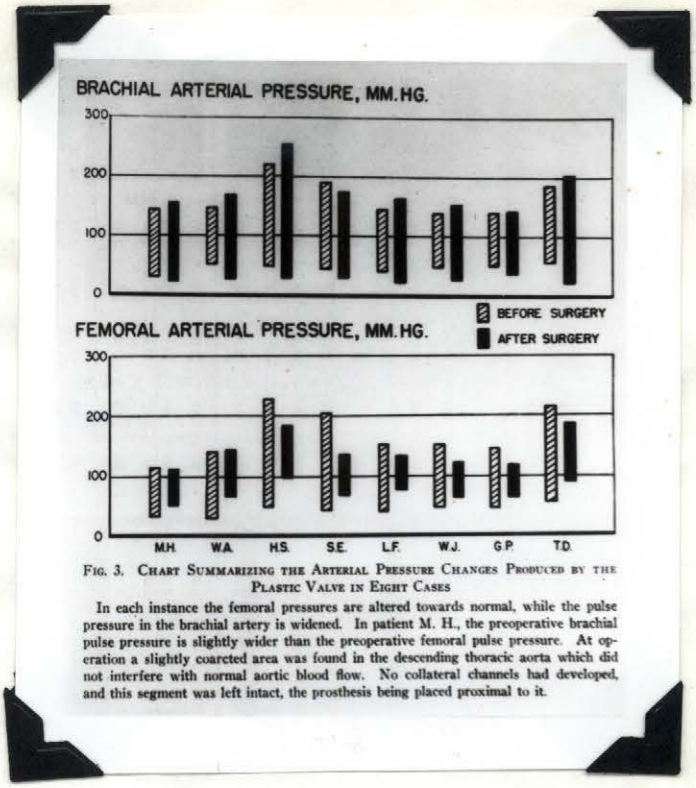


FIG. 3. CHART SUMMARIZING THE ARTERIAL PRESSURE CHANGES PRODUCED BY THE PLASTIC VALVE IN EIGHT CASES

In each instance the femoral pressures are altered towards normal, while the pulse pressure in the brachial artery is widened. In patient M. H., the preoperative brachial pulse pressure is slightly wider than the preoperative femoral pulse pressure. At operation a slightly coarcted area was found in the descending thoracic aorta which did not interfere with normal aortic blood flow. No collateral channels had developed, and this segment was left intact, the prosthesis being placed proximal to it.

Picture -2-

with both an increase in oxygen consumption and decrease A-V oxygen difference. The effective stroke wolume, determined by dividing the average cardiac output by the heart rate, also increased in 6 of 8 cases. The T-1824 dye concentration curves showed that with one exception the postoperative curve was steeper and more rapid than the preoperative curve. The one patient not showing this response also showed no increase in cardiac output post-operatively, or increase in brachial artery systolic pressure. The mean circulation time was calculated from this and was found to be decreased in all 8 patients. See Picture (3) on page 27.

TABLE I

Data obtained pre- and postoperatively in nine patients with a plastic valvular prosthesis for severe aortic insufficiency exclusive of systemic arterial pressures

