Q interval during normal intraventricular conduction is also not unusual.\textsuperscript{16,18} It is conceivable that the prolonged H-Q interval in our patient during normal QRS morphology suggests a diffuse disease of the conduction system. In that case it is possible that subclinical dysfunction of right bundle branch and left posterior fascicle became unmasked in the form of right bundle branch block and left posterior hemiblock as a result of the diminished blood supply during anginal episodes.

**REFERENCES**


**Observations on Myocardial Function During Chronic Catecholamine Oversecretion**

A young patient with pheochromocytoma\textsuperscript{*}

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Observations on myocardial function were made in a young patient in whom a pheochromocytoma was secreting large amounts of catecholamines, primarily norepinephrine. Without history of longstanding or severe hypertension, the patient showed evidence of left ventricular failure. When the heart size had returned to normal as a result of preoperative treatment, there was still a loud atrial gallop and hemodynamic evidence of loss of myocardial reserve.

There are only a few patients in the literature with evidence of myocardial injury due to catecholamines from functioning pheochromocytoma.\textsuperscript{1-4} This patient is particularly appropriate to report because of his young age and, therefore, expected good myocardial reserve.

**CASE REPORT**

A 27-year-old carpenter entered the Houston VA Hospital because of progressive fatigue of five-six months duration. He had experienced excessive perspiration and tachycardia for four-five years. Fatigue and nonproductive cough had begun five-six months prior to admission and became progressive and disabling. Moderate exertional dyspnea was present. There was no orthopnea, paroxysmal nocturnal dyspnea or leg swelling. One month prior to admission, he began coughing up small amounts of white sputum which was occasionally blood-tinted after a severe bout of coughing. Blood pressure and roentgenogram of the chest two years prior to admission were normal. There was no history of headaches, excessive alcoholic intake or poor dietary habits.

On admission, he weighed 162 lb, was afebrile, but appeared moderately ill. He had a regular heart rate of 144 beats per minute and regular respirations of 22 per minute. The blood pressure with the patient in a recumbent position was 135/115 mm Hg, decreasing to 125/95 in the sitting position. The neck veins were not distended. On auscultation, a few rales were heard over the lung bases posteriorly. Cardiac size could not be determined by palpation of the chest. The heart sounds were heard clearly and a prominent atrial gallop was present at the lower left sternal border and at the apex. The liver and spleen were not palpable. There was no peripheral edema. Funduscopic examination was normal. All peripheral pulses were normal.

His admission white blood cell count was 10,300 cells/mm\textsuperscript{3} with a normal differential and a hemocrit of 40

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sitting position. The electrocardiogram remained abnormal 0.14 second. The urinary vanillyl mandelic acid (VMA) voltage was borderline high and the T waves low in suggested biatrial enlargement and left ventricular hypemphy suprarenal mass. Renal arteriograms were performed to fur-

his weight remained stable. Roentgenogram of the chest on tent was 4.450 micrograms (normal less than 75) and albumin and no sugar, 

21 specimens (normal 0.5-7.0); the urinary norepinephrine con-

content was 18, 14, and 22 mg respectively in three =-hour time was 90 seconds. Chest roentgenogram on admission showed moderate, generalized cardiomegaly (Fig 1A) with increase in bronchovascular markings, especially near the hilum suggesting pulmonary infiltrates. The electrocardiogram showed sinus tachycardia of 145 beats/min and suggested biatrial enlargement and left ventricular hypertrophy —the P waves were prominent and diphasic in lead V1; the QRS voltage was borderline high and the T waves low in lead 2, inverted in leads 1, aVL, V4-6.

Treatment by bed rest, digitalization and one injection of meralluride (Mercuzylorin) resulted in an 11-pound weight-loss, by the fifth hospital day complete disappearance of cough and considerable decrease of the cardiac silhouette auscultation and phonocardiogram which diminished in the upper pole of the right kidney. Four and one-half seconds after the appearance of contrast material in the aorta, a mass was faintly outlined over the upper pole of the right kidney.

Blood volume was measured preoperation. By the 131I method, the plasma volume was 3,180 ml (expected 2,770), red cell volume 2,120 ml (expected 2,260) and total blood volume 5,300 ml (expected 5,930). At the time of surgical exploration of the abdomen, both kidneys and adjacent structures were carefully examined. A pheochromocytoma of the right adrenal gland was found and excised. It weighed 56 gms and contained 5.12 mg of norepinephrine and 0.24 mg of epinephrine per gram of wet tissue.

Postoperation, his heart size remained normal, the blood pressure and heart rate returned to normal and the atrial gallop disappeared. Twenty-four hour urinary VMA's and glucose tolerance test were within normal limits. The electrocardiogram 1k months after operation had returned towards normal but remained abnormal—P waves no longer suggested biatrial enlargement, however, T waves were flat in 1, slightly inverted in aVL and inverted in V4-6. One year postopera-

grams via the brachial artery, cardiac evaluation was also carried out.

