Anesthesiology
63:712–715, 1985

Use of a Pacing Catheter to Control Heart Rate in a Patient with Aortic Insufficiency and Coronary Artery Disease

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At least one large study described a high incidence of successful atrial (89%), ventricular (94%), and sequential (88%) pacing using flow-directed pulmonary artery pacing catheters.[1] The groups of patients who might significantly benefit from this ability to be paced are not clearly defined. One common indication is preexisting left bundle branch block because of the potential for sustaining complete heart block during pulmonary artery catheter insertion.[2] A second indication may be patients in whom precise control of heart rate is desirable. Individuals with combined aortic insufficiency (AI) and coronary artery disease (CAD) may be one such patient group. The following case report describes the use of a pacing catheter in such a patient. It also documents progressive PR interval prolongation and transient second degree atrioventricular (AV) block occurring during fentanyl administration in a patient with preexisting first-degree AV block.

REPORT OF A CASE

A 71-year-old man who had previously undergone aortocoronary bypass grafting to the left anterior descending and right coronary arteries in 1975 was scheduled for an aortic valve replacement. Symptoms of angina recurred in 1977, but a repeat cardiac catheterization revealed both grafts to be patent and a lack of new coronary lesions amenable to surgery. A calcified aortic valve was observed at this time, but there was no significant valve gradient and minimal valvular insufficiency. Four years later the patient was rehospitalized with congestive heart failure. This initially responded well to medical therapy, but subsequent worsening symptoms of dyspnea on minimal exertion prompted reevaluation in March of 1983. Medications at this time included digoxin 0.125 mg per day, isosorbide dinitrate 50 mg qid, furosemide 40 mg per day, and quinidine 300 mg qid.

Other medical problems included an ill-defined history of chronic obstructive pulmonary disease, for which he was being treated with aminophylline 100 mg tid, and prior left femoral–popliteal bypass grafting for claudication in 1975. Noninvasive tests also suggested significant bilateral internal carotid artery stenosis, greater on the right than on the left, with an estimated internal diameter of 1.5 mm on the right.

Repeat cardiac catheterization again revealed two patent bypass grafts with significant distal disease in all native vessels (not felt to be amenable to further bypass grafting). Aortic root angiogram, however, now showed 3+/4 aortic regurgitation and moderate aortic stenosis with a peak gradient of 60 mmHg. (estimated valve area of 0.4 cm^2). Pulmonary artery pressures at rest were 88/36 mmHg with a pulmonary capillary wedge pressure of 32 mmHg. Cardiac output was 2.6 l/min, giving a cardiac index of 1.4 $l \cdot min^{-1} \cdot m^{-2}$ and a net forward stroke volume index of 20 ml.

Electrocardiogram demonstrated first-degree atrioventricular block with a PR interval of 0.24 s, occasional blocked premature atrial contractions, old anterolateral myocardial infarction, left atrial enlargement, and nonspecific ST-T wave changes consistent with ischemia or digitalis effect. Preoperative potassium level was 4.6 mEq/l, hematocrit 36%, and creatinine 1.5 mg/100 ml.

He was premedicated with morphine sulfate 4 mg and scopolamine 0.3 mg im about 1 h before his expected time of arrival in the operating room. A large-gauge peripheral iv line, right radial arterial catheter, and right external jugular central venous pressure catheter were inserted without difficulty. A Swan-Ganz TD pacing catheter was passed via the right internal jugular vein; once in position, atrial capture, ventricular capture, and correct "wedging" were all confirmed. Baseline hemodynamics were as follows: blood pressure (BP) 170/70 (mean 105) mmHg, pulmonary artery pressure (PAP) 85/30 (mean 50) mmHg; mean pulmonary capillary wedge pressure (PCWP) 32 mmHg; mean right atrial pressure (RAP) 17 mmHg; and cardiac output (CO) 3.3 l/min. Cardiac rhythm was normal sinus at a rate of 75 beats/min and a PR interval of 0.24 s (first panel, fig. 1).

