by at least 8 h. Symptoms responded well to aggressive potassium supplementation—KCl of up to 40 mEq/h—which was given because lesser infusion rates were ineffective in normalizing the plasma potassium concentration. It should be emphasized that hourly monitoring of plasma potassium concentration may be the only way to detect the disorder before the appearance of muscle weakness. Also, clinical observation of muscular function should be carried out throughout the postoperative period, since some forms of the disease may present paralysis without alteration of the plasma potassium concentration.[7]

In summary, we describe the successful anesthetic management of a patient with hypokalemic familial periodic paralysis undergoing coronary revascularization surgery with extracorporeal circulation. In the postoperative period, hypokalemia preceded by several hours the appearance of muscular weakness and was effectively controlled by supplementation with KCl.

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Ventricular Pacing Can Induce Hemodynamically Significant Mitral Valve Regurgitation

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Mitral valve regurgitation (MR) may develop or worsen suddenly during cardiac operations and result in severe arterial hypotension. Among the common causes are annular dilatation due to left ventricular distention and ischemic papillary muscle dysfunction.[1] The incident reported here highlights an unusual etiology not previously described in a clinical setting—ventricular-pacing-induced MR in the presence of normally timed end-diastolic atrial contraction. Recognition of this uncommon phenomenon is important, since discontinuation of ventricular pacing may produce prompt circulatory recovery.

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Case Report

A 68-yr-old, 58-kg man with a history of hypertension and valvular heart disease was scheduled for aortic valve replacement. Preoperative medications included captopril, digoxin, furosemide, and potassium. An electrocardiogram showed left ventricular hypertrophy, left anterior fascicular block, and right bundle branch block. Cardiac catheterization revealed severe aortic valve regurgitation, moderate MR, mean pulmonary artery wedge (PAW) pressure 15 mmHg (v wave 19 mmHg), cardiac output (CO) 4.5 $l \cdot min^{-1}$, and ejection fraction 59%. Aortic valve replacement was scheduled; MR was believed to be functional and not severe enough to require valvular replacement.

Prior to induction of anesthesia, hemodynamic variables showed blood pressure (BP) 122/56 mmHg, CO 3.5 $l \cdot min^{-1}$, and PAW 12 mmHg without discernible v waves. Anesthesia was induced and maintained with morphine, diazepam, isoflurane, and pancuronium. A 23-mm Hancock porcine aortic bioprosthesis was implanted during cardiopulmonary bypass, along with temporary right atrial epicardial and right ventricular epicardial pacing wires. Separation from bypass was facilitated with a dobutamine infusion and atrial pacing at 80 beats per min to treat sinus bradycardia. At the time, hemodynamic variables showed BP 92/53 mmHg, CO 4.2 $l \cdot min^{-1}$, and pulmonary artery pressure (PAP) 23/8 mmHg with a normal waveform (fig. 1A).

After 15 min of effective atrial pacing with a normal PR interval (200 ms), intermittent complete heart block developed. Atrioventricular (AV) sequential pacing at 80 beats min was instituted and the AV interval maintained at 200 ms. Despite effective pacemaker capture as