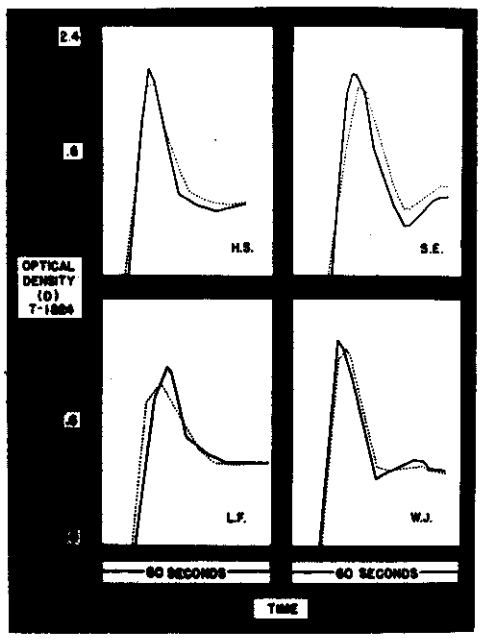
Patient	Days*	Body surface	O_2 consumption	A-V O_2 difference	Fick cardiac output	Average cardiac output	Per cent increase cardiac output	Average cardiac index	Per cent increase cardiac index	Heart rate	Effective stroke volume	Mean circulation time	Arterial hematocrit	Total blood volume	Pressures	
															Pulmonary artery	Right ventricle
								L./min./M.² body surface								
		sq. meters	cc. per minute	volume per cent	liters per min.	liters per min.			postop	per min.	cc.	seconds	%	liters	mm. Hg	mm. Hg
M. H.	1	1.47	148	4.8	3.1	3.25		2.2		66	49	23.5	40	4.08		
	pre		153	4.5	3.4											
	20	1.47	196	3.4	5.8	5.75	77	3.9	77	90	64	16.5	32	3.75		26/0
	post		201	3.5	5.7											
W. A.	8	1.67	175	6.4	2.7	2.6		1.55		68	38	55.0†	39	2.73		50/5
	pre		171	7.0	2.5											
	36	1.64	202	4.7	4.3											
	post		166	5.1	3.3	3.8	47	2.30	48	75	51	40.0†	33	2.61		30/0
C. F.	4	1.89	257	6.3	4.1	3.85		2.0		95	40	28.0	43	4.06		20/0
	pre		163	4.6	3.6											
	14	1.80	250	6.3	4.0	4.4	14	2.4	19	92	48	19.0	35	4.34		20/0
	post		300	6.3	4.8											
H. S.	7	1.93								75		24.0	39	7.14		
	pre															
	18	1.88								90		20.5	36	6.47		
	post															
S. E.	3	1.85	272	5.8	4.7	4.8		2.6		90	53	25.0	37	3.60		55/8
	pre		250	5.1	4.9											
	21	1.72	242	5.4	4.5	4.4	−10	2.55	− 2	100	44	24.5	33	4.33		30/0
	post		244	5.7	4.3											
L. F.	1	1.98	292	7.7	3.8	3.7		1.85		100	37	48.5†	44		27/10	25/2
	pre		314	8.6	3.6											
	21	1.90	260	5.8	4.5	4.45	20	2.35	26	100	45	27.5	40	4.92	25/14	28/4
	post		262	5.9	4.4											
W. J.	3	1.83	209	5.7	3.7											
	pre		205	5.4	3.8	3.75		2.05		80	47	25.0	48	5.50		18/2
	28	1.68	232	4.9	4.8											
	post		225	4.5	5.0	4.9	31	2.90	42	80	61	20.5	32	4.76		20/0
G. P.	9	1.92	238	4.8	5.0	4.9		2.55		86	57	22.5	43	5.09		36/7
	pre		237	5.0	4.8											
	30	1.82	250	4.4	5.8	5.95	21	3.25	28	86	69	17.2	38	5.85		36/4
	post		250	4.1	6.1											
T. D.	26	1.88	288	5.7	5.1											
	pre		306	5.8	5.2	5.15		2.75		80	64		40	5.47	76/30	
	30	1.77	198	5.5	3.6	3.6	−42	2.05	−34	70	52		34	4.36		30/0
	post		200	5.6	3.6											

* pre = Number of days before operation that studies were performed.
post = Number of days after operation that studies were performed.
† Mean circulation time determined with difficulty because of a markedly abnormal dye concentration curve.

Picture -3-

-27-

In four cases postoperatively, dye transit curves were determined simultaneously in both the brachial and femoral arteries. Despite the greater regurgitant flow proximal to the valve these two curves showed a striking correspondence. See Picture (4) below. In all



Picture -4-

FIG. 6. SIMULTANEOUS POSTOPERATIVE DYE CONCENTRATION CURVES DRAWN IN BRACHIAL AND FEMORAL ARTERIES IN FOUR PATIENTS

Brachial curves are the dotted lines, femoral curves the solid lines. Reference to Figure 5 reveals that in each instance the alterations which occur in the shape and duration of postoperative femoral dye curves are simultaneously noted in the brachial dye curves. See text for discussion.

8 cases the hematocrit decreased postoperatively. The reduction varied between 3 and 8 with the exception of one which decreased 16 mm. The blood volume in these 8 cases showed nothing significant in that five showed a decrease and 3 showed an increase.

