THE AORTIC VALVE
AND THE SURGICAL CORRECTION
OF CHRONIC AORTIC INCOMPETENCE.

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D. RAOUL DE VILLIERS.

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INDEX.

INDEX (contd.)

LIST OF ILLUSTRATIONS.

LIST OF ILLUSTRATIONS (contd.)

LIST OF ILLUSTRATIONS (contd.)

LIST OF ILLUSTRATIONS (contd.)

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INTRODUCTION.

Human Intracardiac Surgery, the illustrious protegé of an equally illustrious mentor, General Surgery, has been in existence for only a relatively short while. Only thirty five years have elapsed since first operative attempts were made at correction of intracardiac valvular lesions by "closed" surgical techniques. Much more breathtaking, however, has been the phenomenal development of intracardiac surgery since the first employment of an "open" technique, merely seven years ago.

Approach to the interior of the heart, "the last Great West of Surgery" (Harvey Cushing), so long debarred to the surgeons throughout the era of modern medicine, has at last come within the scope of practicality. The development of hypothermia techniques and extracorporeal pump oxygenator systems, combined with the use of cardioplegic drugs, has enabled the surgeon to work within the cavity of a dry, inactive heart. Accurate visualisation of the pathology is thus possible, enabling adequate correction of the lesion, or lesions.

Whereas the surgery of intracardiac valvular lesions, directed mainly towards the correction of stenotic lesions, has met with success, that of incompetent valve lesions has remained a vexing therapeutic problem. In the sphere of aortic valve surgery this has been the result of various factors

The problem of direct vision aortic valve surgery combined with adequate myocardial maintenance has been overcome by techniques of myocardial oxygenation, and more recently, by the employment of profound hypothermia by total body cooling. Incompetent aortic valves however, are more often than not damaged beyond the scope of plastic repair and for the complete correction of the regurgitant lesion the insertion of a prosthetic valve substitute is required. This entails the introduction of foreign material into the cardiovascular system as well as the correction of a complex haemodynamic abnormality existent in the root of the aorta.

Aortic valvular disease thus having assumed a new and practical importance in the field of operative surgery, demands a consideration of the anatomy and function of the normal valve as well as the pathological anatomy of the diseased valve. It is, therefore, only after a clear understanding of aortic valve function under normal and abnormal conditions has been attained, that an intelligent attack on the diseased valve can be made by the surgeon - to quote Sir William Osler: "as is our pathology, so is our practice".

It is the purpose of this study to consider the normal and pathological anatomy of the aortic valve and review the problems involved in the surgical correction of chronic aortic incompetence with due consideration to plastic procedure corrections of deformed valves, graft replacements and prosthetic valve substitutes or augmentations.

HISTORICAL REVIEW.

Paul Kruger's oft-quoted dictum: "zoek in de verlede al het goed en edele en bouwdt daarop u toekomst" holds good in medicine as well as in politics. It is always of extreme interest, and certainly sometimes of great value, to examine the historical background of any new undertaking, since it is only by a review of previous studies that the investigator can obtain the necessary knowledge and perspicacity to equip him for the task that confronts him. A similar remark was made by Sir Winston Churchill in March 1944 in an address before the Royal College of Physicians: "the longer you look back, the further you can look forward" (Guthrie[52]1945).

The first record of experimental cardiac surgery probably dates back to 1882, when M. H. Block[32] had, after opening the thorax of living rabbits, inflicted injuries upon their hearts and again successfully sutured these wounds. In 1895, at the 11th International Medical Congress in Rome, an Italian surgeon, Del Vecchio[75], presented living dogs with cardiac wounds which he had sutured. A similar experiment was reported by Salomoni[218](1896).

Despite these successes however, medical opinion was still divided as to the merits of the new operations. In 1875 Theodor Billroth[30] wrote: "Paracentesis of the pericardium is an operation which, in my opinion, approaches

very closely to that kind of intervention which some surgeons would term a prostitution of the surgical art, and other madness". Also in 1885[31] he made his famous statement: "Let no man who hopes to retain the respect of his medical brethren, dare to operate on the human heart". Riedinger[207] in 1888 wrote: "The suggestion to suture a wound of the heart, although made in all seriousness, scarcely deserves mention", and in 1896 Paget[198], unconvinced by Del Vecchio's demonstration the year before, stated: "Surgery of the heart has probably reached the limits set by Nature to all surgery; no new methods and no new discovery can overcome the natural difficulties that attend a wound of the heart". Within the year, however, attempts were made to suture hearts in human beings. On September 4th, 1895 Cappelen[45] of Christiana and in March 1896 Farina[92] of Rome attempted this; both were unsuccessful. The latter case died of "some intercurrent disease" (Warbasse[93]1898).

September the 9th, 1896, however, witnessed the successful suture of a cardiac stab-wound by Professor Louis Rehn[203]. A year after this successful operation Rehn[204,205] appeared at the Surgeons' Congress in Berlin and introduced his, by now completely recovered, patient. He closed his account of the operation with the words: "the possibility of performing cardiac suture can no longer be doubted - I trust that this case will not remain a mere curiosity, but that it will

stimulate further work in the field of cardiac surgery, transforming this new field into a life-saving branch of our profession".

Thus the way was paved towards the conquest of that territory which, in more recent times, Harvey Cushing had referred to as "the last Great West of Surgery".

Early experiments in cardiac surgery were directed towards the heart valves. Thus it was in 1872 that Becker[24], an ophthalmologist, in order to investigate the retinal pulsations noted in cases of aortic incompetence by Quincke[202] (1868), produced aortic incompetence in dogs by destruction of one or more of the aortic valve cusps. This was done by the insertion of a glass rod down the left carotid artery. Klebs[145](1876) produced valvular lesions in similar experiments. He passed a "valvulotome" - a tiny knife on a long rod - down the carotid artery to incise the valve cusps. Cohnheim[60](1877) produced aortic incompetence by passing whalebone sounds down the carotid artery and tearing out the valve cusps. He was studying the arterial pulse in aortic insufficiency. Timofejew[249](1888) produced aortic incompetence by introducing sounds and needles through the aorta. He attempted to correlate the size of the defect with the type of murmur produced. Ottomar Rosenbach[212](1878) used the Klebs "valvulotome" to create valvular insufficiency and study the pulse and heart action in aortic insufficiency.

Similar experiments to study the altered haemodynamics of aortic regurgitation were conducted by numerous investigators: Kornfeld[148](1896); Hasenfeld and Romberg[124](1897); MacCallum and McClure[172](1906); Wiggers and Dubois[260](1913) and Wiggers[259](1932).

At the beginning of the 20th century, therefore, research had provided certain methods and instruments for the experimental production of valve lesions. Animal experimentation and clinical surgical experience thus, had proven not only the ability of the cardiac organ to withstand trauma, but also the likeness in healing of cardiac wounds to wounds elsewhere in the body and techniques possible for handling the pulsating vital organ.

The effects on the cardiac action of total or partial occlusion of the great vessels entering or arising from the base of the heart had also been noted. The possibility of surgical procedures to be performed on diseased cardiac valves was, therefore, the next step logically to be investigated. This again commenced with animal experimentation and in 1907 Haecker[119] in Germany and Cushing and Branch[67] in America published first reports of a series of animal experiments in which valves were cut via a transthoracic approach. Thus the first real proof of deliberate surgical approach to the valves was evidenced and, with improved techniques, operative risks could be minimized. Similar experiments were conducted

by Bernheim[26](1909) and Schepelmann[226](1912). Samways[219] (1898) and later Brunton[40](1902) had, however, suggested surgical enlargement of the narrowed orifice in the treatment of mitral stenosis. Samways, a British veterinary surgeon, suggested that "some of the severest cases of mitral stenosis will be relieved by lightly notching the mitral orifice".

In 1913 Ernst Jeger[135] published a monograph entitled "Die Chirurgie der Blutgefässe und des Herzens". He proposed correction of aortic stenosis by use of a segment of the jugular vein to be implanted as a shunt between the left ventricle and one of the main branches of the aorta. Bailey et al.,[10](1950) and Sarnoff and Case[224](1955) have recently revived experimental interest in this method in attempting to bypass the aortic valve. Similar bypass of the mitral valve was also attempted by Bill, Pierce and Gross[29] (1950).

The year 1913 was an important one in the field of cardiac surgery, because in this year Doyen[80,81](1913) performed the first definitive intracardiac operation on a 20 year old female patient. He attempted pulmonary valvotomy by insertion of a small tenotomy knife into the right ventricle. The operation was, however, unsuccessful and post mortem examination revealed a subvalvular stenosis rather than pure valvular stenosis, as had been anticipated. Later that year Tuffier[250]

(1913) successfully dilated the aortic ring of a patient with aortic stenosis by digital invagination of the adjacent aortic wall through the narrowed ring. This is regarded as the first clinical approach to the problem of aortic stenosis. The patient was a young male with signs of marked and progressive aortic stenosis. The thorax was opened and the aortic root exposed. It was the operator's intention to insert a knife above the aortic ring and incise the stenosed valve, but the procedure was changed to dilatation of the aortic valve by invaginating the wall of the aorta just above the valve and pushing this into the stenosis on the forefinger. As late as 1924 this patient was reported as living and improved. Swann and Kortz[241](1956) described this as surgical temerity unequalled until the modern era of cardiac surgery.

Alexis Carrel[47](1914) reported a method for open operations on the pulmonary and aortic valves. He concluded his paper thus: "it is not impossible that some day surgeons will be able to cauterise valvular lesions, or to repair them, as we do today in our experimental operations".

During the First World War (1914 - 1918) cases of successful cardiac suture were recorded (Ballance[17]1920; Lockwood[167] 1929), but experimental work came to a standstill.

The year 1920, however, saw an investigation into the surgical treatment of mitral stenosis by Cutler, Levine and

Beck[69](1924). Initial experiments to correct the mitral valve stenosis under direct vision, after temporary occlusion of the great vessels, was unsatisfactory and the operation was abolished in favour of a blind left ventricular approach with the assistance of small knives mounted on long handles. In May 1923 Cutler and Beck[68] reported the first operation for correction of mitral stenosis. A tenotomy knife was inserted through the left ventricle and each cusp was incised. Their patient, an eleven year old female, survived the operation for 4½ years only and it must be presumed that correction of the valvular lesion was incomplete. Four further operations of a similar nature, attempted over the next two years, were unsuccessful.

In 1922 Allen and Graham[2] of St. Louis reported the use of a cardioscope to be inserted through the left auricular appendage. Graham's only attempt at clinical application of this method was unsuccessful however (1923).

Three further attempts at operative relief of mitral stenosis were made during this period by the employment of the "cardiovalvulotome": Pibram[200](1926); Cutler and Beck[68] (1926, 1928). All these attempts were, however, unsuccessful. Credit for the performance of the first successful mitral commissurotomy by digital manipulation must go to Sir Henry Souttar[238](1925).

Not until 1947 was further interest revived in this

problem of valvular stenosis, when Horace Smithy[235], himself a sufferer of the disease, attempted to solve the problem of relief of aortic stenosis. He pointed out the technical difficulties of a blind trans-aortic approach to the valve with the real danger of producing incompetence. He urged transventricular resection of a portion of the diseased valve. Surgical interest in this difficult problem had now, however, been aroused and the ensuing years saw several reports of blind digital and instrumental techniques to correct valvular abnormalities. In 1947 Brock[34] had actually inserted a cardioscope via the right subclavian artery and obtained a good view of the aortic valve without actually doing anything to the valve.

Holmes Sellors[230](1948) and Brock[33](1948) described the technique of pulmonary valvotomy. During the same year Bailey[5](1948) and Brock[34](1948) also successfully performed mitral valvotomies, thus entering our modern era of direct surgical attack on diseased cardiac valves.

Numerous reports followed on methods to fracture or dilate the stenosed aortic valve, either via the aorta or the left ventricle, either digitally or by instrumentation: Bailey et al.,[6,7,8,10,11,14](1950-1954); Brock et al.,[33,34,35,36,37](1948-1960); Muller et al.,[193](1954); Likoff et al.,[160](1955); Swann et al.,[245,241,243](1954-1958); Pearl et al.,[199](1955); Logan et al.,[168](1954); Urrichio et al.,[251](1954);

Fell[95](1955); Litwak et al.,[166](1952); Glover and Gadboys [108](1958).

Bailey[14](1952) reported a technique of introducing a powerful mechanical dilator through the left ventricular wall upwards into the valve orifice. In 1954[8], however, he recommended the future use of the blind transaortic route.

Harken[121,122,123](1955 - 1959) also approached the valve via the aortic route through a pouch attached to the aorta, as had originally been reported by Swann[245](1954) and Pearl[199](1955) and also used by Bailey[8](1954).

None of these methods however, were found to be really satisfactory. To quote Fell[95](1955): " Surgery for aortic stenosis, at the present, is far from ideal. For the past two years we have dilated stenosed aortic valves by a procedure that likewise is not ideal ....." The dangers and uncertainties of such blind instrumentations and digital manipulations were thus soon realised and a suitable method, whereby the surgeon might actually visualise and directly approach the valvular lesion, was sought after. This becomes essential if a prosthetic replacement or plastic revision of the valve is considered. The solution to the problem came with the discovery of two such procedures to be applied clinically.

The use of general hypothermia with inflow stasis had actually been described much earlier and had also been clini-

cally applied at an earlier date. Bigelow must be regarded as the father of the hypothermia technique (Bigelow et al., [27,28] 1950 - 1954). Other workers in this field were : Delorme[74] (1952); Ross[215] (1954); Gray[115] (1955); Swann et al., [244,242,240,241,243] (1953 - 1958).

The first successful open heart procedure using this technique was performed by Lewis[158] (1953); the lesion corrected was that of an interatrial septal defect.

The other development was that of cardio-pulmonary bypass. This was attained either by means of a pump oxygenator, or by the use of biological oxygenator systems. Clinical application of the former method was first attained by Gibbon[105] (1954). He repaired an interatrial septal defect in 1953 excluding the heart and lungs from the patient's circulation for twenty five minutes.

At present the main artificial oxygenator systems for clinical use are the following:-

1.) The Bubble Oxygenator (DeWall et al., [77,78,79] (1956 - 1957); Lillehei et al., [164] (1956); Brown et al., [38] (1956).

2.) The Film Oxygenator (Miller and Gibbon[187] 1951; Cross et al., [66] 1956).

3.) The Membrane Oxygenator (Kolff et al., [146] 1956).

Various modifications of these basic oxygenator systems

have since been described.

The biological oxygenation system of cardio-pulmonary bypass entails the use of either cross-circulation (Lillehei et al.,[161,162]1955; Warden et al.,[253]1954), heterologous (dog) lungs (Cambell et al.,[42]1955) or a reservoir of oxygenated blood (Warden et al.,[254]1955). First visualisation of the aortic valve for surgery in humans was done by Clowes and Neville[59](1954), using a pump oxygenator with complete cardiac bypass. They did not establish coronary perfusion in the one case reported and the patient failed to survive. In 1956 Julian et al.,[136] reported two cases of aortic commissurotomy under direct vision by the use of hypothermia and inflow occlusion. The first case was done on 10th October 1955. Lewis et al.,[157](1956) likewise presented their experiences with direct approach to the aortic valve using hypothermia and inflow occlusion. They presented a total of three cases with one death - the first case was done on 2nd December,1955.

Kaiser[137](1956) independently reported experiences with direct exposure of the aortic valve using hypothermia and inflow occlusion in dogs and stated that he had found no instances of air embolism. He recommended the application of this approach for operative surgery of the aortic valve in humans.

Further aid is rendered the cardiac surgeon by the induction of cardiac arrest; he is thus able to operate within a dry inactive cardiac organ. This followed the report

by Melrose et al.,[186](1955) on the use of potassium citrate as a cardio-plegic agent. Further experimental application followed (Kolff et al.,[147]1956). First clinical application of this method was in 1956 (Effler et al.,[88,89,90]1956-1958). The use of acetylcholine as a cardioplegic agent for clinical application was also reported: Lam et al.,[149,151,152,153,154] (1955-1958); Gahagan[99](1955); Moulder et al.,[192](1956); and Sergeant et al.,[231](1956). Lam[149](1956) obtained poor results with use of potassium - induced asystole and therefore turned to acetylcholine; but this was because he had used potassium chloride instead of potassium citrate, as had been suggested by Melrose. The use of acetylcholine as an adjuvant to retrograde perfusion of the coronary sinus was also reported: (Gott et al.,[112](1957).

Thus congenital anomalous lesions could readily be corrected and stenosed valves widened, but the problem of valvular regurgitation had remained only partly solved. Methods of correcting incompetence of the mitral valve by various closed techniques have been described: Murray[194](1950); Bailey et al.,[11,13](1951); Davila et al.,[71](1954); Sakakibara[217] (1955) and Glover and Davila[107](1957) and more recently "open" methods for the correction of this lesion: Kay et al.,[142] (1958) and Lillehei et al.,[165](1958).

The problem of aortic incompetence and the surgical correction thereof had been studied since 1950: Cambell[43](1950).

Most acclaim has, however, gone to Hufnagel[126,127,128,129,131,132] (1949-1958), who developed a ball valve prosthesis to be inserted in the descending aorta. Some measure of success is attendant on the insertion of the Hufnagel lucite ball valve in the descending thoracic aorta. This prosthesis is inserted at this site to prevent regurgitation of blood into the heart from the lower body. The workload of the left ventricle is thus decreased, but at the same time coronary perfusion pressure is diminished with resultant effects of myocardial anoxaemia (Ref. paragraph "Essential requirements of a prosthesis - subcoronary placement"). Hufnagel regards the reduction of left ventricular workload following the insertion of this prosthesis into the descending aorta as being in the region of 75%: Hufnagel et al.,[131] (1954); other workers, however, (Taylor et al.,[247] 1958) do not agree with this.

Complications such as embolism and postoperative anaemia due to excessive destruction of the formed elements in the blood (Rose et al.,[211] 1954; Sarnoff and Case[223] 1955 and McKusick et al.,[182] 1954) have been recorded. Aorto-oesophageal fistula as a late complication of the Hufnagel valve has also been recorded: (Kittle[144] 1958). Development of false aneurysm following insertion of the prosthesis has also been reported (Lord and Stone[170] 1955 and Wolcott and Ellison[261] 1957), and even a case in which the ball itself had

split with resultant thrombosis, embolisation and eventual death: Fix and Riker[97](1960). Roshe et al.,[214](1957) also reported an experimental increased susceptibility of dogs to endocarditis after placement of the prosthesis. The Hufnagel prosthesis has therefore never stirred up much enthusiasm amongst the cardiac surgeons: Conklin et al.,[61](1958) and Fawcett and Dhillon[94](1956).

Thus numerous reports of various designs of prostheses, constructed out of a variety of materials, were to appear in medical literature. These prostheses were all designed for subcoronary placement, but most of them were confined to the experimental stage and results were not encouraging enough to allow their clinical usage: Bailey[6,12](1956); Wible et al., [256,257,258](1956-1957); Mallette et al.,[173](1956); Roe et al.,[209](1957); Edwards and Smith[87](1958); Barnard[20](1958) and Long et al.,[169](1959).

Aortic valvular disease has, consequent upon the acquired surgical ability to operate on the aortic valve, whether by closed or open techniques, accordingly assumed a new practical importance in surgery. Consideration must, therefore, be given to the anatomy and function of the normal as well as the pathological anatomy of the diseased valve, before surgical correction is contemplated.

THE NORMAL AORTIC VALVE.

In order fully to appreciate the abnormalities produced by a disease process a complete knowledge of the normal structure and function of the affected part is essential. This holds especially true for the aortic root region where numerous small vital structures are grouped together in a strategic position, being constantly involved in movements, each of which is interrelated with the other. A precise and accurate knowledge of the anatomical structures in the root of the aorta and their functional movements is therefore essential.

A. ANATOMY OF THE NORMAL AORTIC VALVE.

(1) Macroscopy:- The valve is placed in the root of the aorta proximal to the coronary ostia and allows outflow of blood from the left ventricular chamber of the heart. It consists of a valve ring (annulus fibrosis) and three approximately semilunar cusps (leaflets) which are attached by their thickened convex margins to the arterial wall. The free borders of the cusps, slightly concave upwards, project into the lumen of the vessel. The term "valve ring" is applied to describe the area of proximal attachments of the valve leaflets: Gross and Kugel[117](1931). Outward bulgings of the aortic wall immediately opposite and distal to each of the

three cusps are known as the aortic sinuses ("sinuses of Valsalva"). Right and left coronary ostia are sited at the upper margins of the right and left aortic sinuses respectively (Fig. 1).

The aortic sinus is a specialised part of the aortic lumen, being walled by its respective valve cusp medially and by the origin of the aorta laterally. The coronary arterial ostia are related to the aortic sinuses, but since these normally arise at a level just distal to the upper edge of the valve cusps, they do not, in a strict sense, arise from the aortic sinuses, but rather from the aortic wall just above the sinuses (Fig. 2). The names given for the sinuses are the same as those for the valve cusps (See later under paragraph on Nomenclature).

In the body the aortic valve is so orientated that the stream of blood flowing through it is directed to the right and upwards. The valve does not lie in the same transverse plane of the body. The portion of the left aortic cusp nearest the right is the most superior and anterior, while the posterior cusp and the adjacent part of the left cusp lie at the most inferior and posterior levels. The most antero-superior portion of the left aortic cusp lies at the same level as the nearby left cusp of the pulmonary valve. Otherwise the aortic valve lies inferior to the

pulmonary valve. The inferior position of the posterior part of the aortic valve may be appreciated from the fact that, at this level, the aortic valve lies inferior to the " transverse sinus of the pericardium " and anterior to the adjacent portions of the two atria and the interatrial septum (Fig. 3).

Two widely used systems are employed in the nomenclature of the aortic cusps and sinuses. The B.N.A. (Basle Nomina Anatomica) nomenclature views the heart as if removed from the body and held so that the ventricular septum forms the medial plane; the aortic valve cusps are thus named right anterior, left anterior and posterior, or non coronary, whilst the pulmonary valve cusps are named right posterior, left posterior and anterior (Fig. 3).

The other system used names the cusps according to their relative positions with the heart in situ within the thorax; the aortic valve cusps are therefore right posterior, left posterior and anterior (or non coronary), whilst the pulmonary valve cusps thus become right anterior, left anterior and posterior.

The B.N.A. nomenclature seems to be most commonly used: (Gross and Kugel[117] 1931; Edwards and Burchell[86] 1957 and Edwards[85] 1958) and for the purpose of conformity will be employed in this paper. Not only does each aortic sinus have relations that are different from those of the other two

sinuses, but significantly, differences in relationships exist at different parts of each sinus. Hence Edwards and Burchell[86](1957) have divided each aortic sinus into three parts; a central (intermediate) third and two outer thirds, each adjacent to one of the other two cusps and sinuses. The right aortic sinus is entirely related to a cardiac chamber, mainly the right ventricular outflow tract. The central part of the sinus lies against the "crista supra-ventricularis", the prominent ridge seen in the outflow tract of the right ventricle in the upward prolongation of the muscular part of the interventricular septum. The left part of the right aortic sinus is the most anteriorly located of the three parts of the sinus. It abuts against the outflow tract of the right ventricle in the angle between the upper aspect of the crista supraventricularis and the pulmonary valve. The posterior third of the right aortic sinus lies against the right ventricle just posteroinferior to the crista supraventricularis. Below the right aortic cusp and sinus lies the ventricular septum, variably either muscular or membranous portion.

The posterior aortic cusp (non coronary) and sinus are related to the right and left atria and the interatrial septum. Adjacent to the right and intermediate thirds of the posterior aortic sinus is the interatrial septum. Inferior to the right part of the posterior aortic sinus is the muscular

portion of the interventricular septum. Beneath the central part of the posterior sinus is the membranous portion of the ventricular septum. The left part of the posterior cusp shares with the adjacent third of the left aortic leaflet the peculiarity of being continuous with tissue inferiorly of the anterior leaflet of the mitral valve (Figs. 4 and 7). Here is the only part of the aortic wall which is not connected with the ventricular portion of the heart; rather is the aorta connected to the base of the mitral leaflet. Adjacent to the left part of the posterior aortic sinus lies the left atrial wall.

Corresponding triple division of the left aortic cusp and sinus renders the posterior part of this cusp adjacent to the posterior cusp and sinus. Its relations to the anterior leaflet of the mitral valve are therefore identical to the adjacent third of the posterior cusp and sinus. The other extreme of the left aortic cusp and sinus lies in an anterior position and adjacent to the right aortic cusp and sinus. This is the only third of the left cusp related to the ventricular septum and it may be termed "the septal part of the left aortic cusp". This part of the left aortic cusp and the corresponding wall of the aorta are connected inferiorly with the most anterosuperior aspect of the muscular part of the interventricular septum. This is the part of the aortic valve that lies most superiorly in the body. The central

part of the left aortic cusp and sinus is the only region of the aortic root that is not adjacent to a cardiac chamber or the pulmonary trunk. This part of the left cusp and the corresponding part of the aortic wall are connected with the lateral aspect of the left ventricular base. Adjacent to the wall of the sinus in this region is the epicardium. It is above this part of the left aortic sinus that the left coronary artery arises.

The three aortic valve cusps are each an independent unit with two lateral attachments to the aortic wall and an inferior attachment to the root of the aorta. Between these lateral attachments the free edge of the cusp is not a straight line but longer than such a line would be, thus allowing the center of each cusp to extend to the center of the aortic orifice and also allowing considerable overlap of the leaflets with closure of the valve (Refer section: "Function of the normal aortic valve").

Radiating from this central point of the aortic orifice are the contacts of the halves of each cusp with the adjacent halves of their neighbours. The space, or potential space, between the lateral attachments of two adjacent cusps at the aortic wall is referred to as a "commissure". In the normal aortic valve three such junctional zones, or commissures, exist.

A safety margin also exists in the length of the normal

aortic cusp. Thus the cusp is longer in the supero-inferior dimension than is necessary for contact with its fellow cusps. This concept is supported by the fact that the cusps do not touch each other at their free edges; rather, the area of contact extends downwards for a distance of several millimetres from the upper free edge. The lowermost margin of contact is spoken of as the "line of contact" (Fig. 1).

The free edge of each cusp has a round thickening at the center - this is named the "nodulus" or "corpus Arantii". Adjacent parts of the free edge of the cusp on each side of the nodulus are especially thin; these narrow semilunar areas between the free edge and the "line of contact" of each cusp are referred to as the "lunulae". Radiating from the corpus Arantii over the fundus of each cusp and extending to its attachment to the arterial wall are fibrous thickenings of the cusp (Fig. 1).

With advancing age the noduli become increasingly thickened and hardened to form excrescences on the inner surface of the aortic cusp; these are the so called "Lamblian excrescences" (Lambl[155] 1856; Luschka[171] 1856). They are of interest in that these have been confused with healed verrucae.

The lunulae are sometimes fenestrated without any apparent disturbance of the valve mechanism (Quain[201] 1922). Fenestration of the cusps is more common in the male than in

the female and in the aortic more than in the pulmonary valve cusps.

From our studies of the normal aortic valve it also became evident that there was a definite difference in level within the root of the aorta between the level of attachment of the valve cusps to the aortic wall at the commissures and the level where the nodulae meet in the center of the aortic orifice on closure of the valve during ventricular diastole (Fig. 5). This anatomical fact proved to be of importance in the design of a valve prosthesis; (Ref. section "Augmentation prostheses").

(2) Microscopy:- Descriptions of the histological structure of the valve cusps by various workers vary to some extent, but the basic pattern seems to be the same. The backbone of the leaflet is a dense fibrous tissue layer named the "Klappenplatte" - Seipp[229](1896); "Klappenskelet" - Benninghof[25](1930); "Lamina fibrosa" - Tandler[246](1913); "Fibrosa" - Gross and Kugel[117](1931). On each side this layer is covered by ventricular and arterial layers as prolongations of the ventricular endocardium and arterial intima respectively. Veraguth[252](1895) described these two layers as being of identical structure but Mönckeberg[188](1904) showed them to be quite different. He described three layers:-

(i) The central plate (middle fibrous tissue layer) - consisting of longitudinal and transverse collagen fibres. This layer is thicker in the aortic than in the pulmonary cusps.

(ii) The ventricular layer of endocardium consisting of three strata:-

a. a subendothelial fibrous tissue zone with numerous longitudinal fibres,

b. a layer of transversely arranged elastic fibres which remains yellow after van Giesson's stain and is rich in nuclei,

c. a connective tissue layer with longitudinal collagenous and elastic fibres similar to the lamina elastica interna of an arterial wall.

(iii) The arterial layer which is usually the thickest layer of the cusp and shows transverse folds. It is composed of:-

a. a thin subendothelial layer with an elastic network covered by the peripheral endothelium,

b. a broad zone of connective tissue with coarse collagenous bundles arranged transversely and some fine elastic fibres (Fig. 6).

Gross and Kugel[117] (1931) again define four distinct layers in the valve cusp:-

(i) Ventricularis - corresponding to the ventricular

layer described by Mönckeberg.

(ii) Spongiosa - a layer defined on the ventricular aspect of the fibrosa consisting of dense collagenous tissue with fibroblasts and other mononuclear cells.

(iii) Fibrosa - The root of the aorta loses its musculature and most of its elastic fibres thus transforming itself into the Annulus fibrosis and much of the collagenous Annulus curves around the sinus pocket to ascend in the valve leaflet as the fibrosa. In the crypts between collagenous bundles there are to be found delicate elastic fibrillae, as well as mononuclear cells with dense round nuclei and scant cytoplasm.

(iv) Arterialis - corresponding to the arterial layer of Mönckeberg. This is a continuation of the superficial elastic fibres of the aortic intima around the sinus pocket and is covered by the layer of endothelium. It consists of sparse loose connective tissue containing extremely delicate elastic fibrillae as well as mononuclear cells poor in cytoplasm with dark staining nuclei (Fig. 6).

At the lunulae the middle fibrous tissue layer is absent, whilst at the nodulae it is much thickened. At the site of the "valve ring" (Gross and Kugel), zones a. and b. of the ventricular layer (Mönckeberg) are continued into the subendocardial layer of the ventricle, while zone c. (Mönckeberg), or "spongiosa" (Gross and Kugel) is continued into the annulus fibrosis. Thus "valve spongiosa" merges into "ring spongiosa"

(Gross and Kugel). The fibrous tissue of the arterial layer is continued into the fibrous tissue of the tunica media of the arterial wall, and the subendothelial zone passes into the intima (Fig. 7).

