Arterial hypertension in immediate postoperative period after valve replacement

F. GEORGE ESTAFANOUS, ROBERT C. TARAZI, SHERYL BUCKLEY, AND PAUL C. TAYLOR

From the Department of Cardio-Thoracic Anesthesiology; the Research Division; Division of Anesthesiology; and the Department of Thoracic and Cardiovascular Surgery, The Cleveland Clinic Foundation and the Cleveland Clinic Educational Foundation, Cleveland, Ohio, U.S.A.

SUMMARY Paroxysmal hypertension occurred during the first 8 hours after cardiac valve replacement in 15 of 186 consecutive patients. The clinical characteristics of this hypertension were similar to those of hypertension after myocardial revascularisation, except that this complication occurred much less frequently after valve replacement (8.1%) than after myocardial revascularisation (33%) ($P < 0.001$). Hypertension resulting from hypoxia, hypercapnia, shivering, or arousal from anaesthesia was excluded from consideration.

The rise in systemic arterial pressure (average $34/35$ mmHg $\pm 4.9/4.3$ SE) was usually associated with a reduction in central venous pressure (12/15 patients) and a mild increase (2 to 4 cm saline) in left atrial pressure. The incidence of hypertension was not related to the valve replaced (aortic or mitral), type of lesion (stenosis or regurgitation), preoperative level of blood pressure, or use of hypothermia during operation. However, none of the 18 patients who had double valve replacement showed significant rise in blood pressure after operation.

It is suggested that these hypertensive episodes may be related to pressor reflexes from the heart and/or great vessels.

Systemic arterial hypertension, sometimes quite severe and always potentially dangerous, has been reported to occur in the immediate postoperative period after open heart surgery (Estafanous *et al.*, 1973; Estafanous, 1975; Chaptal *et al.*, 1975). It occurs after both coronary bypass surgery (Estafanous *et al.*, 1973; Estafanous, 1975; Chaptal *et al.*, 1975) and cardiac valve replacement (Cleland *et al.*, 1969; Braimbridge and Branthwaite, 1972; McQueen *et al.*, 1972), but has been said to be much less frequent in the latter situation (Estafanous *et al.*, 1973). However, our initial report dealt with fewer cases of valve replacement (38) than of coronary bypass (76). Other reports were concerned only with hypertension after valve replacement and did not include coronary bypass cases (Cleland *et al.*, 1969; Braimbridge and Branthwaite, 1972; McQueen *et al.*, 1972). Further, the numbers involved in the latter series were also relatively small and the criteria for hypertension and the time of its occurrence differed in these reports. It still is not clear, therefore, whether postoperative hypertension is more frequent after myocardial revascularisation than after other

types of open heart surgery. More important, it has yet to be established whether the clinical picture and associated haemodynamic changes were the same in both circumstances. To that end a clinical study by the same group of investigators applying the same criteria was appropriate.

We have, therefore, investigated 186 patients who have had valve replacement operations; the results were compared with our experience in over 5000 myocardial revascularisation procedures (Estafanous *et al.*, 1972, 1973; Estafanous, 1975).

Methods

The postoperative records of 186 consecutive patients who had had an operation for cardiac valve replacement were reviewed. Of these, 85 patients had had mitral valve replacement, 83 aortic valve replacement, and 18 patients had had both valves replaced.

Premedication consisted of Pantopon (0.3 mg/kg body weight) intramuscularly and promethazine hydrochloride (0.25 mg/kg body weight). Anaesthesia was induced with sodium thiopentone (3 to 5 mg/kg body weight), and a muscle relaxant was