It is felt by Rose (18) that because of the results in

the cardiac output and dye injection curves, the cardiac function was improved in 6 of the 8 cases definitely. Because the brachial artery dye injection curve corresponded very closely to the femoral artery dye contour, in all four of the patients tried postoperatively, it was felt that the improvements could not alone be attributed to the decrease in total regurgitant volume. If that was the case then there should have been a definite difference between the brachial and femoral artery dye contours. It was seen that in those patients showing an increase in cardiac output postoperatively, they also showed a shorter duration in dye concentration curves. Because of these findings, the authors (18) felt that the greatest single source of improvement was of cardiac origin. The chain of events leading to this was explained thus: by introducing a plastic valve, the absolute volume of regurgitation was reduced, thus reducing the work load on the left ventricle. This decreased the myocardial need for oxygen. Ventricular contraction then became more effective, permitting better emptying, and with this, there was an increase in cardiac output and stroke volume.

Sarnoff (36) and McKusick (20) feel that more significance should be given to the change in end-diastolic pressure. Sarnoff points out that in the 8 cases reported by Rose (18) there was a drop from 52 to 30 mm Hg. in the end-diastolic brachial artery pressure. This was an average fall of 43%. McKusick (20) reported a similar finding on their one patient. The end-diastolic pressure proximal to the valve fell from 75 to 35 mm Hg. or a fall of 53%.

He contributed even a greater significance to the fall of mean diastolic pressure from 96 to 46 mm Hg. or a fall of 49%.

However, Rose (18) maintains that the more complete left ventricular emptying following the operation provides adequate explanation for this higher systolic and lower diastolic pressure in the regurgitant brachial artery, and that the clinical follow-up produced adequate justification for this impression. Because the ventricle is contracting more efficiently producing an increase stroke volume, the systolic pressure should naturally rise. Since the more efficient left ventricular contractions allow more complete emptying, there is a greater pressure gradient from the regurgitant artery to ventricle and thereby the brachial diastolic pressure is reduced. The justification of the above hypothesis lies in the fact that Hufnagel (17) pointed out that in 17 of the 23 patients surviving 11 months or more following the operation, there has been a 100% increase in exercise tolerance, and the use of nitrites for anginal symptoms has also been cut down universally. Thus it seems that the reduced work load placed on the myocardium allowing more effective left ventricular contraction and thus producing better emptying plays a more dominant role than the reduction of maximum per minute oxygen available to the myocardium.

Another controversial point is the significance of the dye injection curves. Borden (7) showed that in aortic valvular disease with left ventricular dilatation, prolonged mean dye circulation time correlated in part with increase diastolic volume in the heart.

Hufnagel (18) felt that possibly the decrease in mean circulation time seen in 7 of his 8 cases following operation, could be due to the decrease diastolic volume of their hearts. To help corroborate this idea were his follow-up studies which showed a decrease heart size in all surviving cases. This again was attributed to the more efficient ventricular contraction resulting in less pooling of blood in the ventricle so that the passage of dye is increased.

Newman (13) has argued that the character of the downslope of the dye injection curve is chiefly a function of pulmonary blood volume, and thus the alterations of dye injection curves in Hufnagel's patients could be attributed to a decrease volume of the pulmonary vascular bed. The studies and Borden (7) however, showed that prolonged circulation time in left heart failure depends for the most part on the amount of residual blood in the heart and only slightly to the degree of pulmonary congestion. In addition to this, Hufnagel (18) reported that prior to surgery every patient underwent extensive medical therapy in an attempt to decrease or remove all clinical signs of congestive heart failure. With this in mind and with the knowledge that there has been shown to be a decrease in the diastolic volume of the heart following the Hufnagel operation, it seems probable that the studies of Borden (7), are more nearly correct than those of Newman (13).

In the final analysis there is therefore seen following the operation, (1) reduction of work load on the left ventricle, and (2) secondarily a reduction of the maximum per minute oxygen

available to the myocardium. In those patients receiving definite improvement, the decrease in the work load can be thought of as being greater than the decrease in oxygen available to the myocardium. In those patients who did not seem to receive much benefit the reverse is true and in some of these who died following the operation there was found to be definite organic pathological change in the coronaries.

