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Surgical and mechanical support of the failing heart

M. TURINA

Surgical Clinic A, University Hospital, CH-8091 Zurich, Switzerland

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The surgical treatment of acute heart failure is limited to cases of pressure or volume overload. Acute valvular regurgitation due to active endocarditis or to prosthetic dysfunction is a classic example of failure which can be cured by restoring valvular competence. Acute pressure load is mostly caused by prosthetic dysfunction or pulmonary embolism; therapy is aimed at removal of the causative agent. Coronary heart disease can cause heart failure by volume overload: acute mitral incompetence or ventricular septal defect lend themselves to surgical correction. In the surgical treatment of acute heart failure maximal attention is devoted to optimal timing of surgery, anesthetic management and postoperative care. Careful attention to the function of the right and left ventricle and combination of catecholamines, afterload reducing agents and volume loading together with respirator support have considerably improved the surgical results. Acute pump failure due to coronary insufficiency and infarction is less amenable to surgical treatment, with rare exceptions of emergencies during coronary angiography and percutaneous dilatation. The intra-aortic balloon pump is the only method of mechanical circulatory assistance which has reached widespread clinical acceptance. The best results are achieved in conjunction with surgery: either as cardiac support in inherently reversible postoperative heart failure or as the means of circulatory stabilization prior to surgery. Ventricular assist devices are still in the experimental stage: their use has been sharply curtailed by the virtual disappearance of the postoperative low output syndrome. In selected cases of end-stage cardiomyopathy cardiac transplantation is nowadays performed with acceptable survival (70% at one year after surgery). Both orthotopic and heterotopic transplantation (transplanted heart in parallel with the natural one) give comparable results, but the procedure is still very restricted due to the lack of donors, multiple contraindications and lack of suitable heart preservation techniques.

Heart failure is defined as inability of the heart to provide adequate cardiac output to satisfy metabolic demands of the organism[1]. Therefore heart failure is primarily a defect of the contractile pumping component and lends itself only rarely to surgical relief. Nevertheless, several conditions causing acute and chronic heart failure are nowadays amenable to surgical treatment and even total heart replacement has become possible.

Methods

Surgical treatment in acute heart failure is possible when the disease has been caused by a sudden volume or pressure load of the heart—mostly of the left ventricle. Surgical therapy is strictly aimed at the removal of the causative agent which led to the development of the heart failure. The results are very satisfactory in several complications of valvular and coronary heart disease (Table 1).

Table 1 Surgical treatment of acute heart failure

1. Restoring valvular competence:
acute endocarditis
prosthetic dysfunction
papillary muscle dysfunction
aortic dissection
2. Eliminating volume overload:
postinfarction VSD
ruptured sinus valsalvae aneurysm
3. Eliminating pressure overload
prosthetic dysfunction
pulmonary embolism
4. Emergency revascularization.
angioplasty accident
iatrogenic coronary dissection
acute infarction (?)

Closure of *postinfarction ventricular septal defect*

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(*VSD*) has become a standard surgical procedure with acceptable risk[2]. Utilization of large patches, approach to the ventricular septal defect through the infarcted zone of the left ventricle and simultaneous coronary revascularization have enabled survival of a large proportion of patients with otherwise extremely limited life expectancy. Heart failure with a similar pathophysiological mechanism is encountered in *acute mitral insufficiency* caused by papillary muscle rupture in acute infarction; again the surgical treatment with replacement or—rarely—with repair of the mitral valve, combined with revascularization of the myocardium, greatly improves the otherwise dismal prognosis in this group[3,4].

Marked left ventricular failure is sometimes encountered in *sudden aortic* or—more rarely—*mitral valve incompetence* in acute endocarditis. The previous policy of antibiotic treatment followed by valve replacement after sterilization of the infected valve has been completely replaced by active surgical management of the diseased valve[5,6]. In patients with left ventricular failure, peripheral emboli or infection not readily responding to treatment with antibiotics, valvular replacement is performed early, followed by a prolonged (3–6 weeks) course of antibiotic treatment. Both mechanical and bioprosthetic valves can be used for valve replacement; local complications of the infection (annular abscesses, septal perforation and aneurysms of the sinus of Valsalva) are dealt with during valvular replacement.