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selected on the basis of the patient's blood pressure; *d*-tubocurarine (0.7 mg/kg body weight) was given when the mean arterial pressure exceeded 90 mmHg or pancuronium bromide (0.1 mg/kg body weight) when it was below 90 mmHg (Estafanous *et al.*, 1972). In severely ill patients, an initial intravenous injection of 40 mg Pantopon was followed by smaller doses of sodium thiopentone (1 to 2 mg/kg body weight). Anaesthesia was then maintained with 60 per cent nitrous oxide in 40 per cent oxygen and either analgesic concentrations of methoxyflurane (penthrane 0.3 to 0.5 per cent) or intermittent 5 mg doses of Pantopon; the muscle relaxant, pancuronium bromide or curare, was given as required. During operation and for the first 24 hours after operation all patients were ventilated artificially. Arterial blood gas tensions and pH and serum electrolytes were estimated at half-hour intervals during operation and every 6 hours after operation. The amount and pattern of ventilation were continuously adjusted to maintain P_{aO_2} above 150 mmHg and P_{aCO_2} between 30 and 35 mmHg. Any abnormality in pH or serum electrolytes was specifically corrected. Mannitol was administered during cardiopulmonary bypass and after operation whenever urine output fell below 40 ml/hr. Blood losses were measured and replaced as indicated by arterial, central venous, and left atrial pressure levels. Pantopon (5 to 10 mg intravenously) was given postoperatively as needed for analgesia and sedation to ensure adequate synchronisation with the ventilator.

SURGICAL TECHNIQUE

Single valve replacement in patients with unimpaired ventricular function was usually performed under normothermic conditions using a disposable bubble-bag oxygenator and moderate haemodilution. The coronary arteries were not perfused. Anoxic arrest lasted for an average of 30 to 40 minutes. In patients requiring double valve replacement as well as in those with massive left ventricular hypertrophy or with impaired myocardial function, moderate systemic and local hypothermia was used. Systemic cooling was achieved through the extracorporeal circulation to a temperature of 28 to 30°C. The heart was further cooled by a cardioplegic solution made up of 0.893 g KCl, 3.248 g $MgCl_2$, and 0.273 g procaine hydrochloride dissolved in one litre Ringer's solution.

We are currently using the Starr-Edwards (series 1260 or 6120), Cutter-Smeloff, Björk-Shiley, and Hancock bioprostheses for valve replacement. In general interrupted suture technique is used to secure a prosthesis in the aortic or mitral position.

POSTOPERATIVE MONITORING OF BLOOD PRESSURE AND CRITERIA FOR DIAGNOSIS OF HYPERTENSION

In all patients the radial artery was cannulated and the intra-arterial mean pressure recorded was compared with the pressure obtained by sphygmomanometry. The arterial cannula was usually removed after 24 hours and the arterial pressure was then only monitored by sphygmomanometry. Arterial pressure readings were obtained routinely at 15-minute intervals for the first 12 hours after operation and at half-hour intervals for the following day. Central venous pressure was monitored via a catheter inserted percutaneously in the internal jugular vein and advanced to the superior vena cava. The left atrial pressure was monitored during operation via a similar catheter inserted in the left atrium at its junction with the superior pulmonary vein. These catheters were left *in situ* for 24 to 48 hours.

The criteria used to determine hypertension were the same as those used in our previous studies (Estafanous *et al.*, 1973). Postoperative hypertension was defined as an increase in blood pressure to a specified level, not caused by shivering, fighting the ventilator, intolerance to the endotracheal tube, or administration of vasopressor agents. Inotropic drugs (a mixture in varying ratios of isoprenaline and adrenaline) were used during operation; a third of the patients required them after return to the intensive care area. A rise in arterial blood pressure occurring within 60 minutes of discontinuing the inotropic agents was not included. Also excluded was any hypertensive episode that was rapidly controlled by adequate analgesia and, therefore, presumably related to pain or discomfort (Estafanous *et al.*, 1973). Hypertension was defined as follows: (a) for preoperatively normotensive patients, a rise in diastolic pressure to 100 mmHg and/or a rise in systolic pressure to 160 mmHg; (b) for preoperatively hypertensive patients, a rise in either systolic or diastolic pressure to at least 30 mmHg above preoperative levels.