The most serious complication following the insertion of the plastic valve appears to be the so-called "sudden death" syndrome that strikes some patients near the end of the first postoperative week. The exact reason for this is not known, but some insight concerning the problem has been expressed through the following relationships. It is known that the oxygen supply to the myocardium must be increased as the work of the left ventricle is increased. Case (35) has shown the effect of anemia on this relationship. Picture (5) (page 33) shows how they plotted the coronary vascular resistance against coronary perfusion pressure and the changes in this relationship produced by anemia in the dog. From these experiments they concluded that (1) anemia can produce a lowering of the coronary vascular resistance, and (2) marked degrees of anemia stimulate the coronary artery to maximum dilatation. At this point the ability of the left ventricle to function effectively is at its lowest.

Another interesting point concerning this matter is brought out by Picture (6) (page 33) from Case (35). It shows that as

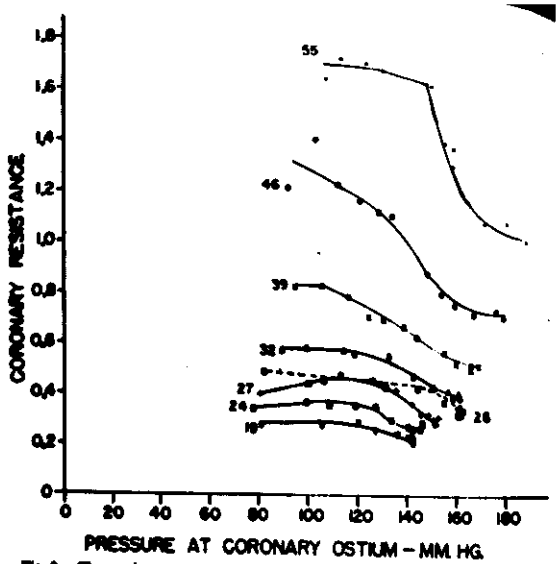


Fig. 2. Changes in coronary resistance with anemia. Coronary resistance expressed as mm. Hg pressure drop across the coronary vascular bed divided by the coronary flow in cc./min. Numbers at left of each curve indicate the hematocrit and the dotted line represents the curve obtained after reinfusion of red cells. (After Case, Berglund, and Sarnoff: Am. J. Med., vol. 18, 1955.)

Picture 5

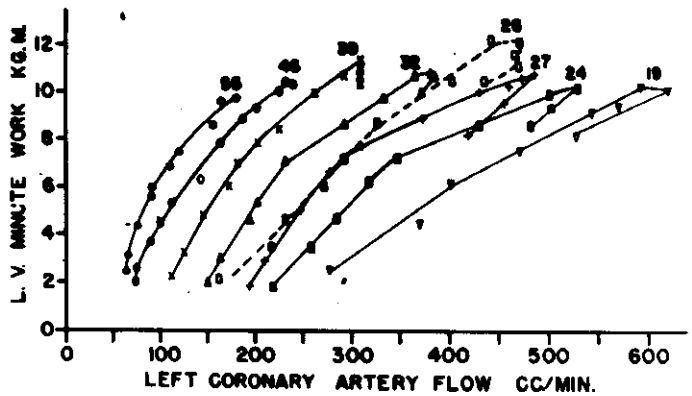


Fig. 4. Work-flow relationships during anemia. Left ventricular work per minute in kg.-m. is plotted against left coronary flow in cc./min. Figures above each curve represent the hematocrit. The dotted line is the curve obtained after the reinfusion of red cells. (After Case, Berglund, and Sarnoff: Am. J. Med., vol. 18, 1955.)

Picture 6

the hematocrit decreased, while the left ventricular work load remained the same, there must be a tremendous increase in the left coronary flow for the myocardium to maintain the same level of work load. For example, at a left ventricular minute work load of 6 kilogram meters per minute, the left coronary flow was 98 cc. per minute at a hematocrit of 55; but at a hematocrit of 32, and the same work level, the left coronary flow must increase to 205 cc. per minute.

