Myocardial damage and valve replacements

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Forty patients who came to necropsy over a period of five years due to myocardial damage sustained during cardiac valve replacements were studied. The clinical presentation of myocardial damage was assessed in relation to the preoperative cardiac status. The cause and nature of myocardial damage were assessed at necropsy. Evidence of clinical, electrocardiographic and aortographic coronary atheroma was correlated with distribution at necropsy, and the value of selective coronary angiography in perfusing the coronary arteries during cardiopulmonary bypass is stressed. The causes of myocardial damage could be classified as (a) thrombo-embolic, (b) iatrogenic damage to the coronary arteries, and (c) poor or absent coronary perfusion during cardiopulmonary bypass. A case is made for the importance of coronary perfusion during cardiopulmonary bypass.

With better understanding of the nutrient requirements of the myocardium, deaths due to myocardial damage sustained during cardiopulmonary bypass are now fortunately infrequent. However, over a period of five years (1965 to 1969) 40 such patients came to necropsy at Sully Hospital, an incidence of about 5% in a yearly output of about 120 valve replacements. The aim of this paper is to study the pathophysiology of myocardial damage in these patients with reference to the incidence of coronary artery disease and to assess the factors adverse to the myocardium during and after cardiopulmonary bypass. In particular, the role of coronary perfusion is outlined, because its use is debatable, and because oxygen requirements and the coronary blood flow demands of the myocardium with non-pulsatile flow and hypothermic perfusate in the beating and fibrillating heart are only now being gradually understood (Sterns, Bonnabeau, and Lillehei, 1966; Vasko, 1967). When coronary atheroma predominates and results in areas of perfusion deficit in the myocardium, the role of coronary perfusion during cardiopulmonary bypass assumes all the more importance. It is in respect of this role that we have looked critically at our coronary perfusion techniques and related them to the myocardial damage sustained in this group of patients. Some of the surgical hazards to the coronary circulation which contributed to the myocardial damage in some of these patients are detailed.

MATERIALS AND METHODS

CLINICAL MATERIAL Table I depicts the age distribution in our patients. The sex ratio was equal. Table II analyses the valve replacements carried out.

TABLE I
AGE DISTRIBUTION

Age (yr)	No. of Patients
Below 30	2
31-40	5
41-50	12
51-60	17
61 and over	4
Mean age 55.3 years	

TABLE II
VALVE REPLACEMENTS

	No. of Patients
Aortic valve	24
Mitral valve	11
Aortic and mitral valves	3
Aortic, mitral and tricuspid valves	2

Cardiac status was appraised by right and left heart catheterization. Table III shows the cardiac index, pulmonary artery pressures, and pulmonary vascular resistance in these patients.

A history of chest pain typical of angina pectoris was present in 16 patients preoperatively. Thirteen of these had aortic valve disease and 3 mixed mitral lesions. Electrocardiographic evidence of myocardial ischaemia, as shown by the presence of 'Q' waves and ST-T changes, occurred in 4 of these patients. Table IV summarizes the findings as regards coronary artery filling in 32 aortograms performed.

PERFUSION DETAILS Perfusion technique in the 40 patients was fairly uniform. A 24-in. Kay-Cross type disc oxygenator was used in most cases, and body temperature was brought down to 30° C. Active cooling of the body to below 25° C. was undertaken in

TABLE III

	No. of Patients
Cardiac index (l./min./m. ²)	
Below 1.5	1
1.6-2.0	6
2.1-2.5	6
2.6-3.0	3
Above 3.0	7
Total	23
Pulmonary artery pressure (mm.Hg)	
Below 40	14
40-60	4
Over 60	8
Total	26
Pulmonary vascular resistance (units)	
Below 4	14
4-12	8
Over 12	1
Total	23

TABLE IV
AORTOGRAPHIC FINDINGS

	No. of Patients
Good coronary filling of both vessels	22
Poor coronary filling of one vessel:	
Left coronary	3
Anterior descending branch	3
Circumflex branch	2
Right coronary	1
Poor coronary filling of both vessels	1

five patients when the perfusion flows fell too far below the predicted values. Haemodilution with PCV of about 30% was usual. Blood gases and serum potassium were monitored every half an hour. In the single patient who developed a base deficit of -10, the low perfusion pressure resulted from proximal obstruction in the left femoral artery, the site of arterial return. In only one patient did the serum potassium level drop below 3.0 mEq/litre for any two readings. Table V shows the total aortic cross-clamp time in the 40 patients studied.