Tests of statistical significance were calculated by standard methods using Student's *t* test and χ^2 values; Yates' correction was used whenever the number of observations was less than 100 (Croxtton and Cowden, 1941).

Results

Hypertensive episodes after valve replacement procedures were documented in 15 of the 186 patients (Table 1). These episodes occurred after mitral valve replacement as well as after aortic valve replacement. None of the patients who had

Table 1 Incidence of postoperative hypertension after valve replacement as related to pathological lesion and type of valve used

	Mitral valve replacement		Aortic valve replacement		Mitral and aortic valve replacement	
	No. studied	Postoperative hypertension	No. studied	Postoperative hypertension	No. studied	Postoperative hypertension
Total	85	5	83	10	18	None
Valve lesion						
Stenosis	25	4	34	5		
Regurgitation	33	1	24	4		
Both	27	0	25	1		
Preoperative status						
Normotensive	73	5	58	7	17	0
Hypertensive	12	0	25	3	1	0
Artificial valve used						
Prosthetic	66	3	59	6	12	0
Porcine	19	2	24	4	4	0

Table 2 Hypertension after mitral valve replacement in 5 patients

Type of valve	Preoperative lesion		Preoperative blood pressure (mmHg)	Increase in blood pressure		Changes in		Onset after operation	Duration
	Stenosis	Regurgitation		Systolic (mmHg)	Diastolic (mmHg)	CVP*	LAP*		
Prosthetic		+	120/80	5	25	↑	↑	Immediate	4 h
Porcine	+		160/80	60	20	↓	↑	45 min	2 h
Porcine	+		110/90	25	20	↓	↑	15 min	2 h
Prosthetic	+		120/60	10	50	↑	↑	½ h	18 h
Prosthetic	+		150/90	30	20	↓	—	6 h	9 h

* ↑ Rise in central venous pressure (CVP) or left atrial pressure (LAP) 3 to 4 cm H₂O; ↓ Decrease in CVP or LAP 3 to 4 cm H₂O.

both valves replaced at the same time had postoperative hypertension.

MITRAL VALVE DISEASE

This group included 85 patients; 25 with predominant stenosis, 33 with predominant regurgitation, and 27 with stenosis and regurgitation (Table 1). Postoperative hypertension occurred in 5 patients in this group (5.9%). The rise in blood pressure started in most within one hour after operation and could persist for as long as 6 hours; both systolic and diastolic pressure levels increased (31 mmHg $\pm$ 10.5 (SE) for the systolic and 27 $\pm$ 5.8 for the diastolic) (Table 2). Despite the rise in arterial pressure, the central venous pressure showed no significant increase and remained normal. In contrast, left atrial pressure increased by 3 to 4 cm H₂O as mean atrial pressure rose, but always remained within normal range for patients on intermittent positive pressure breathing (12–16 cm H₂O). Postoperative hypertension occurred more frequently in patients with mitral stenosis (4/25) than in those with predominant regurgitation (1/33), but this difference was not statistically significant ($\chi^2 = 1.614$, $P < 0.05$). None of the 27 patients with mixed mitral valve disease developed postoperative hypertension. The type of artificial valve

used did not seem to be related to the incidence of postoperative hypertension which was noted as frequently with prosthetic valves as with heterografts. The preoperative blood pressure was also not a factor in the incidence of postoperative hypertension (Table 1).

AORTIC VALVE DISEASE

This group included 83 patients, 34 with aortic stenosis, 24 with regurgitation, and 25 with a mixed aortic stenosis and regurgitation (Table 1). Postoperative hypertension developed in 10 patients (12.05%). As with mitral valve patients, the rise in blood pressure occurred any time in the first 5 hours after operation and lasted up to 6 to 12 hours; both systolic and diastolic pressure levels increased (37 mmHg $\pm$ 4.9 (SE) for the systolic and 40 $\pm$ 6.0 for the diastolic) (Table 3). In all central venous pressure remained within normal limits. Left atrial pressure which had returned to a low normal level (12 to 16 cm H₂O) after operation, indicating rapid improvement in the left ventricular function in these patients, rose slightly (by 2 to 3 cm H₂O), but remained within normal limits (< 18 cm H₂O). Hypertension occurred in patients with aortic stenosis as often as in those with aortic regurgitation. However, as in patients with mitral valve disease,

