Aneurysm of Sinus of Valsalva*

Cause of Dynamic Coronary Constriction after Aortic Valvular Replacement and Bacterial Endocarditis

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A patient who had endocarditis on a prosthetic aortic valve and who had undergone two aortic valvular replacements developed classic angina pectoris. Cardiac catheterization revealed an aneurysm of the left sinus of Valsalva, which constricted a proximal segment of the left circumflex coronary artery during systole. This type of dynamic coronary arterial narrowing has not been previously described secondary to an aneurysm of a sinus of Valsalva and may be responsible for this patient's manifestations of ischemia.

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Figure 1. Unretouched cine frame from late diastolic aortogram (left anterior oblique projection). Catheter is in right side of heart and pulmonary artery. Note aneurysm of left sinus of Valsalva (black arrowheads) adjacent to main left and proximal circumflex coronary arteries (open arrow).

The diagnosis of an aneurysm of a sinus of Valsalva is made infrequently during life, since prior to rupture, these aneurysms rarely cause symptoms. Most cases are acquired and are secondary to infectious endocarditis, with formation of ring abscesses. Most of the remaining cases are apparently congenital, secondary to an absence of media in the aortic wall behind the sinus of Valsalva. The right sinus is most commonly affected, especially in the congenital type, and may cause obstruction of the right ventricular outflow tract.

Although cases of apparently fixed obstruction of the left main coronary artery or its branches by an aneurysm of a sinus of Valsalva have been reported, we are unaware of a report of dynamic coronary arterial narrowing secondary to such an aneurysm. This report describes the findings in a patient who developed typical angina pectoris 2½ years after aortic valvular replacement, which was necessitated by bacterial endocarditis. Cardiac catheterization demonstrated dynamic systolic constriction of a coronary artery by an aneurysm of the left sinus of Valsalva.
found. The findings from coronary arteriographic studies were normal, with a left dominant distribution.

The patient underwent aortic valvular replacement and one year later developed endocarditis due to Staphylococcus epidermidis. Clinical deterioration occurred, with the development of aortic insufficiency and congestive heart failure prompting a second aortic valvular replacement (Starr Edwards 2330). At surgery, valvular and endocardial vegetations were observed, with a friable ascending aorta. Antibiotic administration continued, and the patient recovered uneventfully.

The patient continued free of cardiovascular symptoms until the age of 40 years, when he developed exertional angina and dyspnea. These symptoms progressed rapidly, limited his life style, and prompted hospitalization. Examination showed blood pressure of 140/70/70 mm Hg and a pulse rate of 70 beats per minute and regular. Carotid upstroke was brisk, and the pulse volume appeared normal. The point of maximal impulse was not displaced but was sustained. The prosthetic valvular sounds were normal. A fourth heart sound was heard at rest, and both third and fourth heart sounds were present after exercise. A grade 3/6, early-peaking, systolic ejection murmur was heard best along the left sternal border and radiated well to the aortic area. A grade 2/6 aortic diastolic murmur was heard. The findings from the remainder of the examination were unremarkable.

A chest x-ray film, electrocardiogram, complete blood cell count, and the level of haptoglobin were unchanged. Exercise on a treadmill revealed ST-segment depression indicated by changes in the inferolateral leads at 150 beats per minute accompanying the patient's typical thoracic discomfort. Cardiac catheterization showed normal hemodynamic findings at rest; however, the catheter could be advanced to the left ventricle paravavularly through separations between the valve and aorta. Left ventricular, aortic, and selective coronary angiograms revealed normal motion of the ventricular wall, aortic insufficiency (2+) from several paravalvular regions, an aneurysm involving the ascending aorta and left sinus of Valsalva (Fig 1), and dynamic coronary arterial constriction producing a 90 percent reduction in the diameter of the circumflex coronary artery over a distance of approximately 10 mm (Fig 2). Surgical intervention to correct the aneurysm and paravalvular leak was recommended and is being considered by the patient.