Induction proceeded as follows: atrial pacing was begun at a rate of 80 beats/min (atrial pacing spike [Aspike] to R wave interval of 0.32 s) and an initial dose of 50 μg of fentanyl and 4 mg of metocurarine were administered iv while the patient was breathing 100% O_2 via a mask (second panel, fig. 1). When no significant hemodynamic changes resulted, slow administration of fentanyl and metocurarine were continued. After 750 μg of fentanyl had been administered iv, BP and PA diastolic pressure were stable at 130/60 mmHg and 25 mmHg, respectively, but the Aspike-R interval had gradually lengthened to 0.44 s. As shown in figure 1, this progressed to second-degree block with 2:1 AV conduction after about 1.5 mg of fentanyl, at which time AV pacing was begun. Comparison of hemodynamics with AV pacing, V pacing, and A pacing (2:1 block) are seen in figure 2. With AV pacing the BP was 135/65 mmHg, PAP 74/32 mmHg, mean PCWP 24 mmHg, and thermodilution CO 2.8 l/min. Without pacing the sinus rate was 35 beats/min (conducted 1:1 with a PR interval of 0.28 s), BP 115/40 mmHg, and PAP 65/25 mmHg. After 3.75 mg of fentanyl iv (50 μg/kg body weight) and endotracheal intubation, hemodynamic variables with AV pacing (rate of 80 beats/min throughout) were BP 135/65 (mean 85) mmHg, PAP 70/30 (mean 45) mmHg, mean PCWP 32 mmHg, and CO 3.5 l/min. Atrial pacing with 1:1 ventricular conduction was again present with an Aspike-R interval of 0.38 s (no significant change in hemodynamics between A pacing and AV pacing). Without pacing the patient's underlying rhythm was atrial bigeminy at a rate of 40-75-40-75 beats/min, with a PR interval of 0.24 s; blood

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Received from the Department of Anesthesiology, Massachusetts General Hospital, Boston, Massachusetts 02114. Accepted for publication July 31, 1985.

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Key words: Equipment: pacing catheter. Heart: block, bradycardia; pacemakers, artificial.

