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Pulmonary Edema following Conversion of Tachyarrhythmia*
A Case following Burst Atrial Pacing
Thomas S. Goldbaum, M.D.; James M. Bacos, M.D.; and Joseph Lindsay Jr, M.D.

Restoration of normal sinus rhythm is usually followed by improved hemodynamics. By contrast, pulmonary edema and cardiovascular collapse have been reported following successful electrical reversion of various tachyarrhythmias to normal sinus rhythm. The mechanism for this adverse reaction is not clear but has been thought to relate, at least in part, to electrical myocardial damage from the counter-shock. This report describes a patient in whom this complication occurred on two occasions, first following external countershock and subsequently following burst atrial pacing. Thus, conversion to sinus rhythm may be responsible for this phenomenon independent of the method of conversion.

Although not recently a subject for review in the literature, pulmonary edema with and without cardiovascular collapse following restoration of sinus rhythm has previously been reported in 1 to 3 percent of patients following electrical reversion of atrial flutter, atrial fibrillation, and ventricular tachycardia. This report describes a patient in whom atrial flutter was treated once with cardioversion and subsequently with burst atrial pacing; each time conversion to sinus rhythm was accompanied by pulmonary edema and cardiovascular collapse. We are unaware of a previous report of this complication following overdrive atrial pacing, an observation which appears to exclude electrical injury to the myocardium as the only cause for this syndrome.

Case Report
A 62-year-old man underwent coronary artery bypass surgery in April 1984. Preoperatively, radionuclide ventriculography demonstrated a resting ejection fraction of 43 percent. One month following an unremarkable recovery from surgery, he developed atrial flutter with a ventricular rate of 150. After digitalization, he was cardioverted to sinus rhythm with a single shock of 20 joules. In

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conjunction, he received 15 mg of diazepam orally, 5 mg of morphine sulfate intravenously, and 400 mg of quinidine orally. Immediately after reversion, he became severely dyspneic and cyanotic. His blood pressure was unobtainable, and he had sinus bradycardia at rates between 50 to 60 beats per minute. He was treated intensively with diuretics and pressor agents. A pulmonary arteriogram performed two days after cardioversion failed to show emboli. The pulmonary artery pressure was 43/16 mm Hg, with a mean of 25 mm Hg. Following recovery, pulmonary function studies demonstrated mild obstructive disease. He was discharged receiving theophylline, digoxin, propranolol, and furosemide.

Seven months later, he consulted his physician because of substernal chest pain. Physical examination revealed his blood pressure to be 110/70 mm Hg, his respirations were unlabored at 12 per minute, and his pulse was between 130 and 150 beats per minute. He was an alert, well-developed man in no acute distress. His lungs were clear. Cardiovascular examination demonstrated an S1 of variable intensity, a physiologically split S2, and a grade 2/6 ejection systolic murmur. His ECG demonstrated atrial flutter with variable atrioventricular conduction. His chest roentgenogram showed normal pulmonary vasculature. Blood levels of theophylline and of digoxin were in the therapeutic range, and his electrolyte values were normal.

He was initially treated with intravenous verapamil which increased atrioventricular block, but conversion to sinus rhythm did not occur. Serial ECGs and cardiac enzyme determination failed to reveal evidence of myocardial infarction.

