Current Practice

Tricuspid Valve Replacement in Rheumatic Heart Disease

M. V. BRAIMBRIDGE,* F.R.C.S. ; C. L. SARIN,† F.R.L.S. ; E. YALAV,‡ M.D. ; A. J. CLEMENT,§ M.B., B.S., F.F.A. R.C.S
D. MENDEL,|| M.R.C.P.

British Medical Journal, 1970, 3, 629-631

Summary: The diagnosis of tricuspid valve disease is often difficult; the best treatment is not yet established. Twenty patients had tricuspid valve replacements at St. Thomas's Hospital as part of multiple valve replacement procedures. The hospital mortality was 25%, most deaths being due to a low cardiac output causing hepato-renal failure. Preoperative cardiac cachexia had a fatal outcome in all cases. Except in two instances, surviving patients returned to a satisfactory level of activity.

Involvement of the tricuspid valve is found at necropsy in 25% of patients who suffer from rheumatic disease of the heart and may be present at the time of surgery in 15% of those undergoing mitral valvotomy.^{1,2} The valve is never affected alone by the rheumatic process, and concomitant involvement of the mitral and aortic valves usually overshadows the symptoms and signs of the tricuspid valve disease. The indications for surgery in aortic and mitral valve disease are now fairly uniform, but the indications for tricuspid valve surgery and the best procedure to be used to correct tricuspid incompetence are still disputed. The experience of tricuspid valve incompetence in 20 patients at St. Thomas's Hospital from 1966 to 1969 is reported here and an attempt is made to evaluate the short- and long-term problems. Nine patients had mitral and tricuspid valve replacements, two aortic and tricuspid, and nine triple valve replacements (Table I).

TABLE I.—Tricuspid Valve Replacement Concomitant Procedures in 20 Patients

Group 1.	Aortic valve replacement	1
Group 2.	Mitral valve replacement	9
Group 3.	Aortic and mitral valve replacement	9
Group 4.	Aortic valve replacement and mitral valve repair	1

Five patients in group 2 had minimal aortic incompetence, and one patient in group 1 had mitral incompetence which did not need valve replacement.

Clinical Features

The series consisted of 16 women and 4 men, the age range being from 14 to 58 years. One patient was 14, one 23 years old, 4 were between 30 and 39, 10 between 40 and 49, and the remaining 4 were over 50.

Fourteen patients gave a history of rheumatic fever, and six of these had had more than one attack. Eight patients had had previous cardiac operations, seven having undergone mitral valvotomy or valve exploration and one a thoracotomy for tamponade following transseptal cardiac catheterization.

All but one patient were severely restricted by dyspnoea (Table II). The majority (14) were unable to walk slowly on the flat and five were able to walk slowly but not to hurry. One patient was only slightly dyspnoeic and was operated on for resistant jaundice, but 17 patients were orthopnoeic and nine had paroxysmal nocturnal dyspnoea also.

On clinical examination six patients were abnormally thin and five of these were emaciated (Table III). A malar flush was present in 16. All patients were in atrial fibrillation. The jugular venous pressure was raised from 4 to 9 cm. above the sternal angle in 14 patients at the time of surgery, in spite of intensive medical treatment which had reduced the pressure to normal in the remainder. The liver was enlarged in seven.

TABLE II.—Symptoms in Tricuspid Valve Disease as Part of Multiple Valve Disease

Symptom	No. of Patients
Effort intolerance	19 (5 Grade 2b, 13 Grade 3, and 1 Grade 4*)
Orthopnoea	17 (using 4 to 10 pillows)
Bronchitis	12
Fatigue	11
Paroxysmal nocturnal dyspnoea	9
Peripheral oedema	9 (one with ascites)
Angina on effort	6
Haemoptysis	3
Syncope	2

*Paul Wood's classification.

TABLE III.—Findings in Tricuspid Valve Disease as Part of Multiple Valve Disease

Clinical Examination	No. of Patients
Subnormal habitus	6 (5 were frankly emaciated)
Malar flush	16
Cyanosis	1 (peripheral)
Clubbing	None
Atrial fibrillation	20
Jugular venous pressure raised	14
Liver enlarged	7
Left ventricular hypertrophy	20
Right ventricular hypertrophy	16
Pansystolic murmur left sternal edge	12
Murmur accentuated on inspiration	6
No murmurs at left sternal edge	8
Left axis deviation	10
Chest radiograph: cardiac enlargement	20 (cardiothoracic ratio 58-84%)

Left ventricular hypertrophy was present in all patients due to concomitant aortic and mitral valve disease, and 16 had a right ventricular impulse in addition. On auscultation at the left sternal edge a pansystolic murmur could be heard in 12 of the 20 patients, and in three this was associated with a mid-diastolic murmur. In only six was there definite accentuation of the pansystolic murmur on inspiration.

