Case Reports

The Pitfalls of the Brachial Arterial Pressure Curve in the Evaluation of Valvular Aortic Stenosis

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SUMMARY

Two patients with calcific aortic stenosis were studied with cardiac catheterization and brachial arterial pulse pressures. The ejection time, first derivative and upstroke time of the brachial artery pressure pulse was compatible with severe aortic stenosis. On cardiac catheterization, only minimal aortic stenosis was found. These cases illustrate the importance of cardiac catheterization as the sole determinant in the final evaluation of aortic stenosis.

Additional Indexing Words :

Systolic ejection time Systolic gradient Cardiac catheterization

IT has been found that the direct brachial arterial pulse pressure curve correlates with the central arterial pulse contour in the indirect assessment of the severity of aortic stenosis.¹⁾

This paper presents 2 cases of aortic stenosis which were studied with both cardiac catheterization and peripheral arterial pulse pressures. The ejection time, first derivative and upstroke time of the brachial artery pressure pulse were compatible with severe aortic stenosis. On cardiac catheterization, however, only minimal aortic stenosis was noted.

The purpose of this paper is to stress cardiac catheterization as the only reliable measure in the determination of the severity of aortic stenosis.

Case 1 : A 47-year-old white male was admitted to the Bronx Veterans Administration Hospital on January 8, 1968 because of exertional dyspnea and chest pain.

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Five years prior to admission, the patient first developed fatigue, dyspnea on exertion, and chest pain relieved by rest. Six months before admission, he developed six-pillow orthopnea and paroxysmal nocturnal dyspnea. He was treated by his private physician with an unknown medication. There was no past history of rheumatic fever.

On admission, his blood pressure was 130/100, his pulse was 80/min. and regular. The pertinent physical findings were confined to the heart. The PMI was not palpable. There were no thrills. There was a grade III/VI systolic ejection murmur at the base. On fluoroscopy, there was slight enlargement of the left atrium and the left ventricle with calcification of the aortic valve. There was noted post-stenotic dilatation of the aorta. The EKG revealed sinus rhythm and non-specific ST and T wave changes.

The CBC, urinalysis, BUN, cholesterol, electrolytes, prothrombin time, calcium, and phosphorus were all within normal limits.

It was thought that the patient had severe aortic stenosis and he was digitalized and placed on maintenance digoxin of 0.5 mg. per day.

A cardiac catheterization was performed. A Cournand needle was inserted

Table I. Cardiac Catheterization Data (Case 1)

	Pressures in mm. Hg
Right atrium, mean	1
Right ventricle	24/0
Pulmonary artery	23/9
Left ventricle	170/10
Aorta	150/80
Brachial artery	145/80

Upstroke time : 0.23 sec.

Ejection time : 0.36 sec.

First derivative : 480 mm. Hg/sec.

Fig. 1. A pull through from the left ventricle to the aorta demonstrates a gradient of 20 mm.Hg.

Fig. 2. A brachial arterial pressure tracing in the same patient shows an upstroke time of 0.23 sec. and an ejection time of 0.36 sec. The lower curve is the first derivative which is 480 mm.Hg/sec. (The lines are 0.04 sec. apart).

into the left brachial artery. In order to insure high fidelity tracings, the needle was directly connected to a Satham pressure transducer P23Db. The first derivative of the brachial artery pressure could then be computed by a R-C differentiating circuit. This system provides a uniform frequency response to 40 cycles per second. The right heart was reached via the right antecubital vein, and the left heart via a right brachiotomy. The results of the cardiac catheterization are outlined in Table I. Note a minimal left ventricular-aortic gradient of 20 mm. Hg (Fig. 1). The brachial artery tracing is seen in Fig. 2.

Case 2: A 50-year-old white male was admitted to the Bronx Veterans Administration Hospital for the first time with the chief complaint of shortness of breath which began 3 weeks prior to admission. There was no past history of rheumatic fever.

On admission, the patient was dyspneic. Vital signs were recorded as follows: Pulse was 104/min., blood pressure was 98/50, and the temperature was 98.6. The pertinent physical findings were as follows. There was mild venous neck distention at 45°. The point of maximum intensity of the heart was diffuse between the fifth and sixth intercostal spaces at the anterior axillary line. There was a grade III/VI ejection murmur at the base. Both a presystolic and protodiastolic gallop were heard. The rhythm was sinus at 104/min. There were a few fine rales at both bases posteriorly. The liver edge was palpable 2 cm. below the right costal margin. There was trace edema of both lower extremities. The electrocardiogram revealed sinus rhythm and left bundle branch block. On fluoroscopy, a markedly calcified aortic valve was noted. A chest X-ray revealed cardiomegaly with hilar congestive changes. Laboratory data revealed normal CBC, urinalysis, BUN, FBS, and serum cholesterol.

Table II. Cardiac Catheterization Data (Case 2)

	Pressures in mm. Hg
Right atrium, mean	2
Right ventricle	25/2
Pulmonary artery	25/12
Left ventricle	140/7
Aorta	125/60
Brachial artery	110/55

Upstroke time : 0.22 sec.

Ejection time : 0.32 sec.

First derivative : 350 mm. Hg/sec.

Fig. 3. A brachial arterial pressure recording in the second patient shows an upstroke time of 0.22 sec. and an ejection time of 0.32 sec.