Table 3 Hypertension after aortic valve replacement in 10 patients

Type of valve	Preoperative lesion		Preoperative blood pressure (mmHg)	Increase in blood pressure		Changes in		Onset after surgery	Duration
	Stenosis	Regurgitation		Systolic (mmHg)	Diastolic (mmHg)	CVP*	LAP*		
Prosthetic	+		100/60	50	50	↓	—	1½ hr	12 h
Prosthetic		+	170/60	10	50	↓	—	30 min	2 h
Porcine	+		140/80		10	↓	—	45 min	6 h
Prosthetic		+	130/70	20	40	↓	—	15 min	3 h
Porcine		+	180/50	10	60	↓	—	45 min	2 h
Prosthetic	+	+	118/84	30	16	↓	—	45 min	5½ h
Porcine		+	180/30		70	↓	—	Immediate	3½ h
Prosthetic	+		130/70	30	35	↓	—	Immediate	3½ h
Porcine	+		140/80	40	20	↑	↑	1 h	4½ h
Prosthetic	+		122/68	50	15	↓	—	1 h	1½ h

* ↑ Rise in central venous pressure (CVP) or left atrial pressure (LAP) 3 to 4 cm H₂O; ↓ Decrease in CVP or LAP 3 to 4 cm H₂O.

postoperative hypertension was rare in those with mixed stenosis and regurgitation (only 1 episode in 25 patients). Again, it did not seem that the type of prosthetic valve used influenced the incidence of postoperative hypertension since this occurred as frequently with heterografts as with prosthetic valves.

Hypothermia was used in only one patient with mitral valve disease, but was used in 15 of 83 aortic valve replacement procedures. However, its use during operation in these patients did not seem to influence the incidence of postoperative hypertension (2/15). As in the mitral valve disease group and other groups of patients previously reported (Estafanous *et al.*, 1973; Estafanous, 1975), pre-operative hypertension was not a predisposing factor in the development of hypertensive episodes after aortic valve operations (Table 1).

MITRAL AND AORTIC VALVE DISEASE

Of the 18 patients who had both aortic and mitral valves replaced, none showed a significant rise in blood pressure after operation.

Discussion

Systemic hypertension in the first few hours after open heart operations is being recognised with increasing frequency as a potentially serious complication (Estafanous *et al.*, 1973; Estafanous, 1975; Chaptal *et al.*, 1975). Initial reports from our group were confirmed by others which also stressed the particular hazards associated with the rise in arterial pressure in this initial period. Whereas the frequency of early hypertension after myocardial revascularisation has been well documented (Estafanous *et al.*, 1973), its incidence after other types of open heart operation has not yet been established. This is largely because of the small number of cases reported and the different diagnostic criteria used. Both for practical and theoretical reasons, it is

important to determine whether this postoperative rise in blood pressure occurs only after coronary revascularisation procedures. Therefore, in this study we have compared its incidence after valve replacement in 186 consecutive patients with our recent experience in over 5000 myocardial revascularisation procedures (Estafanous *et al.*, 1973; Estafanous, 1975; Viljoen *et al.*, 1976). By continuous monitoring of these 186 patients a total of 15 hypertensive episodes were recognised, 5 after mitral valve replacement and 10 after aortic valve replacement. This incidence (8.1%) contrasted sharply and significantly ($P < 0.001$) with the 33 per cent observed after coronary artery surgery (Estafanous *et al.*, 1973; Estafanous, 1975; Chaptal *et al.*, 1975). The difference is considered especially significant because the same criteria were used by the same investigators to examine the problem.