Electrocardiography showed left axis deviation in five patients due to the concomitant left heart lesions. In 10 there was T-wave inversion over the left-sided chest leads and in four over the right chest leads.

On chest radiography all patients had cardiac enlargement; the cardiothoracic ratio was increased (mean 70%, range 58-84%). Enlargement of the right atrium was seen in all patients. In 16 it was markedly enlarged and in two it was huge. Mitral valve disease had caused left atrial enlargement in all patients, and in two this was of giant size. Moderate enlargement of the pulmonary artery was seen in all patients.

On cardiac catheterization the mean right atrial pressure varied from 2 to 18 mm. Hg, with a mean of 7 mm. Right atrial "v" waves ranged from 2 to 25 mm. The pulmonary artery oxygen saturation was reduced in all cases and ranged from 30 to 62%. The systolic pulmonary artery pressure was raised in all patients, but in only one was the pulmonary vascular resistance markedly raised. A range of systolic pressures

* Consultant Surgeon.

† Lecturer in Surgery.

‡ Lecturer in Surgery.

§ Consultant Anaesthetist.

|| Consultant Physician.

Department of Cardiothoracic Surgery, St. Thomas's Hospital, London S.E.1.

from 35 to 70 mm. Hg was recorded, the pressure being above 50 mm. Hg in nine patients. An end-diastolic gradient of 3 to 5 mm. of Hg across the tricuspid valve was present in four patients.

External phonocardiography confirmed the auscultatory findings. Intracavitary phonocardiograms were done in 12 patients, but in only one patient was a pansystolic murmur recorded which was not visible on the external phonocardiographic record. A mid-diastolic murmur was recorded in four patients, in two of whom it was also recorded on the external phonocardiograms.

Findings at Operation

The right atrium was much enlarged in all patients. The tricuspid valve was organically diseased in all, with fibrosed shrunken cusps and usually shortened chordae tendineae. All 20 patients had tricuspid incompetence at the time of palpation: in 7 (35%) it was moderate and in 13 (65%) gross. Eight patients had significant tricuspid stenosis also, the orifice of the stenosed valve being 2 to 2.5 cm.

Surgical Management

The Osborn-Bramson-Gerbode disc oxygenator with moderate hypothermia of 30°C. was used for all patients. The chest was opened through a median sternotomy unless the patient had had previous surgery and had a competent aortic valve, in which case the incision was through the fourth right intercostal space. Venous drainage was from separate cannulae in the superior and inferior venae cavae, and arterial return was into the root of the aorta using a special cannula.³

The tricuspid valve was palpated in each patient before the insertion of the venous cannulae. Only in patients in whom there was pronounced organic deformity with a degree of stenosis and with thickening of the cusps was the tricuspid valve replaced.

The tricuspid valve was the last valve to be replaced in each case. The right atrium was opened with a separate incision parallel to the left atrial incision. Starr-Edwards ball-valve prostheses were used for all the multiple valve replacements, the size of the valve being chosen to fit the size of the ventricle rather than the size of the tricuspid valve ring. Of the 20 Starr mitral valves inserted into the tricuspid area 12 were size 2M and 8 size 3M, these smaller sizes being used to prevent unnecessary encroachment on the right ventricular cavity. The valve was sutured in place with vertical mattress Tevdek sutures inserted in the ring except over the bundle of His, where they were placed in the tricuspid valve leaflet itself.

Postoperative Management

Patients with multiple valve disease involving the tricuspid valve have usually been on heart failure regimens for several years and have suffered from prolonged periods with a low cardiac output. In the postoperative period they have almost always needed inotropic agents and assisted ventilation for several days, and the latter was used for between 3 and 14 days in this series. A plastic endotracheal tube was used if artificial ventilation lasted less than a week and a tracheostomy was performed if longer periods of assisted ventilation were required.

The rigid tricuspid prosthesis lies against the conducting tissue of the bundle of His, and pacemaker wires were therefore inserted into the right ventricle in each patient.

Almost all patients had bradycardia and low cardiac outputs, the former in spite of stopping digoxin three days before surgery. Consequently all patients received a continuous intravenous infusion of isoprenaline. Digitalis was given after

surgery when the potassium level was satisfactory and ventricular ectopic beats were few.

Ventricular arrhythmias could usually be abolished by giving potassium chloride continuously in an intravenous drip (100-200 mEq daily), with single increments of 5 mEq when necessary. Lignocaine in the form of a continuous intravenous drip of 1-2 mg. per minute was used in patients with resistant ventricular arrhythmias.