The question as to the existence of blood vessels in the normal semilunar valves has been a controversial one. There are three schools of thought. One group holds that human heart valves have no blood vessels. Another believes that the valves are supplies with blood vessels in at least a considerable proportion of cases and that failure to demonstrate them by many observers is due to a faulty technique. The third group believes that blood vessels occur at times in postnatal human heart valves and that, when they occur, they probably represent foetal remnants. It is now, however, generally agreed that normally there are no blood vessels in the semilunar valves. In pathological valves, however, vessels may be seen throughout the entire extent of the cusps.

B. <u>FUNCTION OF THE NORMAL AORTIC VALVE</u>.

The opening and the closure of the aortic valve is entirely independent of any intrinsic valvular action. Thus movement of the valve cusps is dependent on pressure differences between the aortic and ventricular sides of the valve and the consequent movement of the column of blood. Such conditions

as exist in the root of the aorta can be artificially simulated and the function of normal and diseased aortic valves studied and photographed. Thus in this study normal aortic valves were removed at autopsy shortly after death and set up in such an artificial system. This system will be described later (See section "Testing of Prostheses in vitro - Pulse Duplicator"). The aorta was transected about ½cm. above the level of the valve and the aortic root dissected out carefully so as not to destroy the normal anatomy and supports of the valve leaflets. Flow pressures and rate of action were then adjusted to simulate conditions existing in the root of the aorta during life as closely as possible. The aortic root was sutured to two layers of Polyvinyl sponge (Ivalon) secured about 1½ inches apart - thus allowing free to and fro movement of a water column through the valve (Fig. 8).

(1) Opening of the Aortic Valve. As soon as the cardiac impulse has spread to the ventricular musculature there is the commencement of ventricular systole. Within 0.01 - 0.02 second the rise in pressure within the ventricular cavity forces the atrioventricular valves to close thus constituting the first heart sound. During the next 0.02 - 0.04 second the intraventricular pressure continues to rise and the ventricle becomes more rounded but no actual change of the volume of blood within the ventricle occurs. As soon as

the intraventricular pressure exceeds that of the aorta the aortic valve is forced open and the volume of blood is pumped from the ventricular cavity through the aortic valve into the proximal aorta. On studying the movements of a normal valve in the duplicator one finds that, with the opening of the valve, the free edge of the leaflets become approximated to the side wall of the aorta thus giving a triangular shaped orifice (Fig. 9). This triangular orifice, interestingly enough, was first described by Leonardo da Vinci[156] in Quaderni D'Anatomia (1513); Keele[143](1951). Two important features are thus inherent in this observation. Firstly, the opening between the valve leaflets is considerably smaller than the cross sectional area of the aorta. The cross sectional area of the base of the aorta is approximately 5.3 sq. cms. and the calculated area of the triangle is 2.6 - 3.5 sq. cms.: Quain[201](1922). Secondly, the aortic sinus pockets between the valve leaflets and the aortic wall are not completely obliterated during ventricular systole. This latter finding is of extreme importance in the design of an augmentation prosthesis (See Section "Augmentation Prostheses").

(2) Closure of the Aortic Valve. As ventricular systole comes to an end, movement of the column of blood within the aorta slows and ceases completely with the onset of ventricular diastole. Peripheral resistance of the arterial

system now forces the blood back towards the heart, this occuring within the next few hundredths of a second and constituting the protodiastolic phase of the cardiac action. This column of blood now moving back towards the heart has the same cross sectional diameter as the aorta, this being considerably more than the area constituted by the triangular orifice between the free edges of the valve cusps. Blood will thus collect in the sinus pockets and force the leaflets to close. This gives rise to the second heart sound.

With the aortic valve now in the closed position, it will be seen that the cusps do not merely just approximate at their free edges. Instead there is a considerable amount of overlap of the cusps at their margins of contact (Fig. 9). This is accounted for by the fact that each cusp, in the closed position, covers an area more than one third of the cross sectional diameter of the root of the aorta. The leaflets thus actually meet at their "lines of contact", which are sited a few millimetres below the free margins of the cusps. These few millimetres of leaflet substance thus account for the overlap. The cusps accordingly afford each other a considerable degree of mutual support and the diastolic shock is not borne at the attachment of the cusps to the aortic wall alone, as would have been the case if their free edges had only barely coapted.

Ewald[91](1905) had described the function of the noduli

Arantii as that of interlocking teeth thus to prevent the cusps from slipping on one another. However Barnard[20](1958) in his studies of the normal aortic valve function found this to be unlikely. Our studies confirm the latter contention and mutual overlap of cusps at their margins of contact seems to be of far greater importance in the prevention of cusp prolapse than the possible interlocking action of the corpora Arantii.

Having thus armed ourselves with an accurate knowledge of the anatomy and function of the normal aortic valve, we may pass on to a consideration of the pathological derangements of valvular incompetence.

FIGURE 1.

Normal tricuspid aortic valve showing the posterior (non coronary) cusp, left anterior cusp (with large left coronary ostium) and the right anterior cusp (with smaller right coronary ostium). Note the corpora Arantii with adjacent lunulae. The "line of contact" of each cusp is also clearly visible. The anterior leaflet of the mitral valve is clearly shown illustrating its intimate relation to the aortic valve cusps (see text).

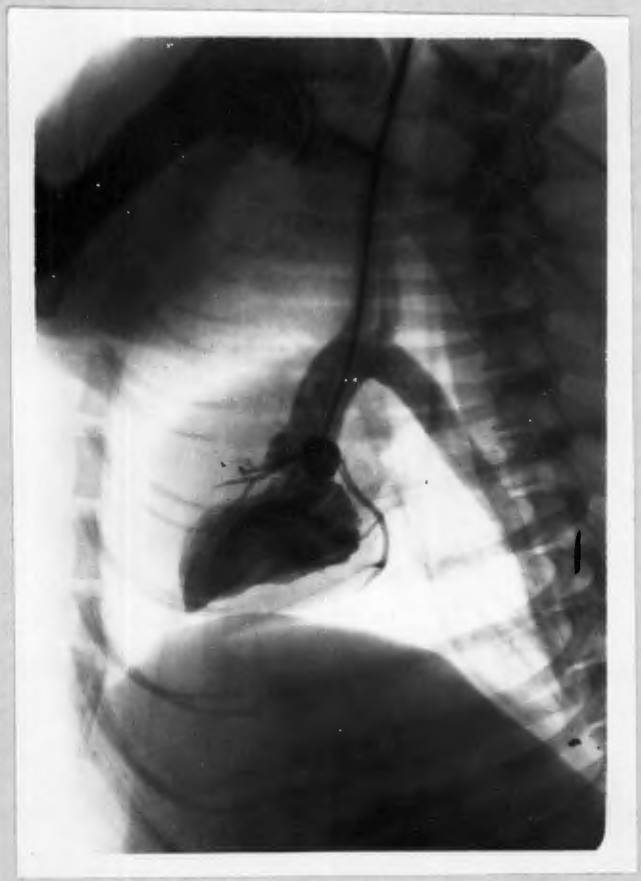


FIGURE 2.

Angio-cardiogram (taken during ventricular diastole) showing left ventricular cavity, root of the aorta and aortic arch. Sinuses of Valsalva and coronary arteries clearly outlined. Dye injection obtained by retrograde carotid artery catheterisation.

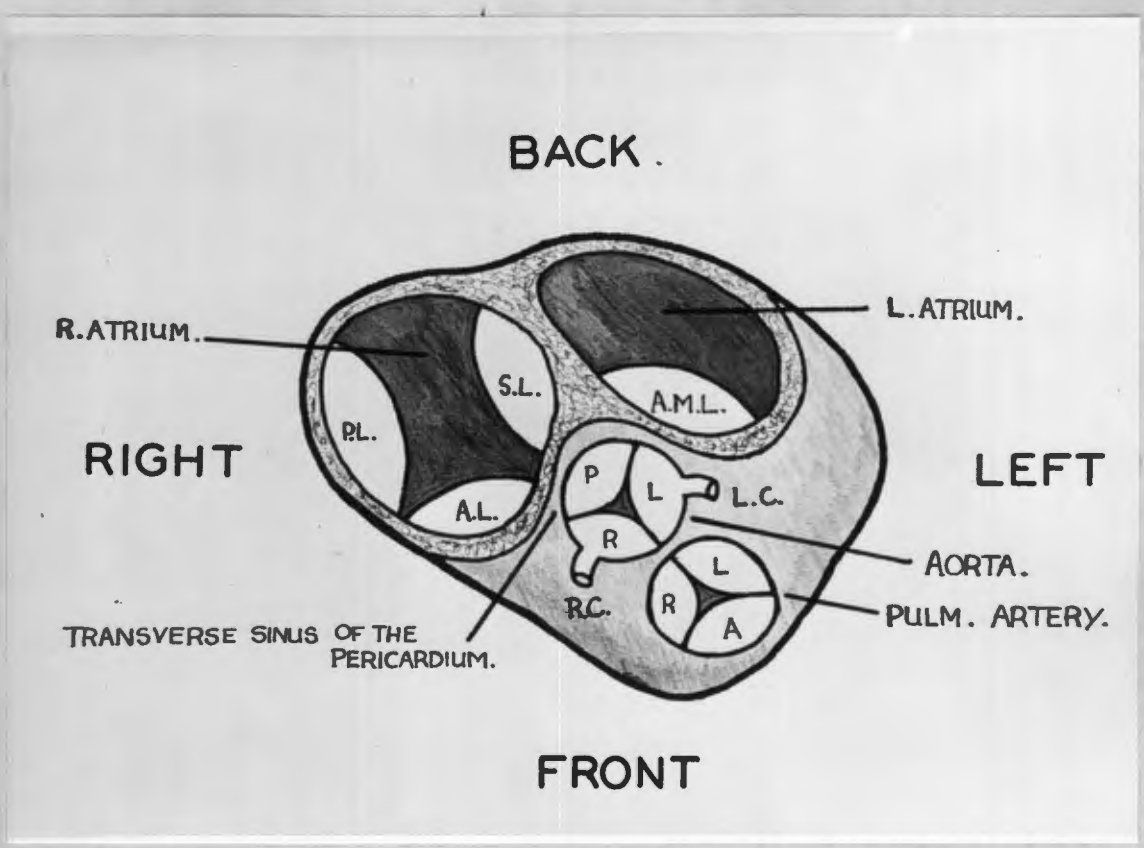


FIGURE 3.

Cross section of the base of the heart. Large type indicates orientation of the heart within the body (Ex Edwards and Burchell).

Aortic valve cusps:	P - Posterior (non coronary) L - Left anterior R - Right anterior
Pulmonary valve cusps:	A - Anterior R - Right posterior L - Left posterior
Coronary arteries:	L.C. - Left coronary artery R.C. - Right coronary artery
Tricuspid valve leaflets:	P.L. - Posterior Leaflet A.L. - Anterior Leaflet S.L. - Septal Leaflet

A.M.L. - Anterior leaflet of mitral valve

FIGURE 4.

Anterior leaflet of the mitral valve showing close relation to posterior and left anterior aortic valve cusps.

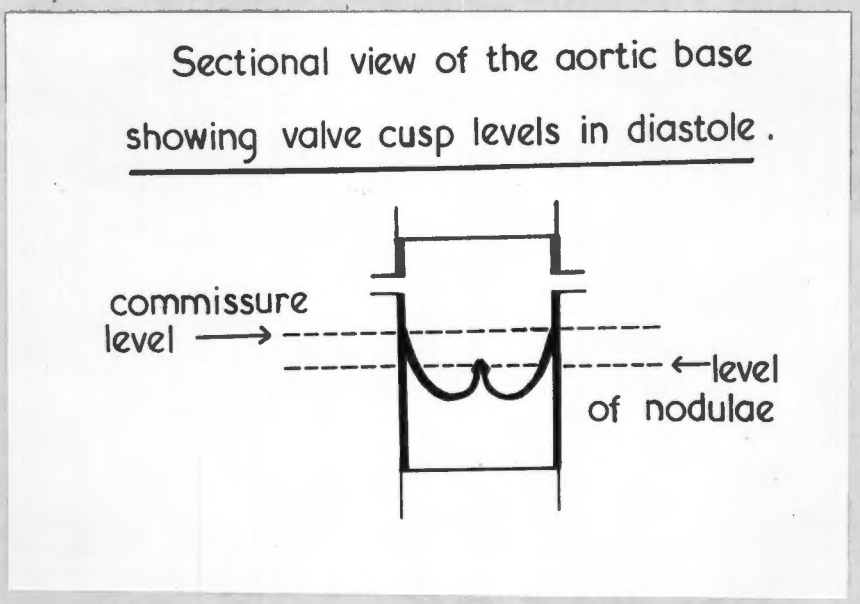


FIGURE 5.

Diagram illustrating difference in "commissural" and "nodular" levels (see text).

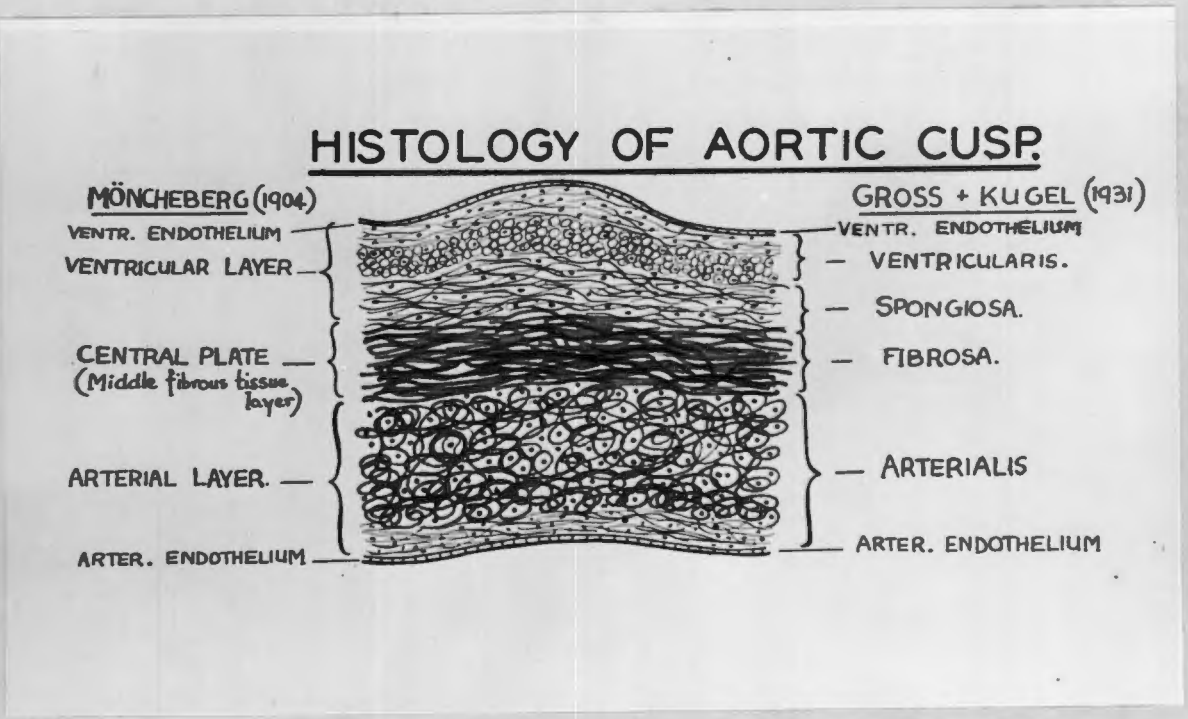


FIGURE 6.

Histological structure of normal aortic valve cusp.

Diagram illustrates difference between various nomenclatures.

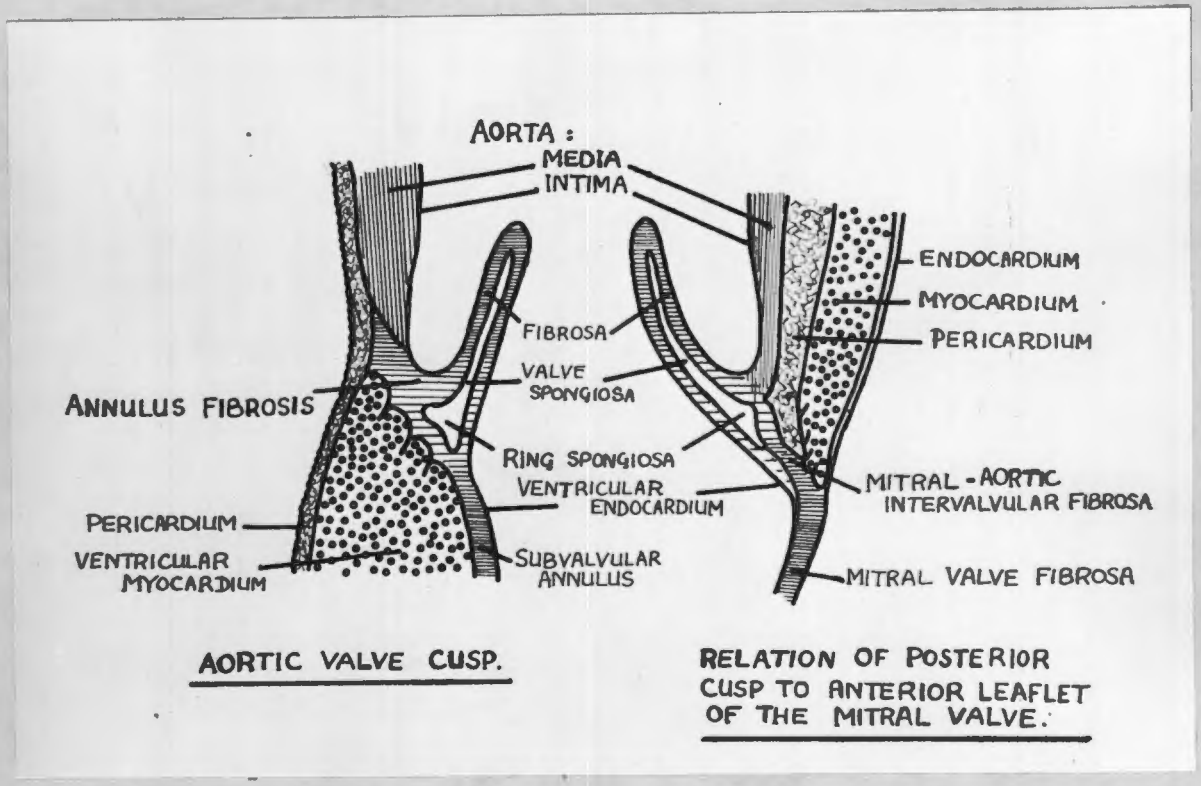


FIGURE 7.

Diagram to illustrate detailed anatomy of aortic leaflet attachment and relation to anterior leaflet of the mitral valve. (Ex Gross and Kugel)

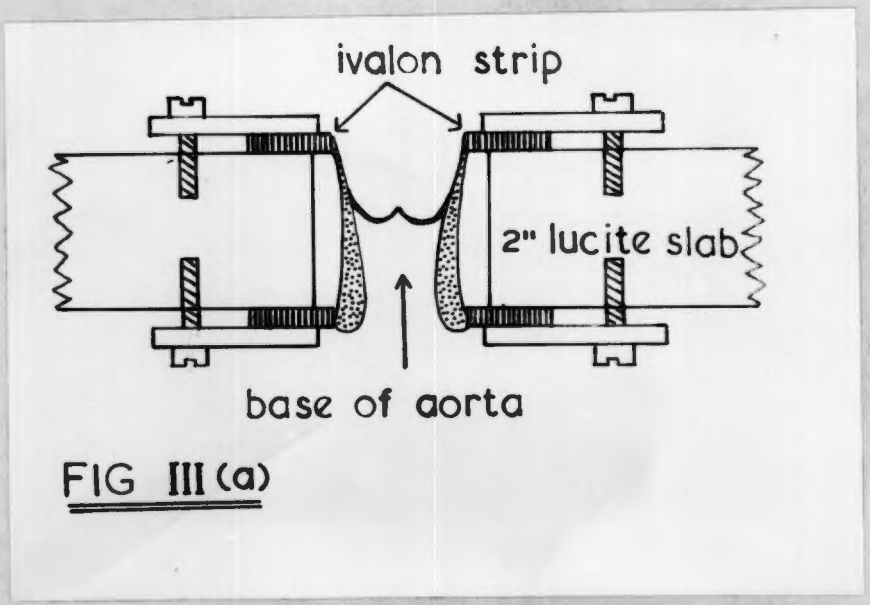


FIGURE 8.

Placement of aortic root, containing intact aortic valve cusps, in the pulse duplicator to enable study of normal valvular function.

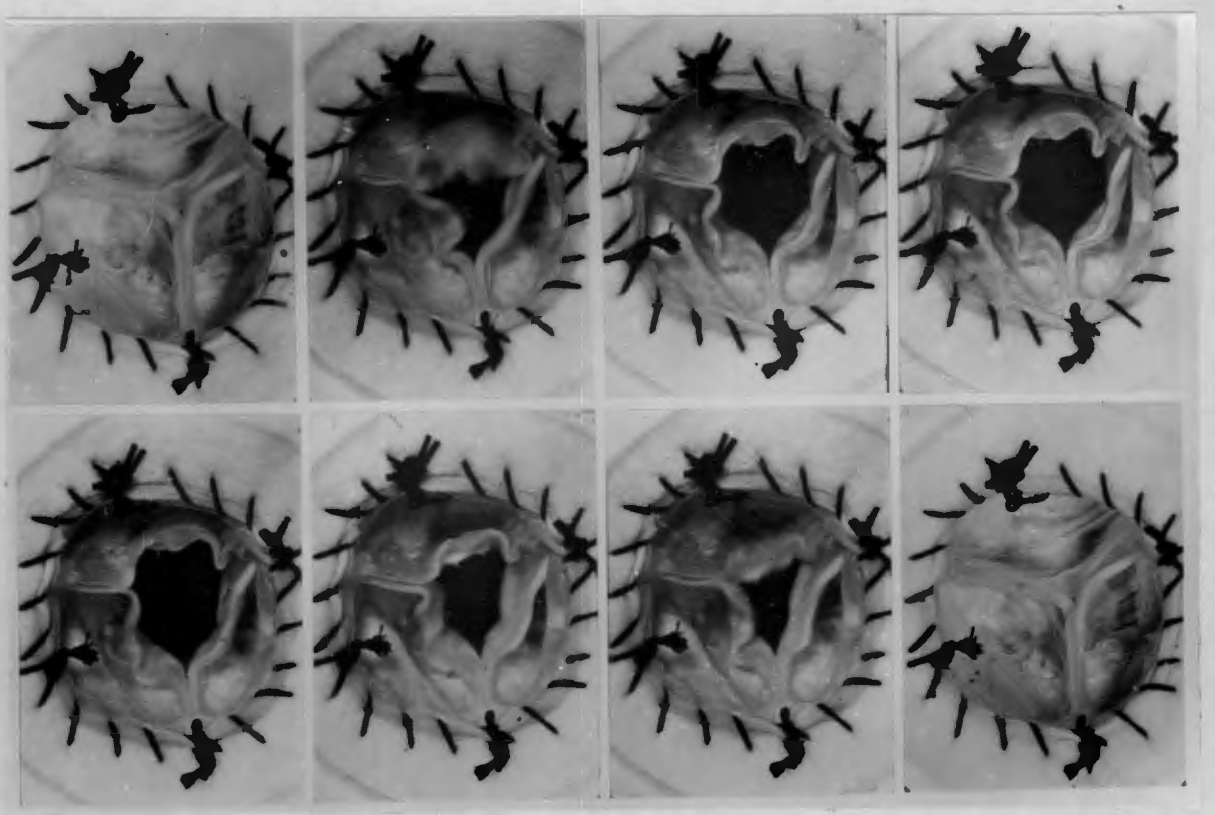


FIGURE 9.

Cycle of normal aortic valve cusp movements. Note incomplete opening of valve cusps (see text).

PATHOLOGY OF AORTIC INCOMPETENCE.

A. NOMENCLATURE - "Incompetence and Insufficiency".

Before considering the pathological process of aortic incompetence in detail a brief consideration of the terminology employed may not be entirely out of place. In medical literature the terms "incompetence" and "insufficiency" are used synonymously. This condition clinically manifests as a fall in the diastolic blood pressure with resultant "splashing" or "water hammer" pulse as was described by Sir Dominic John Corrigan in April, 1832.

The diastolic pressure of the arterial system depends on the following factors:-

(a) volume of systemic flow at a given time,

(b) peripheral resistance to flow and

(c) competence at the aorto-left ventricular junction.

In the presence of aorto-left ventricular competence conditions causing chronically low peripheral resistance include:-

(i) patency of the ductus arteriosus

(ii) systemic arteriovenous fistula

(iii) Paget's disease of bone

(iv) thyrotoxicosis

We are, however, concerned only with the factor of aorto-

left ventricular competence. We propose that in nomenclature a distinction should be made between the terms aortic "insufficiency" and aortic "incompetence", the latter to apply only to lesions affecting the aortic valve and causing regurgitation of the bloodstream from the aorta into the left ventricle. The former may however also be concerned with lesions in structures closely related to the aortic valve that may be responsible for the failure of maintenance of the normal diastolic pressure. Hence aortic "insufficiency" may be caused by aortic valvular "incompetence" but in addition, also such lesions as:-

(i) ruptured sinus of Valsalva

(ii) rupture of acquired aneurysm of the ascending aorta

(iii) traumatic "incomplete" rupture of the aorta

(iv) abnormal communication between a coronary artery and a cardiac chamber or the coronary sinus.

The term "insufficiency" would thus embrace various disease processes affecting all structures related to the aorto-left ventricular junction, whilst "incompetence" would imply pathological processes affecting the aortic valve cusps only. Numerous and various causes of aortic "insufficiency" exist.

B. AETIOLOGY OF AORTIC "INSUFFICIENCY".

Classification:-

1. Acute Aortic Insufficiency.

a). Trauma:

(i) Fenestrated cusp (Barber[18,19] 1938-1944)

(ii) Ruptured cusp

(iii) Traumatic "incomplete" rupture of the aorta: Edwards[85] (1958)

In patients subjected to accidents involving sudden deceleration, the ascending aorta may rupture. In such instances rupture may be through the entire thickness of the aorta, causing sudden death. When, however, the rupture involves the aortic wall only incompletely, dilatation of the ascending aorta may occur. The secondary effects of aortic dilatation on the aortic valve leaflets may be responsible for aortic insufficiency.

b). Syphilis: Hufnagel *et al.*,[132] (1958).

c). Acute Endocarditis - due to erosion of the valve cusps: Wood [262] (1950).

d). Subacute Bacterial Endocarditis with major cusp deformity. When bacterial endocarditis causes significant destructive changes in the aortic valve cusps, the resulting valvular

incompetence is usually responsible for death during the bacteriologically active phase of the disease.

e). Ruptured Aneurysm of Sinus of Valsalva: Ostrum et al.,[197] (1938). Most commonly these aneurysms involve the right aortic sinus. With rupture blood is diverted into the outflow tract of the right ventricle. Such aneurysms are not infrequently associated with an immediately subjacent ventricular septal defect. Less common than aneurysm of the right aortic sinus is that involving the non coronary sinus of the aortic valve. Rupture of an aneurysm in this site is into the right atrium, rarely into the right ventricle. Aneurysms of the left aortic sinus are rare, but, when they occur, they may rupture into the pericardial sac, producing either fatal haemopericardium or a false aneurysm of the left aortic sinus. When aneurysms in this location do not rupture, signs of aortic insufficiency are absent. In a rare instance an aneurysm of the noncoronary sinus may rupture into the left atrium, yeilding aortic insufficiency. Rarely an aneurysm of an aortic sinus may rupture into the left ventricle.

f). Dissecting Aneurysm of the Aorta.

2. Chronic Aortic Insufficiency.

a). Rheumatic Valvular Disease (with or without stenosis - see Para. D.).

b). Congenital:

(i) Bicuspid valve:

1. Uncomplicated - This type of valve often yields no recognisable symptoms of incompetence. Rarely, however, in the absence of stenosis, it may be significantly incompetent. This mostly happens in cases with associated coartation of the aorta (vide infra 5.). A congenital bicuspid aortic valve is not usually stenotic unless diseased. Such stenosis is usually attended by incompetence. Stenosis plus incompetence occur, however, as integral manifestations of the congenital malformation in other types of congenitally deformed aortic valves. The simplest of these forms bears close anatomic similarity to the pulmonary valve when it is involved in congenital dome shaped stenosis. This variety of deformity is termed "simple dome stenosis". A somewhat more complicated variety of dome stenosis exists and may be called "unicommissural dome stenosis" of the aortic valve. In the latter, as the name implies, there is only one commissure in the valve. Such a valve may be viewed as having but one cusp, giving the orifice the shape of an exclamation mark. The narrow part of the orifice is at the single commissure, while the widest part lies opposite. The single leaflet may be said to start at the aortic wall at the commissure; it then extends across the orifice without contacting the aortic wall, bends on itself and then returns to make a second connection

with the aortic wall, thus creating a single commissure.

In both varieties raphes extend from the aortic wall on to the aortic face of the deformed leaflet. Although these may be looked upon as representing abortive commissures, they do not usually extend very far from the base towards the free edge of the valvular tissue. In this way the raphes constitute inadequate lateral supports for the valvular tissues to the aortic wall. In both varieties of dome stenosis it is pertinent to recognise that division of the valve deprives the valvular tissue of proper attachment to the aortic wall. The resulting units thus become redundant and valvular incompetence results. The congenital bicuspid aortic valve may thus be incompetent simply on the basis of its intrinsic structural nature.

2. with associated calcific disease resultant in stenosis or varying degrees of incompetence: Edwards[85](1958).
3. with associated rheumatic valvulitis;
4. with associated subacute bacterial endocarditis;
5. with associated coarctation of the aorta.

(ii) Marfan's syndrome.

In patients with Marfan's syndrome the aorta is congenitally weak. Dissecting aneurysms may occur or the aorta may show uniform dilatation. In patients with this latter phenomenon insufficiency may be associated: McKusick[181](1955).

The aortic incompetence is probably the result of aortic dilatation comparable to the secondary effects on the aortic leaflets of syphilitic aortitis.

(iii) Quadricuspid valve.

(iv) Congenital Hypoplasia and dilatation of the ascending aorta.

(v) Congenital fenestration of the aortic cusps: Matthews[179](1956). Such fenestrations of the valve cusps may, however, exist without any disturbance of the valve mechanism: Quain[201](1922).