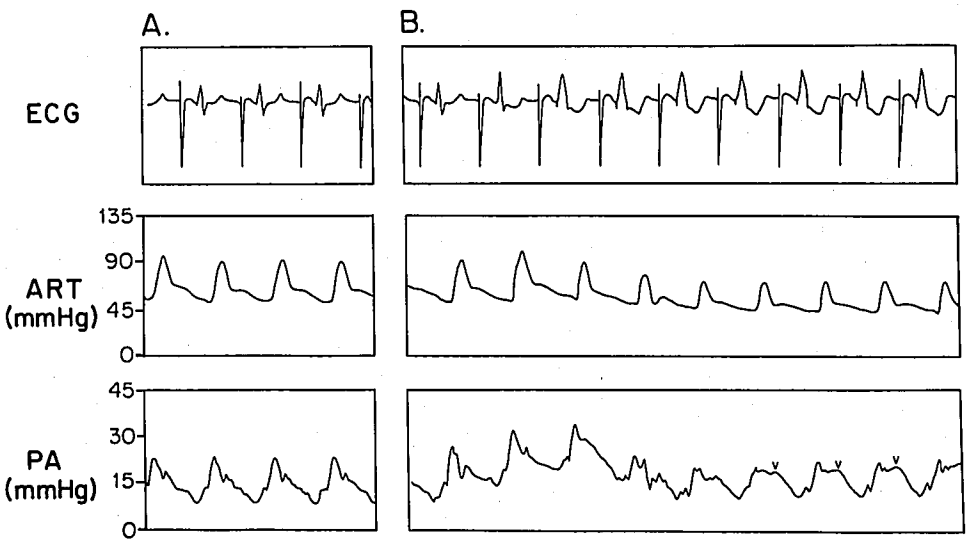

FIG. 1. (*A*) Atrial pacing after cardiopulmonary bypass produced an arterial blood pressure (ART) of 92/53 mmHg and a normal pulmonary artery (PA) pressure of 23/8 mmHg, with a normal PA wave contour. (*B*) Onset of atrioventricular sequential pacing with the third electrocardiographic (ECG) QRS complex shows a widened QRS and sudden arterial hypotension (70/45 mmHg), accompanied by an abnormal PA waveform displaying a V wave (v) of mitral regurgitation. (The atrial pacing spike is large in amplitude compared to the ventricular pacing spike that initiates the third and subsequent complexes.)

evidenced by direct cardiac inspection, arterial hypotension developed suddenly: BP became 70/45 mmHg and PAP 20/10 mmHg with a abnormal waveform (fig. 1B). Adjustment of the AV interval during sequential pacing between 120 and 300 ms did not influence the hypotension or abnormal PA waveform. Pacing was discontinued, and the hypotension immediately resolved; a slow sinus rhythm (60 beats per min) returned. Repeated attempts at AV sequential pacing consistently reproduced these hemodynamic alterations. Consequently, isoproterenol was infused intravenously as an alternative treatment of intermittent bradycardia and AV block over the next hour.

On postoperative day 1, a soft murmur of MR was heard, but hemodynamic consequences were inapparent: BP was 138/74 mmHg; PAP was 35/16 mmHg with a normal wave contour; and no v wave was visible in the PAW tracing. The patient was separated easily from mechanical ventilation, recovered uneventfully, and was discharged on postoperative day 7. An echocardiogram performed before hospital discharge showed slight calcification and thickening of the mitral valve leaflets with moderate MR and a normal appearance of the aortic valve prosthesis.

DISCUSSION

This case report demonstrates that ventricular pacing can induce hemodynamically significant MR and cause severe hypotension in the postbypass period. In this instance, despite maintenance of normal AV synchrony with pacing, the pathway for ventricular depolarization was a critical determinant of normal mitral valve function. Without any change in heart rate or loading conditions, the change from atrial to AV sequential pacing resulted in abrupt hypotension and an abnormal PA waveform, consistent with acute MR. This hemodynamic abnormality was terminated quickly when right ventricular epicardial pacing was discontinued.

In this situation, arterial hypotension occurred abruptly with the onset of AV sequential pacing; simultaneously, PA pressure changed from 23/8 to 20/10 mmHg. These PA pressure changes were minimal, but the PA waveform showed an abnormal contour (fig. 1B). The second discernible peak in the tracing was produced by a left atrial v wave of MR, transmitted in a retrograde manner through the pulmonary vasculature and superimposed upon the antegrade pressure wave (Fig. 2). Tracings of the PAW or left atrial pressures in the immediate postbypass period would have displayed the abnormal v wave, but tracings were not made in this period. Fortunately, the PA tracing was continuously monitored and helped to establish the diagnosis. Although v waves are neither highly sensitive nor specific for MR in other settings,[2] the immediate onset and resolution of these waves in the current case makes other causes far less likely. Transesophageal echocardiographic monitoring of mitral valve motion, contrast echocardiography, or Doppler measurement of transmitral flow patterns might have supplied additional confirmation.[3]

Acute arterial hypotension and an abnormal PA waveform in this clinical setting may have several causes. AV dissociation can induce an elevated left atrial pressure with

