

Thrombo-embolic complications of the cloth-covered Starr-Edwards prostheses No. 2300 aortic and No. 6300 mitral

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The thrombo-embolic complications of the cloth-covered Starr-Edwards prostheses No. 2300 aortic and No. 6300 mitral followed for an average of 14 months in 155 patients are reviewed. There was a high incidence of early fatal and disabling thrombo-embolus in patients having mitral valve replacement. Late emboli were more common after aortic valve replacement. Anticoagulant control was unsatisfactory and not without hazards.

Thrombo-embolism is a serious limiting factor in the success of operations for prosthetic valve replacement. Ball variance, the higher incidence of thrombo-embolism with previous Starr-Edwards prostheses (No. 1000 aortic and No. 6000 mitral), and the difficulties with long-term anticoagulant control (Duvoisin, Brandenburg, and McGoon, 1967; Roberts, Levinson, and Morrow, 1970; Matloff *et al.*, 1969; Chin, Harrison, Blakenhorn, and Moacanin, 1971; Starr, Herr, and Wood, 1967) have prompted modifications and the development of the cloth-covered Starr-Edwards prostheses No. 2300 aortic and No. 6300 mitral (Braunwald and Bonchek, 1967; Hodam, Starr, Raible, and Griswold, 1970; Hodam, Starr, Herr, and Pierie, 1969).

Enthusiastic reports of the reduction in thrombo-embolism with this prosthesis (Hodam *et al.*, 1969) encouraged us to use this valve extensively.

MATERIAL AND METHODS

From June 1968 to December 1969, 164 patients underwent valve replacement with the use principally of the cloth-covered Starr-Edwards prostheses No. 2300 and No. 6300 in 172 operations (Table I). Eight patients had two operations performed. A total of 235 valves were replaced. Ten patients had a Starr-Edwards disc valve inserted (No. 6500) and in five patients it was the only valve inserted. Three patients had a University of Cape Town valve (Barnard, Goosen, Holmgren, and Schrire, 1962) inserted in the tricuspid area in association with Starr-Edwards mitral valve replacement.

In the latter part of the study a modification of the cloth-covered prosthesis—the composite seat valve No. 2310 aortic and No. 6310 mitral—was used, when it became available, in 19 patients. In 12 patients it was

the only valve used and a total of 24 of these valves were inserted.

Our perfusion technique used a disposable bubble oxygenator¹ and Sarns roller pumps² with an arterial flow rate of 2.4 l/min/m² with moderate hypothermia to 30–32° C. The ascending aorta was used for arterial cannulation in all but four patients in whom the right external iliac artery was used.

The mitral valve was replaced using a transseptal approach (Brock, 1956). In our early experience interrupted sutures were used to insert the valve, but in the majority of cases a continuous suture technique was used for fixation of the valve.

For aortic valve insertion, with perfusion of both coronary arteries, the aorta was opened through an oblique aortotomy and the valve was sited with interrupted sutures of 00 Ethiflex³. The tricuspid valve was inserted using interrupted sutures in the area of the bundle of His and a continuous suture for the remainder of the valve ring.

Postoperatively anticoagulant warfarin sodium was started on the third or fourth postoperative day, day 1 being the day of operation. Most patients started therapy on the third day. An attempt was made in consultation with the haematologists to stabilize the prothrombin time at greater than 70 sec, that is, less than 20% of normal, and if possible between 10 and 20%. A control level was reached by the seventh postoperative day in most cases.

All patients received anticoagulants. The records of each patient were reviewed and all patients were seen postoperatively at 6-week, 3-month, and 6-month intervals. The follow-up time ranged from 5 to 26 months, with a mean of 14 months.

In approximately half of the cases anticoagulant control was carried out in the hospital where the operation was performed. The detailed anticoagulant

¹Rygg-Kyvsgaard disposable bubble oxygenator (Polystan, Copenhagen)

²Sarns Inc., 6200 Jackson Road, Ann Arbor, Mich, U.S.A.

³Ethicon Ltd., Scotland

TABLE I
RESULTS OF CLOTH-COVERED STARR-EDWARDS PROSTHESIS

Valve Replacement	No. of Patients	Operative Mortality	Late Death	Total %
Mitral	73	9 12%	6	20
Aortic	39	5 13%	4	23
Mitral + aortic	29	11)	3)	48
Mitral + tricuspid	12	4)	1)	
Mitral + aortic + tricuspid	11	3)	3)	
Total	164	32 20%	17	30

Follow-up 2 to 25 months; mean = 14 months

record was reviewed in those patients seen at this hospital. Control was considered inadequate if more than one out of 10 estimations were outside the limits already mentioned or if anticoagulants had been discontinued for any reason. The status of anticoagulants was available at the time of thrombo-embolism in most patients.

