Successful weaning from mechanical ventilation after coronary angioplasty

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Weaning failure can be caused by myocardial ischaemia during the switch from mechanical to spontaneous ventilation. We report ischaemic left ventricular failure and ischaemic mitral insufficiency during weaning. Angiography showed that the coronary vessels were stenosed. Transluminal angioplasty made weaning possible. We conclude that acute ischaemic mitral insufficiency may contribute to cardiac failure during weaning and that angioplasty, by reversing it, can allow successful weaning.


Keywords: complications, myocardial ischaemia; heart, left ventricular dysfunction; heart, mitral insufficiency; surgery, coronary angioplasty; ventilation, mechanical; ventilation, weaning

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Left ventricular dysfunction can cause failure to wean, and may result from myocardial ischaemia during the change from mechanical to spontaneous ventilation. We report ischaemic left ventricular failure during weaning involving both impaired contractility and mitral insufficiency caused by ischaemia. After transluminal angioplasty, weaning became possible.

Case report

A 71-yr-old woman was referred with septic shock related to pyelonephritis. Three months before, she had an episode of angina pectoris, which was successfully controlled with beta-blockers and calcium channel inhibitors. No coronary angiography was done. Echocardiography at that time showed mild mitral regurgitation associated with inferior hypokinesia, but otherwise normal global systolic function.

After admission, the patient was intubated and mechanically ventilated (Evita 2, Drägerwerk, Lübeck, Germany). Fluid administration failed to restore the arterial pressure, so an infusion of epinephrine (0.4 mg kg⁻¹ min⁻¹) was started. Appropriate antibiotics were given. The ECG showed signs of myocardial ischaemia and troponin Ic blood level was increased (35 ng ml⁻¹). Echocardiography was unchanged.

On the third day of treatment, the patient was afebrile and the circulation was stable so the epinephrine dose was reduced to 0.1 mg kg⁻¹ min⁻¹, and weaning from mechanical ventilation was started. The ventilator was switched from assist-control to a pressure support of 16 cm H₂O, and 5 cm H₂O positive end-expiratory pressure. On day 5, these values were unchanged, and the ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen (Pao₂/FIO₂) was 300 mm Hg. Right atrial pressure was 6 cm H₂O, the circulation was stable, systolic arterial pressure 110 mm Hg, epinephrine infusion at 0.1 mg kg⁻¹ min⁻¹, and troponin was decreasing (9.5 ng ml⁻¹). As the level of pressure support was progressively decreased from 16 to 10 cm H₂O (PEEP being constant), hypotension developed (systolic arterial pressure: 85 mm Hg) and the epinephrine dose had to be increased to 0.4 mg kg⁻¹ min⁻¹. The respiratory state worsened, as respiratory frequency increased from 24 to 36 bpm and Pao₂/FIO₂ ratio decreased from 300 to 150, so we re-started controlled ventilation. Clinical and radiographic signs of acute pulmonary oedema developed and right atrial pressure increased from 6 to 13 cm H₂O. Ischaemic changes were seen on the ECG (Fig. 1A), and troponin Ic increased to 13.6 ng ml⁻¹. These features suggested ischaemic cardiac failure. Echocardiography showed severe antero-lateral-basal hypokinesia and massive mitral regurgitation without left ventricular (LV) dilatation. At coronary angiography, three marked stenoses were shown on the left anterior descending vessel and one on the left circumflex (Fig. 2A). With the Cardiovascular Angiography Analysis System (CAAS), stenosis was greater than 70%, and dilation was recommended. Transluminal angioplasty was done and stents were inserted (four stents on the anterior descending and one on the circumflex; Fig. 2B).
ECG signs of ischaemia decreased (Fig. 1B), and enzyme values decreased (troponin Ic blood level: 8.4 ng ml$^{-1}$ on day 7). The dose of epinephrine was tapered to 0.3 mg kg$^{-1}$ min$^{-1}$ on day 6, and stopped on day 7. The ventilator was switched to pressure support with the pressure level reduced from 16 to 10 cm H$_2$O, with successful weaning and extubation. Later, echocardiography showed less myocardial hypokinesia and no mitral insufficiency.

