reverted back to complete heart block with idioventricular rhythm. The ventricle responded to a stimulus only when it fell in a period extending from the later half of the terminal slope of the T wave to the initial portion of the U wave. The supernormal period was approximately 150 milliseconds in duration.

In our case, the supernormal period was demonstrated at a time when the pacemaker generator was failing and discharging subthreshold stimuli. At this time, the patient's own intrinsic rhythm was atrial flutter with complete heart block and A-V junctional rhythm. Stimuli, all of the same intensity, were visible at varying intervals after the QRS complex (Fig 4). At no time did they elicit a response except during a 50 millisecond interval following the T wave and prior to the next QRS complex, when a stimulus should normally be propagated, the same subthreshold stimulus failed to evoke a QRS response. The following day the generator stimulus failed to evoke a QRS response at any time (Fig 5), but when a new pulse generator was implanted normal pacing took place immediately and continued to do so (Fig 6) indicating that there was no defect in the bipolar endocardial electrode.

This case represents a clear demonstration of the supernormal period in the intact human heart.

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Endocarditis of the Pulmonic Valve Simulating Cardiac Tumor*

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A unique case of calcific bacterial endocarditis involving the pulmonic valve, which simulated cardiac tumor, is reported. The clinical, fluoroscopic and angiographic findings are described and correlate well with the lesions discovered at surgery. In this case, the diagnosis was originally suspected because of the calcification noted during fluoroscopy. However, the unusual auscultatory findings may prove useful in detecting other cases of a similar nature.

Endocarditis of the valves of the right side of the heart is much less common than that involving the left heart and frequently presents difficulties in diagnosis, as many clinical features typical of left-sided involvement are absent. Not infrequently the only signs of right-sided valvular endocarditis are persistent fever and recurrent episodes of pulmonary embolization and infection.1-4 The tricuspid valve is more commonly involved in endocarditis of the right heart. Occasionally both the tricuspid and the pulmonic valves are affected. Only rarely does one encounter isolated involvement of the pulmonic valve.5-13

In this case isolated pulmonic bacterial endocarditis simulated a tumor of the right side of the heart.

Case Report

The patient, a 47-year-old Negro woman with a long history of alcoholism, had been treated for tuberculosis from 1959 to 1961 with isoniazid and cycloserine. There is no history of rheumatic or congenital heart disease. On three

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different occasions she had been hospitalized for episodes of chest pain and fever which were diagnosed as "pneumonia." In June, 1968 she was admitted to another hospital with chills, pleuritic chest pain, and a temperature of 104° F. On auscultation, a gallop rhythm was heard, but no murmurs were apparent. Chest x-ray films revealed fibrotic changes in the left upper lobe. Laboratory findings were compatible with urinary tract infection and therapy with tetracycline was started.

On the fourth hospital day, systolic and diastolic murmurs were noted at the left sternal border with a short apical diastolic rumble. Antibiotic therapy was stopped, and repeated blood cultures proved negative. However, intravenous penicillin was given for five weeks for presumed bacterial endocarditis. The patient became asymptomatic on this therapy and was subsequently discharged.

In September, 1968 she was admitted to Bronx Municipal Hospital Center with hemoptysis and chest pain of one week's duration and complaints of fever, chills, and weight loss. Her temperature was 100° F; her blood pressure was 130/80, and her heart rate 90 per minute. She was tall, thin, underweight and mildly dyspneic. There was slight ankle edema, but no clubbing cyanosis or splinter hemorrhages were noted, and the peripheral pulses were normal. Liver and spleen were not palpable. The lungs were clear on auscultation.

Marked right ventricular heave and third heart sound were present in the second intercostal space near the left sternal border. There were no thrills. A grade 2/6 systolic ejection murmur was heard best at the pulmonic area. A grade 2/6 early diastolic murmur of rather short duration was best audible in the third left intercostal space. The intensity of these murmurs increased on inspiration. A third heart sound varying in its relation to the second sound was audible over the left upper sternal border. The erythrocyte sedimentation rate, blood count, and albumin-globulin ratio were all within normal limits. The electrocardiogram suggested right ventricular hypertrophy with an intraventricular conduction disturbance. Phonocardiogram demonstrated a third sound occurring 0.11-0.16 sec after the aortic component of the second sound; the interval varied with the phase of respiration.

Chest x-ray films revealed linear streaks with minimal retraction of the left upper lobe. There were no significant differences when compared with the previous study. Fluoroscopy revealed a large irregular calcific density in the region of the main pulmonary artery and the right ventricular outflow tract. This density exhibited a complicated cyclic motion synchronous with each heart beat, moving upward into the main pulmonary artery with systole. During diastole, it fell back to the right ventricular outflow tract.

During the hospital course the patient was intermittently febrile, but ten blood cultures were negative. On one occasion she coughed up blood-tinged sputum, which was negative for Mycobacterium tuberculosis. Lung scan at that time was not diagnostic of pulmonary embolization.

Cardiac catheterization revealed pulmonary artery pressure of 29/12 with a mean of 15 mm Hg. The right ventricular end-diastolic pressure was 9 mm Hg. There were no abnormal pressure gradients across the pulmonic or tricuspid valves.

Cineangiograms in both anterior oblique and lateral projections confirmed the presence of a calcified mass that appeared to be attached to the ventricular surface of one of the pulmonic valve leaflets (Fig 1 and 2). During ventricular systole the mass moved with the valve leaflet into the main pulmonary artery.

A repeat lung scan was obtained which demonstrated diminished perfusion in the left lower lobe, indicating a possible embolic episode since the study performed three weeks earlier.