Because the patient had previously developed pulmonary edema following cardioversion, reversion by overdrive atrial pacing was elected two days after admission. Maintenance regimens of digoxin and procaainamide were continued but no other cardioactive drugs were given. After 10 mg of diazepam orally, a bipolar pacing catheter was positioned against the high right atrial wall. Burst atrial pacing converted his rhythm to atrial fibrillation which was followed in three to five minutes by normal sinus rhythm. The patient's blood pressure was 120/70 mm Hg and his heart rate was 70 beats per minute. Over the next 30 minutes, he became progressively short of breath and cyanotic. His blood pressure was unobtainable despite normal sinus rhythm at a rate between 80 and 90 beats per minute. Treatment with vasopressors and oxygen was initiated. The patient's physical examination was now significant for diffuse rales in both lung fields. The murmur was now louder and pansystolic. Poor respiratory effort and hypoxemia necessitated intubation. Blood gas measurement while he was receiving a 40 percent oxygen-air mixture disclosed a partial pressure of oxygen to be 51 mm Hg, the partial pressure of carbon dioxide to be 38 mm Hg, and the pH to be 7.30. His chest roentgenogram demonstrated marked pulmonary vascular congestion and an increase in cardiac size. Episodes of nonsustained ventricular tachycardia were treated with lidocaine, and marked hypotension was treated with dopamine and norepinephrine. As on the first occasion, no enzyme or electrocardiographic evidence of myocardial infarction was detected. The patient was discharged after a four-day recovery period in normal sinus rhythm while receiving digoxin and verapamil. At that time, radionuclide ventriculography demonstrated an ejection fraction of 38 percent.

**DISCUSSION**

Pulmonary edema following electrical reversion of cardiac arrhythmias has been the subject of several reports describing single patients or small series of two to four cases. The mechanism for this acute cardiac decompensation is not clear. Its occurrence in our case following reversion by atrial overdrive pacing, together with the example of pulmonary edema after spontaneous reversion cited by Hollman and Nicholson, provides strong evidence against the hypothesis that is frequently proposed mechanism, left ventricular depression from the electrical shock, as the only operative mechanism.

This hypothesis has previously been considered most plausible since left ventricular injury can be demonstrated after frequent or high energy shocks are delivered to the myocardium. Since virtually all episodes have occurred in patients with either left ventricular dysfunction or mitral valve disease, one can easily conceive that a relatively small deterioration in left ventricular function might produce acute decompensation. Oddly, this complication has been reported only rarely subsequent to unsuccessful reversion. This peculiarity has been the major argument against the hypothesis since large amounts of electrical energy are often employed in unsuccessful attempts.

The case herein reported forces consideration of other possibilities, several of which can be rejected as not being consistent with published observations. No consistent pattern of drug or anesthetic use or abuse has emerged, and neither coronary nor pulmonary emboli have been positively identified in either the four reported necropsies or on the pulmonary angiography of our patient.

The conversion to a sinus mechanism itself may be responsible for cardiovascular collapse. Failure of mechanical atrial systole to return simultaneously with restoration of sinus rhythm has been demonstrated by several investigators. Moreover, right atrial "a" waves can be recorded more frequently than can left atrial "a" waves following conversion to sinus rhythm. These observations suggest that mechanical right atrial systole following cardioversion may augment right ventricular output in the absence of left atrial mechanical systole. It has been suggested that in patients with a poorly compliant left ventricle or with mitral valve disease, left atrial pressure may rise sufficiently to produce pulmonary edema.

It should be recognized that bedside hemodynamic measurements have not been reported in any case of postcardioversion pulmonary edema. The various hypotheses regarding pathogenesis have presumed on clinical grounds that the process of reversion has somehow resulted in an increase in left atrial pressure and an attendant diminution in cardiac output. Direct lung injury from the electrical shock or a shower of small pulmonary emboli, not detected at necropsy or angiography, must still be considered possible.

It is noteworthy that in our case and in previous reports, the expected increase in heart rate in response to hypotension did not occur. Failure of this compensatory mechanism may have aggravated the low output state. Moreover, the possibility exists that this lack of an appropriate chronotropic response reflects a more widespread aberration in the cardiovascular reflex response to the changing rhythm and may underlie this syndrome.

Although the precise mechanism of pulmonary edema in this setting remains elusive, close monitoring of patients with left ventricular dysfunction or mitral valve disease postreversion is prudent. Early and intense therapy directed at optimizing oxygenation and cardiac output can be thus implemented if this potentially fatal complication is recognized.

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Pulmonary Edema after Tachysysthythmia (Goldbaum, Bacos, Lindsay)
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