Hinnen, Kremkau, Rahimtoola

Physical examination revealed a regular pulse at 72 beats/min and blood pressure of 150/90 mm Hg. The jugular venous pulse was normal in wave contour and pressure; the carotids were equal with a normal upstroke. The lungs were clear to percussion and auscultation. Cardiac examination revealed the apex impulse to be in the 5th left intercostal space, 1 cm to the left of the mid-clavicular line. The first and second heart sounds were normal. An S4 was present at the lower sternal border (LSB) and an intermittent S4 was present at the apex. A grade 1/6 systolic ejection murmur was present at the LSB. An electrocardiogram showed normal sinus rhythm with evidence of an anteroseptal scar. ST depression and T wave inversions were present in the inferior and lateral precordial leads.

Right and left heart catheterization, selective coronary cineangiography (Sones technique), and left ventriculography were performed with the patient unsedated and in room air. A temporary transvenous pacemaker was placed in the right ventricle. There was no chest discomfort during the coronary angiography, but after the ventriculogram, the patient developed angina which was relieved with two nitroglycerin tablets. Selective coronary arteriography demonstrated total obstruction of the main LCA 2 mm distal to the ostium and no filling of the LCA branches (Fig 1). The right coronary artery had several 50-75 percent proximal, mid and distal stenoses and supplied collateral arteries to the branches of the LCA via multiple septal vessels (Fig 2). The left ventriculogram demonstrated a large anteroapical aneurysm and paradoxical systolic expansion of the apex and akinesia of the anterolateral wall above the apex.

The cardiac catheterization and angiographic study were well tolerated. After the evening meal, some six hours after the procedure, the patient had a verbal disagreement with a member of his family. He developed severe angina, rapidly became pulseless and expired despite resuscitative efforts. Permission for necropsy was refused.

Discussion

Although complete occlusion of the RCA is frequently demonstrated during coronary angiography, acquired atherosclerotic occlusion of the main LCA with no distal filling of its branches is rarely seen.1-3 This is the first case of complete occlusion in the authors' experience with well over 1,000 cases of angiographically documented coronary atherosclerosis.

Although coronary angiography identifies most patients as right coronary dominant subjects, this dominance refers solely to an angiographic pattern. The dominant coronary artery angiographically is the vessel that crosses the posterior intersection of the atrioventricular and interventricular grooves.4 Although the RCA crosses this point in over 90 percent of subjects, the LCA is the more important coronary artery and supplies most of the left ventricular myocardium and the interventricular septum. It is generally believed that complete occlusion of the LCA is rarely seen angiographically because complete occlusion of this vessel is usually fatal. Indeed, our patient had a cardiac arrest requiring resuscitation during an ambulance trip to the hospital one year prior to his catheterization.

The prognostic significance and surgical treatment of main LCA obstructive lesions have recently been reported by Cohen et al.2 Levine et al.3 Complete occlusion of the main LCA is associated with a poor prognosis, and patients with this lesion should be considered for immediate, emergency coronary bypass graft surgery.

References


Misdiagnosis of Valvular Aortic Stenosis in Isorhythmic Dissociation*

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Failure to recognize isorhythmic dissociation and to record simultaneous ventricular and arterial pressure during cardiac catheterization led to the erroneous diagnosis of

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A 70-year-old man was referred to a cardiologist in 1970 for evaluation of exertional chest pain, dyspnea, and syncope. He had a grade 3/6 systolic ejection murmur which radiated to the carotid arteries. Electrocardiogram showed left ventricular hypertrophy. At cardiac catheterization, aortic systolic pressure recorded during catheter pullback was 45 mm Hg less than left ventricular pressure (Fig 1). Moderate aortic stenosis was diagnosed. Symptomatic bradycardia occurred during that hospitalization and a permanent demand pacemaker was inserted.

