Complete Heart Block Developing during Aortic Valvuloplasty*

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We report the occurrence of CHB consequent to ABV in an 80-year-old white woman with calcific aortic stenosis. The patient underwent ABV with a balloon 20 mm in diameter, resulting in only a modest increase in aortic valve area and a decrease in aortic valve gradient. The procedure was complicated by transient intermittent CHB which required temporary transvenous pacemaking and resolved within 72 hours. A second ABV was performed four weeks later with a balloon 23 mm in diameter. This was complicated by persistent CHB which required placement of a dual-chamber pacemaker. (Chest 1989; 96:1201-03)

CHB = complete heart block; ABV = aortic balloon valvuloplasty; DDD = dual chamber atrial tracking pacing mode

The technique of ABV has recently been introduced for use in patients with calcific aortic stenosis who are elderly or otherwise at increased risk for valve replacement. Initial results suggest that only a moderate increase in aortic valve area and decrease in transvalvular gradient are obtained, and restenosis occurs commonly. Efforts at improving initial results and decreasing the incidence of restenosis have been directed toward the use of larger balloons, either singularly or in combination. Major complications have been reported in as many as 25 percent of cases with ABV. While conduction disturbances are frequently seen, including transient left bundle-branch block and CHB, we know of no report of permanent CHB after ABV. This case is an example of persistent CHB consequent to ABV which required implantation of a permanent pacemaker.

CASE REPORT

An 80-year-old white woman with a history of scleroderma presented with eight months of increasing dyspnea on exertion. The patient denied a history of chest pain or syncope. The ECG disclosed normal sinus rhythm with borderline first-degree AV block and nonspecific conduction delay consistent with left ventricular hypertrophy (Fig 1). Echocardiography showed left ventricular hypertrophy with good systolic function, a calcified aortic valve with limited mobility, and an aortic annular diameter of 18 mm. The estimated mean aortic gradient by Doppler measurement was 56 mm Hg.

On Oct 31, 1988, the patient was admitted to the hospital with acute pulmonary edema. She was successfully treated with nitrates, diuretics, and oxygen therapy. No medication known to interfere with cardiac conduction was administered. The patient was believed to be a poor surgical candidate because of her advanced age, scleroderma, and severe COPD (FEV₁ = 0.8 L) and was therefore referred for aortic valvuloplasty.

On Nov 2, 1988, the patient underwent cardiac catheterization and ABV. Right heart catheterization revealed a mean pulmonary artery pressure of 36 mm Hg. There was no significant coronary artery disease. Fluoroscopy revealed a typically calcified aortic valve and annulus. The mean transaortic gradient was 60 mm Hg, and the aortic valve area was calculated to be 0.35 cm².

Immediately following cardiac catheterization, the patient underwent ABV using a 20-mm (diameter) balloon dilation catheter.

![Image](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21603/ on 06/03/2017)

**Figure 1.** Standard 12-lead ECG done prior to ABV, demonstrating normal sinus rhythm with first-degree AV block, occasional premature ventricular contractions, left atrial enlargement, nonspecific conduction delay consistent with left ventricular hypertrophy and associated ST-T wave changes.
(Mansfield Scientific Co.). Four inflations with pressures to 3 atm lasting approximately 20 seconds per inflation were performed. During the procedure the patient developed intermittent AV block refractory to atropine which required placement of a temporary transvenous pacemaker. After valvuloplasty the aortic valve area increased to 0.59 cm², and the mean aortic valve gradient decreased to 22 mm Hg. The heart block resolved over the ensuing 72 hours, while the patient was monitored in the coronary care unit with a temporary pacemaker in place. Subsequent Holter monitorings showed no heart block, and the temporary pacemaker was removed. The patient was discharged on Nov 11, 1988.

The patient was readmitted on Nov 31, 1988, with worsening shortness of breath. The echocardiogram was consistent with severe aortic stenosis, and she therefore underwent a second ABV on Dec 2, 1988. Prior to the valvuloplasty the patient’s heart was in normal sinus rhythm. A temporary transvenous pacemaker was inserted prophylactically. Initial hemodynamic measurements revealed a mean aortic gradient of 53 mm Hg and an aortic valve area of 0.3 cm². The patient underwent six dilations with a 23-mm (diameter) dilation catheter (Mansfield), which was inflated with up to 3 atm of pressure for an average duration of 20 seconds per inflation. During the first inflation the patient went into CHB which required pacing. After the valvuloplasty the mean aortic gradient decreased from 53 to 35 mm Hg, and the aortic valve area increased from 0.3 cm² to 0.56 cm².

The patient remained in CHB with total dependence on the temporary pacemaker for several days. On Dec 12, 1988, ten days after her second valvuloplasty, the patient was still in CHB (Fig 2) and underwent implantation of a dual-chamber permanent pacemaker. She was subsequently transferred to a chronic care facility. Routine follow-up at two months revealed 100 percent AV sequential pacing, with CHB as the underlying rhythm.

**DISCUSSION**

Aortic balloon valvuloplasty in adults with calcific aortic stenosis remains investigational. Initial reports suggest that the procedure results in a modest increase in aortic valve area, which appears in some patients to be associated with an improvement in symptoms and left ventricular function; however, there is a high rate of restenosis, which occurs early and accrues with time. Complications are frequent, and although procedurally related mortality is relatively low, there is a significant six-month mortality reflecting the patients’ overall condition and perhaps the suboptimal hemodynamic results.