Acute pressure overload of the left ventricle rarely requires surgical treatment: in isolated cases of *aortic prosthesis immobilization* by thrombus or tissue ingrowth urgent surgical treatment represents the only therapeutic modality. Both replacement of the valve or careful removal of the thrombotic material are possible[7–9].

False ventricular aneurysm is a rare complication of acute myocardial infarction[10] requiring prompt surgery[11] because of the large volume of the aneurysm, its potential for rupture and compression of the surrounding structures is great. Myocardial failure disappears almost immediately after resection of the false aneurysm, because the infarction is usually small. *Acute coronary insufficiency* is followed by heart failure when a large area of left ventricular myocardium—more than 40%[4]—is in jeopardy. Acute revascularization of the ischemic myocardium is successful only in rare circumstances, when the acute ischemia occurs under hospital conditions: in accidents of coronary angioplasty[12], and dissection or acute coronary obstructions during cardiac catheterization. The policy of acute revascularization of all myocardial infarctions has been pursued only in a few centers[13,14]; the lack of randomized studies makes an exact appraisal of this aggressive treatment impossible. The ischemic tolerance of a normothermic, ejecting heart is measured in minutes[15] and effective acute revascularization is possible only in patients already in hospital, with operating facilities alerted and coronary anatomy known beforehand. Intracoronary lysis[16] is an interesting non-surgical method which attempts to revascularize the myocardium by reopening the obstructed vessel. It has the advantage of combining pre-operative diagnostic studies with therapeutic intervention, but the results are not always convincing in terms of restoring ventricular function and preventing myocardial infarction[17].

Surgical treatment in chronic heart failure is nowadays possible only in a very limited number of cases. In large *left ventricular aneurysm* with well-delineated border zone and sufficient function of the residual ventricle, aneurysmectomy[18] can improve left ventricular function, decrease filling pressure and left ventricular dimensions, and improve exercise tolerance with elimination of heart failure caused by ventricular dyskinesia[19].

Orthotopic cardiac transplantation with total removal of the diseased organ represents the classic operation for end-stage cardiomyopathy. Pioneered experimentally in the early sixties[17] it was first performed clinically in 1977 in an operation which captured worldwide attention[20]. After an initial widespread application cardiac transplantation was abandoned by most centers due to lack of suitable donors, the high immunosuppression necessary to keep the transplant functioning and a substantial incidence of infections. Continuous clinical application of this method at Stanford[21] has shown that the operation is possible with low early mortality and acceptable late results; improvement in myocardial preservation techniques will widen the application of this method. *Heterotopic cardiac transplantation*[22] was designed to circumvent some of the problems encountered in treating acute rejection episodes in orthotopic cardiac transplants. In this operation, the transplanted heart is attached in parallel to the natural one and it provides a varying degree of circulatory assistance, depending on the condition of the natural heart. The advantages of this elegant operation are obvious but its hemodynamic efficiency has been questioned. *Combined transplantation of the heart and the lungs* has been performed by

the Stanford group[23] in a few selected patients as a final solution to the problem of heart failure with high pulmonary resistance. Although technically simple, this operation probably entails more rejection problems through the presence of two immunologically active organs.

MECHANICAL SUPPORT OF THE FAILING HEART

In the field of *acute mechanical support* considerable advances have been made in the last decade (Table 2).

Intra-aortic balloon pumping (*IABP*) was introduced experimentally in 1962[24] and clinically in 1968[25]. It has emerged as a safe, reliable method of left ventricular assistance which can decrease left ventricular failure by reducing the afterload and by increasing diastolic pressure in the aorta, thereby improving coronary flow. The advent of commercially available pumping devices and non-thrombogenic, reliable balloons, greatly widened the application of IABP and it is nowadays estimated that more than 300 000 implantations have been performed worldwide (D. Bergman, pers. comm.). The percutaneous balloon has eliminated the problems of surgical balloon introduction and has simplified the procedure; it has enabled the cardiologist to apply the device quickly by a simple arterial puncture.