(vi) Aortic incompetence and ventricular septal defect. In some cases of congenital interventricular septal defect, involving the outflow portion of the ventricular septum, aortic incompetence may coexist. Usually the defect occupies a specific location. From the left ventricular aspect it lies inferior to the adjacent portions of the right and posterior aortic leaflets. The defect as seen from the right ventricular side lies posteroinferior to the crista supraventricularis. Since the defect is bordered by the aortic valve, the aorta fails to have a connection with the ventricular septum at the site of the defect. Deviation of the aorta from its normal relationship with the ventricular septum occurs. With this deviation the portions of the aortic cusps

which lie adjacent to the ventricular septal defect, prolapse. These two factors are the essential ones causing incompetence of the aortic valve related to a interventricular septal defect. There are, in addition, fibrous changes in the valvular tissue adjacent to the ventricular septal defect and, with the retraction caused by these changes, the incompetence may be accentuated.

c). Subacute Bacterial Endocarditis - with moderate to minor deformity of the aortic valve. In relatively unusual cases in which the infection is overcome and destructive valvular changes have occurred, the patient survives for some months, or a few years, with chronic aortic incompetence. The lesion is produced by erosions or perforations of the aortic cusps. Preexisting diseases include congenital bicuspid aortic valve, minor degrees of fusion at the commissures, the results of rheumatic fever and calcific aortic valve involvement (Edwards[85]1958). Although the classic teaching is that bacterial endocarditis almost invariably affects valves previously diseased, in some hearts with bacterial endocarditis it is difficult to demonstrate that prior disease had existed: (Edwards[85]1958).

d). ? Unruptured Aneurysm of the Sinus of Valsalva: Hufnagel et al.,[132](1958).

e). Dissecting Aneurysm of the Aortic Root. Under unusual circumstances an aneurysm of the ascending aorta, developing on a basis of acquired disease, may rupture into the pulmonary trunk or, less commonly, into the superior vena cava. Such an aneurysm may then give the clinical picture of aortic insufficiency.

f). Syphilis (Chronic Progressive) (See Para. E.).

g). Atherosclerosis: Fenichel[96](1950).

h). Simple Severe Hypertension - unassociated with atherosclerosis causing dilatation of the aortic valve annulus: Wood[262](1950).

i). Arteriovenous Communication between a coronary artery and the coronary sinus or a cardiac chamber. Such an abnormal, congenital communication is responsible for a run-off of the blood from the aorta and the functional effects of aortic insufficiency.

In this study we are concerned only with chronic valvular incompetence, and not the various causes of "insufficiency" and obviously, clinically, the most important causes of chronic aortic valvular incompetence are rheumatic heart disease and syphilitic aortitis. Only these two pathological processes will therefore be considered in detail.

C. INCIDENCE OF CHRONIC AORTIC INCOMPETENCE.

Campbell and Shackle[44](1932) quote incidences as follows: in a series of cases with disease of the aortic valves the condition was due to rheumatism in 200, to syphilis in 55, to atherosclerosis in 20 and to all other causes in 21 cases. However with the advent of newer antibiotic control of luetic disease this lesion accounts for even less cases of aortic incompetence. Of every 6 rheumatic cases 3 had aortic incompetence plus mitral stenosis, 1 had aortic stenosis plus incompetence combined with mitral stenosis, 1 had aortic stenosis plus incompetence and 1 had only aortic incompetence. When there was no associated mitral disease present there were 2 males for each female but when there was mitral disease present there were 2 men for every 3 women.

Other series record that aortic incompetence predominates in males over females by 3 : 1 (Segal et al.,[228]1956). It is also noted that, in rheumatic aortic incompetence, there is usually a ten year interval between the onset of recognisable auscultatory signs and the development of limiting symptoms. In syphilitic aortitis with incompetence on the other hand, the clinical course is much more rapid. The average survival time after the appearance of the murmur of aortic incompetence due to syphilis is about two years: Montgomery et al.,[189] (1952).

D. RHEUMATIC VALVULAR DISEASE AS CAUSE OF CHRONIC AORTIC INCOMPETENCE.

The relative incidence of valve involvement in rheumatic fever (Clawson[55]1945) is as follows:-

a). aortic and/or mitral (99.8%)

b). pulmonary and tricuspid in only 5.6%; and then the aortic and/or mitral valves always involved: Clawson[54](1940).

Histological examination, however, reveals evidence of rheumatic inflammation in the tricuspid as often as in the mitral and aortic valves: Gross and Friedberg[116](1936).

Rheumatic disease of the aortic valve becomes apparent from childhood to old age; commonest however, in middle and old age.

Rheumatic fever is a disease of recurrent acute attacks rather than a sustained chronic inflammatory process. Thus the surgeon at operation will see the result of repeated attacks of rheumatic valvulitis as either chronic valvulitis or healed valvulitis (valvular deformities). The term "valve defect" was used by Libmann[159](1917) to denote this latter condition.

1. Chronic Valvulitis.

113 of 796 cases of rheumatic heart disease at autopsy showed this change (Clawson[54]1940); in these the valvular deformities are incompletely healed. Of these 113, in 4 the

aortic valve alone was involved, in 28 the mitral and aortic valves and in 65 the mitral alone.

Macroscopy:-

Thickening and irregularity of the surface and gross vascularisation are present. The thickening is maximal at the free edge of the cusp (Fig. 10). Usually thickening and fibrosis have resulted in a loss of elasticity and narrowing of the orifice. Occasionally retraction and curling have led more to incompetence than stenosis (Fig. 11): Clawson et al.,[57](1926). In addition to severe diffuse thickening of leaflets there is deposition of calcium salts. This further distorts the leaflets and is found mostly in the region of the noduli and in the commissures. In addition to distortion the valves are now rigid. Rheumatic verrucae are commonly found and are broad and flat. Occasionally non-specific vegetations are found on such valve leaflets; these consist of non infected thrombi and are not related to active rheumatic inflammation. In addition considerable shortening of the cusp is evident. This is the result of rolling and inversion of the free margins of the cusp towards the sinus pocket. The cusps may also show adhesions at the commissures and verrucae in various stages of activity. The latter may extend from one cusp to another across the commissures. Abnormalities of the sinus pockets include verrucous ridges and folds.

Microscopy:-

In addition to the inflammatory changes in various stages of activity there is evidence of considerable fibrosis and elastic tissue proliferation. The inflammatory cells are predominantly lymphocytes with smaller numbers of polymorphonuclear cells, plasma cells and macrophages. The fibrosis and inflammatory changes involve the rings as well as the leaflets and fibrous and elastic thickening is particularly prominent at the subvalvular angles. With advanced chronicity fibrous connective tissue becomes more homogeneous and hyaline. In the valve rings the annulus fibrosis frequently is hyalinised but may also reveal calcification and even bone formation. The thrombotic vegetations which form on the line of closure of the leaflets and on calcified and ulcerated portions of the deformed valves, have a hyaline appearance and seem to be formed largely of platelets. There is little or no cellular reaction at the base. They rest on the scarlike hyaline connective tissue of the leaflet and the thrombus shows little tendency towards organisation. A well marked feature is the presence of numerous arteries with thick muscular walls in the ring and leaflet - in long standing cases these vessels may even become fibrotic. This increased vascularity is one of the most conspicuous features of recurrent valvulitis, in addition to the various non-specific features of inflammation. Aschoff bodies are frequently found in the fibrosa and

spongiosa layers of the leaflets.

2. Healed Valvulitis.

Not all valvular deformities are rheumatic in origin. It seems that not enough is known concerning the valvular damage in a wide variety of toxic, infectious and metabolic processes to dismiss all etiologic possibilities except rheumatic fever in the interpretation of any valvular deformity. Thus Baldassari[16](1909), Czirer[70](1913) and deVecchi[76] (1931) found histological evidence of acute valvulitis in children in the presence of such diverse diseases as scarlet fever, diphtheria, bronchopneumonia, meningitis and tuberculosis without gross evidence of valvular damage. It thus seems possible that patients, especially children, who survive such infectious processes, also have acute valvulitis which in some instances may heal and result in valvular deformities. The same may be postulated for the healed stage of bacterial endocarditis: Saphir[220](1941-1942); Moore[190](1946). The scarred end stage of any inflammatory process rarely gives pathognomonic signs of the original aetiologic agent. It seems therefore that in instances in which there is neither good clinical nor pathologic evidence of previous rheumatic disease, caution should be exercised in the interpretation of such valvular deformities.

Valvular deformities on a rheumatic basis are mostly the

result of valvular inflammation and only in small part due to the organisation of vegetations. Organisation of thrombi and contraction may be an important explanation of some deformities but the importance of cellular proliferation in the valve itself is often underestimated. The scar forms as the result of fibroblastic proliferation and collagen formation - lipoid deposition and calcification occur later.

Thus, although not all valvular deformities are rheumatic in origin, certainly most of them are. Clawson and Bell[56] (1926) found 55 out of 73 cases to result from rheumatic endocarditis. Of these 55 cases 27 were chronic valvulitis with complete healing of rheumatic lesions.

In the rheumatic heart disease group (total 1796) in 73.5% of deaths valvular deformities were present: Clawson[54] (1940).

Valvular deformities macroscopically may present as adhesions of the cusps at the commissures, thickening, fibrosis and calcification of the ring and cusps causing varying degrees of stenosis. On the other hand scarring, retraction and stiffening of the leaflets will result in regurgitation. Thus the end result of repeated attacks of rheumatic disease may be predominant stenosis, predominant regurgitation or equal degrees of both: White[255](1948).

Cabot[41](1926) reviewed a series of 152 cases of aortic valve disease - 93 manifested stenosis and incompetence of

rheumatic origin and 11 incompetence with little or no stenosis associated; of these 11 only 6 proved to be of rheumatic origin. Most of the cases of incompetence with stenosis (44) were syphilitic in origin.

In a series of 130 cases (Clawson et al.,[57] 1926) of aortic valvular deformities 41 revealed stenosis and incompetence and in 13 of these incompetence was the dominant defect.

It is in the healed valvulitis producing aortic stenosis that an age-old feud exists between pathologists concerning the aetiologic factor. When the lesion is associated with a clear-cut history of rheumatic fever or an associated deformity of the mitral valve, rheumatic inflammation as causative factor appears the most likely. When unassociated with other valvular deformities, however, some investigators (Möncheberg[188] 1904; Sohvol and Gross[237] 1936; Libman[159] 1917; Ribbert et al.,[206] 1924; Geerling[104] 1929; Ashworth[4] 1946; Hultgren[133] 1948) have concluded that degenerative or metabolic factors are responsible. These workers thus hold that the disease is purely degenerative and inflammatory involvement does not exist in connection with it, or is secondary to it. Thus Möncheberg decided that the factors causing the changes in the valve cusps are the same as that found in the intima of the aorta in senile arteriosclerosis. He held that the arteriosclerotic process extended from the aorta by way of the aortic sinuses to the valves. This condition has since

been termed: "Mönckeberg aortic sclerosis"; "primary ascending sclerosis of the aortic valve" or "sclerosis annularis valvulorum".

According to Ashworth[4](1946) the factors to be considered in the development of atherosclerosis of the heart valves are age, hypertension, the physiological decrease in cellularity of the annulus, fibrosis of the aortic and mitral valves and the effects of tension and vibration on certain portions of the valves.

The opposing school of thought, again, conclude that rheumatic fever is always the basis of the valve deformity: Clawson et al.,[57,53,58](1926 - 1938); Christian[51](1931); Anitschkow[3](1913); Hall and Ichioka[120](1940); Karsner and Koletsky[140](1947). This assumption is based on the incidence of rheumatic fever in the history of patients with this deformity, the numerous transitions found between undoubtedly healed rheumatic lesions and calcified nodular valves, and the association of other lesions also probably caused by rheumatic fever.

E. SYPHILIS AS A CAUSE OF CHRONIC AORTIC INCOMPETENCE.

Cowan and Ritchie[63](1935) stated that syphilis accounts for one half of all cases of aortic valve disease between ages 40 - 60 years and for 33% of all cases of aortic valve disease. The frequency and incidence of this disease has, however,

considerably declined, although Paul Wood[262](1950) still stated that it was the most common cause of pure aortic incompetence.

Syphilitic aortitis involving the root of the aorta may cause narrowing or closure of the coronary ostia and, because of involvement of the aortic ring area, incompetence of the valve. Heller[125](1899) and Chiari[50](1904), in their classical descriptions of syphilitic aortitis, described syphilitic involvement of the aortic valve to cause incompetence.

1. Macroscopy:-

Mesaortitis results in weakening of the aortic ring with dilatation of the annulus and secondary valvular changes. Separation of the valve cusps at the commissures and further encroachment on the length of the free cusp edge, because of annular dilatation, result in valvular incompetence. The cusps themselves become classically "rolled, everted and thickened" at their free margins, but many workers regard this as a mechanical effect of the regurgitant stream. This process adds a further element of shortening to the cusps: Edwards[85](1958). Stenosis never occurs and calcification only follows if there is much secondary atherosclerosis. Hyaline plaques, often triangular in shape may be present in the region of the commissures. The adjacent aorta is invariably the seat of characteristic syphilitic lesions with varying

numbers of depressed scars and grooves combined with intimal fibrosis, foci of hyalinisation and calcification. Aneurysmal dilatation of the root of the aorta may be evident.

2. Microscopy:-

Saphir and Scott[221](1927) state that very early adhesions between the intima of the sinus of Valsalva and the corresponding lateral and proximal portions of the aortic cusps occur and these adhesions cause widening of the commissures. Histological changes are those initially of degeneration and later of inflammation. Coincidental with the latter, blood vessels are found to extend through from the adventitia through the intima to the involved commissures and lateral portions of the cusps. Fibroblasts, endothelial cells, leukocytes and lymphocytes are the predominant cell types. At later stage hyalinisation of the intima in the commissural region is encountered. Histology of the more central portions of the cusp, macroscopically showing thickening with rolling, is that of fibrosis and hyalinisation with very few cellular elements and no newly formed blood vessels. This lesion of the cusp center is regarded as due to the continuous mechanical pressure of the regurgitating blood after establishment of insufficiency.

Adhesions between adjacent intima of sinus of Valsalva and aortic cusp may even extend from the commissure to the more central portion of the cusp and corresponding area of aortic

sinus intima. This would result in severe narrowing or even complete obliteration of the aortic sinus with extreme valvular incompetence. Such cases have been described by Saphir and Stasney[222](1933) and Maresch[174](1931).

The reason for syphilitic predilection for the base of the aorta and the region of the commissures is explained by Saphir and Scott[221](1927) on the basis of abundance of vasa vasorum in this region. This is in agreement with Spaltehotz's theory that syphilis is a primary disease of the vasa vasorum.

There are also many instances of syphilitic aortitis with separation of the commissures but with severe dilatation or stretching of the root of the aorta, including the sinus of Valsalva, with resulting incompetence of the aortic valve. This stretching of the aorta is the result of the syphilitic process in the adventitia and media of the aorta itself. In such a case the cusps may actually enlarge to effect adequate closure for some time (Karsner[139]1949), but eventually incompetence of the valve follows.

THE MECHANISM OF CHRONIC AORTIC INCOMPETENCE.

Normal efficient and competent function of the aortic valve depends on a number of factors. The most important of these being preservation of the normal architecture of the valve and free mobility of the valve leaflets. Thus any disease process causing widening of the annulus fibrosis, scarring with resultant shortening and loss of mobility of the cusps, or destruction of leaflet substance will result in defective valvular function.

Two such forms of chronic aortic incompetence are encountered differing markedly in mode of formation and in final functional impairment.

The first and most commonly encountered is the lesion of aortic stenosis plus incompetence.

A. Aortic Stenosis Plus Incompetence ("Mixed Lesion").

This lesion is most commonly the sequel to repeated attacks of rheumatic valvulitis. There is a loss of the normal soft, pliable, translucent appearance of the cusps with resultant hardening, opacity and immobility of the leaflets. These changes may be confined to the free edge of the cusps, (see later "Intermediate Lesion") (Fig. 10) or uniformly distributed throughout the cusps, or in the latter type, maxi-

mally concentrated at the free edge of the cusp. Secondary depositions of atheromatous and calcific material accrue and may attain such proportions that by mere bulk they may interfere with adequate valve function and result in incompetence: Cowan[63](1935).

The main features of this lesion are therefore:-

(i) gross thickening and scarring of the leaflets with a minimal degree of shortening as a result, associated with

(ii) gross secondary calcific deposits (Fig. 12).

Marked loss of cusp mobility and commissural fusion are also evident (Fig. 11). Aortic stenosis is always present in a greater or lesser degree. Fracture of a stenosed valve may further accentuate the degree of incompetence.

The "critical" orifice area in pure stenosis is 0.5sq. cms. and in combined aortic stenosis plus incompetence is 1.5sq. cms. (Gorlin et al.,[110]1955). With such degrees of deformity left ventricular pressures were shown to be 200 - 250 mm. Hg. with transvalvular pressure gradients of 100 - 150 mm. Hg. Because of the large gradient from aorta to left ventricle in diastole, a regurgitant orifice area of no greater than 0.5 sq. cms. is capable of more than doubling the total ventricular workload: Gorlin et al.,[110](1955).

The same degree of aortic stenosis is more serious when incompetence is associated than when it is not. The barrier

to the outflow of blood from the left ventricle by a stenotic valve needs to be measured in terms of the amount of blood that the left ventricle attempts to eliminate in a given time period. It is obvious that with the same effective forward flow the left ventricle has to eliminate more blood when aortic incompetence coexists with stenosis than it does when the stenosis is not associated with incompetence: Gorlin et al., 110(1955).

In many stenotic aortic valves one of the three commissures is obliterated by fusion of two adjacent cusps, usually the right and the left. The resulting fusion of the two leaflets converts the aortic valve into a bicuspid valve (Fig. 11). Fusion of adjacent cusps takes the form of a calcified raphe that joins the aortic wall with the zone of fusion of the two leaflets. The resulting conjoined leaflet is made immobile not only by its intrinsic calcific disease but also because of its rigid fixation to the aortic wall by the calcific acquired raphe. The only chance for the valve to open is thus by motion in the third cusp. In many classic examples of this condition the third leaflet is able, in spite of its intrinsic disease, not only to open a limited amount, but also to close completely, or nearly so, thus preventing any significant regurgitation.

At the opposite end of the scale are those cases of aortic valve stenosis in which adjacent cusps are fused at each of the

three commissures. This fusion may extend for a considerable distance from the aortic wall towards the center of the lumen. Obviously, if the fusion of each commissure extended to the center of the lumen, there would be no aortic orifice. Some opening, however narrow, always exists. This preservation of an opening when tricommissural fusion exists depends on the intrinsic shortening of the valvular leaflets (Fig. 12). This shortening, coupled with the fixation of each leaflet to the others by fusion at the commissures, yields an aortic valve which is no longer a valve in the ordinary sense. The aortic valve has been converted to a structure which may be compared to that of a metal washer with a central perforation which remains the same size throughout the cardiac cycle. It is obvious that such a valve not only causes obstruction to the outflow of blood from the left ventricle but also allows regurgitation (Fig. 13).

The two types of stenosis involving the aortic tricuspid valves which have been described, represent the two purest forms of the disease. In the first type there was fusion at only one commissure, allowing only the third leaflet to act as a flap valve. The possibility of complete closure of the aortic valve during ventricular diastole exists in this valve type. In some instances however, the presence of irregularly deposited calcific spurs may prevent the single functioning

cusp from closing the orifice; thus some aortic incompetence may coexist. In the second type of aortic stenosis mentioned, fusion at the three commissures eliminates any valvular function.

Transitional states between these two types exist. These transitional forms usually consist of fusion at two of the three commissures. In such aortic valves the changes are essentially similar to those in the congenitally bicuspid valve when calcification with fusion has developed at one of the two commissures. Here a small amount of tissue on each side of the commissure between the non-fused leaflets tends to approximate during ventricular diastole, while at the other (fused) commissure, the orifice is held in an open state throughout the cardiac cycle.

In calcific aortic stenosis complicating congenital bicuspid aortic valves the coexistence of aortic incompetence is a common phenomenon. In cases in which fusion has taken place at both commissures, the alteration in the valve is essentially the same as that in the tricuspid aortic valve fused at all three commissures. Thus the orifice is held open throughout the cardiac cycle, and represents a fixed narrow central perforation in a membrane across the aortic orifice.

In some cases of congenital bicuspid aortic valve with

stenosis, fusion occurs at only one commissure. Beyond the fusion, along the line of the commissure, the two leaflets are held apart in an open and fixed state, while at the other commissure where there is no fusion, varying degrees of mobility may be present, with a tendency toward approximation of the two leaflets in this region during ventricular diastole. However closely these leaflets may approximate at this point there is always a persistent opening near the site of fusion.

The other lesion causing aortic incompetence differs markedly from the first and is not as commonly encountered. This is the lesion of pure aortic incompetence.

B. Pure Aortic Incompetence ("Primary" Aortic Insufficiency: Lillehei et al.,[165] 1958)

This lesion is not as commonly encountered for two reasons. Firstly, although this lesion may also be the aftermath of rheumatic heart disease, it is stated that syphilis, even with lessened incidence, is still the most common cause of pure aortic incompetence: Wood[262] (1950). Secondly, by virtue of the fact that direct vision aortic surgery for pure valvular incompetence is only in its infancy, few such lesions have actually been examined during life. Lillehei[165] (1958) has made the observation, that at direct vision surgery, valves clinically producing significant incompetence appear

relatively normal and the lesion may even be often missed at the autopsy table. He also noted the suppleness of the leaflet associated with the usual absence of calcium in "primary" aortic insufficiency.

It is of interest to note that in this paper it was also predicted that "the results of surgical correction of insufficiency either of the mitral or aortic valves is likely ultimately to surpass that possible for stenotic lesions because of the better cusp or leaflet tissue remaining with which to work".

The lesion (Fig. 14) is essentially the result of:-

(i) disease processes causing widening of the annulus fibrosis,

(ii) post inflammatory scarring, shortening and retraction of the free edges of the cusps, or

(iii) a combination of the above two factors.

The cardinal feature of this lesion is therefore a reduction of the available leaflet substance. Thus although the cusps still retain free mobility, it is now evident that the cross sectional area of the rest of the aorta is greater than that area which the leaflets can close with ventricular diastole. The result is therefore a triangular unclosed area remaining between the free edges of the leaflets allowing regurgitation of blood. This constitutes "the aperture of incompetence": Bailey et al.,[9](1955) (Fig. 14).

As the cusp edges no longer overlap and afford mutual support to each other, the full force of diastole is now borne by the attachments of the cusps to the aortic wall at the commissures. Thus weakening may result in prolapse of one or more cusps with further increase in regurgitation.

C. "Intermediate" Lesion (Fig. 10)

Between these widely differing lesions of pure incompetence versus stenosis plus incompetence ("mixed" lesion) various transitional stages may be encountered. Hence thickening, hardening and scarring, even with secondary calcification, may be confined to the free edges of the cusps, thus constituting only a moderate impairment of mobility associated with some shortening of the cusps to result in the production of the central triangular regurgitant orifice (Fig. 15). On the other hand some degree of commissural fusion associated with cusp retraction, with loss of cusp mobility, will result in some degree of stenosis associated with the incompetence.

D. Effects of Aortic Incompetence on the Heart.

Marked aortic incompetence has a more rapidly serious effect than marked aortic stenosis. The heart enlarges grossly and left ventricular hypertrophy and compensatory

dilatation apparently develop simultaneously. With gross left ventricular dilatation the mitral valve becomes incompetent and hypertrophy and dilatation of the left atrium ensue. These are followed in turn by enlargement of the right ventricle and eventually of the right atrium, though death due to left ventricular failure is likely to interrupt the full evolution of these various steps: White[255](1948). In addition to extensive hypertrophy and dilatation with flattening of the trabeculae carneae, endocardial pockets also may result from the regurgitant bloodstream. Apparently the myocardium fails from overwork induced by the valvular deformity. However low diastolic pressure, the result of aortic insufficiency results in functional coronary insufficiency. Apart from focal myocardial fibrosis, however, there is usually no morphologic evidence of myocardial ischaemia: Gould[114](1953). Except for those with the infrequent combination of uncomplicated mitral and aortic stenosis, patients with multivalvular disease are poorer operative risks than those with isolated lesions and offer less promise in functional recovery, particularly when congestive failure has once occurred. The calculated risk in survival and the gravity of the post operative complications emphasise a sober approach to the problem of multivalvular surgery and emphasise the need for careful patient selection: Likoff et al.,[160](1955).

FIGURE 10.

"Intermediate" lesion. Note thickening and calcification confined to free edges of cusps combined with shortening and cusp retraction. Some degree of commissural fusion coexists.

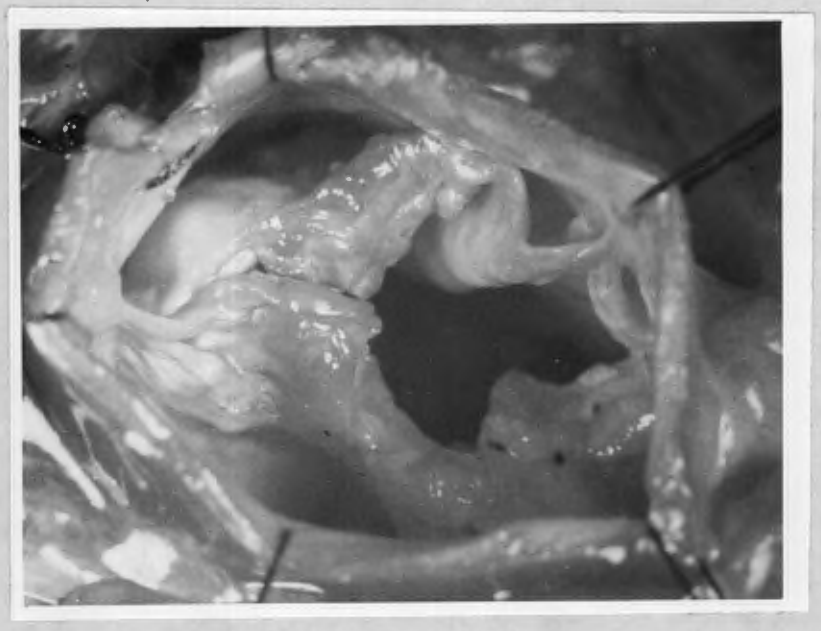


FIGURE 11.

"Mixed" lesion of aortic incompetence plus stenosis (predominant incompetence). Note calcification and thickening of cusps associated with commissural fusion.

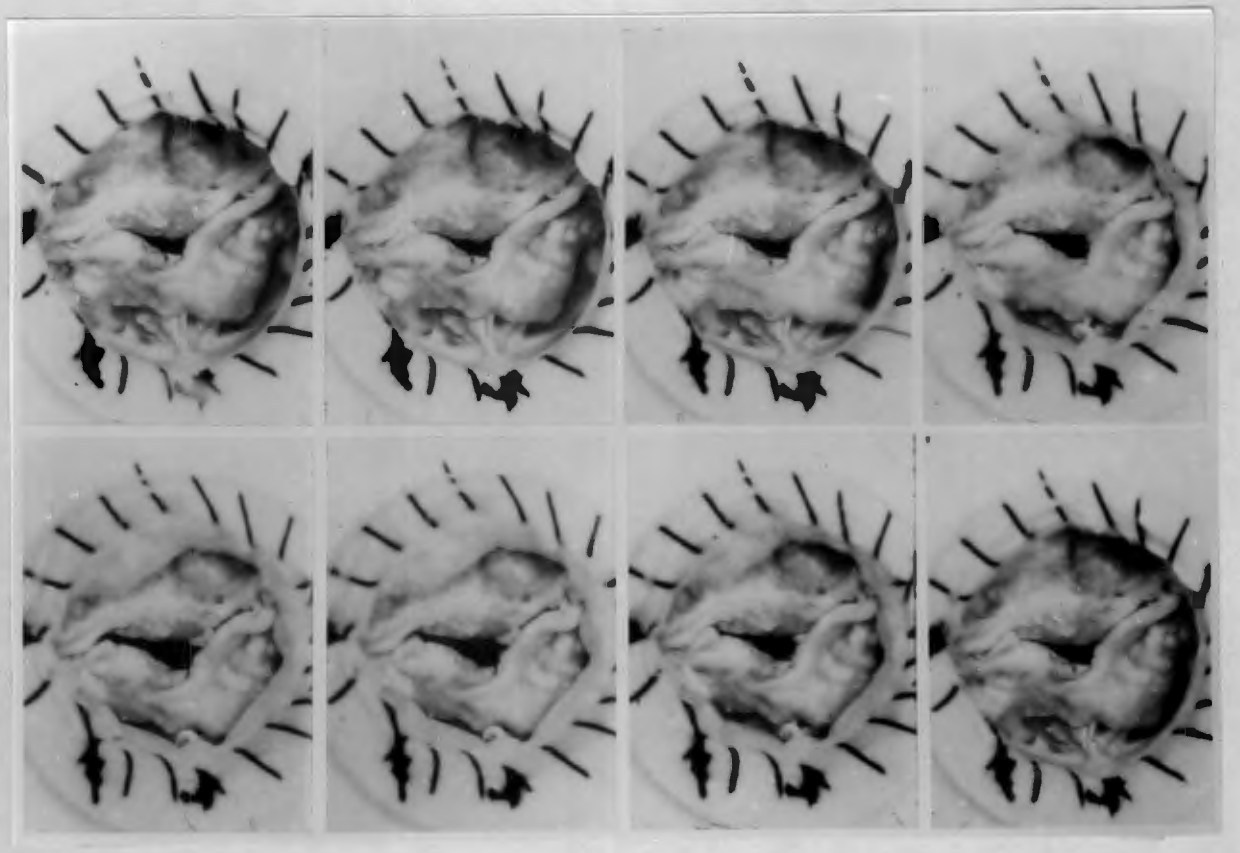
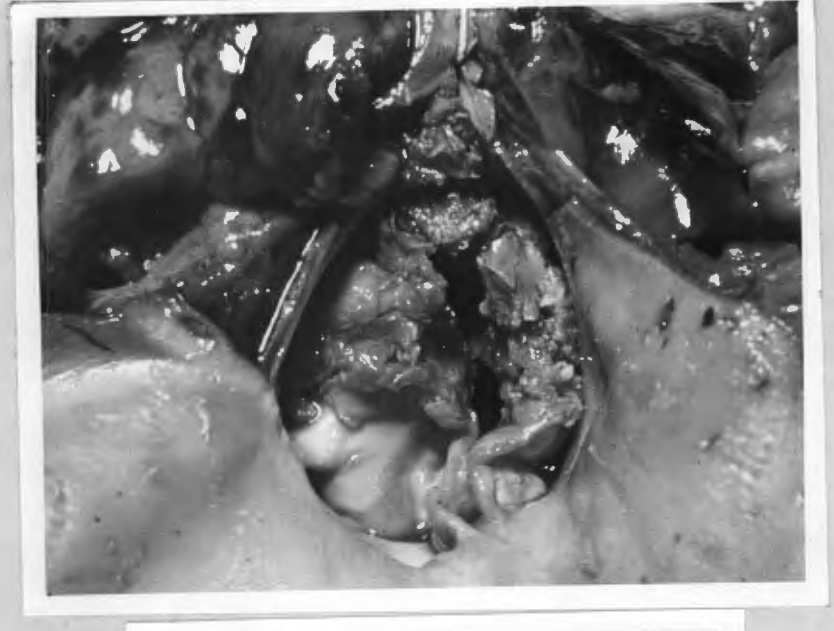


FIGURE 12.