Following the onset of transient ischemic attacks and the recurrence of syncope, he was referred to the University of Oregon Medical School for aortic valve replacement. Electrocardiogram showed normal pacemaker function. At cardiac catheterization, simultaneous pressure measurements from left ventricle and brachial artery showed no systolic gradient. Pressures recorded during catheter withdrawal from the left ventricle to the aorta confirmed the absence of an aortic gradient. Rhythmic variation of ventricular and arterial systolic pressures characteristic of isorhythmic dissociation was present (Fig 2). When p waves preceded ventricular pacemaker beats (atrioventricular [A-V] synchrony) left ventricular end-diastolic pressure was 13 mm Hg and LV and brachial artery (BA) systolic pressures were 152 mm Hg. When atrial depolarization coincided with or followed pacemaker discharge (AV asynchrony), LV end-diastolic pressure was 7 mm Hg and LV and BA systolic pressures were 100 mm Hg. During infusion of isoproterenol the PR interval stabilized and recorded pressures were more characteristic of AV synchrony with diminished phasic variation. Review of catheterization data from 1970 revealed that unrecognized isorhythmic dissociation existed prior to pacemaker implantation. The apparent aortic valve pressure gradient of 45 mm Hg resulted from the presence of A-V synchrony during ventricular pressure recording and from A-V asynchrony during recording of aortic pressure (Fig 1).

**DISCUSSION**

Levy and co-workers have shown that a baroreceptor initiated feedback mechanism is operative in isorhythmic dissociation. Elevated systolic pressure is the trigger of...
baroreceptor discharge which produces sinoatrial slowing. As the p-p interval lengthens, the P wave merges with the QRS and AV synchrony disappears. The resulting decrease in systolic pressure inhibits baroreceptor discharge. Consequently, sinoatrial frequency increases and A-V synchrony reappears. A pattern of phasic systolic pressure variation is established.\(^1\)\(^2\) In addition to baroreceptor reflexes, variations of atrial stretch\(^3\) and distension of the sinus node artery\(^4\) may alter the activity of the sinus node.

A properly timed atrial systole contributes significantly to ventricular filling and ventricular performance on the basis of the Frank-Starling principle.\(^5\) This effect of A-V synchronization has been suggested as the mechanism of fluctuations in arterial pressure with changes in PR interval in isorhythmic dissociation.\(^1\)\(^2\) Our patient demonstrated that elevation of LV end-diastolic pressure, and presumably of LV end-diastolic volume does, in fact, occur in association with proper timing of atrial systole (Fig 2). Ventricular filling is, therefore, determined on a beat-to-beat basis and is an integral part of the feedback control of isorhythmic dissociation.

The coincidence of isorhythmic dissociation and transient ischemic attacks in this case generates speculation that arterial pressure fluctuations in this rhythm disorder may critically diminish perfusion in regional vascular beds and thus produce ischemic symptoms. Reduced perfusion pressure during anesthesia, antihypertensive therapy, and arrhythmia is known to produce cerebral ischemia in regions supplied by severely atherosclerotic vessels.\(^6\)

With increasing use of drugs which alter atrioventricular conduction and more frequent use of ventricular pacemakers, isorhythmic dissociation will become more common. Misdiagnosis of valvular aortic stenosis was the potentially serious consequence of failure to recognize this rhythm disturbance. We recommend inspection of the pressure record for phasic pressure and PR interval variations and simultaneous recording of left ventricular and arterial pressure to avoid this error. Furthermore, isoproterenol infusion may stabilize the PR interval and abolish phasic pressure variations in isorhythmic dissociation.

### References


### Announcements

**Postgraduate Course: Pulmonary Radiology**

The Department of Radiology of Emory University School of Medicine will present a two-day postgraduate course on Pulmonary Radiology, November 2-3, at Grady Memorial Hospital, Atlanta. For details, contact Dr. H. S. Weens, Department of Radiology, 80 Butler Street SE, Atlanta 30303.

**Second National Conference on Effectiveness of On-line Biomedical Computing**

The Second National Conference, sponsored by the Association for the Advancement of Medical Instrumentation, will be held November 29 and 30 at the Ramada Inn, Rosslyn, Virginia (D.C. area). The conference will provide another opportunity for physicians, engineers and other users and industry to survey and evaluate present accomplishments and to explore future effective uses of on-line biomedical computing in major areas of medical use, including multiphasic health testing, general patient care, nuclear medicine, laboratories and monitoring. For information, contact Joy Skillin, AAMI, 1500 Wilson Blvd, Arlington, Virginia 22209.