The diagnosis of embolus was made clinically and confirmed where appropriate by necropsy findings. Embolism occurring as the result of a perfusion accident, that is, air or calcific emboli, or as a result of bacterial endocarditis was excluded from the study. All embolic episodes were arbitrarily divided into definite and probable, depending on the certainty of the episode, but all were included in the calculation of the incidence of thrombo-embolism.

The patient incidence was calculated from the first episode if more than one occurred. All episodes were used in calculating the sites of emboli. In the calculations four patients who died within 24 hours of operation and the five patients who had single Starr-Edwards disc valves inserted in the mitral area were excluded. In those patients who died late postoperatively, the time of death from operation was used in calculating the mean follow-up time. Early thrombo-embolism was that occurring in hospital or during the first postoperative month. The incidence of late thrombo-embolism was calculated from the number of patients alive one month postoperatively.

RESULTS

There were 27 embolic episodes in 23 patients, an incidence of 15% (Table II). Of these, 12 occurred in 11 patients in the early postoperative period (up to the thirtieth postoperative day), an incidence of 7%. In the follow-up period there were 15 episodes in 12 patients, an incidence of 9% or 1 per 115 patient-months of follow-up.

EARLY EMBOLI Of the early emboli, 10 were definite and two probable (Table III). Six patients had mitral valve replacements alone, three mitral and aortic valve replacements, one mitral and tri-

TABLE II
INCIDENCE OF THROMBO-EMBOLISM WITH CLOTH-COVERED STARR-EDWARDS PROSTHESIS IN 155 PATIENTS

Thrombo-embolism	No. of Patients	No. of Emboli	No. of Patients with Emboli	%
Early	155 ¹	12	11	7
Late	128 ²	15	12	9
Total	155 ¹	27	23	15

¹ Calculated by excluding the four patients who died within 48 hours of operation (one aortic valve replacement, one triple valve replacement and two aortic and mitral valve replacements). Excluded also are the five patients who had a single Starr-Edwards disc valve inserted in the mitral area.

² Calculated from the number of patients who were alive one month postoperatively.

cuspid valve replacement, and one aortic valve replacement. The sites of the emboli were cerebral in nine, renal in two, and axillary artery in one. These episodes occurred as early as the fourth postoperative day and all occurred within two weeks with a mean time of seven days. Seven were fatal and represent 22% of an early operative mortality of 32 patients. Two of the non-fatal episodes caused severe residual disability. One patient required an above-elbow amputation of the left arm after two attempts at brachial embolectomy were unsuccessful. The other patient has a residual hemiplegia with dysphasia.

Anticoagulant control was unsatisfactory in nine of the 11 patients. At the time of the four non-fatal episodes, anticoagulant control was unsatisfactory in two. In one of these a therapeutic level had not been reached, and in the other

TABLE III
EARLY THROMBO-EMBOLISM IN 155 PATIENTS

No. of patients	11	6 Mitral 3 Mitral + aortic 1 Aortic 1 Mitral + tricuspid	7%
No. of emboli	12	9 Cerebral 2 Renal 1 Axillary artery Probable—2	
	Definite—10		
Onset	4-14 (mean 7) days		
Outcome	Fatal—7 Non-fatal—4		
Anti-coagulant control:	Satisfactory 2; Unsatisfactory 9		

TABLE IV
EARLY FATAL THROMBO-EMBOLISM

Patient	Valve Replacement	Site of Thrombus at Necropsy	Presenting Symptom	Time in Days		
				Onset of Symptoms	Embolism	Anticoagulant Control Obtained
1	Mitral	Atrium (L)	Cerebral	4	4	Uncontrolled
2	Aortic	Ventricle (L)	Cerebral	12	12	Uncontrolled
3	Mitral + aortic	No necropsy	Cerebral	6	6	13
4	Mitral + tricuspid	Mitral and tricuspid valves	Cerebral	12	12	17
5	Mitral + aortic	Mitral and aortic valves	Pulmonary	2-3	4	6
6	Mitral + aortic	Mitral and aortic valves	Pulmonary	2-3	5	6
7	Mitral	Mitral valve	Pulmonary	2-3	7	5

it had been reached earlier but was uncontrolled at the time of embolism.

