Intra-aortic balloon assistance in cardiogenic shock after myocardial infarction or cardiac surgery

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Sixty-eight patients were referred for consideration of intra-aortic balloon assistance, 55 of whom were accepted. Thirty-one patients were in cardiogenic shock after myocardial infarction and the remaining 24 were cardiac surgical patients. Twenty-three of the myocardial infarct group were established on IABA and all 24 of the cardiac surgical patients. Of the 23 patients with cardiogenic shock after myocardial infarction, 19 showed initial haemodynamic improvement on intra-aortic balloon assistance and 5 (22%) survived to leave hospital. Of the 24 cardiac surgical patients, 15 could not be withdrawn from total cardiopulmonary bypass. With intra-aortic balloon assistance, 11 (73%) could be withdrawn from cardiopulmonary bypass and 5 (33%) were hospital and long-term survivors. The remaining 9 surgical patients were in cardiogenic shock in the early postoperative phase, though 5 showed initial haemodynamic improvement; there was only one hospital survivor in this group. Intra-aortic balloon assistance was, therefore, of most value in patients dependent on cardiopulmonary bypass. The survival in patients with cardiogenic shock after myocardial infarction was marginally improved.

The principle of intra-aortic balloon counterpulsation was first suggested by Moulopoulos et al. (1962), and it was subsequently developed for use in cardiogenic shock after myocardial infarction. A balloon catheter positioned at the top of the descending aorta is inflated in diastole and augments the aortic diastolic pressure, thus increasing coronary artery perfusion. With balloon deflation before the beginning of ventricular systole, left ventricular afterload is reduced, with a consequent reduction in ventricular oxygen consumption. A number of patients with cardiogenic shock have now been treated in this way (Kantrowitz et al., 1968b; Dunkman et al., 1972; Scheidt et al., 1973; O’Rourke et al., 1975). Hospital survival may be improved using this technique but authors have varied in the criteria used for concomitant corrective surgery (Mundth et al., 1971; Leinbach et al., 1973; Windsor et al., 1973). Following its use for patients in cardiogenic shock after myocardial infarction intra-aortic balloon assistance has been used in patients with advanced cardiovascular failure after cardiopulmonary bypass (Berger et al., 1973; Buckley et al., 1973a; Housman et al., 1973; Parker et al., 1974). This paper presents our experience over 24 months with the technique in the treatment of cardiac surgical patients and patients in cardiogenic shock after myocardial infarction.

Patients

Sixty-eight patients were referred, of whom 55 were accepted for intra-aortic balloon assistance: 31 patients in cardiogenic shock after myocardial infarction and 24 cardiac surgical patients. Of the accepted patients 23 were referred from units outside our own, 16 postinfarction and 7 surgical patients.

CARDIAC SURGICAL PATIENTS

Of the 24 cardiac surgical patients, 15 had the balloon catheter inserted in theatre and intra-aortic balloon counterpulsation started there; 10 of these patients were treated in our own unit and the remainder in outside units. These were all patients who could not be weaned from total cardiopulmonary bypass despite a left ventricular vent with total supportive bypass, and maximal
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catecholamine support which was used in at least two attempts to dispense with cardiopulmonary bypass. The patients remained on full supportive bypass while the balloon catheter was inserted. The mean time elapsed between the first attempt to dispense with cardiopulmonary bypass and the initiation of intra-aortic balloon pulsation in the patients treated in our own unit was 65 minutes (range 20 to 85 minutes). The equivalent time in patients treated in the units outside our own was largely dependent upon transportation time and was, therefore, considerably longer. These 15 patients had had a variety of surgical procedures undertaken; there were 9 single valve replacements (5 aortic and 4 mitral valves), 2 double valve replacements and 4 patients who had had coronary artery bypass grafts with the additional resection of a left ventricular aneurysm in 2 patients.

Nine patients were treated for cardiogenic shock in the acute postoperative phase. Five of these patients had undergone coronary artery surgery and had received two or more bypass grafts. One patient was treated after aortic valve replacement, two after double valve replacements, and one after mitral valve replacement with the additional closure of a secundum atrial septal defect. The balloon catheter in these acute postoperative patients was inserted at the bedside in the intensive care unit of the referring hospital.