Artificial heart research has led to the development of *assist ventricles* which could be used either in a paracorporeal position, fixed on the chest of the patient and attached to the heart with canulae; or intracorporeally as an abdominal left ventricular assist device[26]. These ventricles provide much more efficient circulatory assistance than the IABP. Assist ventricles can take over the total output of the diseased heart and they can indeed support the circulation in ventricular fibrillation. Separate assist pumping of the left and right ventricle is possible[27] although careful attention has to be paid to the filling pressures in both atria and to the output of each ventricle. The biggest disadvantage of this method lies in the necessity of thoracotomy for implantation and removal of the devices, even in the paracorporeal pumping mode. Therefore the assist ventricles have been used only in cases of postoperative low output syndrome, i.e. in profound heart failure occurring after open heart surgery[27-30]. Pneumatically driven assist ventricles, which were utilized in these clinical trials, were hand made units, which represented the end result of a long experimental development in the particular center performing clinical trials. Considerably simpler devices—roller pumps normally used in open heart surgery—were also utilized for mechanical support of the failing heart with reasonable clinical success[31]. The use of a roller pump necessitates total heparinization of the patient which seriously limits the application of this method; on the other hand the use of left atrial and aortic cannulae which can be permanently occluded and left in place precludes the need for a second thoracotomy for the removal of the device. All assist ventricles and bypass pumps are 'tethered' devices, the patient being attached to the pumping console with pressure lines needed to drive the ventricles; in the paracorporeal position blood cannulae also penetrate the chest wall, severely limiting the mobility of the patient. Due to numerous difficulties (blood damage, thrombus formation, cannulation problems, infection, bleeding) the ventricular assist devices are used only for a few days, to allow the recovery of the heart from a severe postoperative low output syndrome.

Chronic mechanical support of the circulation is possible with the use of an orthotopically implanted *total artificial heart*. Although much experimental experience has been accumulated in several centers during the last decade, the artificial heart was used only in two patients, and neither of them eventually survived[32]. Both clinical implants were done in a desperate situation, with patients dying after an open heart procedure and other methods of circulatory assistance failing. Cardiac transplantation with removal of the artificial heart has been performed as soon as possible, after a suitable donor was found. The removal of the diseased natural heart, followed by the implantation of the mechanical heart prosthesis is an irrevocable step which most surgeons have been anxious to avoid. In experimental animals mechanical circulatory support is possible in excess of six months[33], but the problems of the long-term biocompatibility of materials in contact with blood have not yet been solved.

Table 2 Mechanical circulatory support

1. Widespread clinical use:
   IABP
2. Clinical trials:
   assist ventricles
   temporary ventricular bypass
3. Experimental stage, isolated clinical applications.
   total artificial heart
   implantable assist ventricles

Clinical indications and results

SURGICAL TREATMENT IN ACUTE FAILURE

In correctable lesions the guidelines for surgical treatment are derived from the recent advances in knowledge of the pathophysiology of acute heart failure. The treatment must be instituted early, and it must be curative in order to ensure the survival of the patient. The early institution of treatment prevents development of late complications which are related both to heart failure and to heart changes caused by the primary disease process. Early elimination of heart failure prevents deterioration in the function of the vital organs commonly seen in profound heart failure: renal failure, coagulation disorders, hepatic failure, pulmonary oedema and onset of septic complications. The timing of the operation is critical: whilst the operation should be performed only when significant hemodynamic deterioration has occurred, it should be early enough to enable total restitution of cardiac function. Heart failure and cardiac enlargement seen in acute volume or pressure overload increase myocardial wall stress and its oxygen consumption; they result in subendocardial ischemia which can progress to subendocardial necrosis, jeopardizing the results of surgical treatment. The operation should be performed before such grave deterioration occurs.