Since none of the 9 patients studied by Rose (18) succumbed to the "sudden death" syndrome, it would be wrong to attempt to directly correlate his findings regarding the changes in hematocrit and blood volume existing postoperatively in these patients. As was stated before, all 9 patients' hematocrit fell from an average of 41.5 to 34.8. These determinations were taken between the 14 and 36 days postoperatively. As simultaneous blood volume studies were also done showing a fall in 3 and rise in 5 it is possible to eliminate hemodilution as a reason for the postoperative fall in hematocrits. As shown previously the effect of anemia in depressing myocardial function depends upon the degree of relative coronary insufficiency already present. It therefore appears that the only pertinent conclusion to be drawn from this is that the hematocrit should be followed closely postoperatively. When an anemic state appears to be developing, a transfusion will relieve the coronary vascular bed from dilating as it would have ordinarily done in response to the developing anemia.

The other possibility contributing to the "sudden death" syndrome is that of red cell destruction by the lucit ball valve prosthesis. Stohlman (31) is currently carrying out experiments concerning the plasma hemoglobin, reticulocyte count, and hematocrit for the first 2-3 weeks after the insertion of the lucite ball valve prosthesis in dogs. As seen from Picture (7) below,

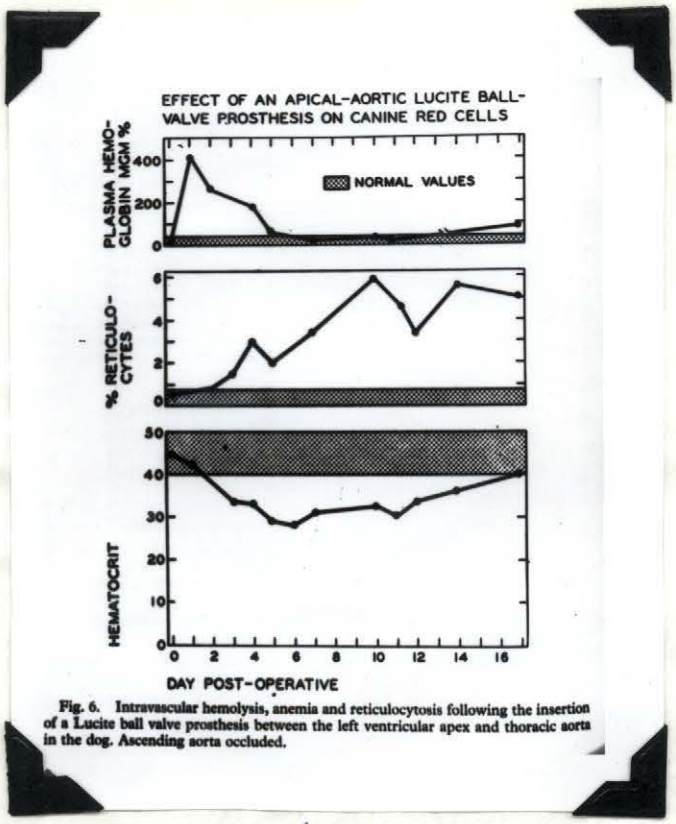


Fig. 6. Intravascular hemolysis, anemia and reticulocytosis following the insertion of a Lucite ball valve prosthesis between the left ventricular apex and thoracic aorta in the dog. Ascending aorta occluded.

Picture 7

to date, he has found the following: (1) plasma hemoglobin rose from 8 to 400 plus mg. per 100 cc. on the first postoperative day and thereafter declined to normal within 6-8 days. (2) The

hematocrit reached a low point on the 7th postoperative day and thereafter gradually returned to normal. (3) The reticulocyte count gradually rose over the period of 16 days. In this it is interesting to note in regard to the "sudden death" syndrome that the lowest hematocrit was registered on the 6th day postoperatively. Of greater significance is the rapid rise of the reticulocyte count which is thought to be the hemopoietic system's response in an attempt to compensate for the increase rate of red cell destruction. Because a straight lucite tube in the aorta did not cause a post-operative elevation of the plasma hemoglobin or anemia, it is postulated by Stohlman (31) that possibly the initial tremendous rise in plasma hemoglobin was due to the ball valve destroying the more fragile red cells.