The clinical characteristics of the hypertension after cardiac valve replacement were similar to those associated with hypertension after revascularisation (Estafanous *et al.*, 1973; Viljoen *et al.*, 1976). Blood pressure began to rise within the first 4 to 6 hours after operation; if not treated, the hypertension lasted for from 75 minutes to 18 hours (median 5 hours). During the hypertensive episode, the central venous pressure decreased by 2 to 3 cm H₂O in the majority of patients (12/15), and increased (though remaining within normal limits) in 3/15. In contrast, left atrial pressure which was monitored in only 5 patients increased slightly in all 5. These variations in atrial pressure were important only in a negative way, indicating the absence of cardiac decompensation, despite the rise in arterial pressure during the critical postoperative period.

Subsequent analysis of possible factors that may predispose to postoperative hypertension disclosed no important or over-riding factor. In our study, we excluded nonspecific rises caused by hypoxia, hypercapnia, pain, or shivering. We found no

significant difference between the incidence of this complication in preoperatively normotensive and in preoperatively hypertensive patients (12/148 or 8.1% and 3/38 or 7.9%, respectively) (Table 1). Our previous experience with hypertension after myocardial revascularisation was similar (Estafanous *et al.*, 1973; Chaptal *et al.*, 1975; Viljoen *et al.*, 1976). It is possible that this paradoxical finding may result from the minor differences in the numerical criteria for the definition of postoperative hypertension in the two types of patients. However, also important in this regard is our policy of continuing most antihypertensive agents until the day of operation. Beta-blocking drugs (usually propranolol) were not often used in these patients and were always discontinued for a variable period (from 4 days to 2 weeks) before operation. Other antihypertensive drugs included methyldopa, diuretics, and reserpine. It might have been reasonable to assume that hypertensive subjects would be likely to respond to pressor influences more consistently and with higher rises in arterial pressure than normotensive subjects. All 38 hypertensive subjects received one or more of the drugs (diuretics, methyldopa, or reserpine) listed above; it is possible that a lingering effect of these medications might have blunted the pressor response.

Neither the type of valve lesion nor its site seem to influence the incidence of postoperative hypertension. Likewise in patients with a single valve lesion there was no difference between those with predominant stenosis and those with predominant regurgitation. Though 4 of 25 patients with mitral stenosis (16.0%) had a significant rise in blood pressure after mitral valve replacement compared with only 1/33 with mitral regurgitation (3.03%), the difference was not statistically significant ($P > 0.10$). These results parallel those of Austen *et al.* (1966) who found no significant haemodynamic difference between patients with mitral stenosis and those with mitral regurgitation immediately after mitral valve replacement. There appeared to be a small difference in the incidence of postoperative hypertension after aortic valve replacement and after mitral valve replacement (12.05% and 5.89%, respectively). This difference is not statistically significant, but, if confirmed by further observations, might indicate the importance of myocardial function and changes in cardiac performance after operation. Rastelli and Kirklin (1967) reported that cardiac output, left ventricular function, and filling pressure improved to a greater extent after aortic valve replacement than after mitral valve replacement. Another observation also seems to substantiate the hypothesis that improvement in myocardial performance may play a role in early postoperative

hypertension. None of the 18 patients who had double valve replacements developed postoperative hypertension. This could be attributed to the relatively small number of patients or to the presumably greater severity of myocardial disease in patients needing replacement of both valves. However, final assessment of this factor must await the direct correlation of the results of haemodynamic and ventricular function studies. Observations in individual patients will be more important in this respect than group differences. Many patients who had mitral valves replaced did develop postoperative rises in arterial pressure, both in our experience and that of Rastelli and Kirklin (1966).