If the cardiac output remained low in spite of the use of isoprenaline, digoxin, and electrical pacing, 50% glucose with insulin was used, potassium being added if its serum level was low.

Postoperative Complications

Five hospital deaths occurred in the patients undergoing tricuspid valve replacement. One of the patients who had had an aortic valve replacement and mitral valve repair in addition to tricuspid valve replacement died on the table owing to haemorrhage from the aorta, which had been torn while attempts were made to replace the aortic valve through the right chest. The other four died during the postoperative period. Two of these patients had had triple valve replacements and two had mitral and tricuspid valve replacements. The patients who died during the postoperative period followed a distinct course. Each had a very low cardiac output immediately after surgery, with a low blood pressure and a cold cyanosed periphery. The cardiac output did not increase after the administration of inotropic agents. Oliguria developed and was followed by jaundice, death ensuing in two to three weeks. In the last phase the cardiac output usually returned to normal but the liver and kidneys did not recover from the damage which had occurred during the low cardiac output phase. The hospital mortality rate was therefore 25%.

Eight patients became jaundiced in the postoperative period. Five developed jaundice within 72 hours of surgery and serum bilirubin levels of up to 50 mg./100 ml. were recorded in spite of the other liver function tests being within normal limits. This type of jaundice was benign and regressed after 10 days. Jaundice developing about five to seven days after surgery followed a prolonged postoperative period of low cardiac output, it was accompanied by progressive oliguria and renal failure, and was invariably fatal. This syndrome of hepatorenal failure was seen in three patients.

Temporary heart block was common. One patient developed heart block on the operating-table, recovery occurring within two hours. Four other patients developed heart block at varying intervals up to 72 hours after surgery, and required pacing. In no case was heart block permanent. The pacemaker was also used in six other patients for control of bradycardia or ventricular ectopic beats.

Late Results

There were three late deaths—one in the triple valve group and two in the mitral and tricuspid valve replacement group. Initially these patients were doing well. The cause of death of the triple valve patient four and a half months after surgery was massive cerebral embolism from a clot on the left atrial wall. The Starr valves were free of thrombus. Heart failure and ventricular arrhythmia was the cause of death in the youngest patient of this series after five and a half months, but necropsy was not permitted. Serum hepatitis proved fatal five and a half months later in the last patient.

Six weeks after surgery one patient had a cerebral embolus which caused a right-sided hemiplegia with temporary loss of speech. He has recovered satisfactorily from it and is leading a normal life, though with slight residual slurring of his speech. Contrary to expectations that a prosthetic valve inserted into the low-pressure right heart chamber might be prone to thrombosis, no clinical case of pulmonary embolism has been seen.

There has been no case of tricuspid or mitral valve dehiscence in this series. One patient, however, has recently developed an aortic regurgitant murmur.

All patients have shown an improvement in their clinical status. Two patients still have moderate (grade 2) dyspnoea—one in the triple valve group and one in the mitral and tricuspid valve group—and both have raised jugular venous pressures, with some peripheral oedema. The remainder of the patients have been followed for periods of six months to three years. All these patients now have grade 0-1 effort intolerance (Wood's grading), and orthopnoea has disappeared. They are back at work or coping with their household responsibilities.

Chest radiography has shown an average reduction in cardiothoracic ratio of 8% (from 70 to 62%). This figure includes three patients in whom there was no change.

Discussion

Tricuspid regurgitation may be functional, due to enlargement of the right ventricle and dilatation of a normal valve ring, or organic, due to involvement of the tricuspid valve cusps themselves by the rheumatic process. The diagnosis of tricuspid valve disease is not always easy.⁴

Tricuspid stenosis can be diagnosed clinically by a slow "y" descent and a tricuspid diastolic murmur and opening snap. At cardiac catheterization an end-diastolic gradient across the valve is measured with a double-lumen catheter and the degree of stenosis can be confirmed at the time of surgery. In tricuspid incompetence it is often difficult to distinguish the pansystolic murmur from that of mitral incompetence. The site of the murmur may be more lateral than the fourth left interspace owing to enlargement of the right atrium, and the inspiratory accentuation that is a diagnostic feature was present in less than a third of the patients in this series. Phonocardiography, both external and intracavitary, did not add much to the clinical auscultatory findings. The mid-diastolic murmur of tricuspid stenosis was heard in only four of the patients who had proved tricuspid stenosis of marked degree.

In the presence of atrial fibrillation "v" waves in the right atrial tracings are of little significance and can be shown to be present when the valve is entirely competent on palpation at the time of surgery. The issue is further confused by the fact that the valve can be made to vary in its degree of incompetence by altering the right atrial pressure, either acutely at surgery or more slowly by medical treatment. The critical decision, which is the estimation of how the tricuspid valve will behave when the other valve disease has been corrected, is therefore difficult to make.