MYOCARDIAL INFARCTION PATIENTS

The patients in cardiogenic shock after myocardial infarction all had evidence of an acute infarct on the electrocardiogram. The other criteria for acceptance were:

1. A peak systolic arterial pressure of less than 90 mmHg confirmed by arterial cannulation.
2. A urine flow of less than 20 ml per hour, measured by bladder catheterisation, in spite of diuretic challenge.
3. Either mental clouding or a significant degree of peripheral skin cooling.

All of the above features had to be present for at least two hours before a patient was accepted on clinical criteria as having cardiogenic shock. They had all received conventional medical treatment which proved ineffective before intra-aortic balloon assistance. The policy was to exclude patients in whom potentially treatable factors could be identified; thus they were excluded if the pulmonary wedge pressure was less than 18 mmHg, indicating potentially reversible hypovolaemia, or if the cardiogenic shock was thought to be the result of a reversible cardiac arrhythmia. In practice none of the patients referred to us had either of these problems. Following confirmation of the acceptance criteria the balloon catheter was inserted under local anaesthetic at the bedside in the coronary care unit of the referring hospital and balloon assistance was started there. Patients treated in peripheral hospitals who required further investigation with a view to operation were transported in an ordinary ambulance on continuing balloon assistance to enable these investigations to be performed.

Methods

A Datascope System 80 was used with a Datascope unidirectional catheter (Bregman and Goetz, 1971; Bregman et al., 1971). A 35 ml balloon was used in the majority of patients though a 30 ml balloon was available for smaller patients. The catheters were all inserted by femoral artery cutdown through an end-to-side 'dacron' graft (Kantrowitz et al., 1968a). The position of the catheter was confirmed by subsequent chest x-ray examination. The recording of arterial pressure from a radial artery cannula and of the electrocardiogram taken from praeordial electrodes, are both essential for the timing of balloon inflation and deflation. When necessary an endocardial pacing system was used. In the cardiac surgical patients any further monitoring was based on the practice of the referring unit. Pulmonary artery wedge pressure was determined in the postinfarction group using Swann Ganz flow-directed catheters. The patients in the postinfarction group were routinely anticoagulated with heparin intravenously in a dose of 1000 units hourly. Anticoagulation was not usually used in the surgical groups until 48 hours after operation. All the patients in cardiogenic shock after myocardial infarction had been receiving catecholamine support before intra-aortic balloon assistance was started. Though this was continued during the first few hours of balloon assistance, the intention was always to dispense with all pharmacological support before any reduction in mechanical support. After 12 to 24 hours the patients' dependence was assessed by temporary withdrawal of balloon assistance. Deterioration in mean arterial pressure, a rise in pulmonary artery wedge pressure, or the recurrence of ischaemic pain were taken as indicators of dependence. No patient was judged to be balloon independent while still on a catecholamine infusion.

Results

The influence of intra-aortic balloon counterpulsation in a patient in cardiogenic shock after cardiac surgery is illustrated in Fig. 1. The augmentation of diastolic pressure and the reduction of systolic pressure during support with the balloon are immediately apparent.
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Intra-aortic Balloon Assistance after Cardiac Surgery

Of the 15 patients who were totally dependent on cardiopulmonary bypass, 11 could be weaned from it with balloon assistance. Five of these patients survived to leave hospital, having required balloon assistance for a mean period of 24 hours (range 2 to 50 hours). They remained fit and well 6 to 24 months later. This represents a salvage rate of 33 per cent in the bypass dependent group who would otherwise have died in theatre. During the period of study, 277 patients were submitted to open heart surgery in King's College Hospital, and 10 (4%) required balloon assistance as they were bypass dependent. Four of these patients had had mitral valve replacements, 3 had had aortic valve replacements, and of the remaining 3, one had an aortic valve replacement with a coronary artery bypass graft, one a double valve replacement and in the remaining patient a left ventricular aneurysm was resected. Of these 10 patients 8 (80%) could be weaned from bypass using intra-aortic balloon assistance and 4 (40%) were hospital survivors.

Intra-aortic balloon assistance produced haemodynamic improvements in only 5 of the 9 patients treated later in the postoperative period. Four of these survived to become balloon independent but only one of them survived to leave hospital. These patients were assisted for a mean period of 42 hours (range 5 to 12 hours).

