Short PR Interval and Narrow QRS Complex Associated With Pheochromocytoma: Electrophysiologic Observations

SHOEI K. HUANG, MD, FACC,* MICHAEL J. ROSENBERG, MD, PABLO DENES, MD, FACC

Chicago, Illinois

A patient with a short (0.10 second) PR interval, narrow QRS complex and palpitation is described. Electrophysiologic studies demonstrated the presence of accelerated atrioventricular (AV) nodal conduction. Subsequently, a pheochromocytoma was found. Surgical removal of the tumor resulted in normalization of the PR interval. These findings suggest that the short PR interval and the accelerated AV nodal conduction were due to the effect of excess catecholamines on the AV conduction system.

In 1952, Lown, Ganong and Levine (1) described a syndrome of short PR interval, normal QRS complexes and paroxysmal rapid heart action. There has been considerable controversy in regard to the mechanism of the short PR interval and the types of tachyarrhythmias associated with this syndrome. A short PR interval has been described in patients with chronic lung disease (2), acute myocardial infarction (3), endocrine disorders (hyperthyroidism and Cushing’s disease) (4), and glycogen storage disease (Pompe’s disease) (5). Other causes of short PR interval include abbreviated atrioventricular (AV) conduction noted in children and young adults, ectopic impulse formation (atria, coronary sinus or AV junction) and erroneous measurement utilizing only one or two electrocardiographic leads (6).

Electrocardiographic changes are frequent in patients with pheochromocytoma (7); these consist of left ventricular hypertrophy, T wave changes and sinus tachycardia. Occasionally, changes suggestive of acute myocardial damage, including marked and diffuse T wave changes and transient ST segment elevation or depression, have been reported (8,9). The acute changes are usually transient and revert to normal after removal of the tumor or after pharmacologic blockade (10).

In the present report, we describe a patient who had a short PR interval and narrow QRS complexes. Electrophysiologic studies demonstrated the presence of enhanced AV nodal conduction. Subsequently, she was found to have a left adrenal pheochromocytoma. After resection of this tumor, the PR interval returned to normal.

Case Report

The patient, a 45 year old black woman, had a history of labile hypertension, palpitation and atypical chest pain. On February 26, 1981, she presented to the emergency room of a local hospital with the chief complaints of severe chest pain and dizziness. Her blood pressure was 234/1140 mm Hg. After admission, she had a cardiopulmonary arrest from which she was successfully resuscitated. A diagnosis of acute myocardial infarction was made based on marked elevation of cardiac serum enzymes and ST-T wave changes. She was discharged on March 28. An electrocardiogram taken on June 25 (Fig. 1A) showed a sinus rate of 75/min, a short PR interval (0.10 second) and an otherwise normal tracing.

On August 23, she was admitted to our center for cardiac catheterization and electrophysiologic study. The blood pressure was 150/100 mm Hg in both the supine and sitting positions. Cardiac examination was normal. Chest X-ray film showed a normal heart size. The electrocardiogram showed a short PR interval and was unchanged from her previous electrocardiogram. Complete blood count, urinalysis and blood chemistry determinations including thyroid function studies were normal. She had a maximal treadmill exercise test that did not show electrocardiographic evidence of ischemia; there were no arrhythmias. A 24 hour Holter electrocardiographic recording demonstrated normal sinus rhythm with occasional ventricular premature complexes and a six beat run of ventricular tachycardia at a rate of 110 to 130 beats/min. Her symptoms of palpitation and chest pain did not correlate with the presence of arrhythmias.
Figure 1. Standard 12 lead electrocardiograms taken before (A) and after (B) resection of pheochromocytoma. The longest PR interval was 0.08 second in A and 0.16 second in B.

Figure 2. Demonstration of 1:1 AV nodal conduction at an atrial pacing cycle length of 280 ms with AH increment less than 100 ms (from 60 ms during basic sinus rhythm to 110 ms) and sinus node recovery time of 540 ms. Both the AH interval (60 ms) during sinus rhythm and the HV interval (35 ms) were borderline short. A = atrial activity; H = His bundle activity; HRA = high right atrium; HBE = His bundle electrogram; RVA = right ventricular apex; S = pacemaker stimulus artifact; V = ventricular activity. Interval between time markers equals 1,000 ms. The paper speed was 100 mm/s.

two-dimensional and M-mode echocardiograms were normal as were the results of cardiac catheterization and coronary angiography.