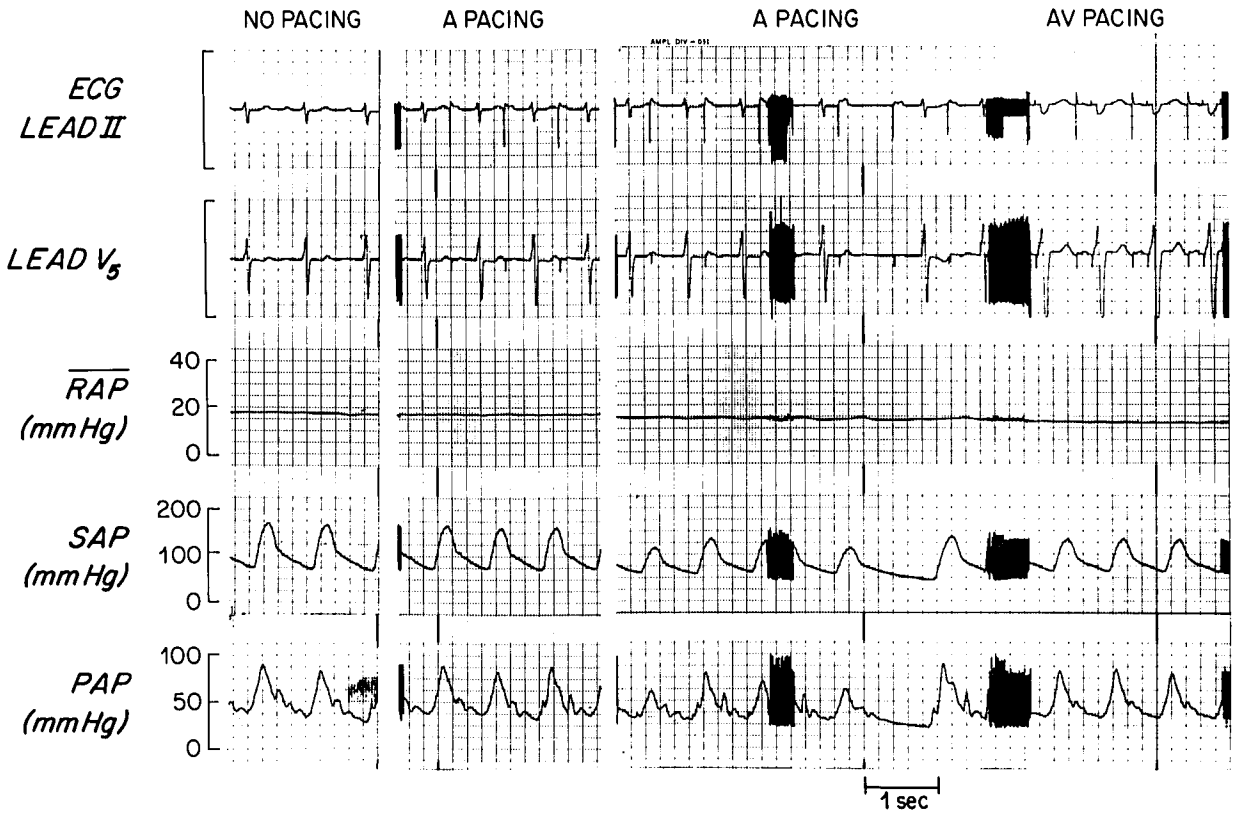

FIG. 1. Progression of AV block with fentanyl administration. *Panel 1:* Baseline hemodynamics with first degree AV block (PR interval 0.24 s). *Panel 2:* Atrial pacing prior to fentanyl administration. *Panel 3:* First section shows prolonged atrial spike to R wave interval following administration of 750 μg of fentanyl iv. Second section shows second degree AV block after 1.5 mg of fentanyl iv. Third section illustrates the effects of sequential AV pacing. RAP = right atrial pressure, SAP = systemic arterial pressure; PAP = pulmonary artery pressure.

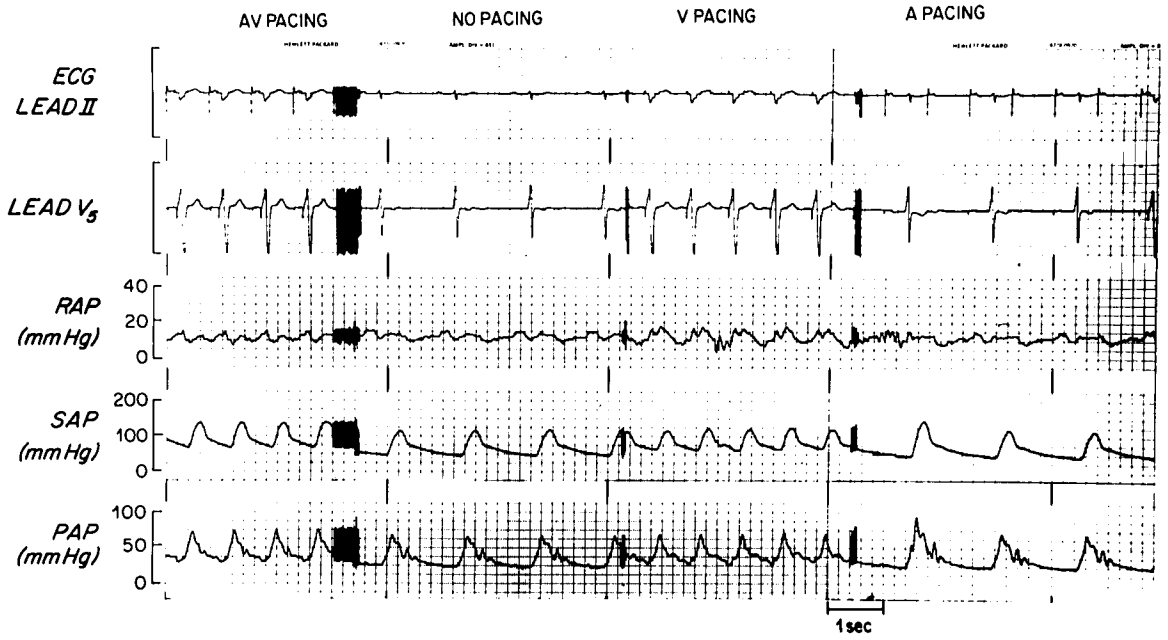

FIG. 2. Comparisons of hemodynamics with different types of pacing. Although 1:1 AV conduction was present at the slowed sinus rate of 35 (PR interval 0.28 s), 2:1 AV block was present at an atrial pacing rate of 80 beats/min. Note improvement in hemodynamics with AV pacing *versus* V pacing.