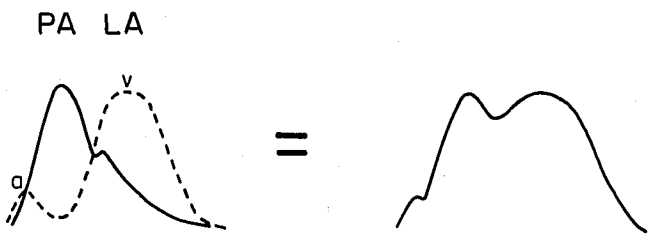

FIG. 2. The pulmonary artery waveform during mitral regurgitation shows the normal antegrade pulmonary artery (PA, solid line) pressure wave generated by right ventricular contraction and a superimposed pressure wave produced by the left atrium (LA, dashed line). These left atrial waves include a small end-diastolic A wave (a) and large regurgitant systolic V wave (v), which distort the normal pulmonary artery waveform and create the composite waveform shown on the right.

an abnormal, "cannon" A wave. Moreover, loss of properly timed atrial contraction at end-diastole can markedly reduce ventricular filling and cause as much as a 40% reduction in stroke volume. The resulting arterial hypotension may be particularly likely in a patient with left ventricular hypertrophy and decreased diastolic compliance.[4] However, AV sequential pacing with effective capture should have prevented this problem in our case. Another common cause of abrupt hypotension immediately after bypass is coronary air embolization and ischemic papillary muscle dysfunction. When the left-sided cardiac chambers are entered surgically, retained intracardiac air can form emboli and cause coronary obstruction. This is unlikely to have caused the events seen in the current case, since the diagnostic electrocardiographic ST elevations were absent.

If abnormal ventricular loading and myocardial ischemia are assumed to have been absent in the current case, how did AV sequential pacing induce acute MR? In the presence of high-grade AV block, sequential pacing is chosen to improve cardiac performance in the immediate postbypass period; atrial contribution to ventricular filling thereby is maintained. An investigation in patients with rheumatic heart disease has shown that CO during sequential pacing remains comparable to that during atrial pacing, and is 20% higher than during ventricular pacing.[5] That study confirmed that aberrant ventricular depolarization associated with sequential endocardial pacing causes little decrease in CO.

In contrast, the origin of ventricular depolarization in the current case was very important. The normal ventricular pathway associated with atrial pacing was not present; rather, AV sequential pacing caused ventricular depolarization to originate at an abnormal, right ventricular epicardial site. We speculate that this pattern of ventricular depolarization altered mitral valve competence, a clinically uncommon phenomenon not previously described in an intraoperative setting. Haas and Strait[6] described a hypertensive 71-yr-old patient in whom endocardial ventricular pacing produced angiographic evidence of MR during cardiac catheterization. However, the hypotension that arose was caused in part by loss of the atrial contribution to ventricular filling. Loss of this atrial contribution was not an issue in our patient. Experimentally, apical right ventricular endocardial pacing has produced significant MR in a dog model.[7] Ventricular pacing from the coronary sinus at the base of the left ventricle minimized the valvular insufficiency.

The mechanism for ventricular pacing-induced MR is unclear, but may relate to the timing of papillary muscle contraction relative to adjacent myocardial contraction or to altered regional myocardial contraction near the pacing site. Perhaps the patient in the current case was at increased risk for developing clinically important MR, since his mitral valve function remained slightly abnormal, as determined by postoperative echocardiographic examination. Furthermore, coexisting conduction pathway disease (left anterior fascicular block and right bundle branch block) may have provided another predisposing factor.

In summary, this report documents that acute MR and consequent arterial hypotension may result from ventricular pacing in the cardiac surgical patient. Moreover, hemodynamic deterioration can occur in the presence of normal AV synchrony. Although cessation of ventricular pacing was immediately therapeutic in this patient, a different site for ventricular pacing may have avoided this problem. If experimental observations in the dog[7] may be extrapolated to the clinical setting, placement of a ventricular pacing wire closer to the cardiac base may allow effective AV or ventricular pacing without the deleterious hemodynamic consequences observed in the current case. In this setting, continuous observation of the pulmonary arterial waveform revealed the regurgitant left atrial v wave and provided critical diagnostic information.

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