In postinfarction VSD the significant left-right shunt with development of heart failure is an indication for surgery *per se*. The operation is performed early, regardless of the time period between myocardial infarction and operation. Reliable coronary angiography is necessary to show the extent of coronary imvolvement in the non-infarcted area; pre-operative implantation of IABP has been particularly beneficial in this group of patients. Mortality of less than 20% can be achieved depending on the timing of surgery, age of the patient, extent of the coronary disease and residual left ventricular function. When the patient has survived the early postoperative period, the long-term outlook is good, although some residual shunting is possible.

In acute valvular incompetence caused by endocarditis the operation is performed in the presence of significant valvular regurgitation with heart failure, peripheral emboli or uncontrollable infection. Operation is also considered earlier if the causative agent is resistant or only mildly susceptible to the antibiotics. With proper timing of surgery, the results are comparable with elective valvular replacement but long-term antibiotic treatment is necessary to prevent the recurrence of infection[5,6].

Sudden prosthetic dysfunction—incompetence or stenosis—in the aortic or mitral position leads to sudden, profound heart failure and even to the death of the patient before treatment can be instituted. Diagnosis is usually difficult and can be made only by excluding other sudden causes of profound heart failure (pulmonary embolism, myocardial infarction or acute dissection). Only a high index of suspicion and immediate operation with minimal diagnostic work-up can assure the survival of the patient. The operation has to be initiated when the patient is still able to maintain some cardiac output to assure the perfusion of vital organs[7].

In acute coronary insufficiency infarction can be prevented only by immediate operation which should start within 30–60 min after acute coronary occlusion. A small increase in CK enzymes still occurs but the appearance of new Q waves can usually be prevented[14]. In sudden coronary occlusion in the course of percutaneous transluminal angioplasty revascularization is possible without mortality or significant morbidity.

DISAPPEARANCE OF POSTOPERATIVE HEART FAILURE

The results of open heart surgery have improved considerably in the last years. The main cause of death after valvular or coronary surgery in the sixties and early seventies was the postoperative low output syndrome, a profound biventricular heart failure resistant to all therapeutic modalities and only sometimes reversed by mechanical circulatory assistance (IABP and assist ventricles). It is now known that this type of heart failure is primarily caused by ischemic damage to the heart which occurs during aortic cross-clamping and cardiopulmonary bypass. Contributory factors are pre-existing heart failure, reperfusion damage after removal of the aortic cross-clamp, low oncotic pressure and low hematocrit during cardiopulmonary bypass, also insufficient aortic pressure in the post-bypass period which provokes subendocardial ischemia, especially in patients with reduced ventricular compliance and high filling pressure. Furthermore, volume loading which was practised earlier usually led to iatrogenic right ventricular failure, which very often precipitated the development of postoperative low output syndrome.

The prevention of this condition is—as usual—more important than its treatment: potassium cardioplegia has practically eliminated postoperative

low output syndrome. Strict adherence to the principle of rapid cardiac arrest (high potassium concentration) and profound myocardial cooling (ice-cold cardioplegic solution) reduces myocardial oxygen consumption and limits myocardial energy utilization during cross-clamping; a sufficient period of reperfusion allows the heart to recover from the ischemic injury and full volume and pressure load can be taken over after a period of 15–20 min. The strict avoidance of hypervolemia, an adequate hematocrit and sufficient arterial perfusion pressure can also prevent the development of this type of heart failure. When the signs of left ventricular failure appear, IABP is used much more aggressively; arrhythmias are strictly controlled and physiological cardiac pacing (AV synchronous) is used instead of simple ventricular stimulation, when the optimal heart rate cannot be maintained spontaneously.

SURGICAL TREATMENT OF CHRONIC HEART FAILURE

Left ventricular aneurysmectomy is an established procedure with known indications. It is not the purpose of this communication to discuss this procedure; however, it can be stated that the early mortality of less than 5% is achieved in patients with suitable indications[13]; that the procedure improves left ventricular function, decreases filling pressure and increases patients work capacity; there is no definite proof that it also increases patients' survival, due to the lack of truly randomized studies.