However, in considering the above, it must be remembered that as yet there is no control data available on the relative mechanical fragility of the red cells of man and dog. It is known that by exerting extra pressure while withdrawing a blood sample from a dog, hemolysis can be produced while the contrary is true in man for the most part. Stohlman (31) is presently undertaking the comparative study of mechanical fragility of dog and human red cells in a standard pumping chamber with and without a Hufnagel valve.

Only the future and the results from the present and future experimentations will be able to prove whether the post-operative fall in hematocrit with its ensuing anemia is the main

cause of the "sudden death" syndrome following insertion of the plastic valve. Regardless of the cause the main biologic need is to afford the myocardium with sufficient oxygen to carry out its work or to decrease the work load so the myocardium can utilize what oxygen it has available. Recently it has been suggested by Sarnoff (35) that "the physiologic gamble involved in the above operation could be converted to a certainty by perfecting the purse string type of operation of the aortic valve". This would be proximal to the coronary ostia and under these conditions there would be no regurgitant volume and the coronary perfusion pressure would be adequately maintained.

The following is the most complete detailed analysis of the "Hufnagel Operation" (17).

Of the first 23 patients undergoing this operation, all showed signs of congestive failure. All have had diastolic gallop rhythms and Austin-Flint murmurs. The heart shadows filled most of the left chest in several instances. No patient had a diastolic pressure above 40 mm. Hg. The majority of patients complained of anginal attacks and some were shown to have conduction defects.

Of these first 23 patients, 17 are living for 11 months or more. One patient who had been bedridden for a year died on the operating table prior to placement of the valve. Another patient died six weeks following the operation of subacute bacterial endocarditis. Blood cultures preoperative were negative on repeated occasions. Two more patients died from acute cardia arry-

thmia. The fifth died of an exacerbation of acute rheumatic fever, and the sixth of congestive heart failure, both several days post-operative. Except for the first death, these represent complication of the basic underlying disease.

No case reported yet presents evidence of clotting or failure of the valve to function. The most serious non-fatal complication appears to be emboli which has occurred in 1/6 or 7 out of 42 cases (19). No major impairment has occurred from this complication.

The author states that about 20% die postoperative before leaving the hospital. Another 20% die during the following two years, the majority of which are due to some intercurrent disease. Hufnagel (17) states that the surviving patients are all able to carry out a relative normal day whereas before the operation they were not. He feels his results are encouraging enough to continue research along the lines of a better valve and a better operation whereby more of the regurgitant volume will be controlled.

SURGERY, BAILEY

Bailey (23), in his continuation of a method to alter the course of aortic insufficiency has come upon two different surgical approaches which come close to that mentioned by Sarnoff (36). For these procedures, Bailey (23) prefers the transaortic approach which permits the direct digital examination of the valve structures. In this way he is able to guide accurately the prosthetic materials to their desired location and is able to determine the degree of

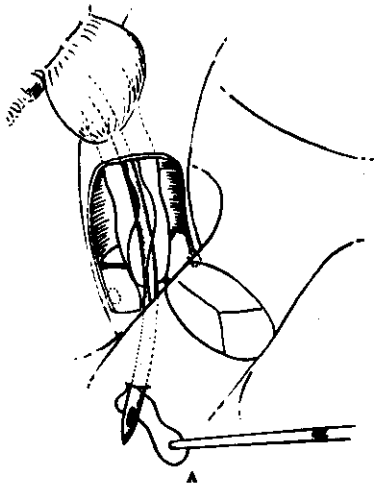
narrowing or constriction of the aortic annulus which is necessary to insure proper valve function.

The patient is placed in a supine position and after being draped and prepared in the usual fashion, an anterior transthoracic incision is made along the 4th intercostal space. The right 3rd and left 4th intercostal spaces are opened. After both pairs of interior mammary arteries are tied and ligated, both 4th costal cartilages are divided and the sternum at this level is transected.

