Late Thrombotic Obstruction of Starr-Edwards Tricuspid Valve Prosthesis*

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Two patients developed late thrombotic obstruction of a Starr-Edwards tricuspid valve prosthesis, one subsequent to restriction of ball motion by interfering subvalvular muscular structures, and the other during Candida parakrusei endocarditis. Late prosthetic tricuspid valve thrombosis is uncommon, but may occur if a preceding event causes the prosthesis to malfunction.

A significant number of patients undergoing surgery for valvular heart disease suffer from rheumatic involvement or functional insufficiency of the tricuspid valve necessitating its replacement with a prosthesis.1,2 Follow-up studies indicate few significant postoperative complications related to the prosthetic tricuspid valve.3,4 Life-threatening obstruction of the prosthesis by thrombus formation, although reported occasionally for mitral prostheses,5,6 is uncommon for the tricuspid prosthesis.7,8 The present report will describe the clinical findings in two patients who developed late thrombotic obstruction of Starr-Edwards prosthetic tricuspid valves.

**Case Reports**

**Case 1**

A 45-year-old woman was admitted on February 18, 1966, with intractable right and left ventricular decompensation. Physical findings of aortic insufficiency, mitral stenosis and insufficiency, and severe tricuspid insufficiency were confirmed by cardiac catheterization. On April 22, 1966, she underwent replacement of the mitral valve with a No. 1M Starr-Edwards prosthesis, replacement of the aortic valve with a No. 2M Magovern prosthesis, and replacement of the tricuspid valve with a No. 2M Starr-Edwards prosthesis. The patient improved gradually so that one year following surgery she was able to do her housework, and was completely relieved of orthopnea and paroxysmal nocturnal dyspnea. She was maintained on warfarin anticoagulation with prothrombin times 2 to 2.5 times the control value.

She continued to do well until August, 1969, when she experienced increased fatigue, peripheral cyanosis with exertion, and increasing pedal edema uncontrolled by diuretic therapy. Examination demonstrated venous distention to the forehead with the patient sitting upright. The chest was clear. Examination of the heart showed normal aortic prosthetic valve sounds; it was not possible to distinguish separately the sounds of the two prosthetic atrioventricular valves. There was no evidence of significant insufficiency of any of the prosthetic valves. The patient refused to consider cardiac catheterization or further cardiac surgery. Despite vigorous medical therapy, she continued in severe right heart failure with increasing evidence of low cardiac output. On December 6, 1969, she became hypotensive and developed bradycardia which progressed to intractable ventricular asystole.

**Necropsy Findings**

Necropsy was confined to the heart which showed mild dilation of the ventricular chambers, moderate dilation of the left atrium and marked dilation of the right atrium. The mitral and aortic prostheses appeared normal. The atrial surface of the tricuspid prosthesis was endothelialized and without clot, but the cage, which was encircled by the papillary muscles and chordae tendineae of the tricuspid valve (not removed at surgery), was enmeshed in organized thrombotic material which occupied much of the interchordal space. Free motion of the ball was marked by so that only a short excursion into the cage was possible (Fig 1).

**Case 2**

This 54-year-old woman was admitted on January 10, 1965, with severe mitral and tricuspid insufficiency. These findings were confirmed by cardiac catheterization and, on January 26, 1965, the mitral and tricuspid valves were replaced with Starr-Edwards 3M prostheses. The patient had
an uneventful convalescence with almost complete resolution of cardiac symptoms. She was maintained on warfarin anticoagulation with prothrombin times 2 to 2.5 times the control value. One year following surgery, cardiac catheterization demonstrated a right ventricular pressure of 30/1.6 mm Hg with a mean right atrial pressure of 6 mm Hg. The mean diastolic gradient across the tricuspid valve was 3 mm Hg with a cardiac output of 3.5 L/min.