Necropsy was performed in six of the seven cases with early fatal episodes (Table IV). In none was muscular restriction of ball movement implicated as a cause of thrombus formation (Spencer *et al.*, 1970). There were four double valve replacements and three single valve replacements. In four patients (Nos 4, 5, 6, and 7) thrombus was present on the valve. In patients 5 and 6, who had mitral and aortic valve replacements, all four valves were almost completely occluded by thrombus. In patient 7 with a single mitral valve replacement, the valve was similarly affected. In patients 5, 6, and 7, the presenting symptoms were those of increasing respiratory distress with bronchospasm and pulmonary oedema and were associated with bizarre dysrhythmias. These symptoms occurred two to three days postoperatively and preceded the onset of embolism. The embolic episodes in two cases were minor.

Of the 12 patients who had composite seat valves inserted, one (patient 5), who had two such valves inserted, had an early embolus. This is an incidence of 8%, which is similar to that of the non-composite seat valves but the figures here are small and of doubtful statistical significance.

In patients 1, 2, 3, and 4 (Table IV) the presenting symptoms were cerebral, the times of onset and of embolism being coincidental. Patient 1 had fresh thrombus in both atrial appendages and on the suture line in the atrial septum. In patient 2, who had an aortic valve replacement, there was thrombus at the site of myocardial infarction resulting from injury to the right coronary artery during coronary perfusion. Patient 4, who had mitral and tricuspid valve replacements, showed a minor amount of thrombus on the mitral valve, but the tricuspid valve was almost completely occluded. This patient had a minor embolus on the twelfth postoperative day but was admitted to hospital four months later in extremis with cardiogenic shock and evidence of tricuspid obstruction. The sounds of the tricuspid

disc valve had disappeared and on screening the poppet was immobile. Emergency operation was carried out and the tricuspid valve was replaced by a Starr-Edwards ball valve prosthesis. This patient died 24 hours later. At necropsy there was histological evidence that clotting on both valves had occurred early, as mature fibrous tissue was present.

In these seven fatalities, anticoagulant control level had not been reached before the onset of symptoms (last column, Table IV). In patients 2 and 4 a satisfactory level had not been achieved. In the other five patients a control level could not have been achieved using our present technique at the time of the onset of symptoms or embolism.

LATE EMBOLI Fourteen of the 15 late thrombo-embolic episodes which occurred in 12 patients were of a minor nature; only nine were definite (Table V). These produced transient symptoms lasting less than 24 hours and left no residual defects.

TABLE V
LATE THROMBO-EMBOLISM IN 128 PATIENTS

No. of patients	12	5 Mitral 6 Aortic 1 Mitral + aortic	9%
No. of emboli	15	11 Cerebral 1 Splenic 1 Renal 1 Coronary artery 1 Femoral artery	1/115 patient-months
	Definite—9		Probable—6
Onset:	5 weeks to 1 year (mean 6 months)		
Outcome:	Fatal—4 Non-fatal—8		
Anti-coagulant control:	Satisfactory 4; Unsatisfactory 7		

There were five mitral valve replacements, six aortic valve replacements, and one mitral and aortic valve replacement in this group. Of these 12 patients, one late embolism occurred in a patient with a composite seat aortic valve (No. 2310) but this patient also had an older type mitral valve inserted (No. 6300). The sites of the emboli were cerebral in 11 and splenic, renal, coronary artery, and femoral artery in one case

TABLE VI
INFLUENCE OF SITE OF VALVE REPLACEMENT ON THROMBO-EMBOLISM IN 155 PATIENTS

Valve Replacement	No. of Patients ¹	Early Emboli (no.) (%)		No. of Patients ²	Late Emboli (no.) (%)		Total (no.) (%)	
Single mitral	68	6	9	60	5	8	11	16
Single aortic	38	1	3	34	6	18	7	18
Total mitral	117	10	9	94	6	6	16	14
Total aortic	75	4	5	60	7	12	11	15

¹ Calculated by excluding the four patients who died within 48 hours of operation (one aortic valve replacement, one triple valve replacement and two aortic and mitral valve replacements). Excluded also are the five patients who had a single Starr-Edwards disc valve inserted in the mitral area.

² Calculated from number of patients who were alive one month postoperatively.

each. The mean time of onset was six months from operation, with a range from five weeks to one year.