Cardiogenic Shock after Myocardial Infarction

Of the 44 patients referred, 31 were finally accepted for balloon assistance, 15 from our own unit and 16 from outside units. Patients without definite electrocardiographic evidence of myocardial infarc-

tion were not accepted, and in practice this involved the exclusion of 2 patients who were subsequently shown to have suffered dissection of the aorta. Secondly, patients over the age of 65 were not accepted and this was the major cause for refusal. Finally there were 2 patients who could not be accepted for treatment as the apparatus was in use elsewhere. Balloon catheter insertion failed in 2 of the 31 accepted patients, because of the presence of iliofemoral atheroma. Both of these patients subsequently died within hours. A further 6 patients died before intra-aortic balloon assistance could be established; all of them had sustained a cardiac arrest before the insertion procedure had started. There were, therefore, 23 patients in whom the surgical procedure involved in inserting the catheter was completed and intra-aortic balloon assistance established.

The control data of the 23 patients established on balloon pulsation are shown in Fig. 2. Only 2 patients were assisted who had been in cardiogenic shock for longer than 12 hours, both of them subsequently died on continued assistance. In the remainder of the patients the mean delay between the onset of cardiogenic shock and the establishment of balloon assistance was 7 hours (with a range of 2 to 12 hours). The time elapsing between the onset of symptoms of myocardial infarction and the diagnosis of cardiogenic shock was variable. Two main groups are apparent; first, 16 patients who developed cardiogenic shock within 24 hours of the first pain of myocardial infarction (mean time 13, range 3 to 22 hours). Secondly, 7 patients who

Fig. 1 A radial artery pressure tracing and electrocardiogram taken from a patient in cardiogenic shock after cardiac surgery. As the balloon pump is switched on there is an immediate augmentation of early diastolic pressure from 53 to 97 mmHg and peak systolic pressure is reduced from 72 to 64 mmHg.

Fig. 2 The control mean systemic arterial pressures, mean pulmonary artery wedge pressure, and urine output during the preceding hour of the 23 patients established on intra-aortic balloon assistance after myocardial infarction.
developed shock from 1 to 7 days after their initial symptoms. Fourteen of the patients had electrocardiographic evidence of anterior wall infarction, 6 of inferior wall infarction, and there were 3 patients who had evidence of new changes in both anterior and inferior leads. Eleven patients had had a previous myocardial infarction. One patient had an acquired ventricular septal defect which was confirmed at subsequent cardiac catheterisation. Acute mitral regurgitation was not responsible for shock in any patient.

The response of mean arterial pressure in the first 24 hours of assistance is shown in Fig. 3. The patients are divided into two groups. First, 9 patients who survived the acute phase of cardiogenic shock and who then either had the balloon catheter removed (7 patients), or who being haemodynamically stable, but balloon dependent, were submitted to corrective surgery. Secondly, those 14 patients who failed to survive the acute phase, of whom half died during the course of the first 24 hours. There was no significant difference in the response of mean arterial pressure between these two groups of patients. However, the response of urine output to balloon assistance provided a reliable prognostic indicator. Fig. 4 shows the urine flow in the same two groups of patients at the same time. There was a uniform and copious increase in the urine output of the acute survivors which is significantly greater than that of the non-survivors at 1, 4, and 24 hours. The non-survivors also showed an increase in urine output at these times which was significantly greater than the control value (P < 0.01).

Diagnostic cardiac catheterisation, including coronary angiography and left ventriculography, was performed in 10 patients. During the first year of the experience presented here, coronary angiography was performed only on those patients who were haemodynamically stable but balloon dependent. In the latter part of the experience, however, all patients who achieved haemodynamic stability were investigated. Emergency revascularisation was only considered in the balloon-dependent group. Of these 8 balloon-dependent patients, only one had an adequate run-off visible on the coronary arteriogram distal to a proximal stenosis and proceeded to surgery. This patient had been haemodynamically stable for 48 hours and underwent coronary artery bypass grafting to two arteries. One further patient with a large anterior septal myocardial infarction developed a ventricular septal defect giving rise to a 3:1 left-to-right shunt. After a period of intra-aortic balloon assistance he was submitted to operation 7 days after the onset of symptoms. Neither of the 2 patients who were operated upon could subsequently be weaned from cardiopulmonary bypass.