Cardiac transplantation (for the purpose of this discussion both ortho- and heterotopic procedures are grouped together) has well defined selection criteria[34]. The procedure should be performed in patients with terminal cardiac disease, with calculated survival of less than 12 months. Patients should be less than 55 years of age, pulmonary vascular resistance should be below 8 U and patients should be able to comply with the complex medical regimen of chronic immunosuppression. The following diseases represent a contraindication to transplantation: insulin-dependent diabetes mellitus, presence of any degree of infection, recent pulmonary infarction, peptic ulcer, substantial cerebrovascular or peripheral vascular disease, any degree of chronic addiction or other significant diseases which might shorten the survival of the patient. With strict adherence to these criteria, a survival rate of 70% at one year after transplantation can be achieved[34]. Although late attrition remains a problem, the largest mortality is encountered in the first three months after transplantation. The results are not uniform: great variability in one year survival rates is encountered among difference centers ranging from 72–38%[34,35]. Cardiac transplantation does indeed provide an almost complete restoration of cardiac function with acceptable long-term results which are due to an improved regimen (Cyclosporin A) and better recognition of rejection by serial biopsy; furthermore the procedure has now a well established list of contraindications (Table 3). Still, most centers have been reluctant to adopt cardiac transplantation after the initial failures. The most common arguments against cardiac transplantation are the relative shortage of donors and inadequate methods for heart preservation and storage. Furthermore, the procedure still has a substantial late attrition rate and it is performed in a disease—cardiomyopathy— with an unpredictable natural course, where late recoveries are possible. The intensive late surveillance and treatment necessary make this therapeutic modality a very costly one; it is also applicable to only a few patients and social-economic reasons have been quoted against cardiac transplantation, i.e. disproportionate allocation of limited means to a very few patients.

Table 3 Cardiac transplantation: pros and cons

Advantages
Cardiac function restored
Long-term results acceptable
Better immunosuppression
Diagnostics of rejection
Contraindications established
Only available treatment
Disadvantages
Lack of donors
Heart preservation deficient
Natural course of cardiopathy uncertain
Late attrition rate
High cost
Social-economic objections

MECHANICAL CIRCULATORY SUPPORT

IABP has gained widespread clinical acceptance in the last decade. It gives the best results (Table 4) when used for temporary stabilization of the patient, either to perform a diagnostic evaluation (coronary angiography) or prior to surgery (postinfarction VSD, unstable angina pectoris, severe left main coronary stenosis with obstruction of the right coronary artery, etc.). The results are mediocre when the device is applied in postoperate heart failure after valvular surgery, where a success rate of

Table 4 IABP · clinical results

High survival:
perioperative use in coronary grafting unstable angina (before surgery)
Moderate success:
heart failure after valvular surgery
Poor results:
congenital heart disease (postoperatively)
Cardiomyopathy and myocarditis
Cardiogenic shock
Toxic and septic heart failure
IABP and transplantation

approximately 50% has to be accepted[36,37]. IABP gives very poor results in postoperative heart failure after correction of congenital heart malformations, in cardiomyopathy and myocarditis as well as in cardiogenic shock when not combined with cardiac surgery. It also fails in toxic and septic heart failure. Recent experience demonstrates that the use of IABP followed by cardiac transplantation[38] has a poor long-term prognosis; from the beginning this method was not accepted by other transplantation centers. IABP provides reliable hemodynamic benefit with a low incidence of early and late complications; blood damage remains minimal and the mechanical assistance can be performed for days and even weeks. Although the overall use of IABP is in decline in postoperative heart failure, it is used more aggressively by cardiologists for pre-operative stabilization of patients.