Cycle of cusp movements in aortic valve "mixed" lesion of combined stenosis plus incompetence (predominant stenosis).



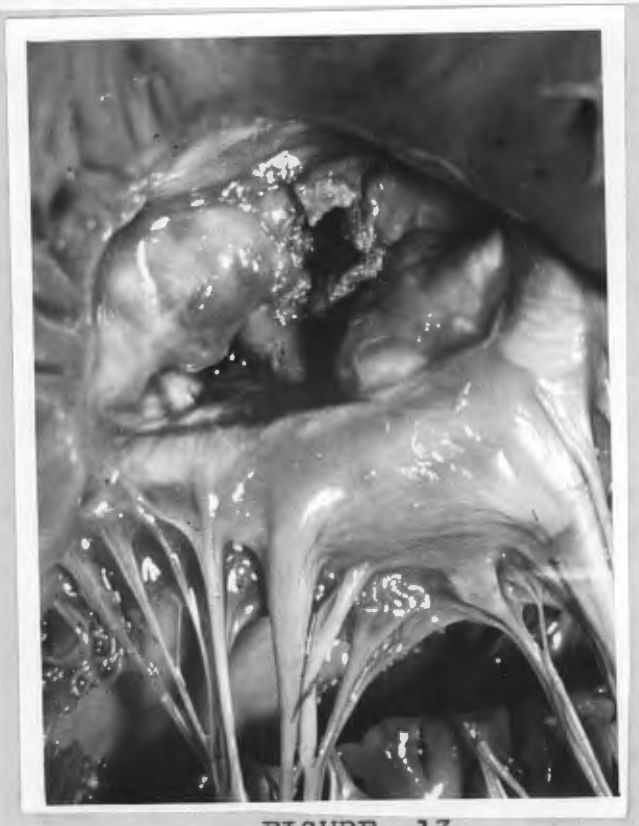


FIGURE 13.

"Mixed" lesion of aortic stenosis plus incompetence (with predominant stenosis).

a). Aortic view

b). Ventricular aspect.

Note gross thickening and calcification of cusps unassociated with any loss of cusp substance but with obvious impairment of cusp mobility.

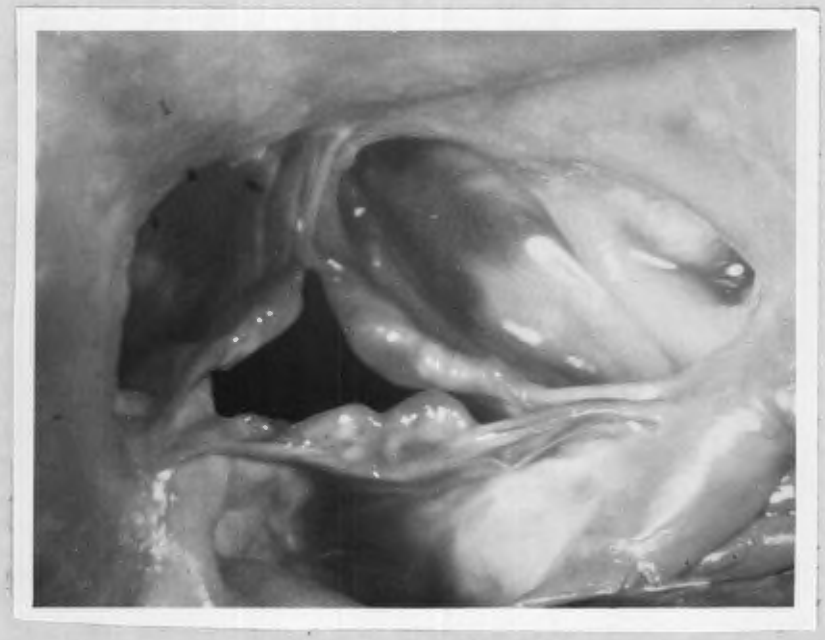


FIGURE 14.

Pure aortic incompetence (Primary aortic incompetence - Lillehei).

The triangular "aperture of incompetence" is clearly shown. Note loss of leaflet substance associated with preservation of cusp mobility.

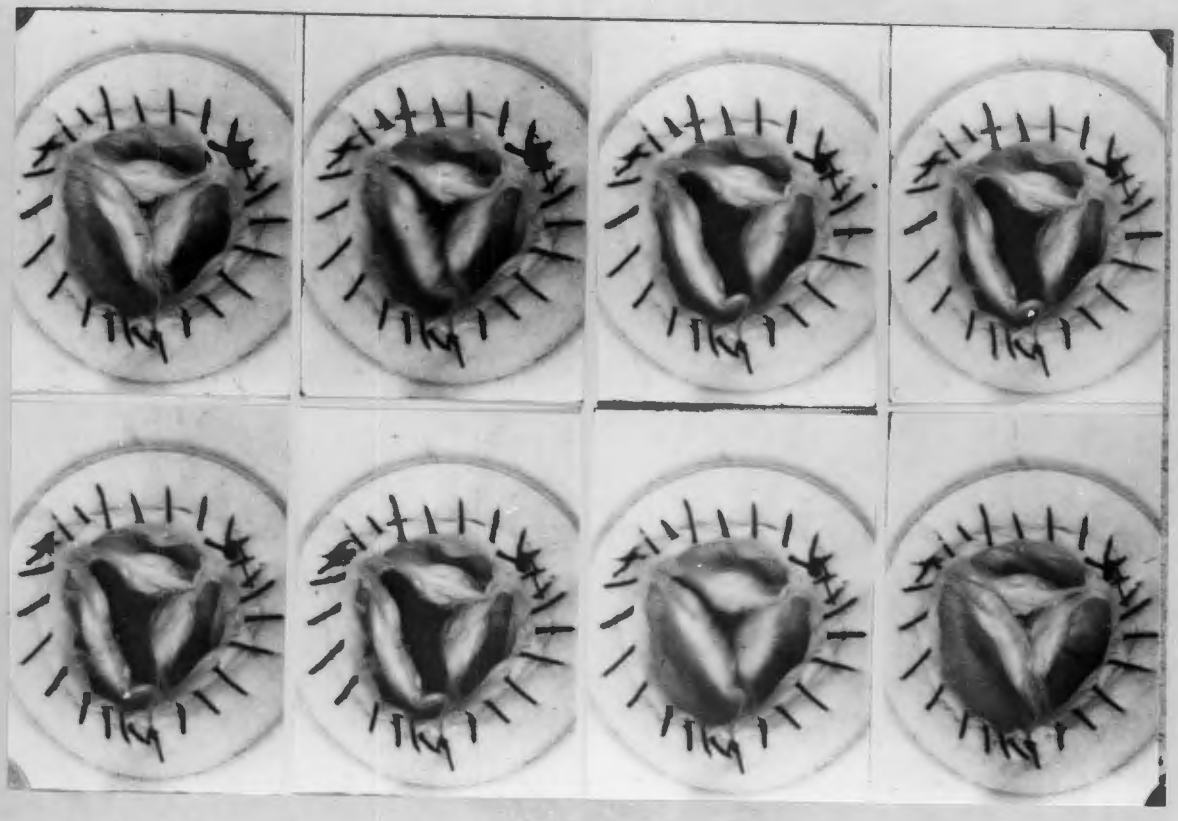


FIGURE 15.

Cycle of cusp movements in aortic valve manifesting "intermediate lesion".

THE SURGICAL CORRECTION OF CHRONIC AORTIC INCOMPETENCE.

Surgical correction of valvular incompetence may be effected by either:-

(a) reconstruction of the diseased tissue to approximate normality, or

(b) replacement of diseased tissue by the use of either autogenous (or homologous) grafts or plastic prostheses.

Experience gained in the field of vascular surgery has shown that, with our present knowledge, reconstructed autogenous tissue, even though diseased, is superior to any foreign material substitute (cf. endarterectomy vs. plastic arterial grafts). This also applies to valvular surgery and it is therefore a sound priciple to surgically approach all forms of chronic aortic incompetence with this in mind.

An attempt should thus always be made at first to obtain as much corrective function as possible from the diseased leaflets by one or more plastic procedures and only then, after this has failed, as it must in some cases, should the insertion of a prosthesis be considered.

A. PLASTIC PROCEDURES.

1. Pure Aortic Incompetence.

Three broad groups of plastic procedures involving the

aortic valve may be considered:-

(a) those aiming at reduction in circumference of the aortic root

(b) those aiming at a plastic reconstruction of the valve leaflets, or

(c) those attaining conversion of the tricuspid valve into a bicuspid structure - this group being closely related to group (a).

In the first group two different operations are described.

(i) Circumclusion (Surgical constriction of the Aortic Annulus Fibrosis).

Bailey's[6,9,12] "sash" operation was first described in 1955. Various modifications have since been elaborated and developed: (Hufnagel[130] 1959). The objections to the operation being displacement of the band, either distally, with subsequent obstruction of the coronary vessels, or proximally, coming to lie over the left ventricular outflow tract and no longer providing corrective constriction in the region of the annulus. Bailey therefore advocated the use of an "internal" and "external" constricting sash. Taylor et al.,[247] (1958) described a similar operation of circumclusion, but stated that direct dissection should be carried out beneath the coronary ostia rather than involving the widespread tissue compression mentioned in connection with the "sash" operation. Proper circumclusion involves placement of this constricting band below the widest girth of the truncated base of the aorta. The

band must be low enough to reduce the area at the very base of the sinuses of Valsalva in order to afford maximum reduction of area to be occluded. Whereas Bailey advocates placement of the encircling band underneath the pulmonary conus ligament thus preventing its displacement distally, Taylor and associates regard division of the conus ligament, to expose the base of the pulmonary artery and aorta and the left coronary artery, as a vital manouevre in this operation. The latter workers do not use a Nylon "sash" but instead a specially double tapered braided silk suture tapering from 000 thickness at the ends to No. 5 thickness in the center with double swedged malleable vascular needles.

(ii) Plication.

A procedure of plication of the aortic wall adjoining the non coronary cusp using a closed technique was described by Hufnagel et al.,[131,132](1954-58). Attainment of decreased circumference of the aortic root at the level of the non coronary cusp permits the leaflets to close by decreasing the cross-sectional area at valve level.

Creech[64,65](1958) reported a clinical case utilising cardio-pulmonary bypass. Plication sutures were placed through the aortic wall at the site of the non coronary cusp thus narrowing the aortic ring with obliteration of the non coronary cusp. Hurwitt et al.,[134](1960) reported that plication of the aortic ring failed to correct aortic incompetence produced

acutely in dogs by avulsion of the non coronary cusp of the aortic valve. These workers elaborated a principle of simultaneous passage of two needles swaged on to opposite ends of a strand of silk thus permitting accurate placement of a horizontal mattress suture in a minimum of time.

The second group of operations aim at plastic reconstruction of the valve leaflets (Valvuloplasty). By suturing, the normally tricuspid aortic valve is converted into a bicuspid orifice and reinforcement and additional support is given to the lips of this bicuspid opening by suturing to its edges small pledgets of compressed polyvinyl sponge ("Ivalon"): Lillehei et al.,[165](1958). Conversion of the valve orifice to a bicuspid aperture is done by interrupted silk sutures. This adds significant support to the leaflet tissue without the concomitant creation of stenosis. The addition of these Ivalon strips corrects the tissue deficiency resulting from the curling of the cusp margins and the added weight of the Ivalon also contributes to their tighter closure during diastole. A favourable observation noted at surgery is the fact that these moderately thickened cusps hold fine silk sutures well and much better than would be true for paper-thin normal valve cusps.

The third group of operations aim at "bicuspidisation" of the aortic valve. Permanent reduction in circumference of the aortic ring by one third is attained thus permitting co-

aptation and improved function of the two remaining coronary cusps. Experimental work along these lines was done by Garamella et al.,[100,101,102,103] (1958-1959). The postero-lateral aspect of the base of the aorta corresponding to the site of the non coronary cusp is incised in the long axis for 2.5 - 3.0 cms. The non coronary cusp is then completely excised at its line of attachment. A triangular wedge of the aortic wall opposite this cusp is then excised and the aortic wall then reconstructed by interrupted sutures approximating the annular lines of attachment of the excised non coronary cusp thus forming a new commissure and a bicuspid aortic valve. Accurate apposition of the two posterior margins of the two remaining coronary cusps is essential. Whereas these studies emphasized removal of the non coronary cusp as an integral part of the procedure for fear that the prolapsed leaflet would cause aortic insufficiency, further studies (Starzl et al.,[239] 1959) suggested that excision of the leaflet was not uniformly necessary. The issue as to whether or not the leaflet must invariably be removed would appear to be of more than academic interest in assessing the potential value of the described procedure in the treatment of clinical aortic insufficiency. In patients with regurgitation the disparity between the available leaflet and orifice area seldom exceeds 25% and is usually less. Restoration of valve competence would often not require such radical rearrangement of valve structure as with total

bicuspidisation. Rather, it would seem that the treatment could consist of excision of a portion of the non coronary annulus with retention of the tricuspid valve as originally suggested by Hufnagel[132](1958). Clinical success with this method of bicuspidisation has been reported by Cooley[62](1958) and Bailey and Zimmermann[15](1959).

2. Aortic Stenosis Plus Incompetence ("Mixed Lesion").

In the mixed lesion of aortic stenosis plus incompetence, the disturbance in valve function is due mainly to the rigidity of the leaflets, without any loss of leaflet substance. The rigidity is due to commissural fusion and deposition of atheromatous and calcific material in the leaflet substance. Plastic procedures aiming at mobilisation of the leaflets by

a). commissural division (commissurotomy) and

b). removal of calciferous and atheromatous material from the leaflet substance

holds great promise. Thus a grossly abnormal valve can be restored to a normally functioning structure by this procedure (Fig. 16).

As mentioned before, the concept of plastic reconstruction of diseased valve tissue to approximate normality being superior to replacement of diseased tissue (by either grafts or plastic prostheses), is of extreme importance. Thus the surgeon should always approach the lesion of aortic incompetence

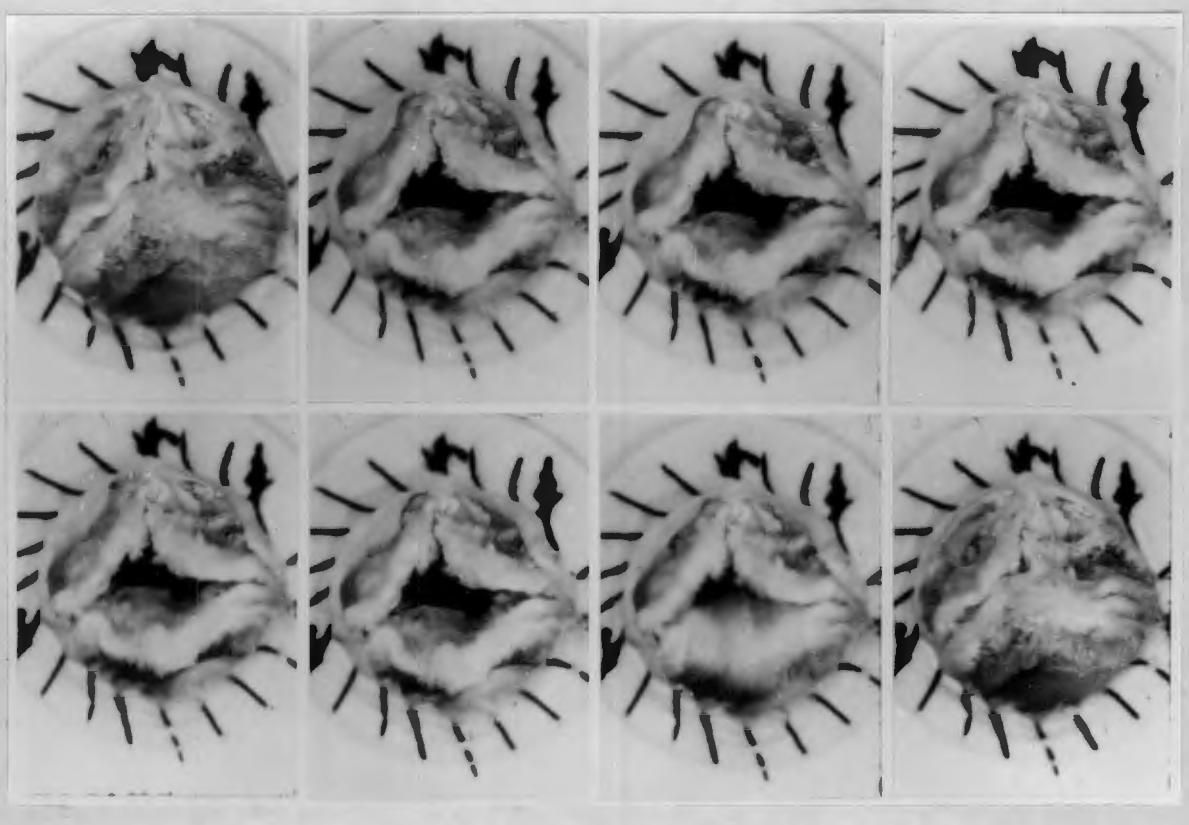


FIGURE 16.

Plastic correction of "mixed" lesion of aortic stenosis plus incompetence. Cycle of cusp movements after such a correction. (The aortic valve is the same as in Fig. 12)

with this in mind. He should attempt, at first, to obtain as much functional correction as possible by plastic procedure and only then, if this method is considered inadequate, should he consider the insertion of a prosthesis.

B. PROSTHESES.

In this chapter consideration will be given to the correction of chronic aortic valve incompetence by the placement of suitable prostheses within the cardiovascular system. Such prostheses, in order to be functionally acceptable, have to comply with certain criteria, thus demanding special methods of construction and design. Before such prostheses can be considered for clinical use extensive laboratory tests and animal experimentation must be carried out.

Various methods of construction and materials used in design will be considered and methods of in vitro, as well as

in vivo, (experimental animals) testing described.

1. Essential Requirements of a Prosthesis:-

(a) Subcoronary Placement.

At present there appears to be general agreement that the "ideal" correction of aortic regurgitation awaits the development of a prosthetic valve suitable for insertion into the root of the aorta in such a way as to be proximal to the coronary ostia thus leaving these exposed to the full force of the diastolic backflow. This latter requirement therefore being the main shortcoming of a prosthesis placed in the descending aorta: Campbell[43](1950); Hufnagel et al.,[127,128,131](1951-1955) and Ryan et al.,[216](1957).

"The creation or placement of a competent valve in the descending thoracic aorta for the treatment of aortic valvular insufficiency is of dubious value since the arterial bed proximal to this level continues to reflux a considerable part of the cardiac output. As a consequence of this inadequate correction of the dynamic disturbances, it does not provide

sufficient relief of the burden on the heart" to quote Michael E. de Bakey[73](1957-1958).

With increased left ventricular workload, due to the regurgitation of blood into the left heart, compensatory left ventricular hypertrophy ensues. Thus an increased coronary bloodflow is actually needed for effective myocardial oxygenation, even if no aortic incompetence had existed (e.g. myocardial hypertrophy associated with systemic hypertension). The causative regurgitation of the aortic valves, however, further decreases diastolic backpressure into the coronary vessels and hence a state of relative myocardial ischaemia ensues.

The placement of a competent prosthetic valve distal to the coronary ostia (in the descending aorta) thus further reduces this diastolic force and consequently further impairs the coronary circulation: Rose et al.,[211](1954) and McKusick et al.,[182](1954). The operation thus assumes the proportions of a "physiological gamble": Sarnoff and Case[223](1955). Schenk et al.,[225](1959) state that "placement of a prosthetic valve (Hufnagel type) in the descending aorta in the presence of aortic incompetence results in only slight improvement in the percentage of regurgitation (18%) and reduces the bloodflow to the brain and in all probabilities the coronary arteries".

Research in the design of an aortic valve prosthesis should thus be directed towards the development of a design suitable

for subcoronary placement. This space, anatomically available, is extremely limited and a bulky prosthesis will not be practical: (see Para. "Replacement Prostheses").

(b) Functional Adequacy.

Such a prosthesis should function "freely" under the conditions existent in the root of the aorta. Hence, a pressure gradient of 10 mm. Hg. between the left ventricular chamber and the root of the aorta should be enough to enable opening of the valve: Dreyer et al.,[83](1959). In diastole again, it should adequately correct the incompetence thus allowing the full impact of the returning blood column to act on the coronary ostia. The valve should thus act immediately there is a pressure change with commencement of return of the blood towards the heart.

(c) Duration of Function.

At 70 beats per minute the heart beats 100,800 times per day and the aortic leaflets thus open and close more than 1,000,000 times over a period of ten days. Such a prosthesis should thus be constructed out of a material which can stand up to extremes of "wear and tear" indefinitely.

(d) Tolerance.

The prosthesis must be tolerated within the circulation and not be extruded as a foreign body.

(e) Blood Destruction.

The prosthesis should not damage the formed elements of

the blood (cf. post operative anaemia with the Hufnagel valve: Sarnoff and Case[223] 1955).

(f) Blind Pocketing.

In design attention must be directed towards avoidance of blind pockets with subsequent stagnation of blood, thrombosis and resultant embolisation.

In determining whether a prosthesis complies with these criteria it is thus obvious that minute attention must be paid to:-

i. material
ii. design
iii. testing.

2. Methods.

(a) Materials.

(i) Grafts.

The use of auto- or homografts, in the correction of valvular insufficiencies, has been investigated by numerous workers.

Alexis Carrel[46] wrote in 1908: "It is proved that the remote results of the transplantation of fresh vessels can be perfect and that arteries kept for several days or weeks outside the body can be transplanted successfully, and that after more than one year the results remain excellent. It has been shown, also, for the first time, that transplanted kidneys

functionate .....". Thus the way was paved and a vast new field of surgical possibilities opened.

The replacement of diseased cardiac valves by grafted homologous tissue has been applied mostly in the field of mitral valve surgery. Thus Murray et al.,[196](1938) experimentally attacked the problem of mitral regurgitation by resection of the mitral leaflet by the cardiovalvulotome of Cutler and replacement with an autogenous vein graft. Murray[194](1950) reported that he had achieved satisfactory results in eight out of ten cases of mitral incompetence by the use of a tendon with a covering vein graft, which acted as a flutter tamponade when inserted into the left ventricle. Bailey et al.,[13](1951) reported satisfactory reduction of mitral regurgitation in six out of seven cases in which pedicle grafts of pericardium were used as artificial valves. Glenn et al.,[106](1954) attained experimental placement of a vascularised pedicle of tissue vertically through the chambers of the left side of the heart between the leaflets of the atrioventricular valve. The graft was composed of internal mammary artery and vein invaginated in a sheath composed of autogenous vein segment. They concluded: "on the basis of these experiments the cautious application of this technique to severely incapacitated patients with mitral insufficiency seems justified". However Templeton and Gibbon[248](1949) used free pericardial and vein grafts experimentally to replace a cusp of the tricuspid valve and they

concluded that the former appeared to be the more satisfactory as revealed by autopsy examination done after three weeks. However in a supplementary note to their paper they added that subsequent changes had occured and that reexamination showed the grafts to be shrunken, contracted and useless.

Production of competent valves in the descending aorta by the use of homologous tissue was attempted by various workers. Silen et al.,[234](1956) constructed a valve in the descending aorta of a dog by intussusception of a segment of the aortic wall utilising the method employed by Eiseman in the construction of a competent valve in the inferior vena cava. This valve allowed only minimal regurgitation and significantly reduced the elevated pulse pressure of experimentally induced aortic insufficiency. Ryan et al.,[216](1957) and Castro-Villagrana[48,49](1957-1958) experimentally created flap valves in the descending aorta. The former by infolding of a full thickness flap of the aortic wall and the latter by the fashioning of a flap composed of intima only. The positioning of such a valve in the descending aorta led to the editorial comment of M. E. de Bakey[73](1957-1958) (See Section "Essential Requirements of a Prosthesis" Para. Subcoronary Placement). Absolon et al.,[1](1959) described an experimental technique of autologous cardiovascular valve construction using the dog's own diaphragm, also sited in the descending aorta. The feasibility of inserting similar grafts into the mitral, tricuspid

and aortic orifices of cadaver hearts was mentioned. Lam et al.,[150](1952) investigated the suture of homologous aortic valve grafts into the descending aorta of dogs. It was technically possible to transplant such a graft into the descending aorta of the recipient animal but unless there was marked permanent insufficiency of the recipient's own aortic valve there was little chance of the grafted valve retaining its function. They found that experimental aortic regurgitation produced by an incision into one cusp was followed by rapid spontaneous restoration of function through morphologic adaptation of the remaining cusps. This phenomenon thus prevents the formulation of definite conclusions as to the fate of the valvular homograft in the presence of persistent valvular insufficiency in the host.

Bypass grafting of diseased valves was attempted by Bailey et al.,[10](1950) for the aortic valve and Bill et al.,[29](1950) for the mitral valve. The former workers attempted to bypass the stenotic aortic valve by double aortic grafts incorporating intact aortic valves bypassing the left ventricle, either directly, or via the left atrium. They concluded: "For technical, physiological and other reasons these grafts were considered inapplicable in human aortic disease". The latter workers attempted to construct a pathway from left atrium to left ventricle from a preserved segment of aorta of a donor animal and fitting it with a valve likewise taken from a donor

animal. Although technically feasible to build such a channel from grafted vessel to be joined to the ventricular wall, it was found wholly unsatisfactory to use grafted valves to allow a one way flow in such a system. Hufnagel[127](1951) reported that his attempts at ventricular-aortic anastomosis "have had little success".

Direct aortic leaflet excision and grafting was experimentally performed by Litwak et al.,[166](1952) and Gadboys et al.,[98](1958). They described a technique of approach to the non coronary cusp of the aortic valve permitting direct vision without circulatory occlusion or bypass employing a "baffle clamp". Through an aortic pouch by use of the "baffle clamp" the non coronary cusp is exposed and excised and a free autogenous flap graft, consisting of xiphoid cartilage within a pericardial envelope, is sutured in place to flap within the circulation and engage the remaining cusps during diastole. No final comment on the fate of these flap grafts could however be made. Bailey et al.,[9](1955) described the application of a "pericardio-cartilaginous stent" in aortic regurgitation, only to be employed when there is an appreciable element of coexisting stenosis, and never when the incompetence is primarily due to annular dilatation; (In the latter case he advised surgical constriction of the aortic annulus fibrosis). On the other hand he also states that such a stent "cannot be tolerated within the aortic orifice unless a significant amount

of valve mobility is present or can be restored".

Moore and Schumacker[191] (1953) however, came to the conclusion, through animal experimentation, that most transplanted tissues undergo macroscopically recognisable fibrosis and contraction in a relatively short time when left in the heart chamber, thus casting doubt on the effectiveness of using transplanted tissue for the correction of valvular lesions. These workers also, whilst agreeing that pedicle grafts (Bailey et al.,[13] 1951) should have its own blood supply and thus be more durable, however conclude that it is "unlikely that a transventricular sling of this type will remain viable and functional on the basis of its own blood supply". This they consider due to anatomic limitations encountered in fashioning the pedicle which necessitates an incision across, rather than parallel to, the principal vessels supplying the pedicle. The already compromised blood supply is further reduced by myocardial constriction at its neck, by the substantial trauma of ventricular activity and by the necessary length of the sling in order to be effective.

The use of autogenous (or homologous) graft tissue for the correction of aortic incompetence therefore seems to be unpractical.

(ii) Plastic Materials.

Following intensive research into the problem of finding suitable material for use in the construction of a prosthesis, Barnard[20](1958) came to the conclusion that a silicone rubber preparation, commercially available as "Silastic", was the best plastic material at present available for this purpose. Other workers have, however, found a substance, "polyurethane", to be conformative to all requirements: Dreyer[82](1958) and Dreyer et al.,[83](1959).

Silastic S-9711, developed in 1952 by the Dow Corning Corporation, is a clear silicone rubber which is particularly inert to body fluids and tissues. In addition, it can be heat sterilised without damage. S-9711 can be easily moulded, extruded or callendered. Recommended pressure vulcanisation conditions are 5 minutes at 260° Fahrenheit. Suggested cure is 2 hours at 480° Fahrenheit.

Typical Properties: (Cured 2 hours at 480° F.)

Colour	Clear
Specific Gravity (at 25° C.)	1.13
Hardness (Shore A Scale)	50
Tensile Strength (psi)	950
Elongation (%)	520
Tear Strength (lb/inch)	100
Compression Set (% after 22hrs. at 300° F.)	100

Brittle Point (^{o}F.) ..... -100

Stiffening Point (^{o}F.) ..... -67

Water Resistance (After 70 hrs. immersion at 212^{o}F.) Durometer change ..... -4

Water Absorbtion (% weight change) ..... +1.10

Electric Strength (Volts/mil.) .. 540

Dielectric Constant,

at 10^2 cycles 2.8

at 10^6 cycles 2.8

Dissipation Factor,

at 10^2 cycles 0.0008

at 10^6 cycles 0.0009

Effect of Oven Cure Time:-

	1 hr. at 400^{o}F.	6 hrs. at 480^{o}F.	24 hrs. at 480^{o}F.
Hardness (Shore A Scale)	45	54	57
Tensile Strength (psi)	1000	900	820
Elongation (%)	570	400	250
Tear Strength (lb/inch)	125	85	70
Compression Set (% 22 hrs. at 300^{o}F.)	100	80	65

A newer Silastic preparation, X-30-146, developed in 1959, can be vulcanised without pressure and has been employed more recently giving even better results.