Of the 23 established patients, therefore, 5 survived to leave hospital. Three of them are alive 6 to 24 months later. This represents a hospital survival of 23 per cent in the patients established on intra-aortic balloon assistance. Acute survivors required balloon assistance for a mean 30 hours (range 19 to 70 hours). The non-survivors died after 2 to 32 hours of assistance.
The only complications encountered were the result of the reduction of arterial flow in the iliofemoral system through which the balloon catheter was inserted. Five patients had evidence of impaired circulation. Two of these failed to show any significant haemodynamic response and both died before impairment of peripheral circulation required any action. One patient had been judged to be balloon independent after myocardial infarction, and the balloon catheter was removed from the affected leg earlier than would otherwise have been intended. Though satisfactory recirculation to the leg was established in this patient, he subsequently suffered further haemodynamic deterioration and died. There were two patients in the surgical group, one of whom had been cardiopulmonary bypass dependent in theatre in another hospital. They both became haemodynamically stable after intra-aortic balloon assistance but required limited amputation (one below knee and one above knee) because of lower limb ischaemia after removal of the balloon catheter. Both of these patients died later in their hospital course. In one death was the result of incidental biliary peritonitis resulting from a rupture of the gall bladder, and in the other an acute myocardial infarction developed because of thrombotic occlusion of the bypass graft. In one further patient there was necropy evidence that the balloon catheter had caused a limited aortic dissection through an atheromatous plaque at the iliofemoral junction subsequently re-entering the aortic lumen, with no clinical evidence of arterial impairment. There were no embolic complications in any of the patients in this series. The insertion of the balloon catheter with subsequent balloon assistance was not thought to have caused death in any patient.

Discussion

Intra-aortic balloon counterpulsation has two major haemodynamic effects. Inflation of the balloon at the start of diastole produces augmentation of the aortic diastolic pressure. In the conditions that obtain in cardiogenic shock, autoregulation probably has little part to play in the control of coronary blood flow (Mueller et al., 1973), leaving coronary artery filling pressure as the major determinant of flow (Powell et al., 1970). Thus Mueller et al. (1971) found that coronary artery flow increased in patients over the first 4 to 6 hours of balloon pumping. However, in patients who had recovered from the acute phase of cardiogenic shock as a result of treatment with intra-aortic balloon assistance and who were investigated 17 hours after the initiation of therapy, Leinbach et al. (1971) were unable to show any significant effect on coronary artery flow. This finding may represent recovery of autoregulatory function in the coronary arteries with haemodynamic improvement. The second haemodynamic effect of intra-aortic balloon assistance results from deflation of the balloon before the onset of ventricular systole, which produces a forward flow in the column of blood between the aortic valve and the balloon at the top of the descending aorta. This results in a lowering of peak systolic pressure generated by the left ventricle which in turn has a favourable effect on myocardial oxygen consumption (Soroff et al., 1963; Mueller et al., 1971).

Cardiogenic shock occurring in the acute phase of myocardial infarction is universally accepted to carry a poor prognosis. However, the exact hospital mortality is variously quoted and depends upon the diagnostic criteria used. Scheidt et al. (1970) found a hospital mortality of 86 per cent in patients managed medically. Scheidt et al. (1973) later emphasised that the mortality in shocked patients refractory to conventional medical treatment increases and probably approaches 100 per cent. All the patients in the present study were refractory to conventional medical therapy.

The criteria used for the acceptance of patients after myocardial infarction in this study were of necessity purely clinical as it was our aim to provide a service in coronary care units which do not necessarily have the facilities for detailed haemodynamic investigation. Furthermore, O'Rourke et al. (1975) emphasised 'the relentless deterioration' occasioned by prolonged haemodynamic assessment. Though the criteria which were applied appear to have successfully identified a critically ill group of patients, in common with other reviews of experience with intra-aortic balloon assistance in cardiogenic shock, there is no satisfactory control group. It is, therefore, difficult to be certain of the true extent to which intra-aortic balloon assistance improves survival in the present study. In a search for suitable controls, the two patients in whom balloon catheter insertion failed and subsequently died would not be representative by virtue of the advanced state of disease in their peripheral arteries. Similarly, those whose age precluded consideration of intra-aortic balloon assistance are also unrepresentative. A more suitable control group would be provided by those patients who fulfilled the acceptance criteria, but could not be offered intra-aortic balloon assistance because the apparatus was in use elsewhere. To date, however, there have been only two such patients, both of whom died.