The right atrial mean pressure was 3 mm Hg (normal 0-8) during a heart rate of 120 beats/minute. The contour of the right atrial pressure tracing showed prominent “a” wave of 5 mm Hg without distinguishable “c” or “v” waves. With only minimal manipulation of the catheter in the right atrium, the patient developed atrial flutter with a 2:1 ventricular response of 164 beats/min which persisted throughout the procedure. During this rhythm, the left ventricular end-diastolic pressure was 24 mm Hg (normal less than 12), the pulmonary arterial wedge pressure was 25 mm Hg (normal less than 13), the aortic pressure was 150/120 mm Hg, the pulmonary arterial pressure was 31/27 with a mean of 30 mm Hg (normal less than 30 systolic and less than 20 mean), and right atrial mean pressure 6 mm Hg. The cardiac index by dye dilution method was 2.25 liters/min/M² (normal 2.5-4.5), the peripheral resistance was 2,440 dyne-sec-cm⁻² (normal less than 1800).

The arteriogram was obtained by injecting radiocontrast material in the abdominal aorta at the level of the diaphragm. Four and one-half seconds after the appearance of contrast material in the aorta, a mass was faintly outlined over the upper pole of the right kidney.

Although the heart size had returned to normal, the blood pressure continued to be elevated and to exhibit a fall in the sitting or standing position. Repeated measurements of blood pressure on the ward during the next two months disclosed a range between 140 and 150 mm Hg systolic, 90 and 110 mm Hg diastolic in the recumbent position. The patient continued to have a sinus tachycardia of 100-120 beats/min, easily audible, sharp heart sounds and prominent atrial gallop by auscultation and phonocardiogram which diminished in the sitting position. The electrocardiogram remained abnormal and on three occasions showed paroxysmal atrial tachycardia of 120 beats/min—P wave inverted in lead 2 and PR interval 0.14 second. The urinary vanillyl mandelic acid (VMA) content was 18, 14, and 28 mg respectively in three 24-hour specimens (normal 0.5-7.0); the urinary norepinephrine content was 4,450 micrograms (normal less than 75) and epinephrine content was 28 micrograms (normal less than 21) in a 24-hour specimen. A nephrotomogram during intra-

venous radiocontrast infusion raised the possibility of a right suprarenal mass. Renal arteriograms were performed to further elucidate the patient’s disease. During renal arterio-
fluctuations but to have increased in the course of five or more years. Myocardial damage by catecholamines is derived from comparative heart rate.7

In animals, there is considerable evidence for myocardial muscle damage following infusion of catecholamines.1, 2-5 In man, most of the evidence for myocardial muscle damage by catecholamines is derived from autopsy studies. The incidence of detection of myocardial muscle necrosis at autopsy appears to depend upon how avidly lesions were looked for. Szakacs and Cannon6 found marked association between catecholamines and pathologic myocardial lesions. Their survey of autopsy material yielded 17 cases of pheochromocytoma, myocarditis being found at autopsy in all of them. Van Vliet, Burchell and Titus7 found evidence of active carditis microscopically in 15 of 22 patients who were found at autopsy to have functioning pheochromocytoma. In two additional patients, fibrosis was found and thought to represent healed carditis.

The incidence of clinically significant myocarditis is probably far less than in the autopsy material. Occasionally, carditis is evident clinically, as exemplified by one of 22 patients of Van Vliet et al.,8 the one patient of Engelman and Sjoerdsmak with malignant pheochromocytoma and probably the one patient of Engelman et al.9 Kline3 states that two of his seven patients studied at autopsy had clinical evidence of myocarditis. More recently, two patients have been reported presenting with cardiomyopathy without impressive elevation in blood pressure due to oversecretion of epinephrine rather than norepinephrine.5, 4

The incidence of exertional dyspnea and congestive heart failure in patients with pheochromocytoma also varies. No detailed clinical study is available of the 22 patients of Van Vliet, Burchell and Titus;8 other than that of the 15 patients with active myocarditis at autopsy; 11 had symptoms and signs of acute left ventricular failure. A clinical report from the same medical center11 in which the clinical features of 76 patients were examined (the diagnosis of 69 patients was confirmed at operation) showed that dyspnea was present in 11 patients (14 percent) and cardiomegaly or congestive heart failure or both in 9 patients (12 percent).

REFERENCES
9 Szakacs JE, Mehlinm B: Pathologic changes induced by 1-Norepinephrine. Am J Cardiol 5: 619-627, 1960

Paroxysmal Coronary Embolism in a Patient with Mid-Systolic Click Syndrome*

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Embolic coronary artery disease is an uncommon cause of death. A paradoxical thromboembolism to the left coronary artery was the reason for death in a 53-year-old man who presented with a mid-systolic click syndrome and chest pain. His clinical course and postmortem findings are presented. Paroxysmal coronary artery embolism is reviewed. An unusual etiology of systolic click syndromes is suggested. Thoughts about therapy are presented.

Coronary embolism is an unusual form of coronary artery disease. Most cases are associated with bacterial endocarditis.1 However, intracardiac thrombus, luetic aortitis, atherosclerotic disease of the aorta, proximal coronary artery thrombus, pulmonary vein thrombus, calcified valves, parasites, cardiac bypass, tumor, coronary angiography, air and paradoxical venous thrombus have been reported as etiologic agents of embolic coronary disease. We recently participated in the management of a man with paradoxical coronary embolism and the unusual association of a mid-systolic click. His case is presented below.

CASE REPORT

A 53-year-old Caucasian man was well until November 11,