The use of Silastic cemented to Ivalon, utilising Dow Corning Medical Adhesive Q-30149, in the construction of a prosthesis has been employed by C. W. Lillehei: Long et al, [169](1959). Such a prosthesis has been placed in the aortic root of a clinical case and fifteen months post operatively this patient was reported as being alive and well.

Bonding of Silastic to Teflon offers many practical problems and the use of Silastic reinforced with cloth offers a better solution. Such a material is available as Dacron Tricot coated with Silastic X-30146 (Dow Corning Corporation) and the possibility of using this material supplemented by Silastic Q-30200 holds great promise for the future of prosthesis construction.

(b) Moulding of Prostheses.

Moulding of the raw material (Silastic) into the required design is effected by a combination of pressure injection into a mould, followed by heat curing of the product.

The raw material is placed into the barrel of the special syringe and, with the aid of a vise, this is injected via a screw connection into the mould (Fig. 17). The mould, containing the raw material, is then heated at 260° Fahrenheit

for 5 minutes and the shaped prosthesis then extracted. The material is then further heat cured at 480° Fahrenheit for 120 minutes. The prosthesis can then be examined and subjected to further tests.

As the manufacturing costs of such stainless steel moulds was found to be extremely high, initial designs of prostheses were constructed in another way out of a different material. A liquid latex rubber compound ("Revultex") was used in such early designs. This milky substance was simply applied by a paintbrush to negative moulds carved by hand out of Plaster of Paris (Fig. 18). Thus different designs and various sizes of prostheses could be easily and inexpensively manufactured and tested prior to assembly of a stainless steel mould used for the Silastic material.

(c) Testing of Prostheses.

(i) In Vitro.

Part of the work in testing fabricated prosthetic valves will necessitate the study of valvular mechanics by dynamic demonstration of the action of these valves. If this is done under hydrodynamic conditions similar to those present during life, such an investigation will determine whether the valve being tested will function freely and adequately when used for the correction of valvular insufficiency. By leaving the prosthetic valve to function under these conditions for long

periods of time at speeds of 120-150 cycles per minute, "fatigue" of the material can be studied, giving useful information on how the valve will withstand the systolic and diastolic forces throughout life.

The action and motion of the valves in living experimental animals was first studied by Smith et al.,[236](1950) and Kantrowitz et al.,[138](1951). McMillan et al.,[185](1952) was the first to adequately demonstrate the value of photographic studies of valvular action in postmortem human hearts by setting up such a preparation in an artificial fluid system; the flow through which was designed to resemble that in the living heart.

McMillan[183,184](1955) and Davila et al.,[72](1956) extended this procedure to embrace studies of diseased heart valves and also to note the effects of certain operative procedures on the abnormal valves. These studies have been proven of inestimable value in judging and assessment of contemplated surgical manoeuvres.

Recently Dreyer[82,83](1958-1959) and Marx et al.,[176](1959) have also described similar type apparatuses to simulate cardiovascular action and allow the study of prosthetic heart valves.

An electrical pulse duplicator (Barnard et al.,[22]1959) is used at this school. It is capable of producing a pulsatile flow of fluid comparable in volume and pressure to the stroke of the left ventricle. This apparatus (Fig. 19a) consists of

a metal T-tube (A) 1 inch in diameter. One limb of this tube is connected through a solenoid valve (B), 1 inch in diameter, to a water tap. Water was found to be the most satisfactory perfusion fluid for photographic purposes (McMillan[185] 1952) and the water pressure in the tap provides inexpensive power for the pulsator. The second limb leads off to a drain through a second solenoid valve (C), of similar diameter. The two solenoid valves are electrically activated. The rate and systole-diastole ratio, corresponding to different rates, are controlled by two switches on control box (D). This control system is constructed in such a manner, that the one solenoid valve is open while the other is shut, and vice versa. The third limb of the metal tube is connected to the testing chambers A and B manufactured out of ¼ inch thick plexiglass (Fig. 19b). These two chambers are separated by a disc C in which the prosthesis to be tested is inserted. It is possible to unscrew chamber A from chamber B thus allowing insertion of disc C containing the test valve.

In order to observe whether the valve to be tested can be secured in the root of the aorta and whether it will function freely in this site, the aortic base with aortic annulus and leaflets together with ½ inch of ventricular musculature and the aortic leaflet of the mitral valve are excised from a post-mortem human heart (preferably from a patient who had died of aortic regurgitation) and attached between two strips of

Ivalon as shown in Fig 8. When the purpose of the test is to see how long the prosthesis will last when subjected to forces similar to those in the circulation, it is set up as shown in Fig. 19c, attached to a strip of Ivalon, overlying an opening approximately the size and shape of the aortic valve in midcycle. After the chambers have been screwed together with disc C in position it is obvious that the only connection between them is through the valve to be tested.

An inverted plastic U-tube D, 1 inch in diameter is attached to the top chamber B (Fig. 19b). The length of the ascending limb of this tube can be adjusted so that, when filled with water, the desired pressure is exerted on the valve. The descending limb is connected by way of a compressible rubber tube to the water drain. The pressure in this tube can thus be adjusted further by partial clamping of the rubber tube. This, combined with the air trapped in the descending limb, will allow of elastic resistance simulating the peripheral resistance in the human circulation.

After the testing chambers and the prosthesis to be tested have been set up as described above and connected to the pulse duplicator the inlet valve B is opened and the water tap turned on, thus filling the system with water and expelling all air. When this is complete the control box D is switched on and the systole-diastole ratio adjusted to give maximum fluid movement. The pressure changes in the two chambers (left ventricle

(chamber A) and aorta (chamber B)) are measured through connecting taps a and b, by a pressure recording device. By adjusting the water inflow, the height of the ascending limb of the inverted U-tube, and the resistance in the descending limb, an output per minute and pressure changes closely simulating those in the human circulation can be obtained. The action of the prosthesis or of a diseased aortic valve to be studied can be observed and photographed through the roof of chamber B.

Adequate testing of such a prosthesis in the pulse duplicator will thus supply answers to questions regarding the essential requirements to be fulfilled.

By simulating conditions existent in the root of the aorta it can be determined whether such a prosthesis will function freely and adequately under such conditions. If an aortic valve showing aortic incompetence can be obtained postmortem from a human heart (as we had managed on a few occasions) then pressure tracings from the two chambers will show equal "systolic" and "diastolic" levels (Fig 20a). If now a corrective prosthesis is added and pressure tracings again made, then that from chamber A will show a fall to zero while the tracing from chamber B will show the same upper "systolic" level but "diastolic" pressure will only descend to a level equal to the pressure exerted by the column of water in the ascending limb of the U-tube and the resistance of the descending limb - thus giving the difference between left ventricle and aorta during

diastole (Fig. 20 b and c). In this way the functional adequacy of the prosthesis may be objectively determined.

Duration of function also can be determined in the apparatus by fixation of the prosthesis, as described above, and then leaving the apparatus to function at speeds of 120-150 cycles per minute for long periods of time (Fig 20 d).

(ii) In Vivo.

The other most important criterion to be complied with, viz., toleration of the prosthesis within the circulation without damage to the formed element of the blood or stagnation with consequent thrombosis and embolism, can only be tested by animal experimentation. The prosthesis was therefore inserted into the descending aorta of dogs. This site was chosen due to relative ease of insertion at this site and also because the proximal flow of blood in the descending aorta during diastole, even with competent aortic valves, will result in adequate movement of the valve.

A Teflon aortic graft was used for this purpose (Fig. 21). In case of a replacement prosthesis (see later) this ring was just simply mounted inside the graft and the tube graft then sutured in the course of the descending aorta. In testing an augmentation prosthesis (see later) a Teflon ring had to be turned on the lathe out of solid Teflon. This was made to have an outer circumference exactly fitting the inner lumen of the

tube graft. In the center of this ring an aperture was cut approximating the opening in the root of the aorta with the aortic valve leaflets in mid cycle. Over this aperture then the augmentation type prosthesis was secured so as to allow a flapping movement (Fig. 21). The ring of Teflon (in the latter case) or the actual prosthesis is secured inside the graft by a circumferential silver wire suture placed around the outside of the tube graft.

The experimental dog was anaesthetised with pentothal, intubated (if necessary with flaxedil addition) and then maintained on N_2O, O_2 anaesthesia. Under strict aseptic conditions a left thoracotomy was performed and about three inches of descending aorta distal to the left subclavian artery mobilised by ligation and division of the relevant intercostal vessels. Through an incision on the left side of the neck the left common carotid was then exposed and a similar procedure completed with the right femoral artery. After heparinisation (1½ mgm/kgm body weight) the left carotid artery was cannulated proximally (i.e. towards the heart) while the distal section was clamped. Similar retrograde cannulation of the right femoral artery was performed and these two catheters then connected, via a Sigmamotor pump calibrated to flow at 300-400 cc./min., to flow in the direction from the carotid artery towards the femoral artery. This is done to perfuse the lower body of the dog during the period of aortic occlusion, to allow

insertion of the graft. Damage to the lower spinal cord, due to ischaemia, is thus prevented. Carotido-femoral bypass was then commenced and the aorta cross clamped with two clamps placed about one inch apart.

A one half inch segment of the aorta was then excised and the Teflon graft, containing the prosthesis to be tested, was sutured between the two transected ends of the aorta, ensuring that the graft was filled with saline before placing the final sutures. Release of the distal clamp prior to final completion of sutures will also allow filling of the graft lumen thus ensuring that all the entrapped air is expelled. Gradual release of the proximal clamp will allow visualisation of all grosaly bleeding areas and these can then be resutured, if necessary.

The carotido-femoral bypass was then stopped and the heparin effect neutralised with protamine sulphate given in a dosage calculated on the basis of 1 mgm protamine sulphate per 1 mgm heparin. Resuture of the carotid artery was effected after removal of the catheter but the femoral artery was ligated in continuity. The thoracotomy incision was closed in layers in routine fashion with intercostal underwater drainage.

Such animals were then daily inspected for evidence of embolisation eg. monoplegias or paraplegia. Evidence of blood destruction can be obtained by regular examination of the constituents of the blood. Sacrifice and autopsy examination

of such dogs done after variable time periods will then allow inspection of the prosthesis for signs of material "fatigue" or evidence of thrombosis.

(d.) Placement of Prostheses in Aortic Root of Experimental Animals.

The largest available mongrel dogs were employed for these experiments, as it was found that only in these was the root of the aorta of a suitable size to permit the performance of a surgical procedure. The dog was anaesthetised with I.V.I. pentothal and intratracheal intubation performed. A 5% Dextrose/Water intravenous infusion was maintained during the operation. The dog was placed to lie on its left side and a right thoracotomy approach employed resecting the 4th or 5th rib. Careful haemostasis was ensured during these early stages of the operation. After opening the pericardial sac anterior and parallel to the right phrenic nerve the pericardium was divided cephalad until $\pm 1\frac{1}{2}$ inches of aortic base became visible. Careful haemostasis of the pericardial edge at this stage is essential. The adventitial fat pad overlying the anterior aortic surface is also dissected off at this stage. A tape is passed around the aorta proximal to the roots of the big vessels and an occlusive clamp is placed around the aorta, but this is not yet tightened at this stage.

The azygos vein is then ligated and the pericardial edges

retracted with traction sutures. The left femoral artery is then exposed high in the groin and after heparinisation (1.5 mgm/kgm body weight) a Bardic catheter No. 14 or 16 was passed in a retrograde fashion upwards into the femoral artery. This was then connected to the arterial limb of the cardio-pulmonary bypass oxygenator. The type of oxygenator employed in all experiments was the Helical reservoir bubble oxygenator (DeWall et al.,[77,78,79] 1956-1957).

Venous catheters were then placed. Firstly the superior vena cava and inferior vena cava are encircled with tapes and occlusive rubbers as described by Barnard et al.,[21,23] (1958-1959). The superior vena caval catheter in these cases however, was passed through the right auricular appendage and the inferior vena caval catheter through the side wall of the right atrium. In addition a coronary sinus suction catheter, into the right atrium, was placed through a stab incision encircled by a purse string suture in the wall of the right atrium.

Cardiopulmonary bypass (Fig. 22) was then commenced and, after cross clamping of the aorta, an arteriotomy incision was made along the anterior surface of the aorta. Myocardial oxygenation was effected by a variety of methods (see later). 6 0 black silk sutures were then placed along the margins of the aortotomy incision to be used as stay sutures, thus allowing visualisation of the aortic valve leaflets. The aortic valve cusps can then be accurately defined and the posterior (non

coronary) sinus identified. Aortic incompetence was then produced by plication of the free margins of the leaflets at the site of the lunulae (Fig. 23).

A suitably sized prosthesis is then selected and preloaded with 3 black silk double-needled 3 0 sutures . The sutures are then passed through the aortic wall adjacent to the posterior (non coronary) sinus of Valsalva, and carefully tightened. The sutures are then carefully tied over strips of Ivalon outside the aortic wall. In these experiments only "augmentation" type prostheses (see later) were inserted. These were placed in such a way that the "base section" of the prosthesis (vide infra) was positioned within the posterior (non coronary) aortic sinus (Fig. 24). Having ensured accurate and correct positioning of the prosthesis, the aortotomy incision is then carefully resutured with 6 0 black silk sutures ensuring that all entrapped air is expelled from the left ventricular cavity and the proximal aorta.

Decompression of the left side of the heart at this stage is extremely important and performed through, either a left auricular catheter, or a catheter through the upper end of the aortotomy into the left ventricle. Ventricular fibrillation was usual at this stage and was corrected by electrical defibrillation. After resumption of normal sinus rythym the aortic clamp occluding left ventricluar outflow is released. If heartbeat continues satisfactorily, a final phase of "partial

cardiopulmonary bypass" (i.e. release of occluding tapes around the venous catheters) is maintained for about three to four minutes, and then, if aortic pressure is satisfactorily maintained bypass completely discontinued. After meticulous haemostasis the thoracotomy incision is carefully resutured in layers with intercostal underwater drainage of the pleural cavity.

Various methods of myocardial maintenance were employed during the period of aortic occlusion (by cross clamping):-

(a) Anoxic arrest

(b) Retrograde coronary sinus perfusion

(c) Prograde coronary artery perfusion

(d) Local myocardial hypothermia

(e) Total body cooling

Potassium induced asystole was not used in the series as canine myocardium is known to be notoriously sensitive to potassium.

(a) Anoxic arrest - no survivors were obtained beyond 2-3 hours post-operatively and the incidence of ventricular fibrillation was 100%. In some cases too, myocardial activity would not return at all after resumption of oxygenation. Periods of aortic cross clamping varied from 20-30 minutes.

(b) Retrograde coronary sinus perfusion was used in most cases. A No. 12 or 14 Foley's catheter is inserted into the coronary sinus opening in the right atrium and the bulb

inflated with 5ccs. water. Perfusion is maintained by gravity flow from a well ±40 - 50cms above the level of the heart: Gott et al.,[112,113](1957); Lillehei et al.,[163](1956). Immediately the aortotomy is closed this catheter must be removed and right atrial suction instituted. Retrograde coronary sinus perfusion however reaches principally only the left side of the heart, and the problem of cannula fixation remains incompletely solved: Shumway[232](1959). Distortions in the heartbeat induced by reversal of the coronary circulation also detracts from the use of this technique: Massimo and Boffi[177,178](1958).

(c) Prograde coronary artery perfusion (Kay et al.,[141](1958) is performed by cannulating the left coronary ostium only; adequate communications between left and right coronary systems ensure that total mycardial oxygenation is adequate. A disadvantage of this method is the addition of a catheter into an already confined operating space. Also, securing the catheter in position and maintaining it in position is a problem; however, this has been subsequently solved. Shumway[232](1959) states that "individual cannulation of the coronary ostia after prompt emptying of the heart by means of a left atrial catheter supplies an acceptable method for maintenance of cardiac circulation during direct approaches to the aortic valve".

(d) Local myocardial hypothermia: Gott et al.,[111](1959); Grow et al.,[118](1956) and Shumway et al.,[233](1955).

After commencement of cardiopulmonary bypass a right atrial catheter,to allow of coronary sinus drainage, is placed. The aorta is then cross clamped proximal to the large vessel roots. By means of a sharply curved needle as described by Roe[208] (1958) a puncture is made in the anterior aortic wall and perfusion of cold blood (2^{o} - 5^{o}C.) through the coronary vessels commenced (Fig. 25). Cold blood was obtained by circulating blood through the cooling Circuit A for about ten minutes before use, i.e. after cross clamping of the aorta. Cardiac slowing and eventual arrest, invariably occured within the space of ten minutes. Coronary perfusion was then stopped, the curved needle removed, and anterior aortotomy performed to expose the aortic leaflets.

After closure of the aortotomy and release of the aortic clamp the flow of warm oxygenated blood through the coronaries will again reactivate myocardial activity. Initial ventricular fibrillation invariably reverted again to sinus rythym on regainder of preoperative body temperature. Again we were unable to obtain long term survivors in dogs operated on in this manner.

(e) Total body cooling to extreme low temperature ranges (8^{o} - 10^{o}C. body temperature) was obtained by use of a heat exchanger in the arterial circuit of the heart-lung machine

(Fig. 26). Myocardial standstill, obtained within about 15 - 20 minutes, allowed cross clamping of the aorta. Cardiopulmonary bypass was then ceased completely. This was followed by anterior aortotomy and insertion of the prosthesis. After resuture of the aortotomy, the aortic clamp was removed and warm blood (39° - 41°C.) again perfused through the total body by cardiopulmonary bypass oxygenator. This rewarming was effected by again using a warm fluid in the heat exchanger.

The heat exchanger used is a modification of the Brown-Smith-Emmons type heat exchanger[39](1958), (Seally, Brown et al., [227]1958), and consists of 16 straight stainless steel seamless tubes with a wall thickness of 0.5mm throughout and length 24 inches having been highly polished on the outer surface. These are joined at the ends by two reservoirs to allow inflow and outflow. The entire unit is then encased in a length of Tygon tubing with inner diameter of 1½ inches and this clamped securely at both ends. Blood now flows inside the Tygon shell around the stainless steel tubes inside of which a coolant (or heating fluid) is circulated. Blood flow occurs in only one direction and if stood upright, the upper portion will serve as a bubble trap to collect all possible gases released on reheating (Figs. 27 and 28). By these various experimental methods then, employed in vitro as well as in the living animal, prostheses may be adequately tested and assessed with a view to functional adequacy.

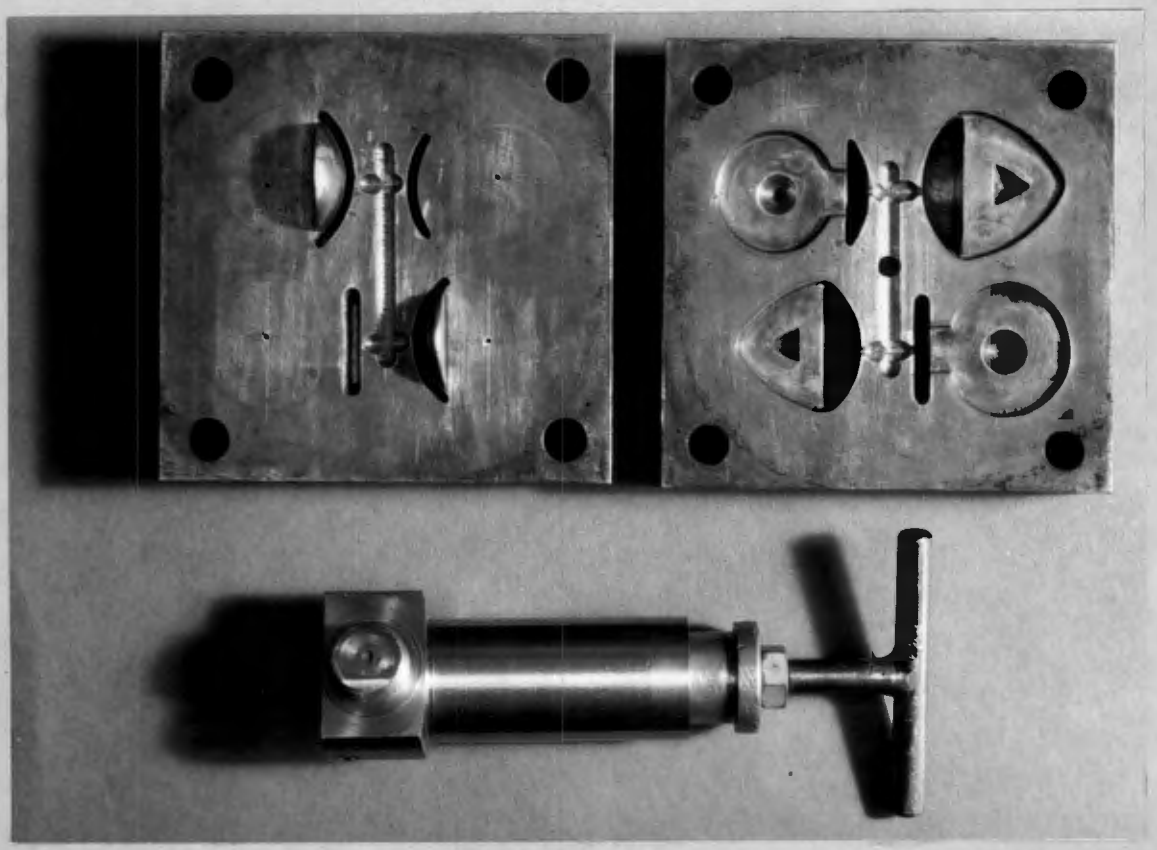


FIGURE 17.

Stainless steel mould and syringe for manufacture of Silastic Prostheses.

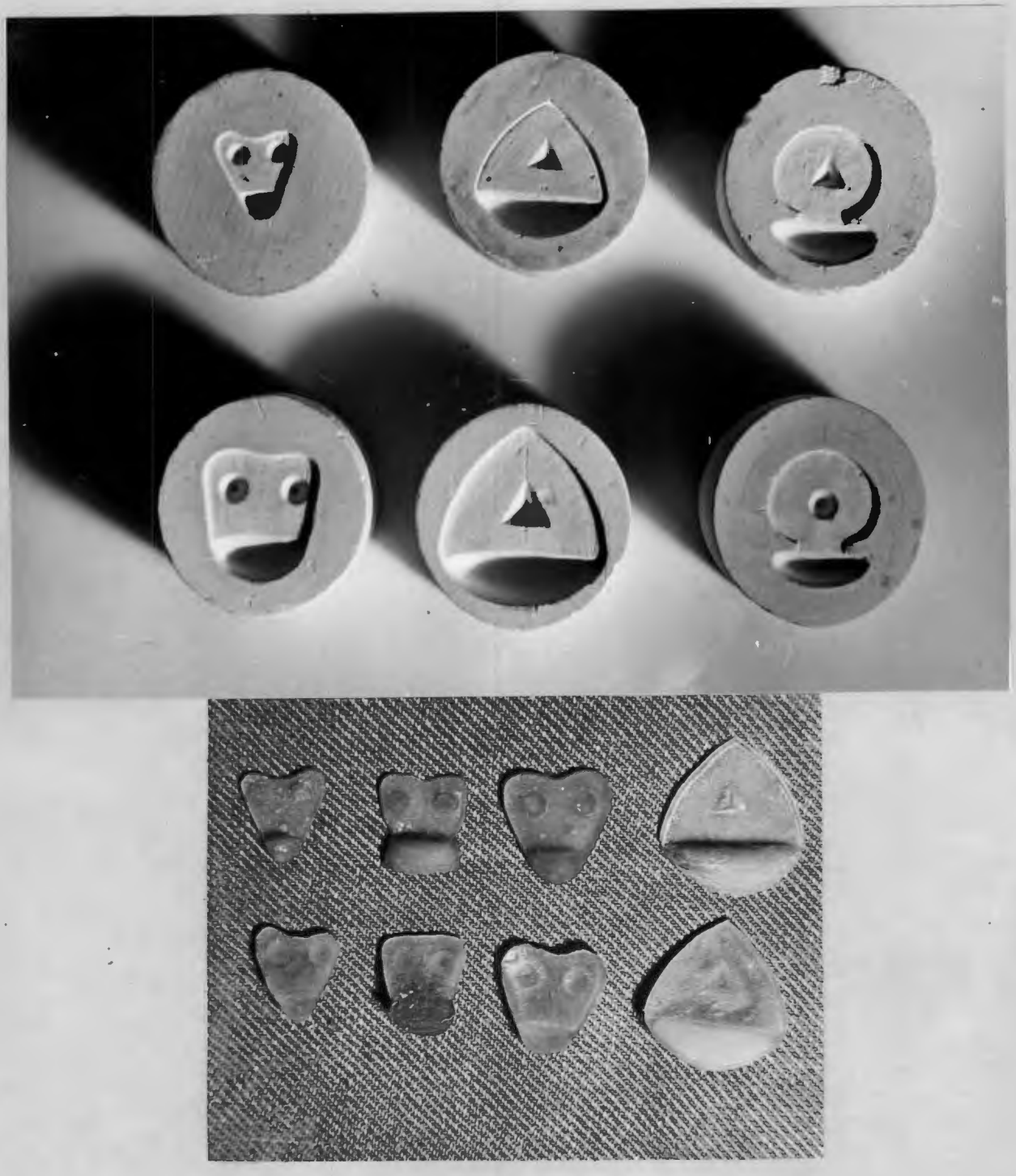


FIGURE 18.

(a) Plaster of Paris moulds for manufacturing of "Revultex" prostheses.

(b) Early designs of "Revultex" augmentation prostheses.

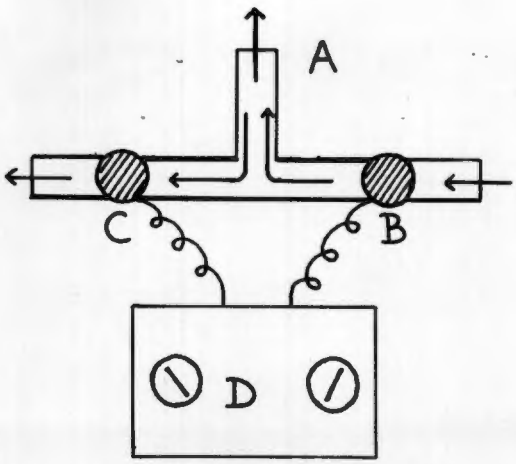


FIG.1.

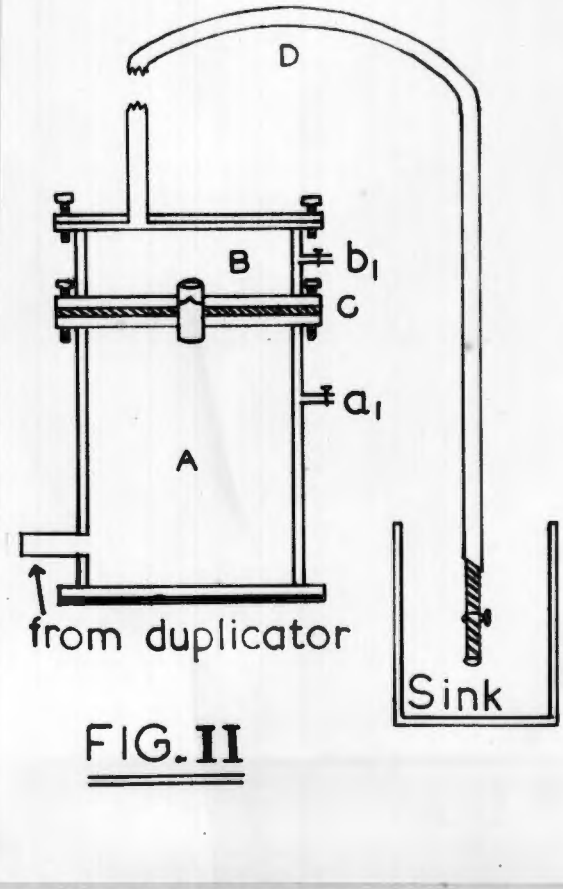


FIG. II

FIGURE 19.

The electrical pulse duplicator.

(a) Solenoid valves and control box.

(b) Testing chambers - left ventricle (chamber A); aorta (chamber B).

P.T.O.

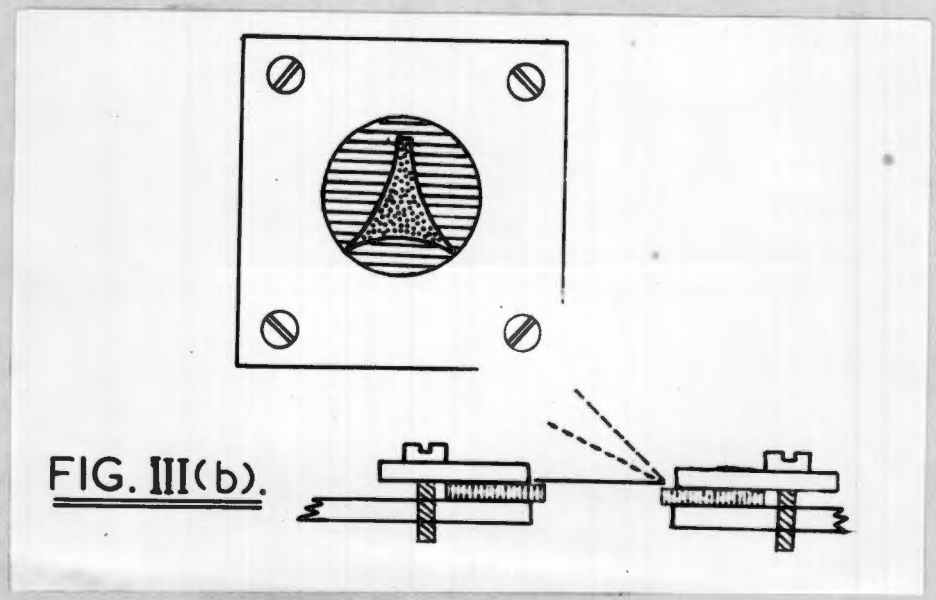


FIGURE 19. (contd.)

(c) Fixation of augmentation type prosthesis to assess effects of prolonged use.