Assist ventricles have been used by numerous groups in the recent years, almost exclusively in cases with profound heart failure after open heart surgery. By adherence to a strict list of criteria (all surgical problems solved, blood pressure below 80 mm Hg, left atrial pressure above 20 mm Hg, full catecholamine support, IABP implanted, pH and electrolytes normalized and all arrhythmias controlled) the results were poor: only a few patients are long-term survivors, and the program of mechanical ventricular assistance has been suspended in many centers which previously worked in this field. Again, the disappearance of postoperative low output syndrome has eliminated the major area of application for this type of circulatory support; it remains to be seen if a higher success rate can be obtained in patients with profound cardiogenic shock after myocardial infarction, when the device is employed after acute revascularization. The total artificial heart is still in an experimental stage and all clinical applications were unsuccessful. It is questionable if the present artificial heart should be offered to patients in endstage cardiomyopathy and if patients will be able to accept a 'tethered' device with a limited life span, which will furthermore severely limit their mobility and reduce their quality of life.

References

(1) Braunwald E, Ross J, Sonnenblick EH. Mechanism of contraction of the normal and failing heart. Boston: Little, Brown and Company, 1967: 139.

(2) Hill JD, Lary D, Kerth WJ, Gerbode F. Acquired ventricular septal defects. Evolution of an operation, surgical technique and results. J Thorac Cardiovasc Surg 1975, 70: 440–50.

(3) Friedberg CK. Disease of the heart. 3rd ed. Philadelphia: WB Saunders, 1969: 857.

(4) Mundth ED, Buckley MJ, Daggett WM, Sanders CA, Austen WG. Surgery for complications of acute myocardial infarction. Circulation 1972; 45: 1279–91.

(5) Krayenbühl Ch, Turina M, Kugelmeier J, Rothlin M, Senning Å. Chirurgische Behandlung der aktiven infektiösen Endocarditis. Thoraxchirurgie 1978; 26: 241–4.

(6) Stinson EB, Griepp RB, Vosti K, Copeland JG, Shumway NE. Operative treatment of active endocarditis. J Thorac Cardiovasc Surg 1976; 71: 659–65.

(7) Björk VO, Henze A. Encapsulation of the Björk-Shiley aortic disc valve prosthesis caused by the lack of anticoagulation treatment. Scand J Thorac Cardiovasc Surg 1973; 7: 17–21

(8) Byrol CL, Yahr WZ, Greenberg JJ. Long-term results of 'simple' thrombectomy for thrombosed Björk-Shiley aortic valve prosthesis. Ann Thorac Surg 1975; 20: 265–73.

(9) Cooper DKC, Sturridge MF. Acute massive mitral regurgitation from prosthetic valve dysfunction. Br Heart J 1976; 38: 701–5

(10) Roberts WG, Morrow AG. Pseudoaneurysm of the left ventricle. An unusual sequel of myocardial infarction and rupture of the heart. Am J Med 1967; 43: 639–44.

(11) Gueron M, Wanderman KL, Hirsch M, Borman J. Pseudoaneurysm of the left ventricle after myocardial infarction. A curable form of myocardial rupture. J Thorac Cardiovasc Surg 1975; 69: 736–42.

(12) Turina M, Grüntzig A, Krayenbühl Ch, Senning Å. The role of surgeon in percutaneous transluminal dilatation of coronary stenosis. Ann Thorac Surg 1979; 28: 103–12.

(13) Phillips SJ, Kongtahworn C, Zeff RH, *et al.* Emergency coronary artery revascularization: a possible therapy for acute myocardial infarction. Circulation 1979; 60: 241–50.

(14) Berg R Jr, Kendall RW, Duvoisin GE, Ganji JH, Rudy LW, Everhart FJ. Acute myocardial infarction, a surgical emergency. J Thorac Cardiovasc Surg 1975; 70: 432–9

(15) Ebert PA, Greenfield LJ, Austen WG, Morrow AG. Experimental comparison of methods for protecting