TABLE V
TOTAL AORTIC CROSS-CLAMP TIME

Minutes	No. of Patients
Nil	7
20-60	5
60-90	2
90-120	6
120-180	14
Over 180	6

The coronary arteries were cannulated in 29 patients. Coronary perfusion was by two separate Watson-Marlow flow inducers (type MHRE). Arterial blood was taken direct from the oxygenator

via a heat-exchanger. By prior pump calibration the flow to each coronary artery was known. It was usual for the myocardium to be cooled to 28° C. In 16 patients, however, myocardial hypothermia of around 15° C. had to be undertaken when coronary arteries could not be satisfactorily perfused and hypoxic myocardial damage seemed to be imminent. Coronary flows and line pressures are shown in Table VI.

TABLE VI

Total Coronary Flow (ml./min.)	No. of Patients	No. of Patients with Line Pressure above 120 mm.Hg
Below 200	10	Nil
200-400	11	4
Over 400	8	4

Coronary perfusion was deemed satisfactory on the basis of the following stringent criteria: (a) that both coronaries were perfused and that the period of total myocardial ischaemia was restricted to less than 20 minutes during a particular cardiopulmonary bypass procedure; (b) that total flow through both coronaries, commensurate with the size of the myocardium, was above 300 ml. per minute; and (c) that to allow this flow through the coronary bed the line pressures were below 120 mm. of mercury.

Based upon the above criteria, three distinct groups of patients emerged, as shown in Table VII.

TABLE VII

Group	Coronary Perfusion	No. of Patients	Valve Replacements
I	Coronary perfusion satisfactory	16	8 aortic 7 mitral 1 aortic + mitral
II	Right coronary perfu- sion unsatisfactory	8	9 aortic 2 aortic + mitral + tricuspid
III	Left coronary perfu- sion unsatisfactory	3	7 aortic
	Coronary perfu- sion unsatisfactory	13	4 mitral 2 aortic + mitral

CLINICO-PATHOLOGICAL ASPECTS Clinical presentation of myocardial damage was studied in all cases. Biochemical, electrocardiographic, and radiological data were analysed. An attempt was made to correlate the preoperative cardiac status with the particular mode of presentation. At necropsy, distribution of coronary atheroma in relation to preoperative assessment of coronary artery disease was studied. Causes of myocardial damage were delineated and an attempt was made to correlate a particular type of myocardial damage with a particular causative factor.

RESULTS

As shown in Table VIII, more than half the deaths occurred on the day of the cardiopulmonary bypass.

TABLE VIII

Time of Death	No. of Patients
1 (a) At the end of perfusion	10
(b) 2 hours later when the chest was being closed	7
(c) Within the first 24 hours	7
2 Within 24-72 hours	7
3 During the first week	6
4 During the second week	3

In the 16 patients who survived longer than 24 hours, evidence of low cardiac output, despite intermittent positive pressure respiration and isoprenaline, was obtained. Seven of these patients showed electrocardiographic evidence of fresh posterior myocardial infarction within 24 to 48 hours of the cardiopulmonary bypass procedure. Four were subsequently found to have a stitch around the circumflex branch of the left coronary artery while the remaining three had sustained dissections of the right coronary artery. Three of the 16 patients also showed changes suggestive of pulmonary oedema on serial daily chest radiographs. In almost all patients the final episode was a ventricular fibrillation which could not be reverted. In 5 patients, however, fulminating infections contributed to death. In addition, 2 patients had gross hepatic necrosis as well.