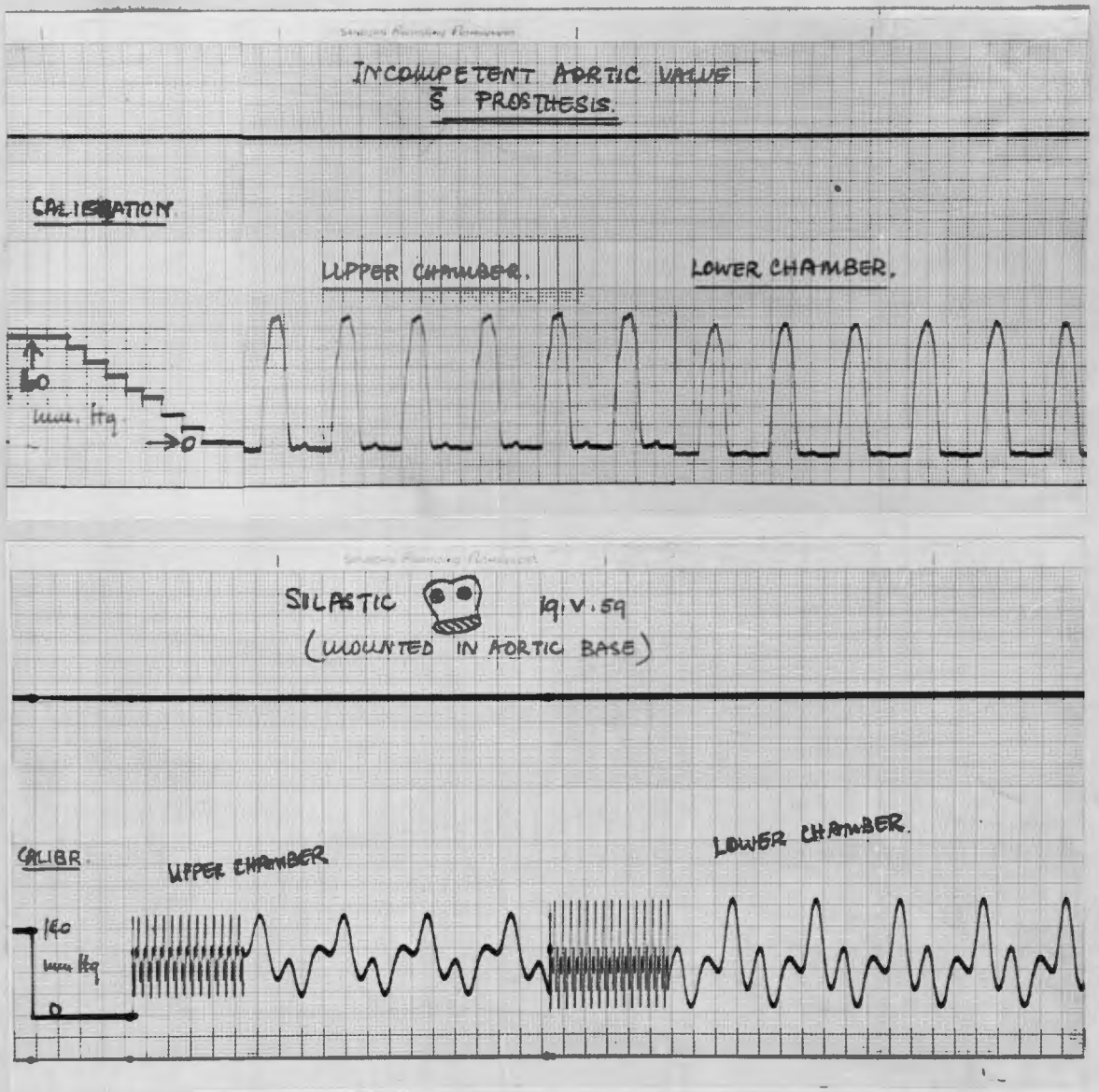


FIGURE 20.

Pulse duplicator tracings:

(a) Incompetent aortic valve

(b) Incomplete correction of incompetence by prosthesis of an early design.

P.T.O.

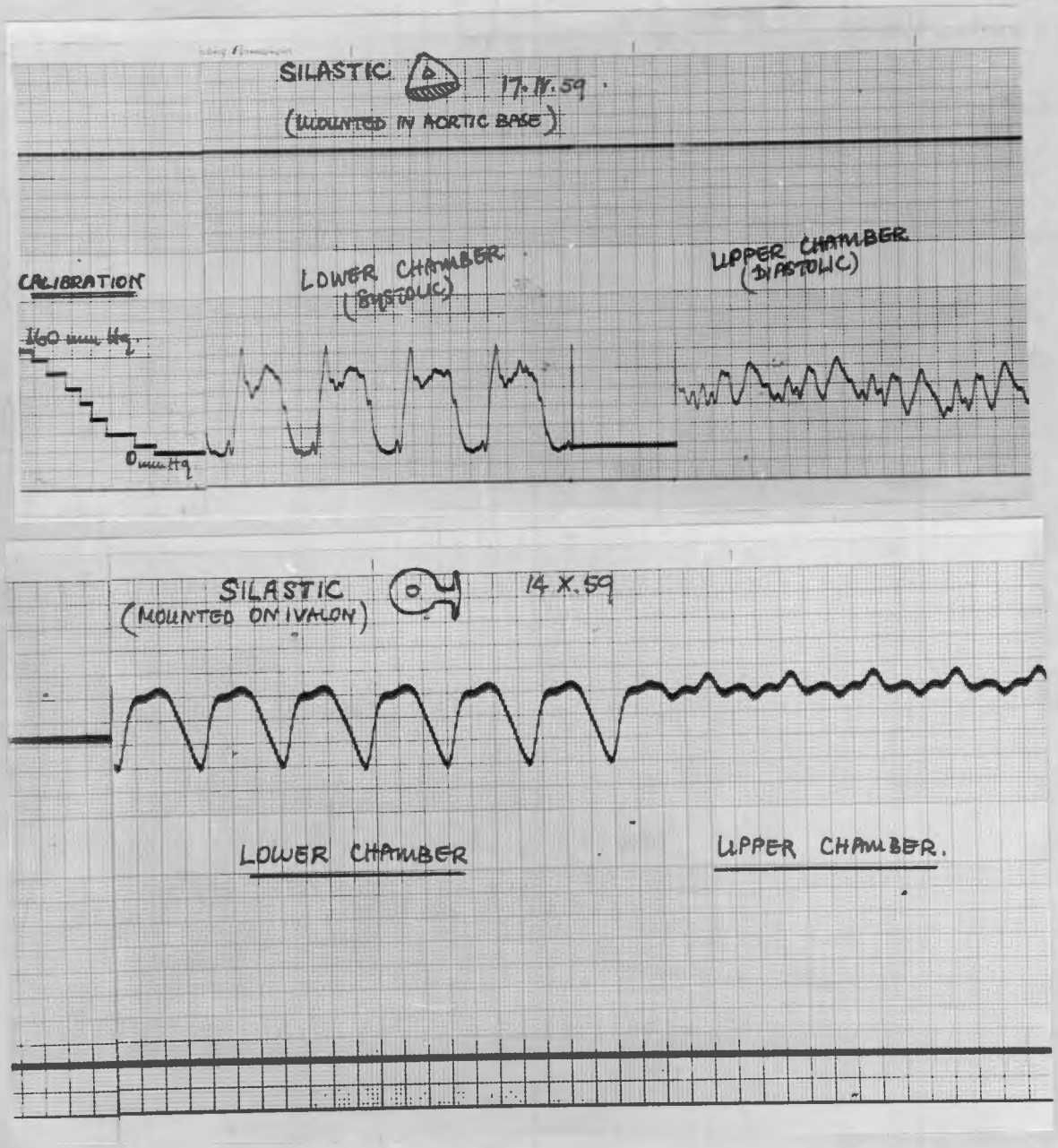


FIGURE 20. (contd.)

(c) Complete correction of incompetence by prosthesis of triangular design.

(d) Theoretical absolute correction obtained with prosthesis mounted on Ivalon base.

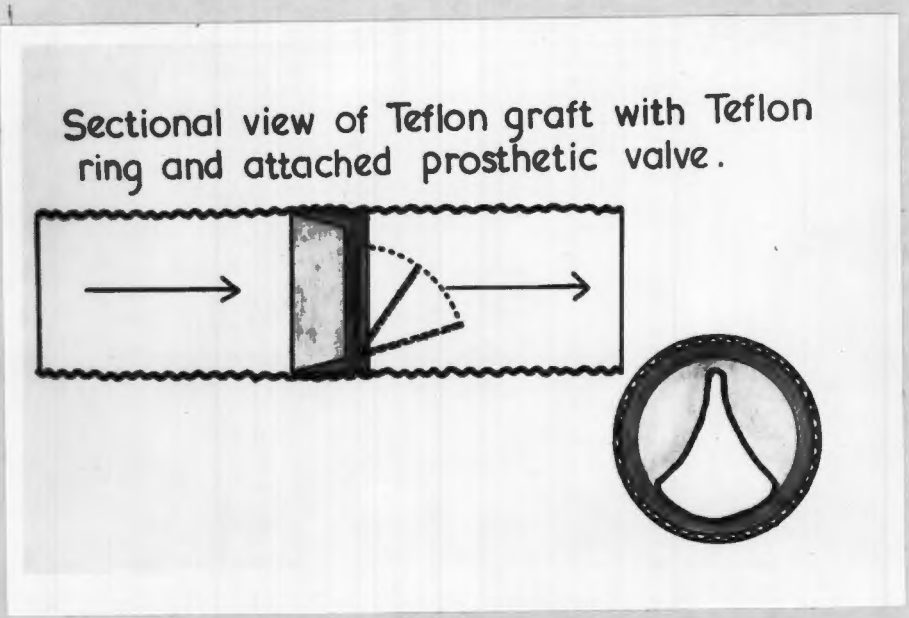


FIGURE 21.

In vitro testing of prosthesis by incorporation into Teflon graft to be sutured in the descending aorta.

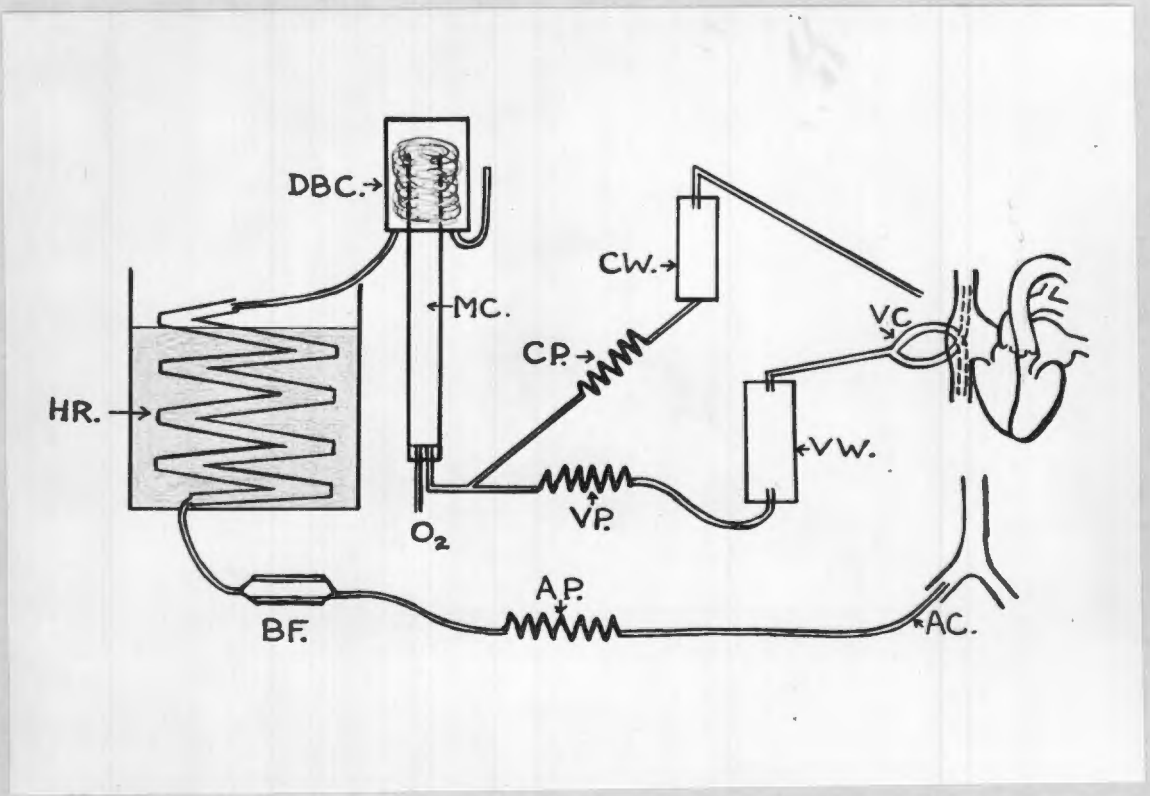


FIGURE 22.

Diagram of total cardiopulmonary bypass circuit.

V.C. - Venous catheters
C.W. - Cardiotomy well
V.P. - Venous pump
M.C. - Mixing chamber
H.R. - Helical reservoir
A.P. - Arterial pump
V.W. - Venous well
C.P. - Cardiotomy pump
O_2 - Oxygen supply
D.B.C. - Debubbling chamber
B.F. - Blood filter
A.C. - Arterial catheter

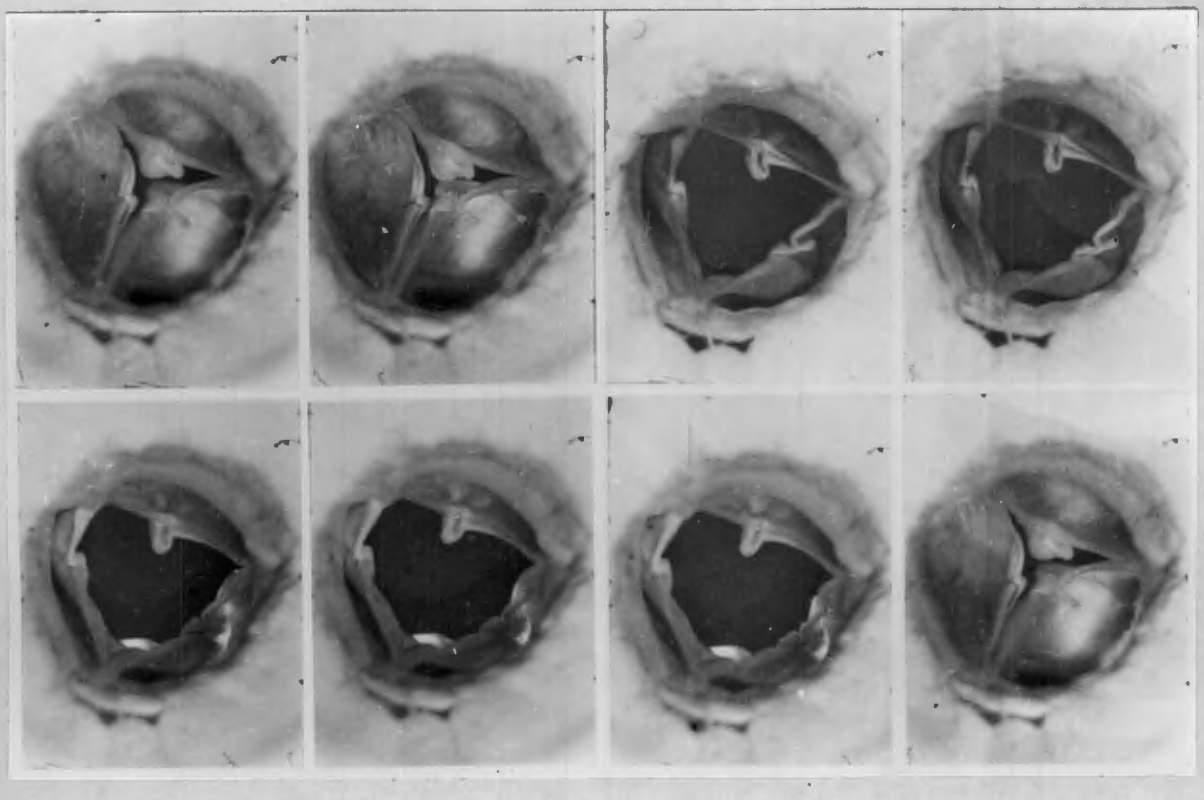


FIGURE 23.

Cycle of cusp movement in experimentally induced aortic incompetence by plication of leaflets.

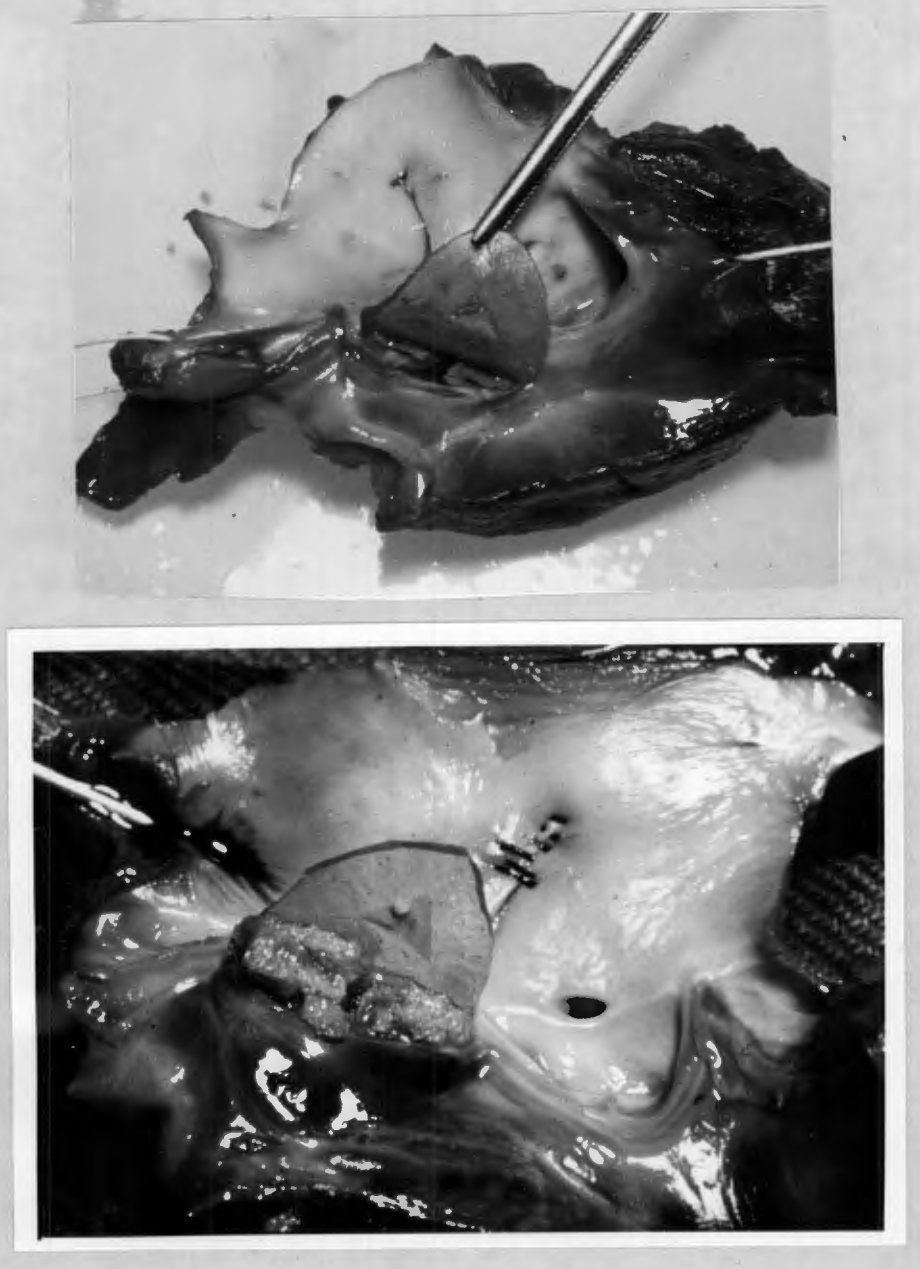


FIGURE 24.

Augmentation type prostheses experimentally placed within the aortic root of dogs.

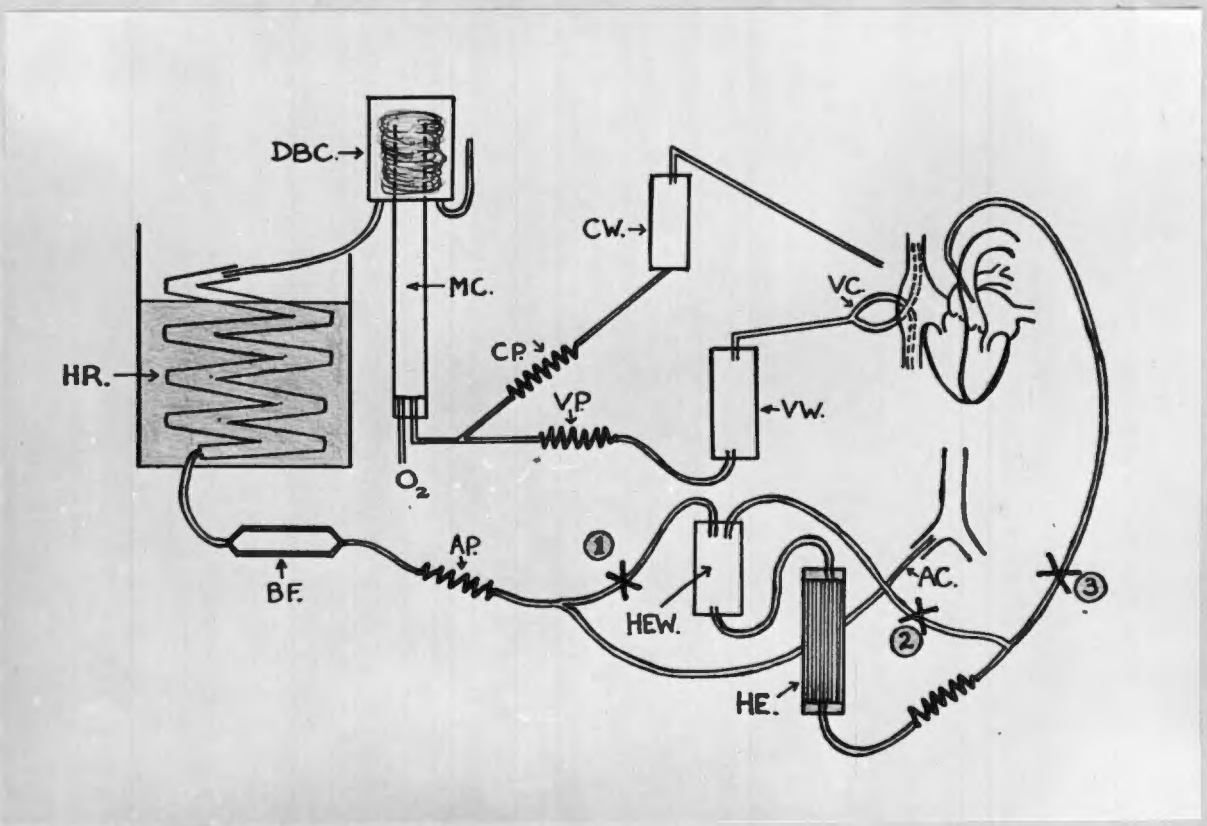


FIGURE 25.

Diagram of cardiopulmonary bypass circuit employed to obtain local myocardial hypothermia.

Legend similar to Fig. 22.

(1), (2) and (3) control sites of coronary perfusion circulation.

H.E.W. - Heat exchanger well

H.E. - Heat exchanger

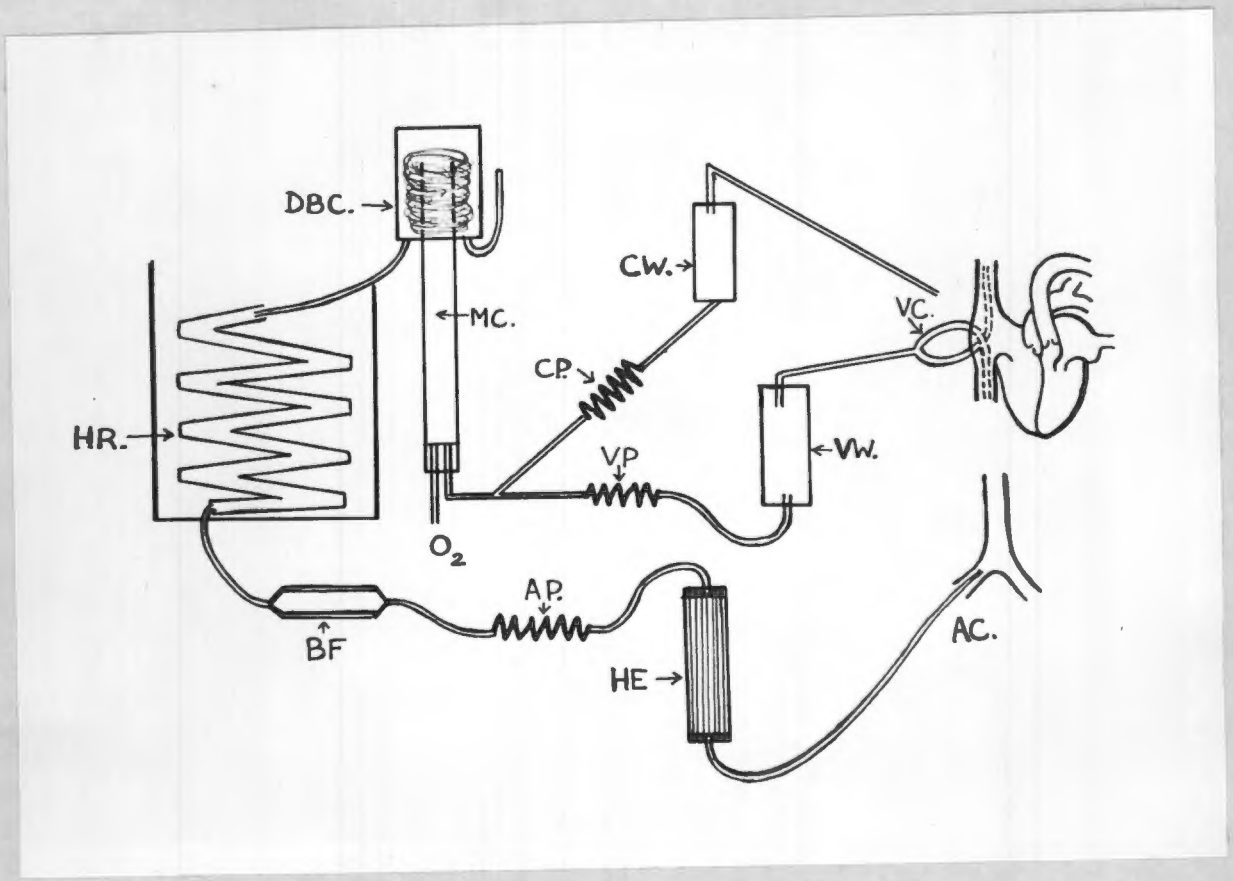


FIGURE 26.

Diagram of cardiopulmonary bypass circuit employed to obtain total body cooling.

Legend similar to Figs. 22 and 25.

Note absence of water bath (T. 39-41°F.) usually employed with helical reservoir.

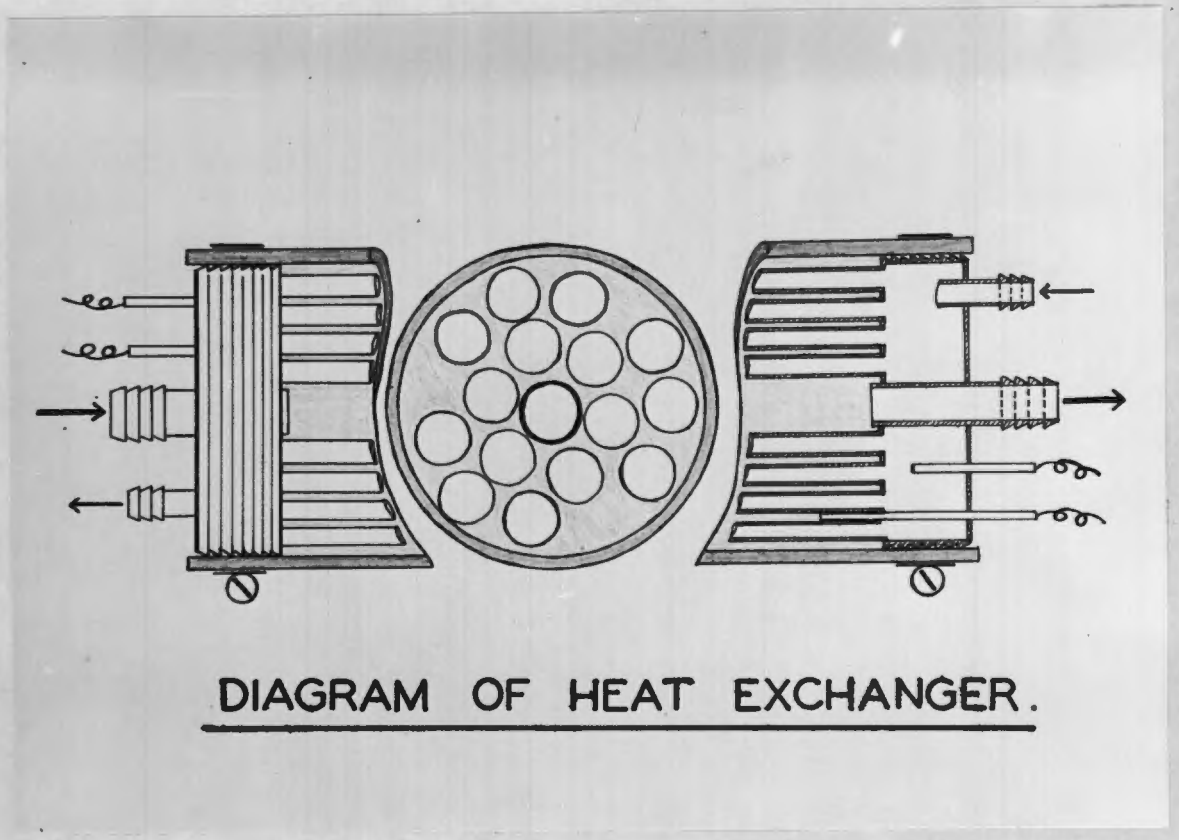


FIGURE 27.

Diagram of Heat Exchanger unit.