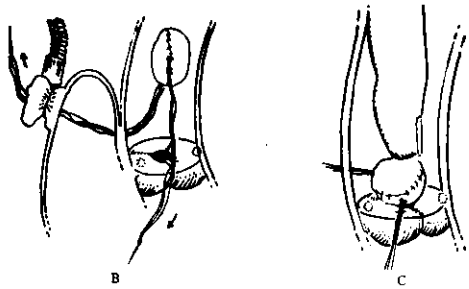
Following this, a large sheet of pericardium is obtained, and an incision 4 cm. in length is made in its center. A "poncho" is created by purse-stringing the periphery of this patch. Using a special excluding clamp, the anterior portion of the ascending aorta is picked up and clamped excluding a sizeable portion from the main outflow stream. After making a similar incision in the aorta the pericardium is sutured to the aorta by imbrication.

The pouch is flushed with heparin and rinsed with saline. The ends of the purse-string suture are incorporated into a Rumel-Belmont tourniquet. The valve is then explored and an attempt to correct the stenosis, if present, by using the finger is employed before instrumentation is tried.

The surgical correction of the insufficiency is then begun. See Picture (8) (page 40). A curved surgical probe threaded with nylon suture is guided by the intra-aortic finger through the right anterior sinus of Valsalva. The suture is grasped as it emerges from the right ventricle. The probe is then passed



A



B C

FIG. 19. Placement of two-tailed valve. A. Passage of probe anteriorly to emerge from the right ventricular outflow tract on its anterior surface. B. Drawing nylon tails through aortic wall and out of surface of the heart. Posterior tail emerges from the aorta to traverse the right atrial chamber. C. Adjustment of location and mobility of prosthetic valve by guidance of intracardiac finger.

Picture 8

so as to penetrate the right posterior sinus of Valsalva and emerge from the right atrium.

The tails of the sutures emerging from within the aorta are attached to tails of the nylon tampon. As the ball is directed down the aorta, it begins to move with the blood currents. The exact placement is guided by the intra-aortic finger and the extent of the arc of the tampon's mobility is determined by traction upon the tails. When this has been adequately determined, the tails

are secured by threading them through cone disks and tying them in place.

If on the other hand, the examining finger finds that the insufficiency is due to a dilated annulus and that the valve cusps are normal the following procedure is used. The same method for exposure is used. Two similar "ponchos" are made as described before only this time they are imbricated to the atria.

After exploring the interatrial septum, the lowest portion of the intra-cardiac aorta is determined. A malleable probe with heavy nylon suture attached is then guided into the left atrium. From here it courses close to the aortic wall and is pressed on into the right atrium to emerge from the right auricular appendage. A nylon sash 30" long, 4" wide, and tapered at each end is attached to the nylon suture and by traction at the opposite end is pulled into position. See Picture (9)(page 42).

The main pulmonary artery is then dissected from the aorta to its origin. Another malleable threaded probe is passed from the pulmonary artery's root through the ventricular musculature well below the right coronary artery and emerged from the right auricular appendage. One of the trailing ends of the sash is attached to the suture and drawn along the above mentioned course so that the sash emerges from the anterior surface of the heart. Another threaded probe is passed between the separated aorta and pulmonary artery, through ventricular musculature, under left coronary artery and emerged from the left atrial chamber. The

FIG. 22. A. Threaded probe passed between aorta and pulmonary artery under left coronary artery to enter left atrial chamber and emerge from appendage. B. Drawing left side of sash along course of probe.

Picture 9

other trailing end of the sash is attached to this suture and drawn back through the left atrium, under the left coronary artery, and out between pulmonary artery and aorta.

The sash is overtightened so that by the end of the first 24 hours the loosening which always occurs will maintain valve competence. The opening in the aorta and the auricular appendages are then repaired and the chest wall is closed in the usual manner.

Using the nylon sash and silicone rubber prosthesis, there

have been two deaths and five survivors. Four of the five survivors have shown evidence of objective benefit by (1) elevation of the diastolic blood pressure, and (2) characteristic changes in the arterial pressure waves.

Surgical constriction of the annulus has been applied in 23 patients with a total of seven deaths. The authors state that "most of the survivors have shown marked benefit."

SUMMARY

This paper has dealt with the problems of aortic insufficiency. Corrigan (1) was the first to describe at length this condition, its course, and physical signs and symptoms. In recent reports of Hufnagel (17) and Bailey (23) it has been stated that the leading cause of this condition is now rheumatic in origin.