Grondin *et al.* reported a series of 13 patients who had no treatment of their tricuspid valve disease at the time of mitral valve replacement.⁵ Seven patients died and four of the six survivors had poor functional results. Starr advocated replacing even anatomically normal tricuspid valves for the same reason but has since changed his mind.⁶ Braunwald *et al.* support the policy of replacing only organically diseased valves in the belief that the incompetence will eventually, though slowly, regress, regardless of the degree of the incompetent jet found at operation.⁷ Twenty-eight of their patients had functional tricuspid incompetence, due in 25 to pulmonary hypertension. No valve was replaced and all patients subsequently assessed were improved.

Some surgeons advocate valvoplasty to accelerate regression of such functional tricuspid incompetence.^{5,8} Our experience of tricuspid valvoplasty has been as unpromising as that of many other cardiac units. Our policy, therefore, like Braunwald's, has been to replace all tricuspid valves which are

organically damaged and have moderate or severe stenosis or incompetence, but to leave all valves that are anatomically normal, disregarding the degree of incompetence in the latter instance.^{9,10} The reasons for this policy are that organically damaged valves must necessarily be less likely to become completely competent after surgery of the aortic or mitral valves but that normal valves may well do so. In addition, the replacement of anatomically normal valves is technically difficult due to the flimsiness of the cusp tissue over the area of the bundle of His.

All three recognized types of postoperative jaundice were seen in this series. The early benign type occurred in 25% of patients, and the late malignant hepatorenal type in 15%. This was the most common cause of death in the series and was associated with a very low cardiac output in the immediate postoperative period, which damaged the liver and kidneys irreversibly, death occurring after two or three weeks when the cardiac output had returned to normal. Serum hepatitis proved fatal in one patient after discharge from hospital.

The results of tricuspid valve replacement at St. Thomas's Hospital are difficult to assess because the lesion causing most of the symptoms was the aortic or mitral valve disease. The operative mortality of 25% is probably reasonable, considering that it includes our entire series of multiple valve replacements beginning in 1966, when operative and postoperative management was less effective than it is today. Starr⁵ reports a 10% mortality in his early series, but few other surgeons can achieve this level of success.

The main cause of death in the present series was a low cardiac output presenting as hepatic and renal failure in the immediate postoperative period and as cardiac failure after discharge from hospital. This was presumably the result of a damaged myocardium because all their prosthetic valves were competent in this group. It is significant that five of the eight deaths in this series were emaciated before surgery. Cardiac cachexia in multiple valve disease would seem therefore an indication of poor myocardial function.

REFERENCES

- 1 Bailey, C. P., and Bolton, H. E., *New York State Journal of Medicine*, 1956, **56**, 649.
- 2 Kitchin, A., and Turner, R., *British Heart Journal*, 1964, **26**, 354.
- 3 Braimbridge, M. V., in press, 1970.
- 4 Cairns, K. B., Kloster, F. E., Bristow, J. D., Lees, M. H., and Griswold, H. E., *American Heart Journal*, 1968, **75**, 173.
- 5 Grondin, P., Lepage, G., Castonguay, Y., and Meere, C., *Journal of thoracic and cardiovascular Surgery*, 1967, **53**, 7.
- 6 Starr, A., Herr, R., and Wood, J., *Surgery, Gynecology and Obstetrics*, 1966, **122**, 1295.
- 7 Braunwald, N. S., Ross, J., and Morrow, A. G., *Circulation*, 1967, **35**, Suppl. No. 1, p. 63.
- 8 Kay, J. H., Maselli-Campagna, G., and Tsuji, H. K., *Annals of Surgery*, 1965, **162**, 53.
- 9 Braimbridge, M. V., Clement, A. J., Brown, A. H., Sabar, E., and Mendel, D., *British Medical Journal*, 1969, **3**, 683.
- 10 Lillehei, C. W., Gannon, P. G., Levy, M. J., Varco, R. L., and Wang, Y., *Circulation*, 1966, **33**, 34.

B.M.J. Publications

The following are available from the Publishing Manager, B.M.A. House, Tavistock Square, London WC1H 9JR. The prices include postage.

<i>Today's Drugs/1 (New Series)</i> ...	Price 20s.
<i>Road Accidents and the Family Doctor</i> ...	Price 10s.
<i>Diseases of the Digestive System</i> ...	Price 40s.
<i>B.M.J. Annual Cumulative Index,</i> 1967, 68, 69 ...	Price 30s. each
(15s. each to B.M.A. members)	
<i>Practical Psychiatry</i> ...	Price 15s.
<i>Porphyria—a Royal Malady</i> ...	Price 13s. 6d.