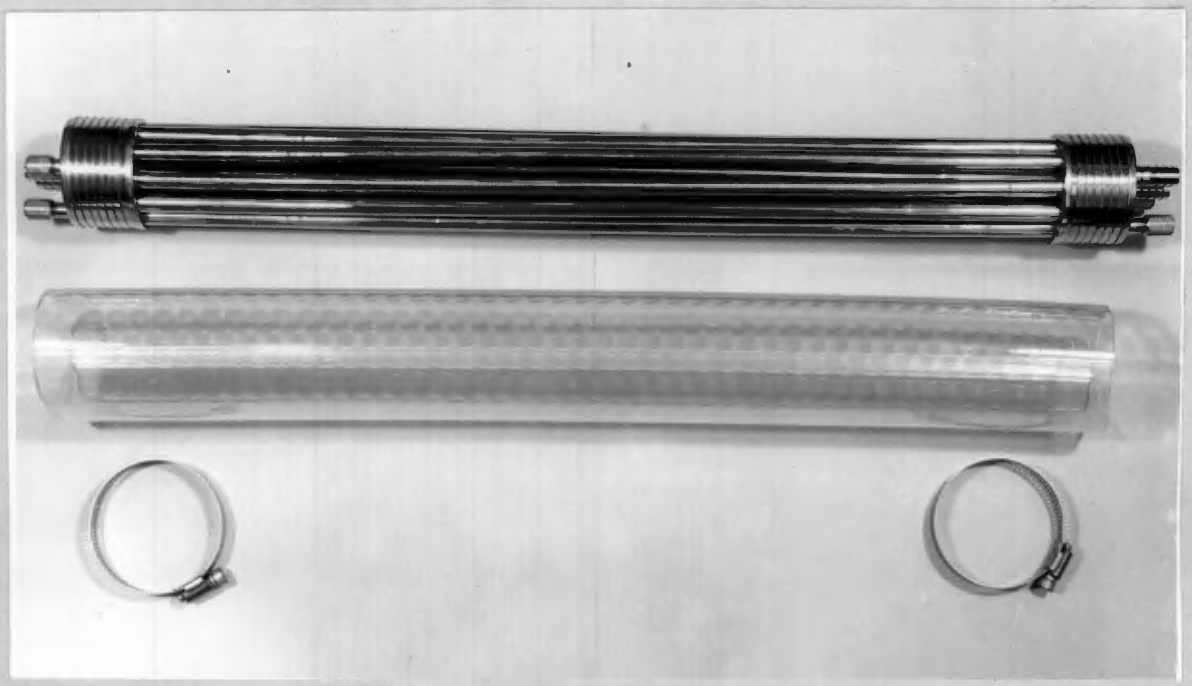


FIGURE 28.

Photograph of Heat Exchanger unit.

3. Prosthesis Designs.

Prostheses designed to be placed in the root of the aorta proximal to the coronary ostia may be of two types; either (a) replacement or (b) augmentation prostheses (Fig. 29).

(a) Replacement Prostheses.

Such prostheses aim at completely replacing the aortic valve leaflets anatomically and taking over their function. This can be attained by either excising the diseased aortic valve cusps or by placing the prosthesis within the root of the aorta inside the aortic leaflets to compress these outwards. Excision of aortic valve cusps is a rather hazardous procedure as the aortic leaflet of the mitral valve, forming an integral part of the ventricular outflow tract, is intimately attached to the two posterior aortic valve cusps (Fig. 4). In excising these aortic leaflets therefore, damage to mitral valve action seems inevitable. In the latter procedure again one can conceive that in such a case as depicted in Figs. 11 and 13, with some thickening and calcification of the valve cusps associated with some stenosis, a prosthesis considerably smaller than the aortic root will have to be employed thus constituting the real danger of producing some degree of aortic stenosis.

Numerous such replacement prostheses have been described

being of three main types (Fig. 30):-

i) Cusped and Flutter valves: Malette *et al.*,[173](1956) and Long *et al.*,[169](1959)

ii) Flap type valve eg. Neuman-Bailey Polywog valve: Bailey and Likoff[12](1955)

iii) Ball valve: Hufnagel[127](1951) and Edwards and Smith [87](1958).

Three types of replacement prostheses of type i) were manufactured and tested by Barnard[20](1958), these being:-

1. Mylar 3 cusp valve
2. Silastic 2 cusp butterfly valve
3. Silastic 3 cusp valve (Fig. 31)

The Silastic 3 cusp valve was found, on testing in the pulse duplicator, to be the most successful and resembling closely the architecture of the normal aortic valve. Major disadvantages were, however, encountered in placing this prosthesis in the aortic root proximal to the coronary ostia:

a) due to mere bulk of prosthesis, difficulties in placement and securing in position were encountered;

b) pocketing of artificial valve cusps allows of blood stagnation with resultant clotting and embolisation phenomena;

c) prolonged tests invariably produced tearing of the leaflets.

Presently available materials and designs of replacement prostheses therefore tend to contraindicate this type of

prosthesis and another method of correction of incompetence must be attempted: Barnard[20](1958).

(b) Augmentation Prostheses.

This possibility of a prosthesis to augment rather than replace the function of some diseased valves was considered after it had been observed at autopsy that the valves of patients dying from pure incompetence still possessed a large amount of mobile functioning leaflet substance (Fig. 14). It therefore appeared logical in such cases to augment the function of the valve rather than to replace it completely. The leak during diastole in these cases occurred from the triangular shaped opening, "the aperture of incompetence", which exists between the cusps and, to produce competence again, the augmentation prosthesis only had to close this orifice. Thus valve incompetence was produced in the pure form of aortic incompetence by reduction of the amount of functioning leaflet substance with a resultant central regurgitant opening during diastole and correction of this regurgitant jet would attain complete competence. A flap type valve anchored to the aortic wall above the aortic cusps would thus seem to fulfil all requirements (Fig. 32).

An original design of such a flap valve prosthesis consisted of a central round disc thicker in the center than at the periphery joined by a flat arm to a cross bar, this latter

to be attached to the inner aspect of the arterial wall immediately above the non coronary (posterior) cusp. In diastole the flap section would be seated on the partially closed aortic leaflets thus allowing the central convex section to be pushed into the existing defect by diastolic back pressure in this way preventing blood from regurgitating backwards into the left ventricle.

Measurement of this flap valve type prosthesis depends on the size of the defect to be corrected and on the diameter of the root of the aorta. The thinner periphery of the disc should be wide enough to overlap the defect so that it may be caught by the cusp margins thus preventing prolapse of the flap through into the ventricle during diastole. The thinner periphery would also to some extent aid in sealing off the slit-like defects extending along the cusp margins towards the commissures.

The distance from the center of the flap section to the cross bar of the prosthesis, to be attached to the aortic wall, should be exactly equal to the radius of the root of the aorta, thus ensuring accurate positioning of the flap over the defect. Adequate correction of regurgitation can be obtained by a flap valve measuring in length (i.e. from cross bar to tip of flap section) three quarters of the diameter of the root of the aorta. This allows free and adequate flap action and in diastole exposure of a major portion of the sinuses of Valsalva to

allow free bloodflow into the left and right coronary arteries. Fixation of the cross bar to the aortic wall was effected by sutures passed through the wall and tied over Ivalon strips outside the wall (Fig. 38). Longevity of this type of valve will depend on whether the hinge arm will allow millions of flexions. In testing these flexions it was shown that valves flexed up to 100,000,000 times showed no signs of wear and tear and it was concluded that the flap valve would probably last for many years without breaking: Barnard[20](1958).

Adequacy of function was tested in the pulse duplicator by suturing such a prosthesis within a postmortem aortic root specimen of a patient who had died of aortic incompetence (Fig. 8).

From these results it was concluded that the regurgitant leak could be adequately corrected by this type of prosthesis (Fig. 20 c). The valve could last for a sufficient time period to make its insertion of practical value.

In practice however, precise placement of such a crossbar to allow accurate flap action with overlap of the defect was found to be extremely difficult. If the crossbar is not positioned correctly the flap will close at an angle with resultant incomplete correction of regurgitation.

Further designs along these lines were developed, resulting in the formation of a base section, the shape of the non

coronary cusp in the diastolic position. If this base can then be positioned and secured within the non coronary sinus, the flap section of the prosthesis must seat correctly during diastole. This correct placement is simplified by careful suturing of the corners of the base at the cusp commissures. Several flap section designs were expounded and tested (Fig. 33 a and b) but finally a triangular flap section was found to correct the regurgitation most adequately (Fig. 20 c) - thus also including correction of the peripheral slit-like defects between the cusp margins. This prosthesis therefore consisted of a solid base section moulded to fit inside the non coronary aortic cusp with a triangular shaped flap projecting from the upper aspect of the base section. Centrally placed on the lower surface of the flap, positioned opposite the regurgitant aperture, was a projection which would serve to guide the flap into the correct position during diastole (Figs. 33 a, 34 and 35).

Studies of normal aortic valvular function revealed that when the leaflets are approximated (as in diastole), the center of the free edge of each cusp (i.e. the area of the nodulae) is at a slightly lower position than the commissures (Fig. 5). Due to prolapse and downward retraction of the cusps in free aortic regurgitation this difference is further accentuated. It follows therefore that if a flap type prosthesis is designed with the flap section arising from the upper aspect of the base

section, and this prosthesis is placed as described above, the flap will move past the horisontal position of the valve during diastole, before it can seat on the aortic cusps, and this leaves an opening at the base of the valve through which the blood can leak (Fig. 36). The flap section should therefore join the base section at nodular level and not at commissural level.

Further, studying the action of such prostheses with solid base sections in the pulse duplicator, it was soon apparent that the upward movement of the flap during systole was inadequate and would cause some interference with propulsion of blood from the ventricle. This led to the construction of a prosthesis with a hollow base section the shape of the non coronary cusp (Figs. 34 and 37). Adequate placement could still be ensured by suturing the outer margin of the base to the inner aspect of the aortic wall as before (Fig. 38). In practice, however, the advantage of increased flap action was outweighed by the disadvantage of the non-emptying of this pocket, allowing stagnation of blood and subsequent thrombosis and embolisation. It was thus decided to fabricate a prosthesis with a thinner base, thus allowing more movement of the flap section yet still maintaining the advantage of positioning the base in the non coronary sinus.

Further observations revealed that movement of this triangular type of prosthesis, when the flap is molded to join

the base at a lower level (i.e. nodular level), will disturb the rounded contour of the free margin of the non coronary cusp. This will further distort the already abnormal anatomy and further interfere with valve closure. A narrow shoulder to the flap section thus seems essential and a triangular design was found impractical.

Another design of flap type prosthesis was then embarked on, remembering all the requirements to be fulfilled viz.:

a) a thin solid base section slightly rounded to fit the contour of the aorta

b) a flap section joining the base at a lower level (remembering the difference between commissural and nodular levels) consisting of a shoulder and a circular flat section with a central downward projection (Fig. 39).

Testing of such prostheses in the pulse duplicator (Fig. 40) revealed that:-

i) the valve functioned freely under conditions similar to those existent in the root of the aorta of a patient suffering from aortic insufficiency

ii) the regurgitant leak could be adequately controlled by this type of prosthesis

iii) the valve would last indefinitely

iv) accurate subcoronary placement was possible allowing free bloodflow into the coronaries during diastole

v) insertion of such valves mounted on a Teflon ring in the descending aorta of dogs produced no evidence of blood destruction or thrombosis and embolisation phenomena.

This design of augmentation prosthesis, although functionally adequate, did not, however, fulfil all requirements mainly because the base section composed of Silastic had to hold sutures. Even though these could be placed over small Ivalon pledgets, as indicated in Fig. 38, it was not considered safe. The possibility of an Ivalon base cemented to a Silastic flap section was considered but early supplies of Dow Corning Medical Adhesive were ineffective.

A further design in flap type augmentation prosthesis was then developed (Fig. 42). This consists of a base section which can be sutured directly to the arterial surface of the posterior (non coronary) aortic leaflet. This design is favoured by several features already noted in the lesion of pure aortic incompetence, viz.:-

a) Cusp retraction and thickening of the free edge result in the production of a cusp margin which is relatively straight (cf. curved free cusp margin of normal leaflet). This feature is well illustrated in Figs. 10 and 14. Thus, if the base section of the prosthesis is directly attached to the cusp, easy flap movement is allowed (Fig. 42).

b) Adequate valve opening, during ventricular systole, will be allowed by flap movement combined with mobility of the leaflet.

c) Fibrotic changes occuring in the pathological valve cusp renders "better cusp or leaflet tissue remaining with

which to work": Lillehei et al.,[165](1958). Whereas normal leaflet tissue is paper thin and tears readily when sutured, these diseased leaflets will hold sutures easily.

d) Closer proximity of the "aperture of incompetence", to be corrected, to the site of prosthesis fixation renders the chance of incorrect prosthesis placement more unlikely.

A prosthesis design, as depicted in the diagram (Fig.42), was therefore experimentally tested. The base section, shaped slightly at an angle to the flap section, is designed to be sutured to the diseased non coronary cusp by three or four mattress sutures of 5 0 or 6 0 black silk. The convex projection, on the undersurface of the flap section, designed to fall into the incompetent aperture, was placed at a distance of 0.3 - 0.5 cms. from the line of junction between flap and base sections.

Theoretically a circular - shaped prosthesis would be ideal, but final shaping of the flap section should be done by the surgeon at the operating table after accurate and critical assessment of the size of the diseased aortic valve.

As pointed out previously, the use of Silastic alone in the construction of a prosthesis has the disadvantage in that this material does not effectively hold sutures. The possibility of bonding Teflon to Silastic was considered, but this entails numerous complex chemical problems: (S.A. Braley - Dow

Corning Medical Research Center). Construction of such a prosthesis out of Dacron Tricot coated with Silastic X-30146 supplemented by Silastic Q-30200 holds great promise and is the subject of further experimentation.

An augmentation prosthesis of the type illustrated in Fig. 42 has been used clinically at Groote Schuur Hospital, Cape Town (Dr. C.N.Barnard). Fig. 43 illustrates the pre- and postoperative pressure tracings.

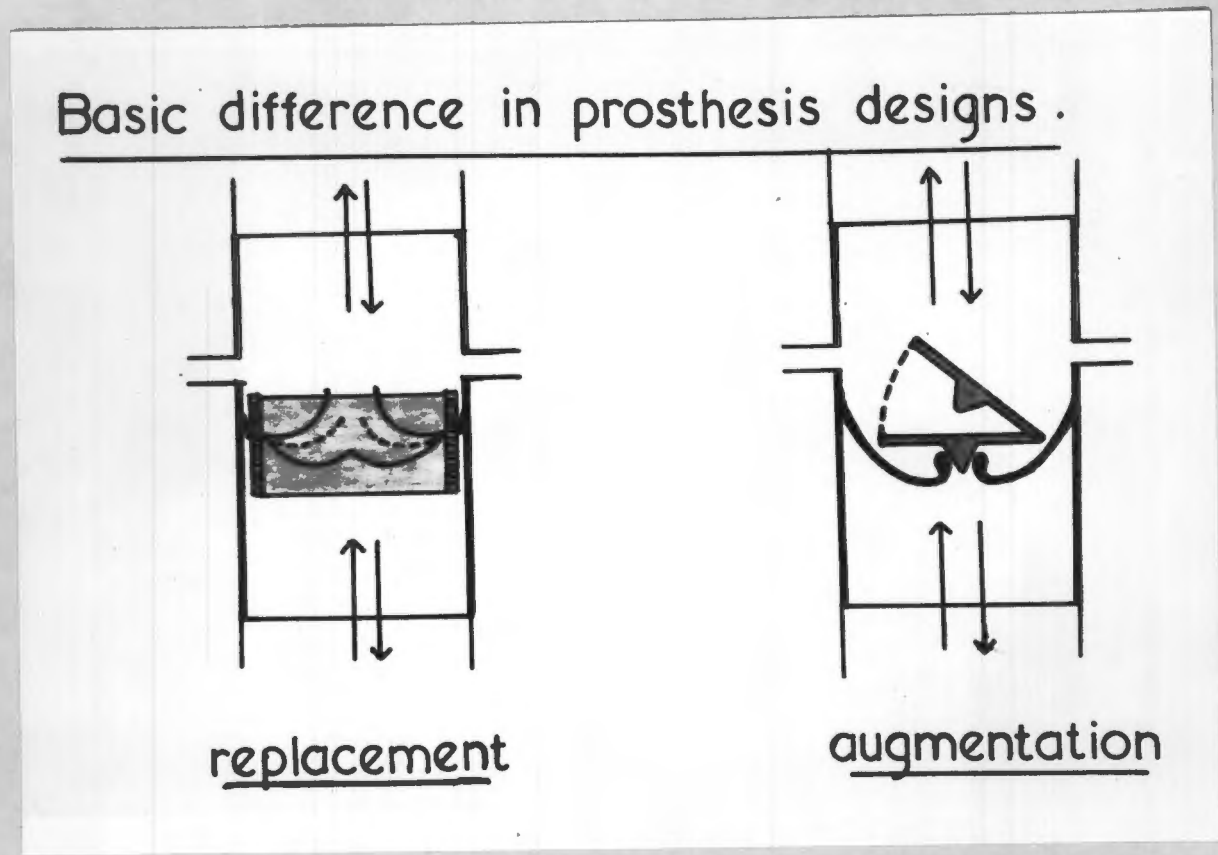


FIGURE 29.

Basic differences between

replacement and augmentation prostheses.

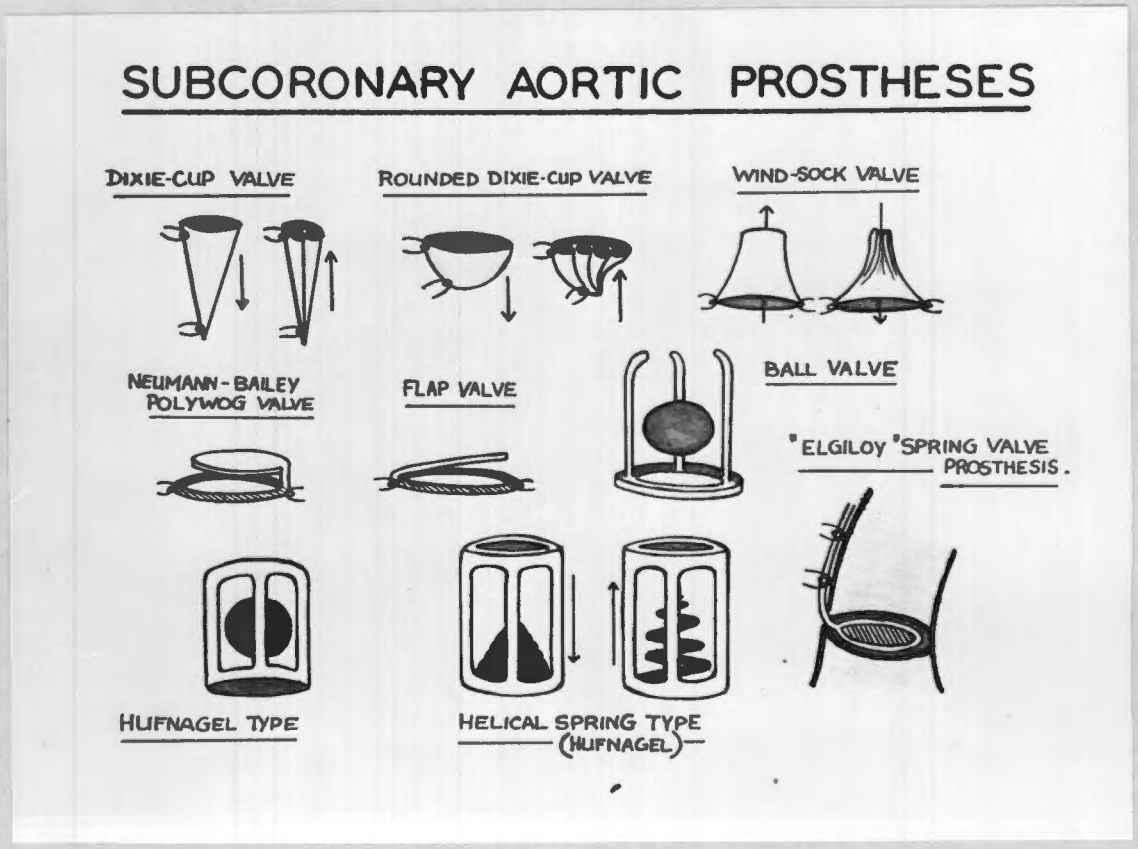


FIGURE 30.

Various types of subcoronary aortic prostheses.

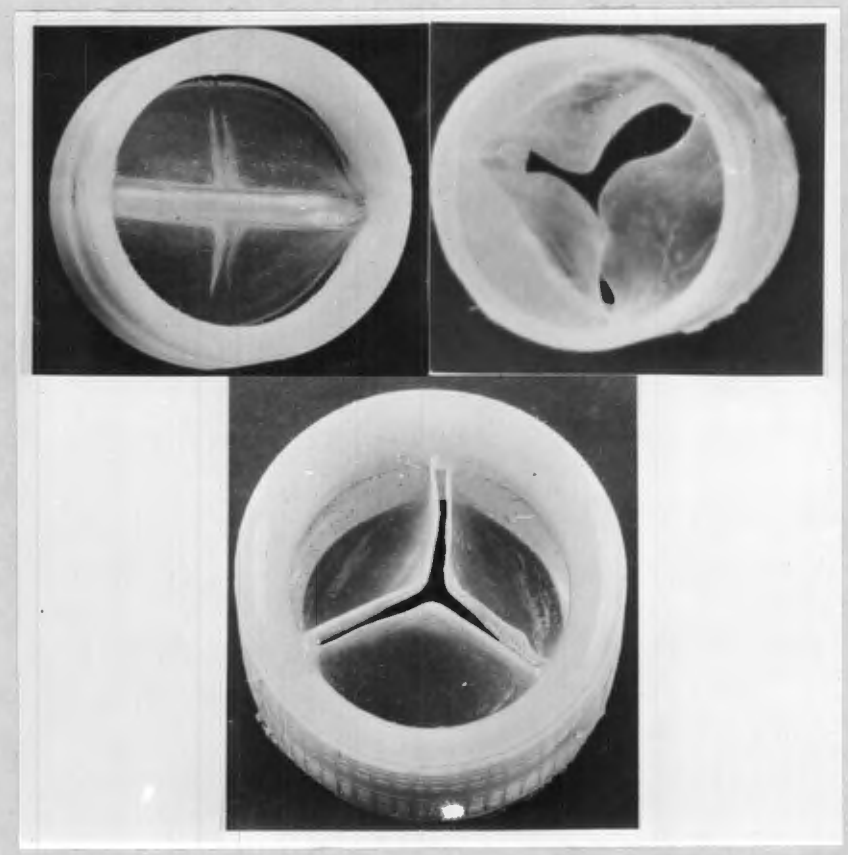


FIGURE 31.

Replacement type prostheses (Barnard):

(i) Silastic butterfly valve

(ii) Mylar three cusp valve

(iii) Silastic three cusp valve

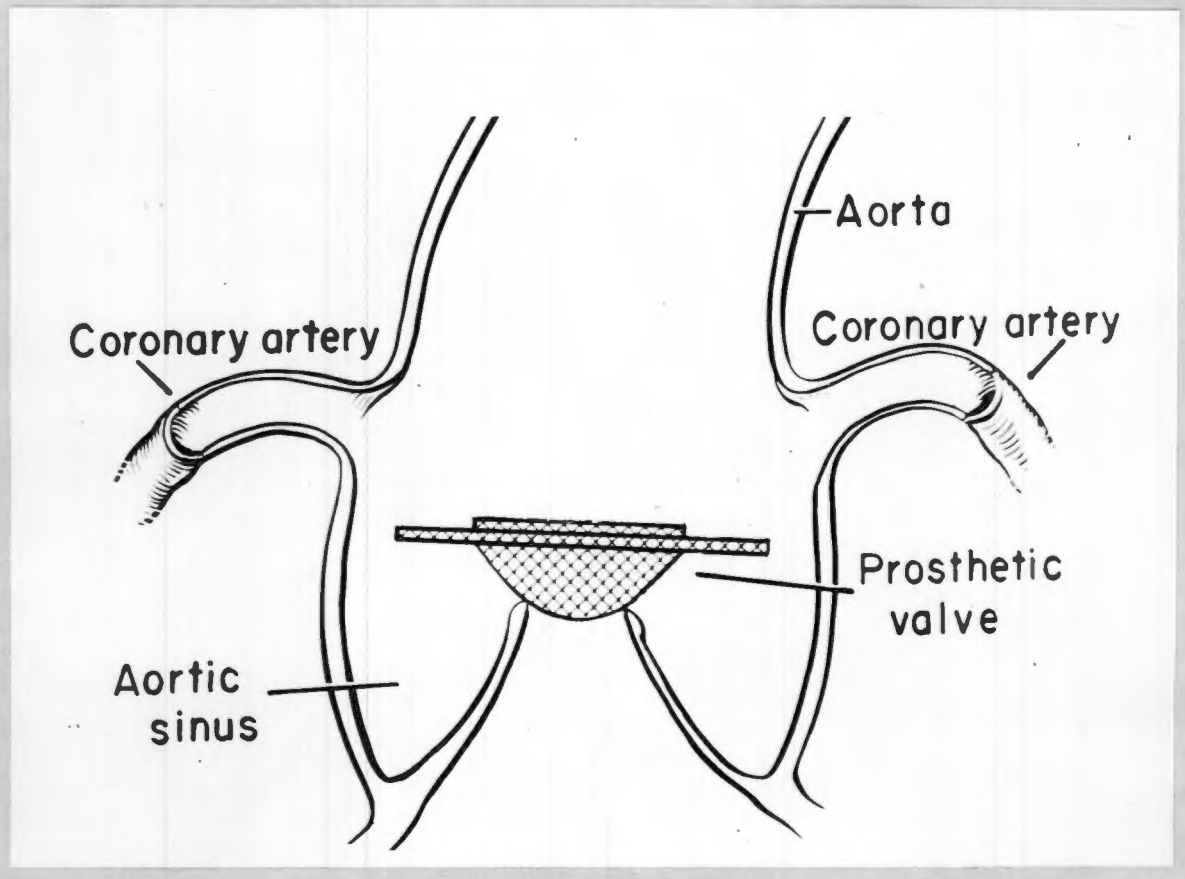
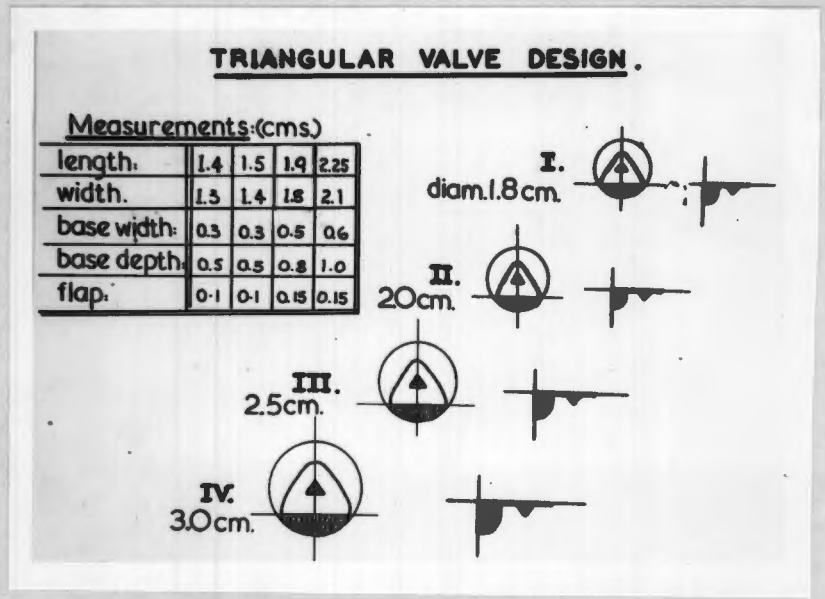


FIGURE 32.

Flap type prosthetic valve designed to cover the central regurgitant aperture.



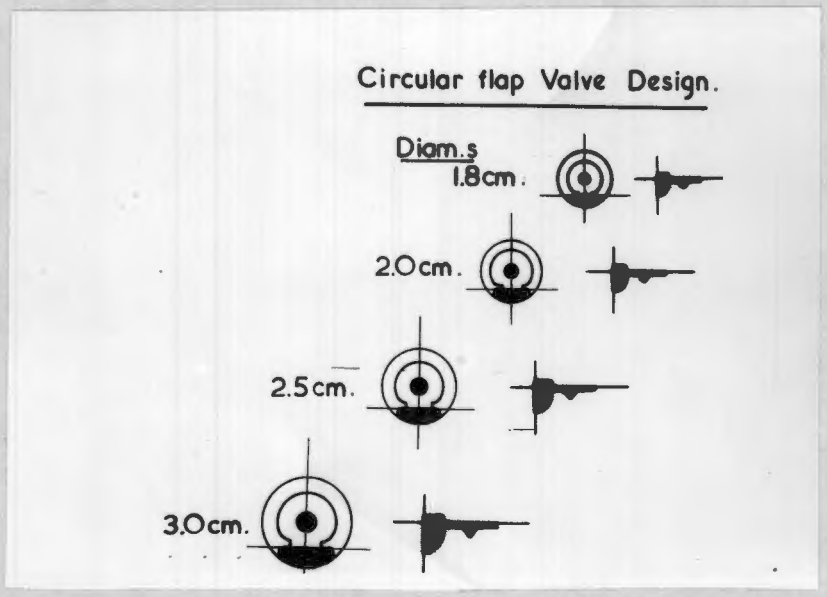


FIGURE 33.

Augmentation Prosthesis designs and sizes:

a). Triangular flap valve

b). Circular flap valve.

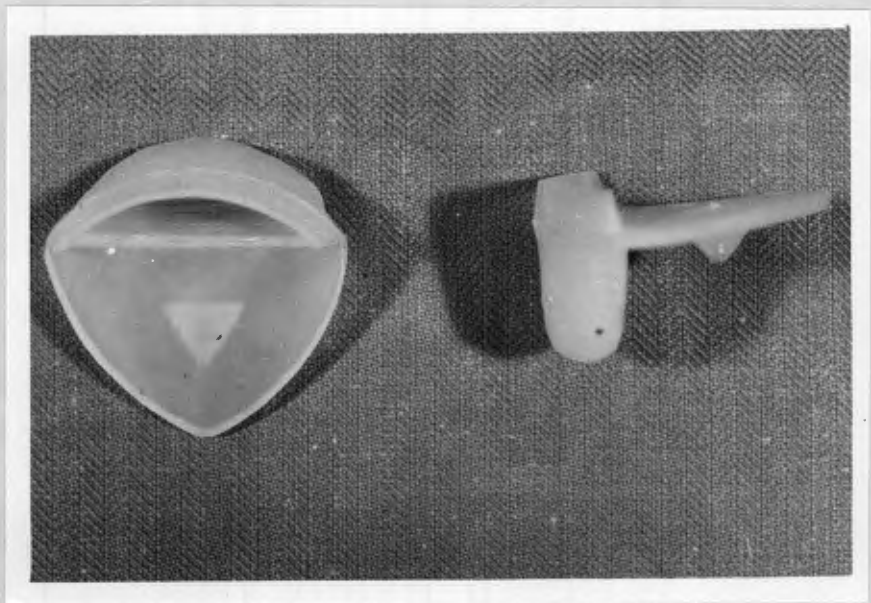


FIGURE 34.