Electrophysiologic study was performed using previously described methods (11) because of the history of cardiac arrest and recurrent palpitation. All cardiac medications were discontinued for 48 hours before the study. At the time of electrophysiologic testing, her sinus rate was 92 beats/min. The AH interval was 60 ms, the HV interval 35 ms and the
PA interval 25 ms. Incremental atrial pacing from the high right atrium (cycle length from 600 to 300 ms) demonstrated a gradual but small increment in AH interval from 60 to 110 ms (ΔAh = 50 ms). At an atrial pacing cycle length of 280 ms (rate 214 beats/min), 1:1 AV nodal conduction was still present (Fig. 2). The AV nodal effective (<260 ms) and functional (<300 ms) refractory periods at the driven pacing cycle of 500 ms were short. Similarly, 1:1 retrograde ventriculoatrial (VA) conduction was present up to a ventricular pacing rate of 200 beats/min (cycle length 300 ms) with gradual prolongation of VA interval. There was no inducible supraventricular or ventricular tachycardia by programmed single and double extrastimulus technique.

The possibility of pheochromocytoma as a cause for the patient’s hypertension and symptoms was suggested. This diagnosis was substantiated by the elevation of urinary catecholamines (675 μg/24 hours, normal 38 to 100), metanephrine (1.9 mg/24 hours, normal 0.3 to 0.9) and vanillylmandelic acid (VMA) (8.4 mg/24 hours, normal 0.7 to 6.8). Subsequently an abdominal aortic angiogram demonstrated a 5 to 6 cm circumscribed hypervascular mass in the left suprarenal region (Fig. 3). Two months later, a left adrenal pheochromocytoma weighing 50 g was removed. An electrocardiogram taken 1 week after the operation as well as 4 months later while the patient was not taking any cardiac medication showed a normal PR interval (0.16 second) (Fig. 1B). The patient had no further complaints of palpitation or chest pain. Blood pressure returned to normal. The patient denied consent for repeat electrophysiologic study. The two follow-up 24 hour Holter monitor recordings did not show any ectopic rhythm.

**Discussion**

Disorders associated with short PR interval and narrow QRS. In their original article, Lown, Ganong and Levine (1) commented on the possible relation between their findings and the endocrine system (particularly the adrenals) and autonomic nervous system. They quoted several endocrine disorders that may affect the PR interval. Hyperthyroidism and Cushing’s syndrome are associated with short PR interval and Addison’s disease is occasionally associated with first degree atrioventricular (AV) block. They also described several other conditions (neurosis, emotional in-stability, hypothalamic overfunction, standing position) associated with increased sympathetic tone that may result in shortening of the PR interval and paroxysmal rapid heart action. It is surprising that pheochromocytoma has not been previously associated with this syndrome.

Electrophysiologic studies. Many electrophysiologic studies (12–19) have been reported in subjects with a short PR interval and narrow QRS complexes, with or without documented tachyarrhythmias. Most studies (12–17) found that the short PR interval reflects a shortening of the AH interval, although in some (17–19) there was a shortening of the HV interval. A short PR interval associated with a short AH interval may be explained by the presence of a “partial” AV nodal bypass (James fibers) (20), a total AV nodal bypass (Brechenmacher tract) (21), a small or immature AV node or an intranodal “fast pathway” (22). Gallagher et al. (23) introduced the term “enhanced AV nodal conduction” to describe the following electrophysiologic phenomena: 1) an AH interval during sinus rhythm of 60 ms or less; 2) 1:1 atrio-His bundle conduction during high right atrial pacing up to a cycle length of 300 ms, and 3) an increment in the AH interval that does not exceed 100 ms with rapid atrial pacing up to a cycle length of 300 ms.

**Present case.** We describe a patient who had pheochromocytoma and a short PR interval, narrow QRS complex and episodes of palpitation. Electrophysiologic studies before surgery demonstrated a short AH interval (60 ms) during sinus rhythm, with an increment of only 50 ms in the AH interval during rapid atrial pacing, and 1:1 AV conduction at a paced cycle length of 280 ms. These findings fulfill the criteria for the presence of enhanced AV nodal conduction. We also demonstrated short AV nodal refrac-

**Figure 3.** Abdominal aortic angiogram demonstrating a hypervascular mass approximately 5 to 6 cm in the left suprarenal region (delineated by **solid arrows**).
tory periods. There was excellent retrograde ventriculoatrial (VA) conduction through the AV node (1:1 VA conduction at a ventricular pacing rate of 200/min). After resection of the tumor, the PR interval returned to normal and the symptoms subsided. These observations confirm the role of catecholamines, particularly epinephrine and norepinephrine, in the genesis of short PR interval and enhanced AV nodal conduction.

The short PR interval in our patient was not dependent on the presence of sinus tachycardia, but was recorded during normal sinus rates. We hypothesize that chronic sympathetic hyperstimulation resulted in a short PR interval secondary to enhanced AV nodal conduction. This accelerated AV nodal conduction was not accompanied by a concomitant chronic increase in sinus nodal discharge rate, suggesting a differential adaptation process between the sinus and AV nodes to chronic catecholamine excess. Resection of the pheochromocytoma resulted in normalization of AV conduction time.

We deeply appreciate the secretarial assistance of Cheryl Bahrychuk.

References