On November 16, 1970, the patient underwent total colectomy for adenocarcinoma. She received no warfarin for two days preceding surgery. One day prior to surgery, she received 8 gm of kanamycin orally for suppression of bowel bacteria, and 25 mg of vitamin K$_1$ intramuscularly. On the day of surgery she received 3 gm of cephaloridine intravenously in three divided doses, and 25 mg of vitamin K$_1$ intravenously. Her prothrombin time, which previously was in the therapeutic range, fell to control values by the time of surgery. She tolerated the surgical procedure well, and on the second postoperative day, anticoagulant therapy was re instituted with 20 mg of warfarin orally followed by 5 mg daily until her prothrombin time returned to the therapeutic range (22.8 sec with a control value of 11.5 sec on the fifth postoperative day). Thereafter, her usual dosage of 3.75 mg of warfarin daily maintained a therapeutically satisfactory prothrombin time.

During the first week following surgery, she was intermittently febrile with rectal temperatures to 102°F. Blood cultures were sterile, but urine cultures demonstrated greater than 100,000 colonies per milliliter of E coli and, following appropriate sensitivity testing, she was treated with an eight day course of intravenous ampicillin, 1 gm four times daily, beginning on the fourth postoperative day. At the conclusion of this therapy, she had become afebrile. Although one subsequent blood culture grew Candida parakrusei, seven additional specimens were sterile. Since the patient remained afebrile and appeared to be recovering satisfactorily from surgery, the isolated occurrence of fungal growth was thought to have resulted from contamination of the blood specimen, and she was discharged from the hospital on the 19th postoperative day.

The patient was readmitted on January 21, 1971, with a three-week history of exertional dyspnea and pedal edema without orthopnea or paroxysmal nocturnal dyspnea. She had experienced chills for one week. Physical examination demonstrated neck veins distended to the angles of the jaw with the patient sitting. The face appeared plethoric and there was pedal edema which had not previously been present. The chest was clear. Auscultation of the heart revealed two prosthetic valve opening sounds, but only a single closing sound. The chest x-ray picture showed no pulmonary vascular congestion and no cardiomegaly. Because these findings suggested acute obstruction of the tricuspid valve, right heart catheterization was performed. The catheter was advanced across the prosthetic tricuspid valve. Right ventricular pressure was 38/0.4 mm Hg while mean right atrial pressure was 20 mm Hg with a mean diastolic gradient across the tricuspid valve of 18 mm Hg (Fig 2). A cine right atrioogram showed very slow passage of contrast material from a markedly dilated right atrium into the right ventricle, which did not appear dilated. No specific structural abnormalities of the prosthetic valve could be seen.

Immediately thereafter, the patient underwent exploratory cardiotomy. The right atrium was markedly dilated. The prosthetic tricuspid valve was in good position, but a large

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21540/)
LATE THROMBOTIC OBSTRUCTION OF STARR-EDWARDS PROSTHESIS

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FIGURE 2. Pressure recorded from the right atrium and right ventricle during cardiac catheterization in patient 2. The right ventricular diastolic pressure is normal, but right atrial pressure is markedly elevated, demonstrating obstruction to blood flow at the level of the prosthetic tricuspid valve.

amount of friable thrombotic material extended over the struts of the valve from the atrial side, obliterating approximately 90 percent of the orifice and immobilizing the ball. Microscopic examination of the thrombotic material showed budding yeast which grew Candida parakrusei on culture. The patient was treated with a ten-week course of intravenous amphotericin B. Although she initially sustained a good response, she has again developed evidence of recurrent Candida endocarditis three months after completing amphotericin B therapy.

DISCUSSION

Thromboembolic complications have been reported to occur in 20 percent to 30 percent of patients having prosthetic mitral or aortic valves. In contrast to this, thromboembolism involving prosthetic tricuspid valves has been reported very infrequently. Nevertheless, it is not clear whether the incidence of thromboembolic complications is actually less for tricuspid prostheses than for other prosthetic valves, or whether this complication has been reported less frequently merely because there is a smaller population of patients at risk (tricuspid prostheses being much less frequently used than mitral or aortic prostheses). Also, a small embolus shed from a tricuspid prosthesis into the lungs may well go unnoticed, while a similar embolus originating from the left side of the heart and occluding a systemic artery would be unlikely to escape attention.