Triangular flap augmentation prosthesis with hollow base.

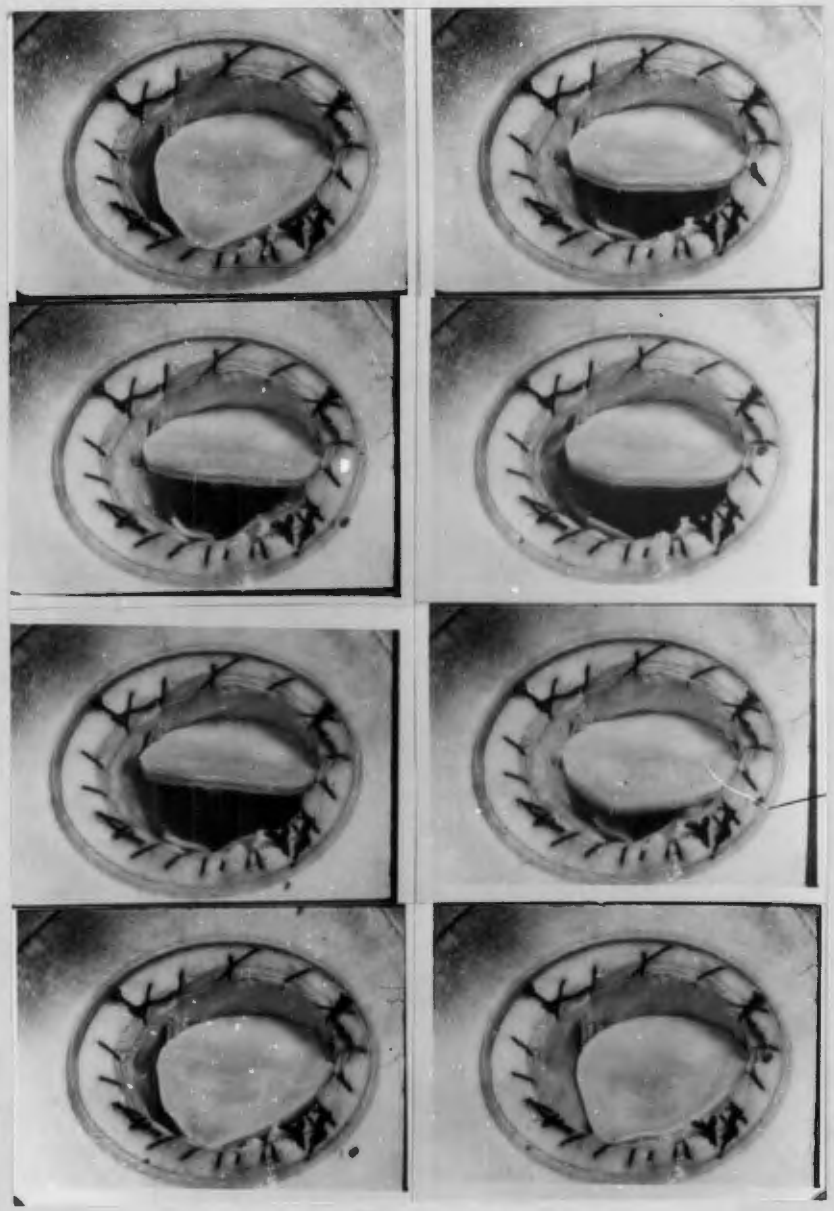


FIGURE 35.

Cycle of movements of triangular flap augmentation prosthesis with correct action (cf. Fig. 41 - incorrect action).

P.T.O.

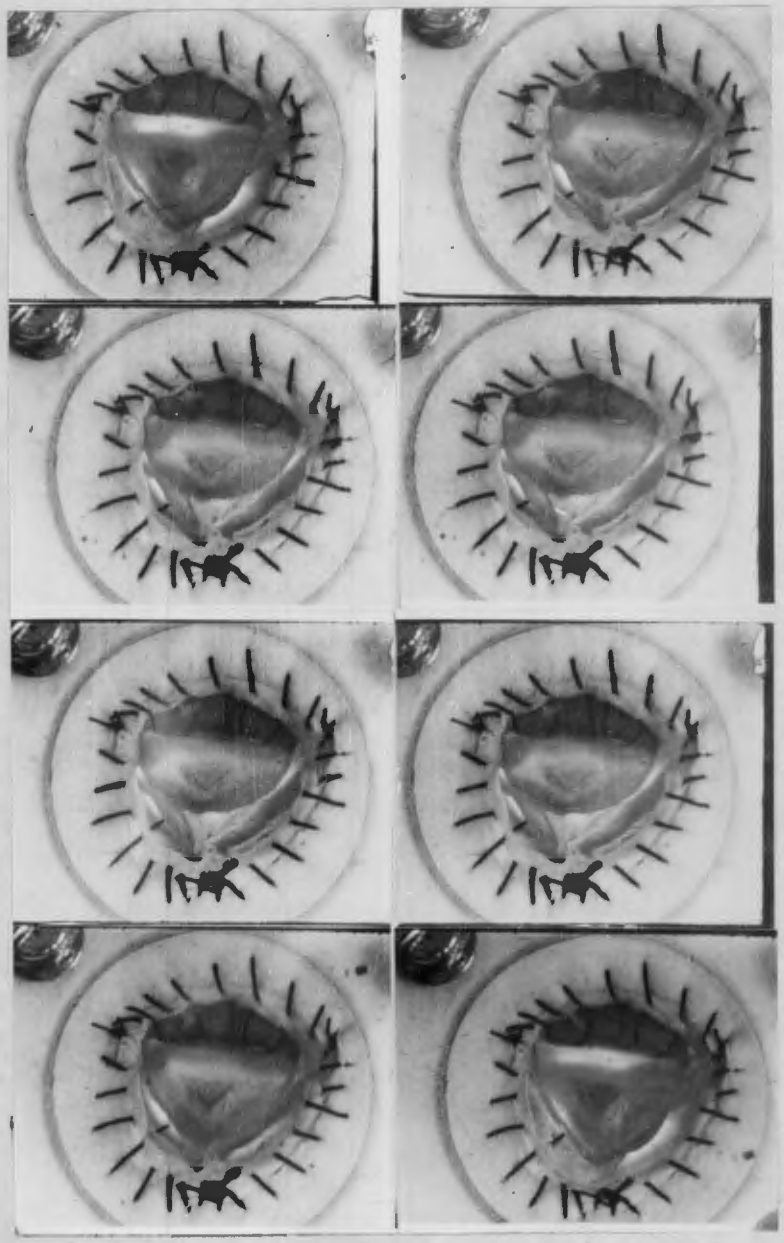


FIGURE 35. (contd.)

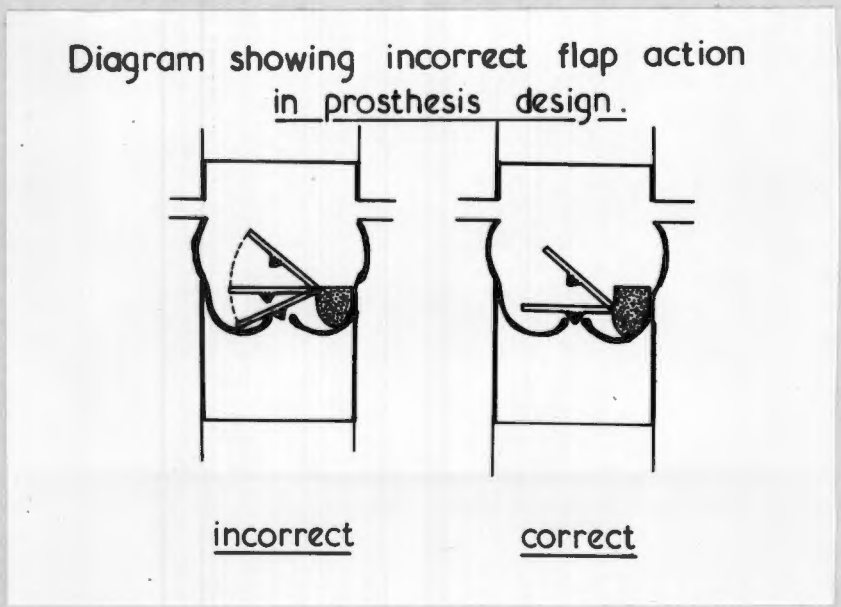


FIGURE 36.

Correct and incorrect flap action of augmentation prostheses.

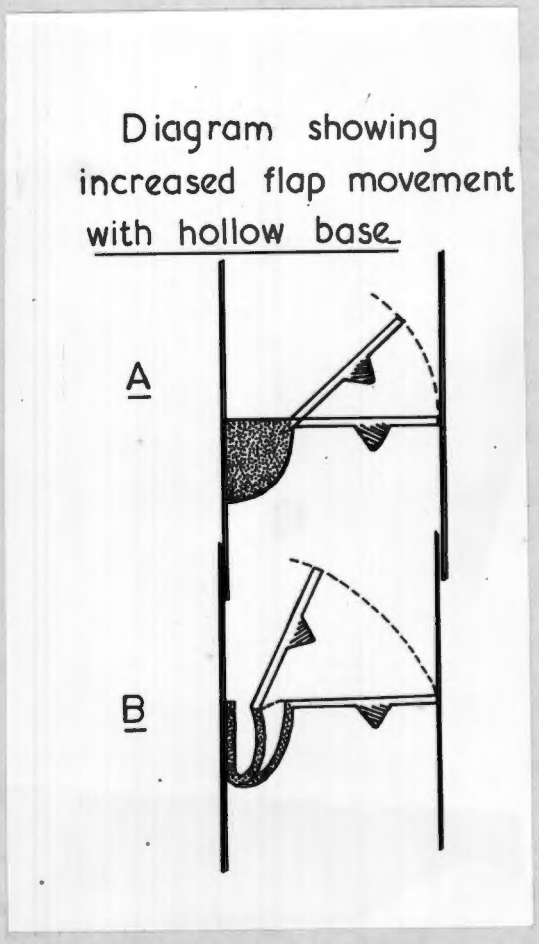


FIGURE 37.

Hollow based augmentation prosthesis.

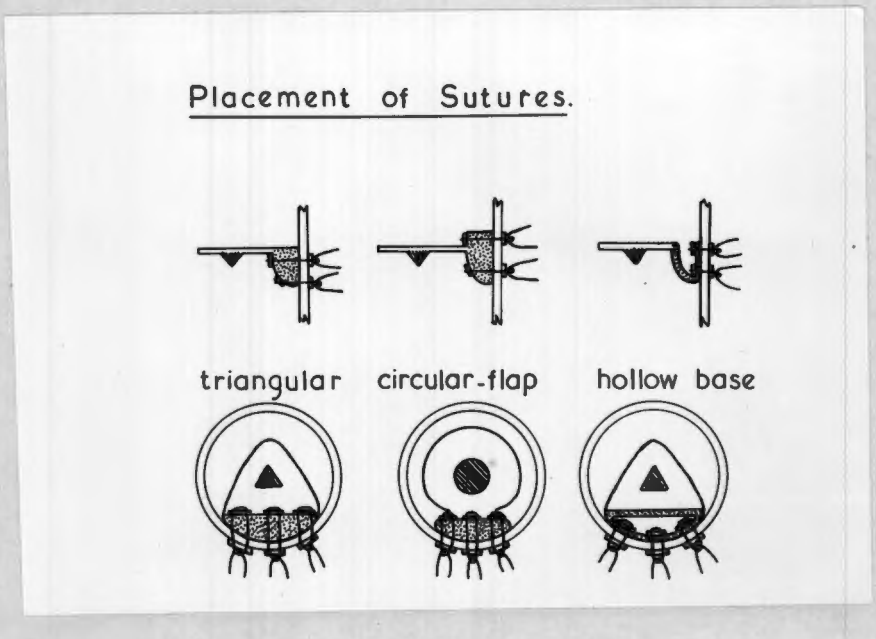


FIGURE 38.

Placement of sutures to secure prosthesis within the aortic root.

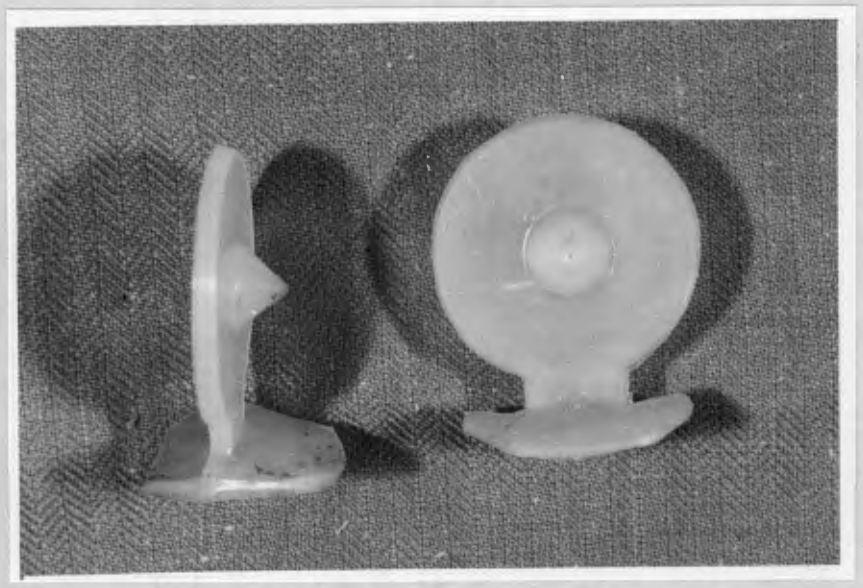


FIGURE 39.

Circular flap augmentation prosthesis.

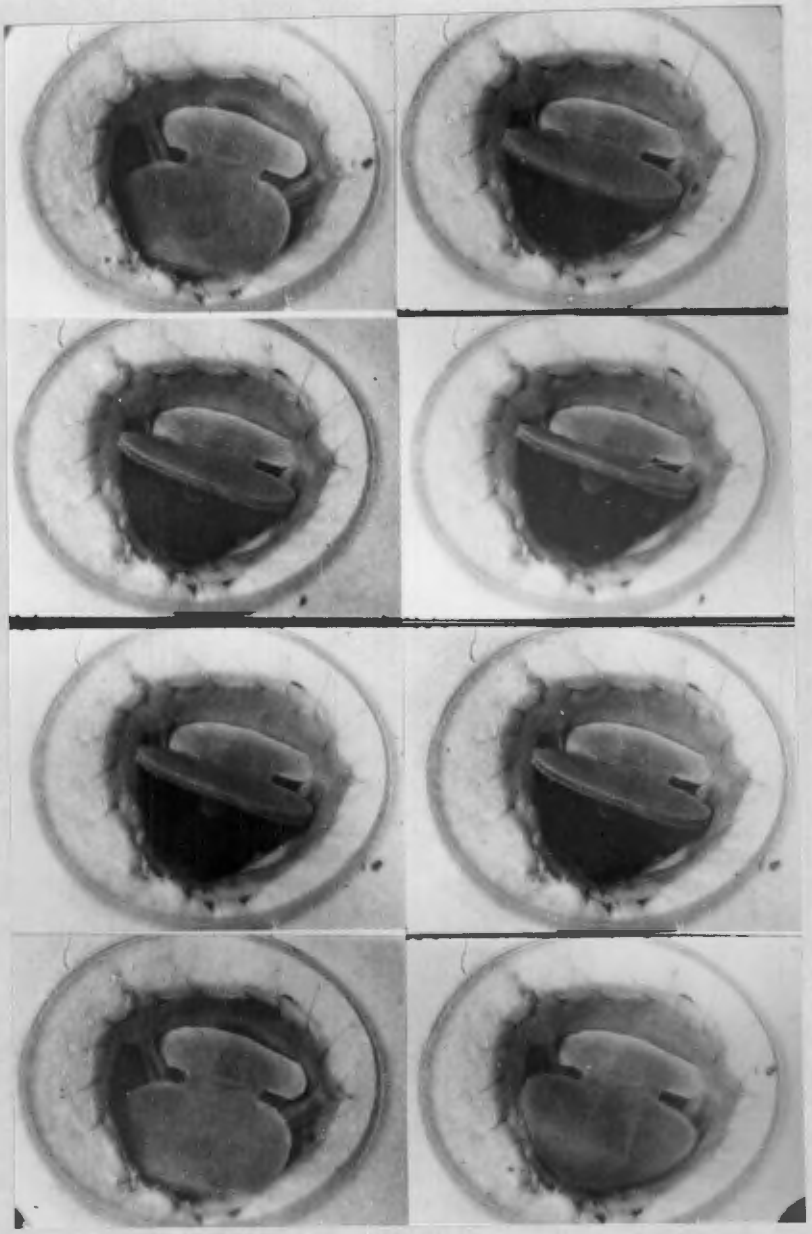


FIGURE 40.

Cycle of movement of circular flap design augmentation prosthesis.

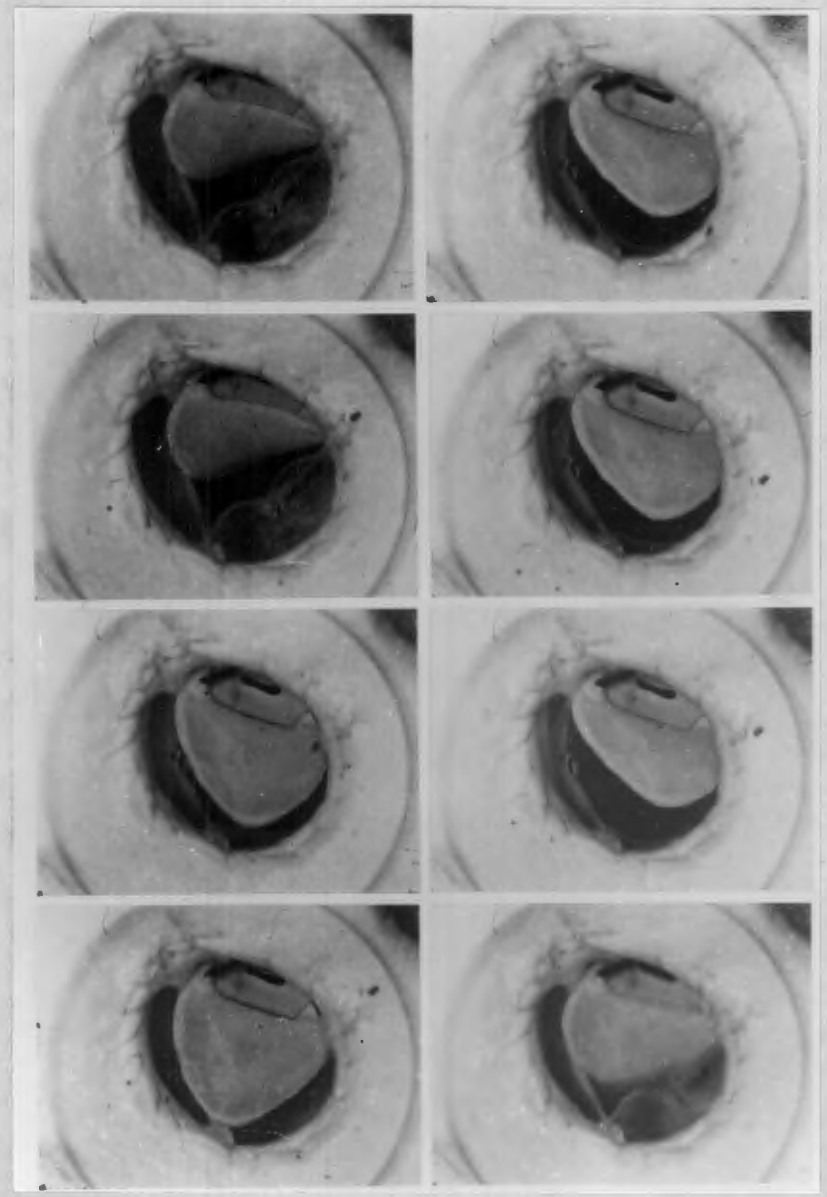


FIGURE 41.

Cycle of movements of triangular flap augmentation prosthesis acting incorrectly due to faulty placement.

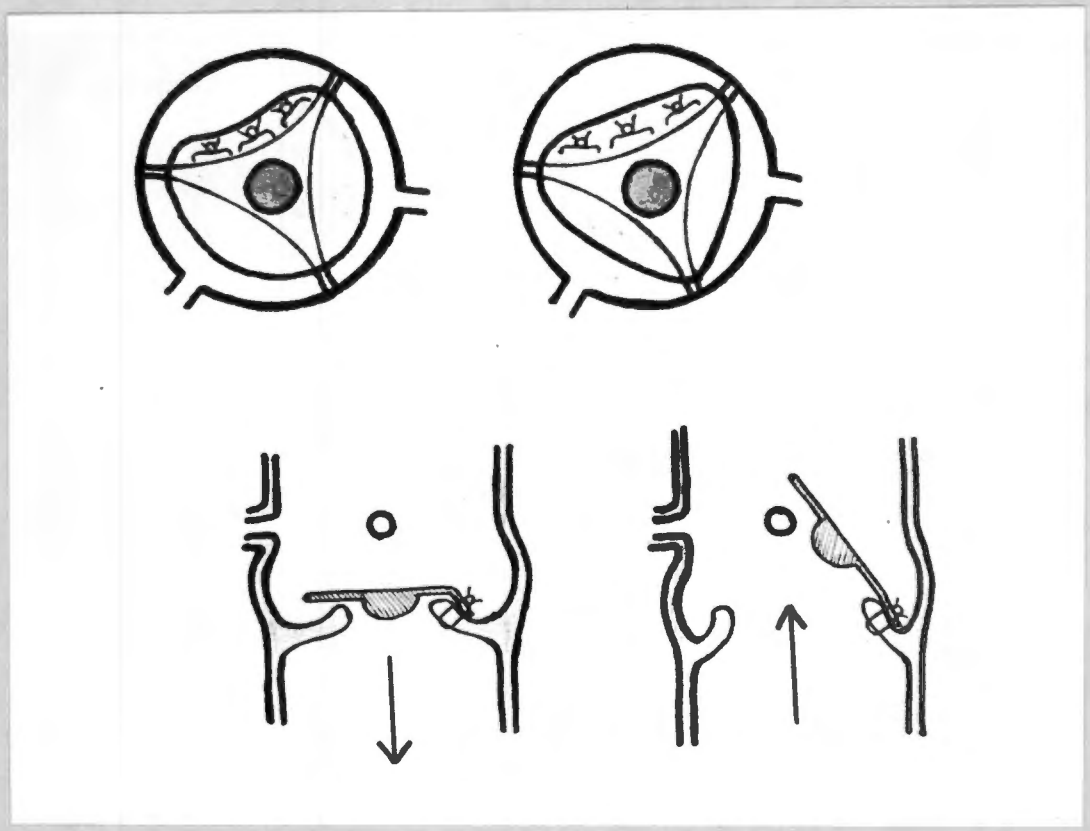


FIGURE 42.

Diagram of augmentation prosthesis designed for direct suture to non coronary aortic leaflet.

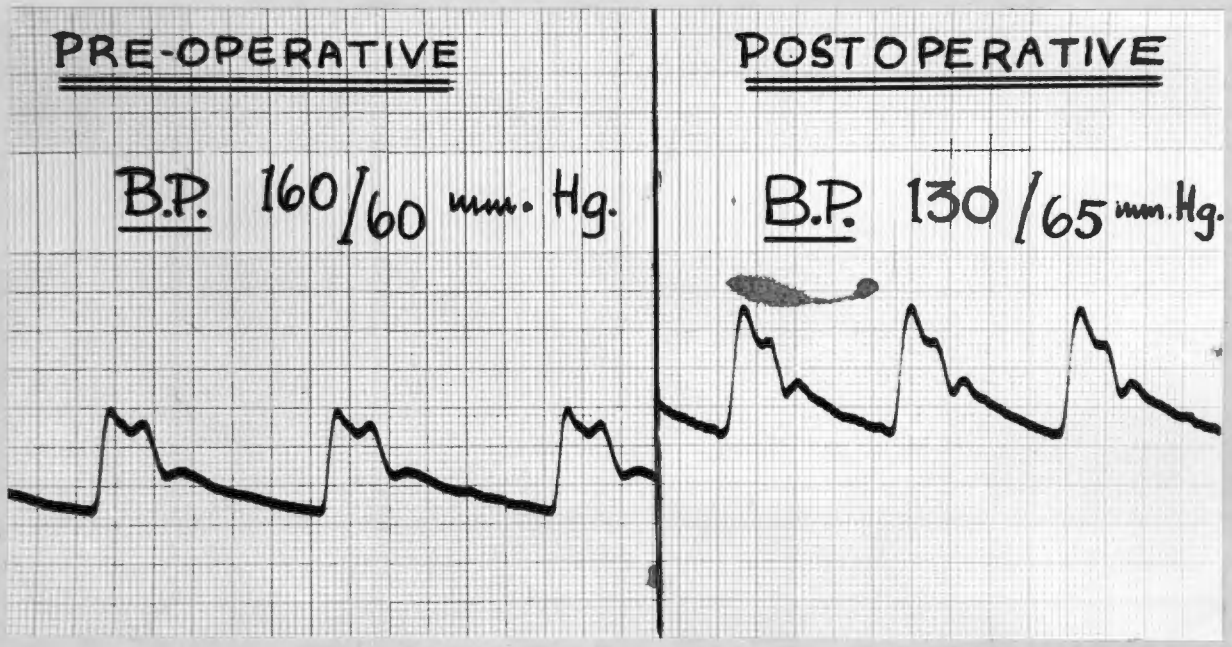


FIGURE 43.

Arterial pressure tracings of clinical case.

SUMMARY AND CONCLUSIONS.

Surgical approach to the aortic valve has become practical with the introduction of hypothermia and extracorporeal oxygenation techniques. Methods of myocardial maintenance allow direct exposure of the aortic valve, thus enabling surgical correction of lesions in this structure. Corrective surgery for aortic incompetence has, however, not yet reached the stage of clinical perfection. This is due in part to the near complete destruction wrought in some valves by the disease process, rendering plastic revision impossible, thus necessitating prosthetic replacement of the structure. Various such operative procedures have been described, either as plastic reconstructions or valve substitutions (autogenous graft or plastic material).

Before considering the surgical correction of chronic aortic incompetence, a detailed knowledge of normal anatomy and function, as well as of pathological anatomy and dysfunction, is essential. This was acquired by a detailed study of aortic valves, both normal and diseased, set up in an artificial pulse duplicator system. Various aetiological factors concerned in the production of "chronic aortic incompetence" are considered and only the two most important disease processes are discussed in detail. The mechanism whereby incompetence results ("Mechanism of Chronic Aortic Incompetence"), irrespec-

tive of aetiological factors, is analysed. Three basic lesion types can be differentiaied viz.

a). "mixed" lesion - either aortic stenosis plus incompetence (predominant stenosis), or aortic incompetence plus stenosis (predominant incompetence).

b). "pure" (primary) aortic incompetence

c). "intermediate" lesion - as a combination of mild features of both a). and b).

Surgical correction of the incompetent lesion may be effected by either reconstruction or replacement of the diseased valve. Surgical experience thus far gained in the field of vascular surgery has unequivocally shown that, at present, with currently available plastic materials, reconstruction of autogenous diseased tissue is superior to foreign material substitution. Thus, in aorto-iliac surgery, endarterectomy has gained preference over arterial grafting. Similarly, in aortic valvular surgery does this principle apply and should always be borne in mind. The surgeon must attempt to attain as much functional correction as possible by a plastic revision procedure and only then, if this is considered impractical, should the insertion of a prosthetic valve substitute be resorted to. Numerous operative procedures have been described to attain correction of pure aortic incompetence by plastic reconstructions with variable success.

The wide variety of methods employed, however, is indicative of the fundamental shortcomings of these procedures and it seems that it is just in this lesion that the insertion of a prosthesis of the augmentation type holds the greatest promise. In the "mixed" lesion, on the other hand, the insertion of a prosthesis seems contraindicated. This is for two reasons:

a). because of cusp thickening and hardening associated with only minimal retraction, excision of leaflets followed by insertion of a replacement prosthesis, is indicated. Such excision presents extreme dangers because of the close proximity of the anterior leaflet of the mitral valve.

b). marked loss of cusp mobility renders insertion of an augmentation type prosthesis impractical.

Numerous attempts at correction of valve regurgitation by use of autogenous or homografts have been described, but these have all been unsuccessful.

The use of plastic valve prostheses has been investigated in many centers. Subcoronary placement of such a prosthesis seems physiologically essential. Such prostheses may be used either for replacement of the diseased valve or to augment the impaired function of the diseased valve. For various reasons an augmentation prosthesis is superior to a replacement prosthesis. Methods used in design, manufacture and testing of such augmentation prostheses, in vitro as well as in vivo, are described. From results obtained it would appear that a flap

type (circular or triangular) augmentation prosthesis, sutured directly to the posterior (non coronary) aortic leaflet, holds the greatest promise. A material of Dacron Tricot coated with Silastic will prove of greatest use in the future of prosthesis construction. The types of pathological valves ideally suited to the insertion of this prosthesis are the lesions of either pure aortic incompetence or the "intermediate" lesion.

The surgeon should thus approach the operative correction of chronic aortic incompetence armed with a detailed knowledge of the normal anatomy of the aortic valve. If the lesion encountered is one of combined aortic stenosis plus incompetence ("mixed" lesion), a form of valvuloplasty is indicated. If, on the other hand, the lesion is one of either pure ("primary") aortic incompetence or "intermediate", he should then consider the insertion of an augmentation prosthesis. This must be of a flap type, sutured directly to the posterior (non coronary) cusp, and final shaping of the flap segment must be performed at the operating table after careful assessment of the size of the diseased valve.

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