The overall hospital survival in patients estab-
lished on intra-aortic balloon assistance with shock after myocardial infarction which was refractory to conventional medical therapy was 22 per cent of the present series. This compares favourably with 17 per cent of 87 reported by Scheidt et al. (1973), 16 per cent of 44 patients reported by Dunkman et al. (1972), 24 per cent of 80 patients reported by Leinbach et al. (1973), and 11 per cent of the 18 patients reported by Willerson et al. (1975). It is less favourable than the 35 per cent achieved in patients in shock by O’Rourke et al. (1975). Clearly one of the problems is to identify the appropriate place of angiography and corrective surgery. The role of intra-aortic balloon assistance in the management of acquired ventricular septal defects and acute mitral regurgitation has been described and recently emphasised (Buckley et al., 1973b; Slama et al., 1975). Though the only such patient reported here subsequently died at operation, he was at least operated upon while well perfused and polyuric, having been anuric and in a state of profound cardiogenic shock 48 hours previously in a peripheral hospital. We now regard the presumed presence of an acquired ventricular septal defect associated with anything more than mild haemodynamic impairment as a positive indication for intra-aortic balloon assistance after myocardial infarction (Slama et al., 1975). The role of emergency revascularisation with or without infarctectomy is more problematical. It seems reasonable to confine attempts at emergency revascularisation to the group of patients who are haemodynamically stable but balloon dependent (Leinbach et al., 1972, 1973). In the series reported here, however, there were few patients who were balloon dependent and who survived for a significant period of time. It is for this reason that few attempts at emergency revascularisation were made. The question remains as to whether the balloon independent group should be submitted to coronary angiography. On the one hand is the obvious reluctance to submit this group of patients to any further risk. On the other hand there is a disquieting situation which occurred in 2 patients who became balloon independent but who deteriorated and died later in their hospital course after removal of the balloon catheter. Had an assessment of their operability been available as a result of prior angiography, a decision to reinstitute intra-aortic balloon assistance and perhaps undertake late surgery would have been reached more easily.

Despite the difficulty, referred to above, in assessing the benefit of this treatment in the absence of control patients, there appears to have been an increased survival in the shocked group of patients after myocardial infarction shown here which, albeit marginal, certainly justifies the continued use of intra-aortic balloon assistance in cardiogenic shock. However, this is a relatively rare condition. Kuhn (1974) found only 28 shocked patients in 636 consecutive cases of myocardial infarction, only 10 (1.5%) of whom were aged less than 65. Most of these patients will, of course, present to district hospitals and it is there that the attempt to salvage them must be made. We have shown that intra-aortic balloon assistance can be satisfactorily provided on a regional basis using no more specialised equipment than the system itself. Presentation to a peripheral hospital did not affect the mortality adversely but rather the reverse, since all our survivals after myocardial infarction initially came from outside our own unit.

It is particularly pertinent to consider what role intra-aortic balloon assistance might have in the management of patients whose cardiovascular failure occurs after infarction but in whom deterioration and the development of frank cardiogenic shock have not yet occurred. Since even in cardiogenic shock haemodynamic improvement occurred in 19 of the 23 patients established on intra-aortic balloon assistance it is attractive to consider using it in a less severe group. The hope would be that the augmentation of coronary blood flow, together with the reduction of left ventricular afterload and therefore myocardial oxygen consumption, would reduce or limit infarct size and so improve mortality. The difficulties of designing a suitably controlled prospective trial of this invasive therapy in less-sick patients has to date limited its application. It is likely that in the future such an application will be more extensively studied.

In the management of cardiac surgical patients there was a clear distinction in the efficacy of intra-aortic balloon assistance between the two categories; those who were totally dependent on cardiopulmonary bypass and treated in theatre, and those who were treated later in the intensive care unit. In the second group 3 patients were treated in the first few months of our experience and a further 3 in units outside our own. Treatment may, therefore, have been unduly delayed. Nevertheless, the failure of 4 of the 9 patients to show any haemodynamic response to intra-aortic balloon assistance and the hospital survival of only one cannot be considered encouraging. In contrast there were 15 patients who were cardiopulmonary bypass dependent and presumably without intra-aortic balloon assistance they would all have died. The fact that 10 were subsequently weaned from cardiopulmonary bypass and 5 (33%) survived to leave hospital shows the effectiveness of intra-aortic balloon assistance in this category of patients. The balloon pump,
therefore, finds its major application in the resuscitation of patients totally dependent on cardiopulmonary bypass and it must be considered an essential adjunct to the practice of cardiac surgery.

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References


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