The leading pathologic manifestation is felt to be a widening of the commissures when due to syphilis. If rheumatic in origin, the valves are thick, distorted and retracted, and the annulus fibrosis is overstretched through hypertrophy and dilatation of the left ventricle. When these pathological changes are prominent over 50% of the cases present anginal symptoms.

The characteristic physical findings are a rumbling diastolic murmur heard best over the third and fourth left intercostal spaces, visible peripheral pulsations, water-hammer pulse, and Durozież's sign. The physiologic mechanisms producing these are decompetent aortic valves allowing regurgitation of blood back into the left ventricle. The ventricle then has to contract harder to pump

out this increased diastolic volume to maintain the peripheral blood flow. This increased cardiac output with increased stroke volume produces the increased systolic pressure. The decompetent valves and sino-aortic reflex allow peripheral vasodilatation which accounts for the decreased diastolic pressure. These two changes result in the production of the characteristic increase pulse pressure.

Left heart catheterization is as yet experimental; however, present results indicate that in the future it might help in determination of the degree of stenosis or insufficiency. Findings seen on the electrocardiogram, ballistocardiogram and roentgenograms are discussed. The medical treatment at present with some results of the past are summarized.

After 300 plus experimental operations on dogs, Hufnagel (15) carried out the first insertion of an aortic prosthetic valve on a human in September 1952. This patient at one time bed-ridden is now carrying out an altered daily routine. This plastic prosthesis works on a ball-valve mechanical basis. It is inserted just distal to the left subclavian artery and held in place by two multiple point fixation rings. After some 90 operations, Hufnagel (19) has 24 living patient with a 2½ year follow-up. Webster (16) has shown that patients with a similar condition handled on a medical basis have 18-24% - 2½ year survival rate. It is therefore believed, by those advocating surgery, that in selected cases surgical treatment can improve the patient's prognosis

over that which could be obtained by medical treatment.

Bailey (23) has devised an operation which tamponades the aortic valves by inserting a nylon disk over them. He uses this technic when he finds pathologic valves to be the underlying cause of the aortic insufficiency. If the aortic insufficiency is a result of the dilatation of the annulus, he purse stings the root of the aorta. These types are differentiated by direct digital examination of the aortic valve. He has carried out these two procedures in a total of 30 patients. In the 21 patients surviving the operation, Bailey feels that there has been definite subjective and objective improvement.

CONCLUSIONS

One should certainly view the recent surgical attempts with a very open mind. This is because medical treatment for this condition has been to no avail after severe symptoms of congestive failure are present. This refers to the patient in Class III and IV.

After reviewing the recent literature, it is apparent that as yet a definite operation has not been found to correct aortic insufficiency. Some disadvantages of the Hufnagel procedure are the following: (1) It fails to relieve regurgitation proximal to the valve. (2) There is as yet a high operative mortality rate (40%). (3) For a period following operation there is a disturbing clicking sound. However, patients do not complain about this after 3-4 months. This is because the valve has become surrounded by a thin film of fibrous tissue. Some advantages of the operation which

are important enough to stimulate further investigative work are the following: (1) A certain percentage (50-60%) have a good prognosis postoperatively. (2) The operation decreases the work load on the left ventricle. (3) Anginal symptoms are relieved by the decreased oxygen demand of the ventricle. (4) In those that survive there is a definite sense of well-being. They are able to carry out altered daily routines whereas before operation they were bedridden for the most part.

In comparing Hufnagel's operation with Bailey's purse-stringing the aortic root, it seems the latter stands the greatest chance of fully correcting the basic pathologic mechanism. By the latter operation, the total prevention of regurgitant volume seems possible. The main drawback with this operation at this time is that manipulation of the aortic valve and aortic arch area greatly increases the possibility of cardiac arrythmias.

Thus, as in many cardiac conditions which were once thought to be hopeless, these recent surgical operations have provided a glimpse into the future which may provide a definite treatment for aortic insufficiency.

I wish to thank Dr. Delbert D. Neis for the help he gave me in preparing this thesis.

Appendix III

BIBLIOGRAPHY

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