At the time of embolism, anticoagulant control was satisfactory in only four patients and unsatisfactory in seven. In the other patient it was unknown.

There were four fatalities in the group, but only one was the result of embolism. Anticoagulants were temporarily discontinued two weeks before the fatal coronary artery embolism, which was confirmed at necropsy, as was a slightly older renal embolus. There was a minor amount of thrombus on the aortic prosthesis at necropsy. A second patient, who had two minor cerebral embolic episodes, was negligent about taking his anticoagulants, having discontinued them on several occasions. He died suddenly one year postoperatively. Permission for necropsy was not obtained, but his death was presumed to be embolic in origin. An embolism occurred in a third patient whose anticoagulant control was unsatisfactory. His dosage was increased and he died two weeks later from a cerebral haemorrhage as a direct result of anticoagulant overdosage. The fourth patient died of bacterial endocarditis one year after a probable minor embolus. Two further patients died as a result of overdosage with anticoagulants, both suffering a cerebral haemorrhage.

The influence of the site of valve replacement on thrombo-embolism was investigated (Table VI). Patients who had a mitral valve only replaced had a 9% incidence of early emboli, whereas patients who had an aortic valve only replaced had a 3% incidence. When all patients who had a mitral valve replaced alone or in combination with other valves are compared with those who had an aortic valve similarly replaced, the relationship is similar at 9 and 5% respectively.

With late emboli the relationship is reversed. Eight per cent of patients with single mitral valve replacements and 18% of patients with single aortic valve replacements had emboli. When all

mitral and aortic valve replacements are compared, the relationship remained 6% and 12% respectively.

The incidence of early embolus was similar in single and multiple valve replacements, 7% and 8% respectively, but late emboli were less common with multiple valve replacements (3% compared to single valve replacement (12%).

Factors that may have influenced the incidence of thrombo-embolism were investigated. These included a past history of thrombo-embolism, a previous mitral valvotomy, the size of the valves inserted, the presence of thrombus in the left atrium at operation, valve calcification, atrial fibrillation, marked enlargement of the left atrium and postoperative haemorrhage.

In the patients who had a single aortic valve replaced and a postoperative embolus, only valve size, valve calcification, and atrial fibrillation were relevant as the other factors were absent. One patient had an early embolus with the onset of atrial fibrillation. Ten other patients having aortic valve replacements developed transient atrial fibrillation early after operation without embolization. Two of the six patients with late emboli who had aortic valve replacements were in atrial fibrillation, but in the non-embolic group there were no patients with atrial fibrillation. Neither valve size nor calcification had any influence on the incidence of thrombo-embolism.

In patients who had their mitral valves replaced atrial fibrillation did not appear to influence the incidence of thrombo-embolism. Eighty-three per cent of patients with early emboli had atrial fibrillation, 82% of all patients with emboli were in atrial fibrillation, and 84% of patients who did not have emboli were so afflicted (Table VII).

A previous history of thrombo-embolism was just as common in patients with or without emboli. In the three groups mentioned, the percentages were 17%, 18%, and 16% respectively. Although nine patients were found to have left atrial thrombus at operation, no atrial clot was

TABLE VII
FACTORS INFLUENCING THROMBO-EMBOLISM IN 68 SINGLE MITRAL VALVE REPLACEMENTS

Group	No.	Atrial Fibrillation (no.) (%)	Past History of Embolism (no.) (%)	Previous Mitral Valvotomy (no.) (%)	Valve Calcification (no.) (%)	Left Atrial Enlargement (no.) (%)	Postoperative Haemorrhage (no.) (%)
Early emboli	6	5 83	1 17	0 0	1 17	4 66	3 50
All emboli	11	9 82	2 18	3 27	2 18	8 73	3 27
No emboli	57	48 84	9 16	22 40	21 37	23 40	10 18

TABLE VIII

FACTORS INFLUENCING THROMBO-EMBOLISM IN 117 PATIENTS WITH SINGLE OR COMBINED MITRAL VALVE REPLACEMENTS

Group	No.	Atrial Fibrillation (no.) (%)	Past History of Embolism (no.) (%)	Previous Mitral Valvotomy (no.) (%)	Valve Calcification (no.) (%)	Left Atrial Enlargement (no.) (%)	Postoperative Haemorrhage (no.) (%)
Early emboli	10	9 90	3 30	2 20	3 30	5 50	3 30
All emboli	16	14 87	5 31	6 38	5 31	10 63	3 19
No emboli	101	83 82	18 19	45 45	45 45	36 37	21 21

found in any patient who subsequently had an embolus. Calcification of the mitral valve or a previous valvotomy did not predispose to embolism; these conditions were present in 40% of patients without embolus but in only 27% of those with emboli.