A few days after catheterization, which was performed under penicillin coverage, the patient's temperature rose to 105° F with shaking chills. Secondary bacterial endocarditis of the damaged valve resulting from the catheterization procedure was suspected and treatment with parenteral oxacillin and streptomycin was initiated. Despite massive antibiotic therapy, the patient remained febrile and her condition deteriorated. It was assumed that fragments of the mass were embolizing to the lungs and surgical intervention was planned.

Two days before surgery the patient complained of sudden right-sided pleuritic chest pain. Chest x-ray films revealed an infiltrate in the right lower lobe and lung scan showed a possible perfusion defect of the right lower lobe in addition to

**Figure 1.** Enlarged frame from cineangiogram (right anterior oblique projection). Opacified right ventricular outflow tract showing round defect related to ventricular surface of the pulmonic valve.

**Figure 2.** Schematic outline of Figure 1.
FIGURE 3. Fragments of pulmonic valve lesion. The upper three fragments were received from the lung and are irregular masses of yellow-tan, friable, and focally calcified tissue. The two lower fragments are gray glistening portions of pulmonic valve cusp with irregular outgrowths (arrows) of calcified tissue along the free margins.

the left lower lobe defect noted previously. Cardiac examination at this time no longer revealed the loud third heart sound with palpable shock in the left parasternal area. It seemed probable that part or all of the mass had embolized to the right lower lobe.

Surgical Procedure

At operation there was moderate dilatation of the right ventricle and pulmonary artery. A systolic thrill was present over the pulmonary artery and a diastolic thrill over the right ventricular outflow tract. There was no thrill and no systolic expansion palpable over the right atrium. The endocardium of the right ventricle was quite normal between the base of the pulmonary valve and the papillary muscle of the conus. However, the left cusp of the valve was torn, and attached to its free edge was a small calcific mass 1 cm in diameter, which was excised. The pulmonary arteries were explored with a balloon catheter and a piece of grossly uncalcified tissue, 1.5 x 1 cm in size, was retrieved from the artery to the right lower lobe.

Following surgery the patient's condition improved. Her temperature fell to 100-100.5° F and eventually returned to normal with further antibiotic therapy. The infiltrate in the right lower lobe slowly resolved.

Postoperatively the patient developed congestive heart failure manifested by a protodiastolic gallop, distended neck veins, and a congested liver which improved with rest and small doses of digoxin. The only pertinent physical finding on discharge was the pulmonic valve diastolic murmur.

Pathologic Findings

The specimen from the lung consisted of three irregular masses of yellow-tan friable tissue, which was composed of irregular masses of fibrin with immeshed neutrophils and histiocytes. Several miniature abscess-like zones were present. In addition, focal calcification was seen. No bacterial or fungal organisms could be identified on periodic acid-Schiff and Brown-Brenn stains. Two portions of pulmonary valve cusp, with irregular outgrowths of yellow-tan calcified tissue along the free margins, were also excised and showed hyalinization, focal calcification, vascularization, and frank granulation tissue. Fresh fibrinoid material was also present on the valve surface (Fig 3-5). The gross and microscopic findings were consistent with the diagnosis of a healing bacterial vegetation proceeding calcification.

FIGURE 4. Pulmonic valve cusp with adherent vegetation. The tissue on the left is vascularized and hyalinized pulmonic valve cusp in which numerous inflammatory cells are evident. A circular area of increased acidophilia (arrows) suggests early necrosis of stroma. In the upper right is an adherent polypoid mass of fibrin with immeshed neutrophils. Hematoxylin and eosin x20 (original magnification).

FIGURE 5. Vegetation on pulmonic valve surface. The arrow marks a layer of fibrin covering a meshwork of neutrophils, necrotic debris, and fibrin. Hematoxylin and eosin x80 (original magnification).

DISCUSSION

The rarity of endocarditis of the right side of the heart is already well documented.1-3,5-7 The relative immunity of the right side of the heart, and particularly of the pulmonic valve, has been explained by the hypotheses that low pressures existing in the right heart chambers tend to cause less mechanical stress7 and that lower oxygen saturation in the blood of the right heart discourages the growth of aerobic bacteria. Also, the mitral and aortic valves are more subject to rheumatic or congenital deformity, which is an important predisposing factor for bacterial endocarditis.8 Right-sided endocarditis, usually...
with virulent organisms, such as *Staphylococcus aureus*, has been described mostly in heroin addicts using unsterilized syringes and needles.\(^1,11,12,14\) Even in addicts, right-sided endocarditis is less frequent than left-sided disease, with isolated pulmonic involvement quite rare.\(^1,13,15\)

A reported case with some similarity to ours is that of Garcia and Taber\(^9\) who described a case of endocarditis of the pulmonic valve in a patient with an atrial septal defect who had recurrent episodes of pulmonary infection. At surgery, there was a large, partially calcified vegetation on the right cusp of the pulmonic valve which measured about 1 cm in diameter.

The etiology of our patient's disease is not clear. We cannot rule out the possibility of a minor undetected congenital malformation in the pulmonic outflow tract or valve. She denied using narcotics. Also, the responsible organism could not be isolated from the blood or sputum. Numerous blood cultures and cultures of the surgical specimen all proved negative, perhaps because she was treated with antibiotics throughout most of her hospitalization. This patient's history of recurrent episodes of chest pain and pulmonary infection probably represented repeated septic pulmonary emboli which may well suggest that the involvement of the pulmonary valve was present for years prior to diagnosis.

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