Investigation of other factors also yielded negative results. Thus, the type of artificial valve used made no significant difference to the incidence of postoperative hypertension. The hypertension reported after homograft aortic valve replacement (Layton *et al.*, 1973) is a delayed complication of that operation probably different from the immediate postoperative condition described here. The anaesthetic techniques used were similar for all our patients except for the occasional use of hypothermia. Hypothermia was used in only one of our mitral valve patients, but in 15 of 38 patients who had aortic valve replacement. There was however no statistically significant difference in the incidence of postoperative hypertension related to the use of hypothermia. The postoperative rise in blood pressure was also not related to hypervolaemia, at least as indicated by changes in central venous and left atrial pressure as hypertension developed. These results confirm our previous experience with myocardial revascularisation operations (Estafanous *et al.*, 1973; Estafanous, 1975).

These clinical similarities indicate that early postoperative hypertension may be a complication of many types of open heart surgery. This is not surprising, since there are several factors common to all patients undergoing such procedures. Cardiac output and left ventricular function can improve after valve replacement (Kloster *et al.*, 1966) and also, in some patients, after myocardial revascularisation (Moran *et al.*, 1973). Peripheral resistance usually increases after the use of nonpulsatile flow during cardiopulmonary bypass, particularly in the first few hours after operation (Dammann *et al.*, 1963). These haemodynamic sequelae would result in a rise in systemic arterial pressure because of the increase in both output and resistance. Changes in blood volume may be important but do not appear to be relevant to this form of hypertension, since there was no evidence of hypervolaemia or hypovolaemia in association with the rise in blood pressure (Estafanous *et al.*, 1973; Estafanous, 1975;

Viljoen *et al.*, 1976). Obviously, other factors must be involved in addition to those mentioned above, since not all patients develop postoperative hypertension. Furthermore, disturbances related only to open heart surgery cannot by themselves fully explain the difference in the incidence of this complication between myocardial revascularisation and valve replacement procedures.

We have previously suggested that the rise in blood pressure after myocardial revascularisation might be related to pressor reflexes from the heart and/or great vessels (Peterson and Brown, 1971; Estafanous *et al.*, 1973; Liard *et al.*, 1975; Viljoen *et al.*, 1976). This suggestion was supported by the subsequent observation that hypertensive episodes frequently occur during total bypass when the aorta is clamped and systemic flow artificially maintained at constant levels. Under these conditions the rise in pressure must reflect changes in peripheral resistance possibly secondary to such pressor reflexes. The frequency of hypertension after myocardial revascularisation might then be related to the greater likelihood of alterations in coronary haemodynamics. Preliminary trials of stellate ganglion block to interrupt such reflexes have shown that this rapidly controls postoperative hypertensive episodes in many patients (Tarazi and Gifford, 1975). These observations are obviously no more than pointers to possible factors. Serial haemodynamic measurements are needed to establish their relevance.

Whatever the basic mechanism, these hypertensive episodes are potentially dangerous; they increase the incidence of bleeding both from thoracotomy and aortotomy incisions and may predispose to cerebrovascular accidents or to cardiac or renal failure. The demands imposed on the left ventricle by the rise in systemic pressure may be deleterious after operation (Layton *et al.*, 1973). These potential dangers of hypertension after heart surgery necessitate careful monitoring, prompt recognition, and immediate treatment. The special circumstances associated with open heart surgery preclude the use of many of the potent antihypertensive agents (Tarazi and Gifford, 1975). We found sodium nitroprusside to be the most effective and reliable drug at this stage; it was administered while monitoring arterial and central venous pressure, and also left atrial pressure when possible. The infusion was usually begun at an initial rate of 20 $\mu\text{g}/\text{min}$ and then adjusted to maintain mean arterial pressure between 80 and 100 mmHg; the median dose needed was 40 $\mu\text{g}/\text{min}$. After the initial reduction in pressure, the drug had to be continued for 2 to 18 hours (median 5 hours). However, this approach is reserved for those patients

with particularly severe hypertension with unrelenting rise in blood pressure. The insistence on early diagnosis and prompt treatment has effectively prevented significant complications in any of the patients followed in the past 2 years.

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Requests for reprints to Dr F. G. Estafanous, Department of Cardiothoracic Anesthesia, Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44106, U.S.A.