Marked left atrial enlargement occurred in 66% of patients with early emboli, in 73% of the all embolic group, but in only 40% of the non-embolic group.

Postoperative haemorrhage requiring re-operation was more common in the group with early emboli than in the control group—50% as compared to 18%.

In comparing the non-embolic group of patients who had a mitral valve replacement either alone or in combination with other valve replacements with the embolic group the relationship still holds (Table VIII), except that patients with a past history of embolus are more common in the embolic group (30% and 31% respectively) than in the non-embolic group (19%). This discrepancy may be due to the small number of patients in the embolic group.

The influence of valve size in the mitral area on thrombo-embolism was investigated, as it was thought that the smaller valves might cause stasis

TABLE IX

INFLUENCE OF VALVE SIZE ON THE INCIDENCE OF THROMBO-EMBOLISM IN 68 SINGLE MITRAL VALVE REPLACEMENTS

Thrombo-embolism	No.	Size 2 or 3 (no.) (%)	Size 4 (no.) (%)
Early	6	4 67	2 33
Early and late	11	5 46	6 55
No embolus	57	25 44	32 56

and hence thrombus formation (Table IX). But valve size did not appear to be important except with early emboli when patients who had had a smaller valve inserted appeared to be more prone to embolus formation. This was not statistically significant.

Anticoagulant control in the patients without emboli who were followed at our own clinic (approximately half of all the patients) was unsatisfactory in all patients followed as none fell within the strict limits described. At the time of embolism only six patients out of 22 were in satisfactory anticoagulant control.

TABLE X

INFLUENCE OF VALVE SIZE ON THE INCIDENCE OF THROMBO-EMBOLISM IN 117 MITRAL VALVE REPLACEMENTS

Thrombo-embolism	No.	Size 2 or 3 (no.) (%)	Size 4 (no.) (%)
Early	10	4 40	6 60
Early and late	16	6 38	10 62
No embolus	101	54 53	47 47

DISCUSSION

The cloth-covered Starr-Edwards prostheses No. 2300 aortic and No. 6300 mitral, specifically designed to reduce the incidence of thrombo-embolism and to eliminate the need for anticoagulants (Braunwald and Bonchek, 1967; Hodam *et al.*, 1970), do not fulfil their promise with or without satisfactory anticoagulant therapy. The high incidence of early and disabling thrombo-embolism is similar to, and in some cases greater than, that reported for the earlier ball valve prosthesis (Hodam *et al.*, 1969; Kahn *et al.*,

1966; Duvoisin *et al.*, 1967). The episodes are more severe and produce greater disability. The fatalities from embolism represent 22% of the operative mortality. Only two patients out of 11 with early emboli had transient symptoms with minimal disability. The incidence of early thrombo-embolism in our series (7%) is similar to that of Hodam at 6% (Hodam *et al.*, 1969), using the same prosthesis. The incidence of early embolism after aortic valve replacement is low (3%) and similar to that of the earlier Starr-Edwards prostheses No. 1000 and No. 1200 (Hodam *et al.*, 1969). The mitral prosthesis appears to be at greater risk as a site of thrombus formation early postoperatively. Our incidence of 9% is similar to that with the earlier No. 6000 prosthesis but less than the 11% reported by Hodam *et al.* (1969) with the cloth-covered prosthesis No. 6300. The incidence is overestimated because there is a significant risk, although small, of thrombo-embolism after both open and closed mitral valvotomy and repair operations on the mitral valve when a prosthesis is not used (Kahn *et al.*, 1966; Olinger, Rio, and Maloney, 1971; Ellis and Harken, 1961; Greenwood, Aldridge, and McKelvey, 1963).

The reduction in flow ratios with the newer valve introduces another factor in promoting stasis in the left atrium and thrombus formation (Kloster, Herr, Starr, and Griswold, 1969; Reis *et al.*, 1970; Hodam *et al.*, 1969). In our series early emboli were more common in patients with size 2 and 3 Starr-Edwards prostheses than in those with a size 4. This early incidence has been improved with the introduction of the composite seat valve, which has a comparably greater orifice (Hodam *et al.*, 1970; Reis *et al.*, 1970; Kloster *et al.*, 1970). One patient of the 19 who had a composite seat valve inserted late in the study had a fatal thrombosis of both aortic and mitral valves. A further 16 patients not included in the present study have shown no embolic episodes following composite seat valve insertion, giving a total embolic incidence of 3% for the 35 patients.