**Discussion**

We report weaning failure induced by acute cardiac ischaemia, which was treated by emergency coronary angioplasty. When weaning was started the patient developed hypotension, tachypnoea, and acute pulmonary oedema. Cardiac ischaemia was shown by ECG changes, increased troponin levels, increased hypokinesia, and severe mitral regurgitation. These features developed when the pressure support was reduced, suggesting a close relationship between the two events.

Cardiac ischaemia results from an imbalance between oxygen supply and demand.$^1$ Oxygen supply is reduced by hypoxaemia, which is common during weaning, because of tachypnoea and worsening of ventilation-perfusion matching,$^2$ and can be made worse by pulmonary oedema. Myocardial oxygen delivery is also reduced by catecholamine-induced tachycardia, which limits the diastolic perfusion time. In addition, increased LV end-diastolic pressure may reduce diastolic sub-endocardial blood flow, and coronary narrowing could also limit coronary blood flow, reducing oxygen supply to the myocardium. An increase in cardiac afterload, caused by changing from positive pressure mechanical ventilation to negative pressure spontaneous breathing,$^3$ can also impair myocardial function. Mitral regurgitation has been described during weaning$^4$ and may have worsened the cardiac failure. The sequence of events suggests an ischaemic cause.

However, during weaning, pleural pressure decreases in inspiration, and diastolic transmyocardial pressure and systemic venous return will increase. This will increase the volume of the left ventricle and dilate the mitral valve ring. If the efficiency of apposition of the valve leaflets was impaired, this would contribute to the mitral regurgitation we observed.

Several studies have reported cardiac ischaemia during weaning, and greater risk of weaning failure (Table 1). Räsänen and colleagues$^5$ observed angina and electrocardiographic evidence of myocardial ischaemia in six of 12 patients during weaning from the ventilator after acute myocardial

![Fig 1](image1.png) Changes in leads V4 to V6 of the ECG before (A) and after (B) coronary angioplasty. Angioplasty is followed by ST segment depression.

![Fig 2](image2.png) Anteroposterior projection of the left coronary system. (A) Before angioplasty, shows marked stenoses of the left anterior descending (arrows). (B) A better coronary flow after transluminal angioplasty and stent insertion.
infarction. These changes were concomitant with increased pulmonary artery occlusion pressure. Lemaire and colleagues described patients with ischaemic heart disease who had scintigraphic evidence of abnormal left ventricular wall motion during mechanical ventilation (MV), which worsened during spontaneous ventilation. Treatment of the cardiac failure allowed weaning. Hurford and colleagues reported abnormal scintigraphic perfusion images suggesting ischaemia in five out of 15 patients. During weaning, ECG changes suggesting ischaemia were found in six of 17 ventilated patients and silent myocardial ischaemia, shown by S-T segment analysis, was noted in 9–14% of patients during weaning after non-cardiac surgery. Others have reported similar findings.

Treatment of weaning-induced ischaemic LV failure is described in two studies, with diuretics or changes of cardiac medication. Such treatments allowed weaning for most subjects. In our patient, the sequence of cardiac ischaemia, left ventricular hypokinesia, mitral regurgitation, acute pulmonary oedema, and weaning failure was reversed by emergency transluminal coronary angioplasty. After this, pressure support was reduced safely and weaning accomplished. The sepsis had already resolved, so the final successful weaning is not likely to have been because of resolving sepsis. The reversion of the ischaemic LV failure and the successful extubation following angioplasty suggest that coronary narrowing affected myocardial oxygen supply in our patient. Our report is similar in some ways to a case reported by Boussarsar and colleagues, in which weaning was possible after percutaneous balloon mitral commissurotomy for severe mitral stenosis.

We suggest that coronary angioplasty should be considered when cardiac ischaemia and left ventricular failure occur during weaning. Ischemic myocardial dysfunction during weaning may be associated with acute ischaemic mitral insufficiency and may contribute to weaning failure. If ischaemic heart failure can be demonstrated, then appropriate treatment can allow successful weaning and reduce the possibility of cardiac damage during weaning attempts.

### References