Fig. 4. A pull through from the left ventricle to the aorta demonstrates, in the second patient, a gradient of 15 mm.Hg.

The patient underwent right and left heart catheterization. A summary of values is found in Table II. Note the left ventricular-aortic systolic gradient of 15 mm. Hg (Fig. 3). The brachial artery tracing is seen in Fig. 4.

DISCUSSION

In aortic valvular disease there is an obstruction to the flow of blood from the left ventricle into the aorta. The left ventricular pressure increases as a consequence of the reduction in the aortic valve orifice, thereby producing an increase in left ventricular work. Hemodynamically, this results in a prolongation of the systolic ejection time with the establishment of a systolic pressure gradient across the aortic valve.

Clinical investigators have demonstrated that: 1. The direct brachial arterial pressure-pulse, by measuring the ejection time, upstroke velocity, and upstroke time, provides a fairly accurate measure in the determination of the degree of severity of aortic valvular disease.¹⁾ 2. Calcified valves are always the rule when an 80 mm. gradient or more exists in aortic valvular disease; however, calcified valves have also been reported in those instances where the pressure gradient was below 80 mm.Hg. Rockoff and Austen²⁾ report calcification of the aortic valves with peak aortic systolic gradients of 31–80 mm.Hg. Hence, calcification may be present with minimal aortic stenosis.

Our first patient presented as a typical case of valvular aortic stenosis with a history of exertional dyspnea, paroxysmal nocturnal dyspnea, and chest pain. Clinically, a grade III/VI systolic ejection murmur was heard at the base with radiation to the neck. On fluoroscopy, a calcified aortic valve was noted.

The brachial arterial tracing in itself was characteristic of severe aortic stenosis with an upstroke time of 0.23 sec. (normal 0.06–0.11 sec.),³⁾ an ejection time of 0.36 sec. (normal 0.28–0.34 sec.)¹⁾ and a first derivative of 480 mm.Hg/sec. (normal 811 ± 185 mm.Hg/sec.).⁴⁾

These findings, therefore, indicated a moderately severe stenosis of the aortic valve. The striking feature, however, was the pull-through gradient of 20 mm.Hg.

In the second case, a history of dyspnea and chest pain was obtained. Clinically, a systolic ejection murmur was auscultated, and on fluoroscopy, a markedly calcified aortic valve was noted.

The ancillary diagnostic measures widely employed to evaluate aortic stenosis were consistent with severe aortic stenosis and were as follows: an upstroke time of 0.22 sec., an ejection time of 0.32 sec., and a first derivative of 350 mm.Hg/sec. A left ventricular-aortic gradient of 15 mm.Hg was noted.

The analysis and correlation of the central and brachial arterial pulse

pressures have been reviewed by many investigators. They have found that there is a definite resemblance between the two and have, therefore, considered the brachial artery pulse study a normal diagnostic procedure in the evaluation of valvular disease. Many authors consider the upstroke time, systolic ejection time, and the brachial artery upstroke velocity important in determining the severity of aortic stenosis. They suggest that careful evaluation of these values may, in some instances, render cardiac catheterization unnecessary in the final determination of the severity of aortic valve disease.¹⁾ Delman and associates¹⁾ in their series concluded that valvular aortic stenosis could be identified on the basis of the upstroke time, upstroke velocity and the systolic ejection time. They also stated that those patients with significant obstruction could be distinguished from those without significant disease. We, however, have found that while the brachial arterial pulse pressure and its corresponding systolic ejection time, upstroke time and upstroke velocity are useful adjuncts in the evaluation of aortic stenosis, they must be complemented with cardiac catheterization to obtain a true measure of the severity of aortic valve lesions. The reason for this is that in some instances, highly abnormal brachial arterial upstroke time, upstroke velocity, and systolic ejection time determinations are found in the presence of only minimal pressure gradients on cardiac catheterization. Peripheral pulse contours in our patients revealed very abnormal findings which might have been indicative of severe aortic stenosis had they been interpreted alone. However, cardiac catheterization in both patients revealed minimal aortic stenosis.

Our findings are complemented by other case histories recently reported by Reeve.⁵⁾ He cited 3 cases in which the upstroke velocity, upstroke time, and systolic ejection time determinations were within normal limits in the presence of severe calcific valvular aortic stenosis. This was proven at operation in 2 cases and on autopsy in the third case.

In addition, both our patients presented with moderately calcified aortic valves on fluoroscopy. This clinical finding alone might correlate with the abnormal brachial artery findings, thus erroneously indicating a severe degree of aortic stenosis. Rockoff and Austin²⁾ correlated chest films of 50 patients with aortic stenosis with cardiac catheterization data and found that all patients who had radiologically uncalcified aortic valves had peak aortic systolic gradients of 85 mm.Hg or less. All females who had gradients greater than 80 mm. Hg had radiologically calcified valves. This was not found to be the case in the male representatives of the study, in which calcified valves were found with a wide range of aortic gradients.

Our cases serve to illustrate the importance of cardiac catheterization as the sole determinant in the final evaluation of aortic valvular disease. These

findings also emphasize the fact that hazardous conclusions may arise from the sole determination of the degree of aortic valvular stenosis by analysis of the arterial pressure pulse.

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