We and others have shown that better hemodynamic results are obtained with a larger-diameter dilating force achieved with multiple balloons or larger balloons. Indeed, there is a current trend toward oversizing the measured annulus, as was done in this patient; however, there is no evidence that the use of larger balloons decreases the incidence of restenosis or improves prognosis. In addition, the use of larger balloons carries with it increased potential for complications subsequent to injury of the periaortic tissue.

We have previously demonstrated that damage to the periaortic conduction system appears to be related to the balloon’s size in normal dogs undergoing valvuloplasty with single and double balloons. Dogs dilated with double balloons had evidence of injury to the conduction system, as demonstrated by contraction band necrosis of cells of the left bundle branch. While no dog developed heart block, there was prolongation of the QRS complex in one half of the animals dilated with double balloons.

Disturbances in the ECG are frequently observed during clinical ABV. Conduction abnormalities are also not uncommon, with transient left bundle-branch block being reported in 13 percent and transient CHB in approximately 1 percent of the cases. To our knowledge, this is the first report of ABV-induced persistent heart block requiring a permanent pacemaker.

The coexistence of scleroderma and the electrocardiographic findings of borderline first-degree AV block with an intraventricular conduction delay raises the possibility that our patient had some underlying disease of the conduction system; however, there was no evidence of second-degree or third-degree AV block on Holter monitorings before valvuloplasty. Even if occult conduction system disease were present, the association of CHB with catheter manipulation and its persistence subsequent to the use of a larger balloon suggests that valvuloplasty caused the significant injury. In view of the proximity of the conduction system to the aortic valve apparatus, it is surprising that heart block is not more frequently encountered with ABV. Heart block requiring permanent pacing is a recognized complication of aortic

**FIGURE 2.** Rhythm strip from lead MCL₁, demonstrating CHB persisting ten days after valvuloplasty. Atrial rate is 90 bpm, and ventricular rate is 42 bpm.

**FIGURE 3.** Simultaneous left ventricular (LV) and aortic (AO) pressures measured during intermittent heart block associated with first ABV. Paced ventricular rhythm gives way to sinus mechanism with corresponding increase in aortic and left ventricular pressures.
valve ring abscess and aortic valve replacement. Our animal data suggest that irreversible damage to cells of the conduction system may be brought about by balloon dilatation and that this may be more frequent with larger balloons. While our patient had reasonably maintained blood pressure with ventricular pacemaking, the loss of atrial function in a patient with a hypertrophic noncompliant ventricle may result in a marked decrease in blood pressure and cardiac output (Fig 3). The utilization of dual-chamber pacing in the DDD mode, which provides for an appropriate AV contraction sequence, as well as rate augmentation, seems appropriate under these circumstances.

In summary, we present a case of CHB requiring permanent pacing subsequent to ABV. The irreversible damage consisting of contraction band necrosis in the cells of the conduction system in the canine model may underlie the mechanism of heart block in this patient. The physician performing ABV should be aware of the need for urgent pacemaker therapy and may wish to prophylactically insert a temporary pacemaker in these cases.

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Cocaine-induced Acute Aortic Dissection*

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While cocaine-induced myocardial infarction has been frequently documented, the differential diagnosis of chest pain should include aortic pathology. The successful management of acute aortic dissection secondary to cocaine abuse has not been previously reported to our knowledge. In a 45-year-old man who presented with typical chest pain and wide mediastinum, the successful management of this disease included early and accurate diagnosis and replacement of the aortic valve as well as the torn portion of the ascending aorta.

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Clinicians are aware of the more commonly occurring cardiac and cerebrovascular consequences of cocaine abuse: ventricular arrhythmia, myocardial ischemia and infarction, cerebrovascular accident, and subarachnoid hemorrhage. However, the differential diagnosis of chest pain associated with cocaine abuse should include aortic pathology. Acute rupture of the ascending aorta has been reported as a fatal consequence of smoking cocaine (freebasing). This article is the first description, to our knowledge, of a successful antemortem diagnosis and repair of a cocaine-induced acute aortic dissection.

CASE REPORT

A healthy 45-year-old male executive, at the end of a 36-h drinking and cocaine-snorting session, presented with severe, sudden, unrelenting, crushing substernal chest pain without radiation. There was associated upper back pain, along with diaphoresis and palpitations, but without lightheadedness or leg, abdominal, or groin pain. The patient was initially evaluated at a nearby hospital and referred to North Shore University Hospital for definitive care.

Past medical history was significant for mild, untreated hypertension. The patient smoked two packs of cigarettes and drank one quart of vodka per day. He had had a recent emergency room visit for drug overdose (IV heroin and cocaine—speedball). There was no history of Marfan’s syndrome or connective tissue disorder, nor were other drugs or other forms of cocaine used during this time.

Physical examination revealed moderate distress. His vital signs were as follows: heart rate, 100 bpm and regularly irregular; blood pressure 149/60 mm Hg; and respiratory rate, 26 breaths/min. Cardiac examination showed a normal S1 and S3 with a decrescendo diastolic murmur at the left sternal border. There was no rub or gallop. The left radial and left femoral pulses were diminished, with no palpable pulses distal to the left common femoral artery. The right radial and femoral pulses were full and bounding. His urine output and laboratory values were within normal limits. The ECG showed sinus rhythm with frequent atrial premature contractions, normal axis, and no evidence of acute infarction nor ischemia. A chest x-ray film showed clear lung fields with a widened mediastinum.

Echocardiogram and CT scan suggested a Stanford type A acute aortic dissection. Angiography revealed a tear starting in the

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