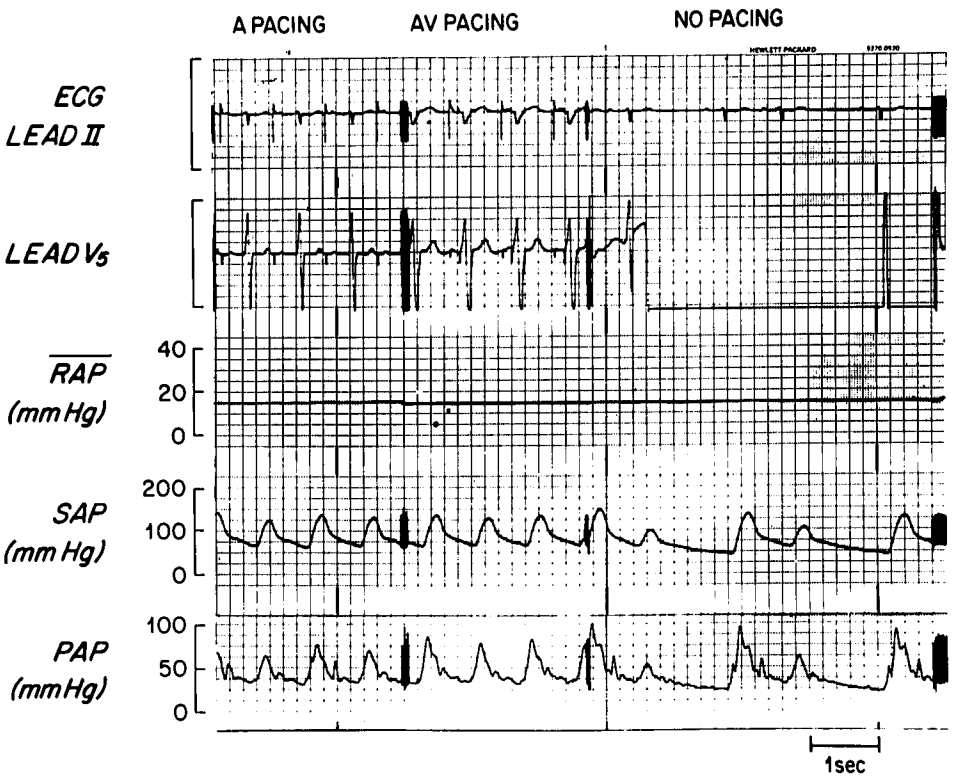

FIG. 3. Hemodynamics following endotracheal intubation. Note that 1:1 AV conduction is again present at a paced rate of 80 beats/min. The underlying rhythm without pacing is atrial bigeminy, conducted with a PR interval of 0.24 s.

pressures generated by this sequence were 135/60 mmHg, 105/40 mmHg, 135/60 mmHg (fig. 3). With continuing sinus node dysfunction, atrial pacing at a rate of 80 beats/min was continued up until bypass with excellent hemodynamic stability.

DISCUSSION

The beneficial effects of moderate (75–85 bpm)[3–5] to fast (>100 beats/min)[5,6] heart rates on the hemodynamics of patients with AI have been demonstrated previously to include consistent, significant decreases in left ventricular diastolic pressures, systolic volumes, and wall stress[3,4] and elevation in systemic arterial diastolic pressure.[4] Effects on forward cardiac output and regurgitant volume per minute are somewhat more variable. As heart rate increases, regurgitant volume per beat decreases, but total regurgitant volume per minute may change only slightly. Forward cardiac output has been variably reported to decrease (infrequent),[4,5] remain unchanged,[3] or increase.[4–6]In general, cardiac output appears to significantly increase when going from bradycardia to rates of 75–85 beats/min, change less predictably as rate increases from 85 to 110 beats/min (trend towards a small increase), and change little or decrease with rates in excess of 110–120 beats/min. A high degree of patient variability in response to rates in the range of 75–110 beats/min makes prediction of changes in forward cardiac output in any individual patient difficult.