Most thromboembolic complications involving prosthetic mitral and aortic valves occur relatively early after surgical placement of the prosthetic valve, while very few patients have their first evidence of thrombus formation more than two years after surgery. This is in contrast to the present two patients who manifested tricuspid valve thrombosis three and one-half and six years after surgical insertion. In both of these patients, however, a preexisting condition compromised prosthetic valve function prior to thrombosis. Patient 1 had a papillary muscle remnant and chordae tendineae entangled in the valve cage, severely limiting excursion of the ball, while in patient 2, the fungal infection appeared responsible for thrombus propagation. Similarly, the patient reported by Samaan and Murali had a papillary muscle entangled in the cage of the prosthetic valve. Likewise, six of seven patients reported by Vander Veer and co-workers had a strut of the valve cage embedded into or impinging upon the right ventricular musculature, thus preventing free movement of the ball. Thus, it appears that prosthetic tricuspid valve thrombosis rarely, if ever, occurs de novo, but may occur if normal valve function is compromised by some independent event. The most common preceding event appears to be impingement of subvalvular muscular structures upon the prosthetic valve which may occur as the hemodynamic improvement following surgery results in a decrease in the size of the right ventricular cavity so that the prosthesis no longer has adequate space to function.

The Candida parakrusei infection which resulted in prosthesis malfunction in patient 2 almost certainly began during her hospitalization for colectomy, since the rarity of this organism precludes its isolation from blood during that hospitalization to have been coincidental. Ellis and Spivack have reported that transient candidemia is not likely to represent tissue invasion or to require antifungal therapy if the patient is clinically improving and subsequent attempts to isolate the organism are unsuccessful. However, this case demonstrates that in patients with abnormal or prosthetic heart valves, even a single isolation of such organisms is of the gravest significance and must be pursued until the presence or absence of endocarditis is unequivocally established. Most patients previously reported with fungal endocarditis have had three predisposing conditions: (1) an abnormal heart valve, (2) broad spectrum antibiotic therapy which suppressed normal bacterial flora and thus encouraged fungal overgrowth, and (3) an obvious portal of entry for the organism. All three conditions existed in the present patient: (1) the prosthetic valve formed a nidus for fungal growth; (2) she received antibiotic therapy for urinary tract infection; (3) abdominal surgery provided a portal of entry, either from the bowel which commonly harbors Candida species or from the skin via the intravenous infusion apparatus. In addition, it is possible that the reversal of anticoagulant therapy, even for the briefest
interval thought to be compatible with a safe surgical procedure, might have resulted in fibrin deposition on the tricuspid prosthesis, thus enhancing the nidus for fungal growth. After the infection was established, it appears that continuing fungal growth resulted in propagation of thrombus to eventually obstruct the prosthesis.

The diagnosis of tricuspid prosthesis obstruction is based upon findings of severe right heart failure with increased systemic venous pressure, dependent edema, and often facial plethora and cyanosis in the absence of left ventricular failure. Diminution or disappearance of the tricuspid prosthetic valve sounds is of great significance but is difficult to appreciate if the patient also has a mitral valve prosthesis. Appearance of a tricuspid diastolic flow murmur would be of great clinical significance but was not apparent in either of the present patients. Finding a large diastolic gradient across the prosthetic tricuspid valve at cardiac catheterization confirms the diagnosis. If the prosthetic valve cannot be crossed with a cardiac catheter, the diagnosis is supported by a right atrioogram showing dilation of the right atrium with slow, prolonged emptying of the right atrium into a nondistended right ventricle. Since obstruction of a tricuspid valve prosthesis is a catastrophic event which may be rapidly fatal, early recognition of this complication with confirmation by cardiac catheterization is essential to permit surgical correction.

References

A Critique of Galen

Born in Pergamum, Galen (130-200) made a local reputation for himself as a physician to the local gladiators before setting out in his middle thirties in the hope of making an even bigger one in Rome. His ambition was quickly fulfilled—mainly, if his own account can be trusted, because of his remarkable diagnostic flare: he had the knack not simply of sensing what was wrong with patients (which was not necessarily much help to them, if there was no suitable remedy) but of sensing what would cure them. For some reason—fear of catching the plague, has been suggested; or fear of the animosity of his rivals—he suddenly left while his reputation was at its peak, to return to Pergamum. Not though for long; at the bidding of the Emperor Marcus Aurelius, he set out again for Rome, where he was physician to five emperors, spanning thirty years. That he was persuaded to go back has been widely considered one of the greatest misfortunes that medicine has ever suffered.