The incidence of early embolism was similar in patients who had multiple valve replacements as in those with single mitral valve replacements, confirming that patients with mitral valve replacement are at greater risk in the early postoperative period.

Our method of using warfarin to prevent these early episodes is unsatisfactory as embolus or thrombus on the valves had occurred before anti-coagulant control could have been achieved and

emboli occurred in two patients who appeared to be adequately controlled at the time. A trial using heparin early postoperatively did not appear to make any difference to the incidence of thrombo-embolism (Starr *et al.*, 1967), but the administration of the drug was delayed until the fourth postoperative day, and even earlier administration may be necessary, although the risk from haemorrhage may be prohibitive. It is thought that valve thrombus occurs early after operation and the preventative measures, as reported by Matloff *et al.* (1969), should begin as soon as possible postoperatively, that is when bleeding has ceased, to be effective. Platelet thrombi on the rough cloth covering of the prosthesis, before pseudo-endothelium covers the cloth, may be the most significant single cause of thrombo-embolism with the prostheses studied. The administration of dipyridamole in association with warfarin to prevent platelet aggregation may be worth while (Sullivan, Harken, and Gorlin, 1968). Unless the problem of early embolism is overcome, this prosthesis appears to offer no advantage over the older ball valve prosthesis and has the disadvantage of a larger gradient across the valve (Kloster *et al.*, 1969; Reis *et al.*, 1970; Hodam *et al.*, 1969).

Our findings confirm that the incidence of embolism is not influenced by the presence of left atrial thrombus at operation nor by a previous mitral valvotomy (Matloff *et al.*, 1969; Duvoisin *et al.*, 1967). Neither did atrial fibrillation appear to influence the incidence, which is in contrast to other reported series (Yeh, Anabtawi, Cornett, and Ellison, 1967). The number of patients with atrial fibrillation in our series was very high, 83% compared with 52% in the series of Matloff *et al.* (1969). Transient atrial fibrillation was present in the one patient with embolism who had had an aortic valve replacement. Postoperative haemorrhage, severe enough to require re-operation, occurred in 50% of patients with early emboli, all of whom had had mitral valve replacements. The overall incidence of embolism, 15% was greater than that reported by Reis *et al.* (1970) at 12%.

The reduction in serious late thrombo-embolism when compared to other series with the older prosthesis is encouraging. Our 9% incidence with the prostheses studied is more than the 6% reported by Hodam *et al.* (1969). For mitral valve replacement alone the incidence is 8% in our series but only 3% in Hodam's. There was a higher incidence of late embolism after aortic valve replacement (18%) in our series compared to 6% in Hodam's larger series. The incidence of

late emboli is more common after aortic valve replacement in both series. Two of the six patients who had aortic valve replacement (Table V) were in atrial fibrillation at the time of embolism whereas none of the aortic patients without emboli were in atrial fibrillation. Fourteen of the 15 late emboli were minor with rapid recovery of the patient and no residual disability.

Stable anticoagulant control was difficult to achieve throughout. Many factors are involved, not least patient unreliability. This emphasizes the inherent difficulty of outpatient anticoagulant control and stresses the need for prostheses which will not require postoperative anticoagulants.

Anticoagulants are not without hazard. Death directly attributable to their use occurred in three of our patients; all died from massive cerebral haemorrhage.

When the cloth-covered prostheses No. 2300 and No. 6300 are used, valve occlusion or malfunction due to thrombus formation should be considered in the early postoperative period in patients in whom there is the slightest suggestion of minor embolism, persistent pulmonary oedema, or persistent dysrhythmias, and should be investigated promptly. Failure to do so may account for the high early mortality from thromboembolism, although the results of re-operation have been disappointing (Vander Veer, Rhyneer, Hodam, and Kloster, 1971; Gunstensen, 1971; Horsley, Rappoport, Vigoda, and Vogel, 1970). The solution lies in the prevention of thromboembolism by a change in valve design and a more effective medical regime to abolish platelet thrombi in the immediate postoperative period. Our early experience of the use of the composite seat valve is encouraging.

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