The detrimental effects of AI on myocardial oxygen supply–demand balance are related to decreased systemic diastolic pressure, increased diastolic ventricular pressures, and increased systolic volumes, wall tension and stroke work.[7] Laboratory experiments suggest decreased coronary blood flow reserve (as measured by reactive hyperemia) in dogs with controlled AI and coronary narrowing.[8] Measurements during catheterization in humans have shown a reduced myocardial supply–demand ratio (as measured by the DPTI/SPTI index) related to the severity of regurgitation.[9]

The effect of rate on myocardial oxygen balance in the presence of AI and concomitant CAD has not been systemically evaluated. Such effects may be analyzed conceptually in terms of resultant changes in the determinants of oxygen demand and oxygen supply on a *per-beat* basis. A relative bradycardia (heart rates of 55–65 bpm) usually is considered beneficial in patients with CAD due to the relative increase in diastolic time for coronary perfusion (increased O_2 supply with lesser changes in per beat O_2 demand). Relative bradycardia may not be beneficial with concomitant AI, however, as the increase in diastolic time also results in increased per-beat regurgitant volume. This in turn leads to increased ventricular volumes, with significant increases in per-beat stroke work, wall tension, and O_2 demand. The beneficial effects of increased diastolic perfusion time are also partially offset by greater than normal decreases in coronary perfusion pressure (from both decreased systemic pressure as well as in-

creased intraventricular pressure). Important determinants in overall response to rate changes would be predicted to include severity of regurgitation, extent of coronary disease, and presence and severity of any combined aortic stenosis. Clinically, severe bradycardia may be tolerated poorly,[10–12] even in the absence of CAD. Although induced tachycardia was well tolerated in AI patients in one study, these were patients without concomitant CAD.[7]

Since the effects of rate on myocardial ischemia and cardiac performance were felt to be less predictable in this case than in CAD patients without AI, the anesthetic technique was designed to allow complete control of heart rate. In this manner, if ischemia and/or hemodynamic deterioration did occur, one would be able to precisely increase or decrease the rate in attempt to find this particular patient's "optimum rate." We administered a high dose of fentanyl to slow the sinus rate and avoided certain muscle relaxants and other drugs that would be likely to increase the sinus rate. We could then precisely control actual heart rate by cardiac pacing. In this patient we selected a rate of 80 beats/min before induction because this approximated his own sinus rate when he was comfortable and angina free. We maintained this rate with good hemodynamic results during the entire prebypass period. While some control of heart rate may be obtainable by the judicious use of other anesthetic regimens and/or chronotropic drugs (*e.g.*, pancuronium, fentanyl, gallamine, propranolol, or atropine), such pharmacologic means of rate control are less predictable (in both magnitude and duration of effect) and may have other detrimental side effects, including nodal dysythmias, myocardial depression, and pharmacologic "overshoot." These might be poorly tolerated in patients with combined AI and CAD.

PR interval prolongation and second degree AV blocks have not been described previously with the use of fentanyl. We speculate that in this patient the slowed conduction resulted from a fentanyl-induced change in autonomic nervous system "tone" (*i.e.*, increased vagal tone relative to sympathetic tone) acting upon the AV node.[13] Lengthening of the atrial pacing spike to R wave interval from 0.32 s to 0.44 s was noted following administration of 750 μg of fentanyl. This progressed to 2:1 AV block with further fentanyl administration. Although 1:1 AV conduction with a prolonged PR interval (0.28 s) was present at the slowed sinus rate of 35 beats/min, second-degree AV block was reproducibly present at an atrial pacing rate of 80 beats/min. This was a transient phenomenon (approximately 5 min duration), as the PR interval did subsequently shorten back to its baseline value of 0.24 s and 1:1 AV conduction returned. This patient did have first-degree AV heart block before induction of anesthesia, and patients with aortic valvulopathies are known to have a much higher than normal incidence of conduction disturbances.[14] In patients such as this with known conduction abnormalities, it would seem advisable to ensure ventricular (sequential) pacing capability before the use of the above described technique, employing high-dose fentanyl in conjunction with intentional cardiac pacing.

In most cardiac surgical patients, pacing capability is usually obtainable early during the procedure, as pacing wires may be directly applied to the heart as soon as the pericardium is open. In "redo" procedures,[16] however, such as the case described here, it may take some time to open up the chest because of the presence of adhesions. A pacing catheter makes pacing capability available from the beginning of anesthesia. Patients for "redo" procedures who have a history of AV block or hemodynamically significant bradycardia may thus constitute another group in whom pacing pulmonary artery catheters could offer significant benefit.

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