the heart during aortic occlusion. Ann Surg 1962; 155: 25–32.
(16) Rentrop P, Blanke H, Karsch KR, *et al.* Wiedereröffnung des Infarktgefässes durch transluminale Rekanalisation und intrakoronare Streptokinase-Applikation. Dtsch Med Wochenschr 1979; 104: 1438–40.
(17) Gangadharan V, Ramos RG, Hauser AM, Westveer DC, Gordon S, Timmis GC. Natural history of left ventricular function following intracoronary thrombolysis. Am J Cardiol 1982; 49: 962.
(18) Cooley DA, Henley WS, Amad KH, Chapman DH. Ventricular aneurysm following myocardial infarction: results of surgical treatment. Ann Surg 1959; 150: 595–612.
(19) Speiser K, Goebel N, Hess O, Rothlin M. Left ventricular function and course after left ventricular aneurysmectomy. Eur Heart J 1981; 2 (suppl A): 141.
(20) Lower RR, Stofer RC, Shumway NE. Homovital transplantation of the heart J Thorac Cardiovasc Surg 1961; 41: 196–204.
(21) Barnard CN. The operation. A human cardiac transplant: an interim report of a successful operation performed at Groote Schuur Hospital, Cape Town. S Afr Med J 1967; 41: 1271–4.
(22) Barnard CN, Losman JG. Left ventricular bypass. S Afr Med J 1975; 49: 303–12.
(23) Reitz BA. Heart and lung transplantation. Heart Transpl. 1982; 1: 82–3.
(24) Moulopoulos SD, Topaz S, Kolff WJ. Diastolic balloon pumping (with carbon dioxide) in the aorta—a mechanical assistance to the failing circulation. Am Heart J 1962; 63: 669–75.
(25) Kantrowitz A, Tjønneland S, Freed PS, Phillips SJ, Butner AN, Sherman JL Jr. Initial clinical experience with intra-aortic balloon pumping in cardiogenic shock JAMA 1968; 203: 113–8.
(26) Norman JC. An intracorporeal (abdominal) left ventricular assist device (A-LVAD), XXX. Clinical readiness and initial trials in man. Cardiovasc Dis Bull Tex Heart Inst 1976; 3. 249–88.
(27) Turina M, Bosio R, Senning Å. Clinical application of *paracorporeal uni- and biventricular artificial heart* Trans Am Soc Artif Intern Organs 1978; 24: 625–31.
(28) Berger RL, Merin G, Carr J, Sossman HA, Berhard WF. Successful use of left ventricular assist device in cardiogenic shock from massive postoperative myocardial infarction J Thorac Cardiovasc Surg 1979; 78: 626–32.
(29) Wolner E, Deutsch M, Losert V, *et al.* Clinical application of the ellipsoid left heart assist device. Artif Organs 1978; 2: 268–72.
(30) Pierce WS, Migliore J, Donachy J, *et al.* Prolonged mechanical support of the left ventricle. Circulation 1977; 56 (suppl III): 249.
(31) Litwak R, Koffsky R, Jurado R, *et al.* Use of a left heart assist device after intracardiac surgery: technique and clinical experience. Ann Thorac Surg 1976; 21: 191–202.
(32) Cooley DA. Staged cardiac transplantation: report of three cases Heart Transpl 1982; 1: 145–53.
(33) Hastings WL, Aaron JL, Deneris J, *et al.* A retrospective study of eight calves surviving five months on the pneumatic total artificial heart. Trans Am Soc Artif Intern Organs 1981; 27: 71–6.
(34) Copeland J, Solomon NW, Mammana RB, Siroky K, Larson D. Cardiac transplantation, a two-year experience. Heart Transpl 1981; 1: 67–71.
(35) Cabrol Ch, Gandjbakhah J, Guiraudon G, *et al.* Cardiac transplantation: our experience at La Pitié Hospital. Heart Transpl 1982; 1: 116–9.
(36) Bregman D. New advances in aortic counterpulsation. Proc Card Surg Update, Jerusalem, May 1982.
(37) Bardos P, Turina M, Meier W, Gattiker R, Rothlin M, Senning Å. Intraaortale Ballongegenpulsation bei Myokardversagen nach herzchirurgischen Eingriffen. Intraaortale Gegenpulsation. Stuttgart: Thieme Verlag, 1977: 132–5.
(38) Reemtsma K, Drusin R, Edie R, Bregman D, Dobelle W, Hardy M. Cardiac transplantation for patients requiring mechanical circulatory support. N Engl J Med 1978; 298: 670–1