NECROPSY FINDINGS At necropsy 17 patients had moderate to severe coronary atheroma (Table IX).

TABLE IX

Distribution of Coronary Atheroma	No. of Patients
Triple vessel disease	5
Double vessel disease:	
a. Right coronary and anterior descending branch of left	2
b. Circumflex and anterior descending branches of left coronary artery ..	1
Single vessel disease:	
a. Right coronary artery	4
b. Anterior descending branch of left coronary artery	4
c. Circumflex branch of left coronary artery	1

Table X describes the nature of myocardial damage.

The causes of myocardial damage were defined and were found to be almost equally divided between (1) thrombo-embolic episodes, (2) iatrogenic damage to the coronary circulation, and (3) poor or absent coronary perfusion during cardiopulmonary bypass.

TABLE X

Nature of Myocardial Damage	No. of Patients
Infarction	
Posterior myocardial	13
Postero-lateral myocardial	4
Lateral myocardial	1
Septal myocardial	1
Anterior myocardial	2
Patchy fibrosis with necrosis of papillary muscles	21
Small focal areas of necrosis	11
Haemorrhagic discolouration of posterior myocardium with disintegration of muscle fibres and haemorrhage ..	5
	3

TABLE XI

Group	No. of Patients	No. of Patients with Causes of Myocardial Damage
Group I: satisfactory coronary perfusion	16	7 Thrombo-embolic causes 7 Iatrogenic damage to coronary vessels 3 Patients had severe triple vessel disease
Group II: coronary perfusion of one artery deficient	11	6 Thrombo-embolic causes 5 Iatrogenic damage to coronary vessels 4 Patients had severe right or left coronary artery disease
Group III: unsatisfactory coronary perfusion	13	3 Iatrogenic damage to coronary vessels 2 Patients had severe triple vessel disease

Table XI summarizes the causes of myocardial damage in the three groups of patients, divided on the basis of adequacy of coronary perfusion during cardiopulmonary bypass.

Thus most patients in group III sustained myocardial damage because of unsatisfactory coronary perfusion.

DISCUSSION

Our original method of coronary perfusion on bypass was by taking a coronary supply line off the systemic arterial line via a Y-piece, flow to the heart being controlled by a gate-clip. Calibration of the apparatus before clinical use was by adjusting the gate-clip to allow delivery of 200-300 ml./minute at the calculated systemic flow with a systemic line pressure of 250 mmHg. However, variations in systemic flow or line pressures resulted in alterations in coronary flow of uncertain degree. It is almost certain that some of our patients succumbed from irreversible myocardial damage from 'over-perfusion' of the myocardium. With our present method of coronary perfusion, using two separate coronary artery

pumps, delivering blood from the oxygenator to each coronary artery separately via a heat-exchanger, over-perfusion is eliminated. There is little difference in the pressures in the coronary line and coronary artery (Shaw, Mosher, Ross, Joseph, and Lee, 1962), and mean pressures of 80 to 100 mmHg in the coronary line are relied upon to provide 'physiological' coronary flows. For the time usually involved in aortic valve replacement the temperature is reduced to 28° C.

However, the problem of myocardial perfusion in the presence of coronary atheroma remains. In our series one-third of the patients had coronary atheroma severe enough to jeopardize coronary perfusion. Furthermore, preoperative evidence of coronary artery disease based on clinical symptoms, electrocardiograms, and aortograms was largely negative and correlated poorly with the post-mortem findings. This agrees with the work of Sones and Shirey (1962), and we rely increasingly on selective coronary angiograms not only to delineate more fully the pathological processes in patients undergoing valve replacements, but also to anticipate the problem of myocardial perfusion during cardiopulmonary bypass.