**DISCUSSION**

This patient had an acquired aneurysm of the left sinus of Valsalva, which produced dynamic compression of the circumflex coronary artery. He also had angina and ischemic electrocardiographic changes with exercise, reflecting the distribution of the compressed coronary vessel. Previous endocarditis and two aortic valvular replacements presumably weakened the aortic wall. No abnormalities of the coronary arteries, aortic root, or sinuses of Valsalva were noted during the first catheterization or both thoracotomies. It is likely that the patient's manifestations of ischemia were related to dynamic constriction of the affected coronary artery.

Dynamic coronary arterial entrapment and constriction have recently been reported by Noble et al as the basis for symptoms and other more objective findings of myocardial ischemia. Myocardial muscle bridges create coronary arteriographic findings similar to those observed in our case. The ischemia is thought to be "due to entrapment" of the arterial segment within ventricular muscle and muscle shortening with systole. Patients with dynamic coronary arterial narrowing secondary to muscle bridges may have angina pectoris as the initial symptom, as in our patient. In our case the dynamic...
coronary arterial constriction seems to be secondary to external pressure from the aneurysm of the left sinus of Valsalva. During systole the greater distending pressure within the aneurysm could "trap" and constrict the circumflex coronary artery, while diastolic pressure (reduced because of aortic insufficiency) may not be sufficient to create arterial constriction.

During exercise, electrocardiographic changes and exertional angina suggest that the coronary constriction had physiologic significance. The mechanism for production of myocardial ischemia by systolic coronary arterial compression is not clear. Catecholamines, excitement, tachycardia, and exercise have been shown to shorten the diastolic filling period and also increase systolic coronary flow proportionately more than diastolic flow.\(^*\)\(^*\) We hypothesize that the potential for myocardial ischemia to develop in the distribution of the circumflex coronary artery could increase with exercise, possibly due to limitations of this systolic reserve mechanism. This unusual cause of dynamic coronary arterial constriction with manifestation of ischemia represents both a rare acquired anatomic defect and a rare cause of myocardial ischemia.

**REFERENCES**


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**An Electrophysiologic Study of Swallowing-Induced Tachycardia**

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Electrophysiologic studies were performed on a 73-year-old man with swallowing-induced supraventricular tachycardia, in order to define the characteristics of this unique dysrhythmia in this patient. Swallowing reliably provoked an automatic atrial focus type of atrial tachycardia, which usually changed into an atrioventricular nodal reentrant tachycardia when a critical delay in atrioventricular nodal conduction (atrio-His interval \( \geq 340 \) msec) was achieved. The atrioventricular nodal reentrant form of tachycardia did not occur spontaneously. The ease of induction and the duration of the episodes of supraventricular tachycardia were facilitated with the intravenous administration of atropine and ouabain and were decreased with administration of procainamide hydrochloride.

Only eight case reports\(^1\)\(^8\) of deglutition-induced tachycardia have appeared since Sakai and Mori\(^9\) first described the entity in 1926. The proposed theoretic mechanisms range from a vagovagal reflex arc to mechanical stimulation of the left atrium by a transiently distended esophagus. An electrophysiologic study (with pharmacologic manipulations) performed on a patient with deglutition-induced tachycardia forms the basis of this report.

**CASE REPORT**

A 73-year-old man described a 30-year history of infrequent palpitations, which were documented to be secondary to paroxysms of supraventricular tachycardia and were generally well controlled with therapy with quinidine sulfate. More recently, the tachyarrhythmia was precipitated only by swallowing solids or liquids. The therapy with quinidine sulfate was changed to oral administration of propranolol (40 mg every six hours), with little or no improvement. The cardiac physical findings were a nonejection midystolic click and a grade 2/6 mid to late systolic apical murmur.

On admission, the chest x-ray film, electrocardiogram, and echocardiogram were within normal limits. Continuous 24-hour ambulatory electrocardiographic monitoring performed prior to admission demonstrated premature atrial contractions with varying coupling intervals between the P wave and the premature atrial contractions and paroxysms of supraventricular tachycardia consistently initiated by swallowing solids or liquids. The results of cardiac fluoroscopic studies with a barium esophagram were normal. The patient noticed a short period of palpitations five seconds after the

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