Most of the group III patients and three of the group II patients in whom the cardiopulmonary bypass was unduly prolonged died as a result of absent or poor coronary perfusion, thus contributing to mortality in almost one-third of the patients. The causes of inability to perfuse the coronary arteries in this group ranged from the narrowness of the coronary ostia or their damage at subsequent cannulation to the involvement of the coronary arteries with atheroma. When coronary atheroma was present to any degree of severity, the coronary cannulae flicked out of the coronary arteries with even the most modest of coronary flows; so frequently as to be pathognomonic of atheromatous coronary arteries. In this situation, despite further cooling of the myocardium to about 15° C. and coronary perfusion with minimal flow rates to keep the coronary line pressure from rising, the outcome was fatal if the cardiopulmonary bypass was at all prolonged. Neither was the situation helped in eight patients by perfusing the coronary bed at higher line pressures than 120 mm. with 'stitched in' coronary cannulae to ensure better coronary flows. In fact, with Vasko (1967) and Brown, Braimbridge, Miles, Gerbode, and Aguilar (1969), we believe this might have contributed to 'over-perfusion' of the coronary bed, culminating in a fatal outcome in at least three of the patients. At these line pressures we have

not observed myocardial capillary rupture and haemorrhage (Hirose and Madden, 1964) at necropsy as a direct result of coronary perfusion, although in three unrelated patients in whom coronary perfusion was at physiological pressures disintegration of myocardial muscle fibres with haemorrhage was in fact the only necropsy finding.

The surgical hazards to the circumflex branch of the left coronary artery as it courses from its origin to form the anatomical relation of the mitral ring are now well recognized. In the six patients in our series the occlusive damage to the vessel by a surgical stitch occurred at a distance of 6 to 10 mm. from its origin, particularly when the anterior cusp was grossly calcified and calcium had to be cleared from its bed in the mitral ring. Damage to the coronary ostia, leading to endothelial blockage of the ostia, or dissection of the coronary artery occurred in nine patients in the earlier series but is now infrequent due to the use of softer tipped coronary cannulae (Ramsey, de la Torre, Linhart, and Wheat, 1967). Again, early cannulation of the coronary arteries now undertaken during replacement for calcific aortic valve disease has reduced the incidence of coronary calcific embolism (Glotzer, Shaw, and Scannell, 1962). The risks to the myocardium from air and particulate embolism, particularly when there is gross enlargement of cardiac chambers, still remain and can only be prevented by meticulous care.

More than half of our patients died following cessation of cardiopulmonary bypass or soon after. In the 16 who survived longer than 4 hours, the haemodynamic situation gradually deteriorated despite intermittent positive pressure respiration, isoprenaline and cardiotonic drugs. In all there was only a marginal correlation between the length of survival after cardiopulmonary bypass and the preoperative cardiac status. Neither did the degree of gross myocardial damage influence the length of survival in these patients. Thus, in the cases where coronary perfusion was inadequate, death usually occurred at the end of cardiopulmonary bypass, and gross damage to the myocardium was minimal. Again, the type of myocardial damage did not correlate with any particular causative factor in our series. Our own view is that if particular care is taken to keep the coronary line pressure between 80 and 100 mm. Hg with a moderate hypothermic myocardial perfusion, the heart being kept gently beating, or at worst allowed to fibrillate, myocardial damage

due to coronary perfusion itself in the absence of coronary atheroma does not occur.

It is conceded that there is evidence to suggest that the healthy human myocardium subjected to complete ischaemia for 45 minutes at body temperature will recover sufficiently to support circulation, and that cooling to 30° C. will prolong this period (Kay, Nogueira, Suzuki, Postigo, and Mendelsohn, 1961; Glotzer *et al.*, 1962). In a heart damaged by previous chronic ischaemia, rheumatic fever and coronary atheroma with hypertrophy and dilatation of its chambers, the problem is different. As it is difficult to quantitate this damage to the myocardium against the ischaemic period which might be tolerated in an individual patient, our view is that ischaemia of the heart for anything but the shortest period of time is less safe than good coronary perfusion.

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