Successful use of biventricular pacing to facilitate weaning from mechanical ventilation in a patient with severe left ventricular failure

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Introduction

Biventricular pacing is increasingly used in the management of severe heart failure. We report the successful use of biventricular pacing to aid weaning from mechanical ventilation in a patient with severe left ventricular dysfunction.

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Case report

A 62-year-old man presented to the emergency department of his local hospital with ischaemic chest pain. He had a past medical history of ischaemic heart disease, including two myocardial infarctions, and had undergone two coronary angioplasty procedures with stent placement, one 10 years and the other three months previously. In addition, he had diabetes mellitus (treated with insulin), chronic renal impairment (his serum creatinine was 180 µmol/L), peripheral vascular disease and hypertension. He had stopped smoking three months previously and had a 190 pack-year history.

An electrocardiogram (ECG) showed chronic left bundle branch block. His troponin I level was elevated at > 50 µg/L. He developed pulmonary oedema and subsequently suffered a cardiac arrest. He was resuscitated after about five minutes of cardiopulmonary resuscitation (CPR), during which he underwent endotracheal intubation, and emergency coronary angiography was performed. Angiography demonstrated a alocked stent in the left anterior descending artery (LAD), moderate disease of the circumflex artery and an unobstructed right coronary artery. He subsequently underwent disobliteration of the LAD artery, with insertion of three further stents. Post procedure, he was admitted to the intensive care unit. Transthoracic echocardiography demonstrated poor left ventricular function with anterior and inferior wall motion abnormalities. The following day he was extubated but developed pulmonary oedema within 30 minutes

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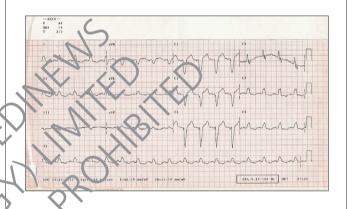
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Figure 1. Electrocardiogram of the patient prior to insertion of a biventricular pacing system



and had to be re-intubated and mechanically ventilated. He was transferred to the intensive care unit of this hospital for further management.

On arrival he was in sinus rhythm, heart rate 90 beats per minute; mean arterial pressure was 70 mmHg supported with adrenaline infusion (0.2 µg/kg/min). He had cool peripheries, with no pulses palpable below the femoral arteries. The central venous pressure was 13 mmHg, cardiac index (LidCO, LidCO Ltd, Cambridge, UK) was 1.9 L/min/m² and systemic vascular resistance 1500 dyne.cm.sec⁻⁵. He was being mechanically ventilated with inspired oxygen of 50%, resulting in PaO₂ of 17.0 kPa. Chest X-ray showed cardiomegaly and pulmonary oedema. Examination of the abdomen was unremarkable. He was oliguric and had a metabolic acidosis (base excess -8 mmol/L). An ECG (figure 1) showed left bundle branch block (PR interval 142 ms, QRS duration 147 ms). Transthoracic echocardiography showed severe left ventricular impairment with anterior and septal wall akinesia and inferior, posterior and lateral hypokinesia.

A milrinone infusion (0.3 μg/kg/min) was started and the adrenaline discontinued. Use of an intra-aortic balloon pump was contra-indicated because of his peripheral vascular disease. Continuous veno-venous haemodiafiltration (CVVHDF) was initiated. The next day CI was 2.5 L/min/m² and base excess was -1.9 mmol/L. He subsequently developed evidence of sepsis and blood cultures grew multi-resistant *Staphylococcus aureus*

(MRSA): he was treated with vancomycin and rifampicin. CVVHDF was stopped on day four. On day nine he was extubated but required re-intubation for acute pulmonary oedema despite optimal pharmacological management of impaired left ventricular function, including milrinone 0.7 µg/kg/min and an infusion of glyceryl trinitrate (GTN). Percutaneous tracheostomy was carried out on day 10. Further attempts at weaning him from positive pressure ventilatory support again resulted in pulmonary oedema.

In view of his persistent cardiac failure and established left bundle branch block, cardiac resynchronisation was considered. A biventricular implantable cardiac defibrillator (ICD InSync III Marquis 7279, Medtronic) was implanted on day 12. Three leads were introduced via the left subclavian vein. A Guidant EasiTrak II lead was introduced into the coronary sinus and was placed in a lateral coronary vein. Satisfactory positions were obtained for all three leads.

When biventricular pacing had been established, an immediate haemodynamic response was noted. The mean arterial blood pressure rose by 15 mmHg. The procedure was well tolerated and uncomplicated. Echocardiography confirmed an improvement in left ventricular function immediately after synchronised pacing.

Subsequently, weaning of the patient from mechanical ventillation was achieved without further episodes of pulmonary oedema, inotropic support was weaned and he was discharged from the intensive care unit on day 25. He was transferred to the referring hospital six days later for continuing rehabilitation.

Discussion

End-stage cardiac failure is a common teaton for admission to the Intensive Care Unit (ICU). Patients have often been established on maximum medical therapy (angiotensin converting enzyme [ACE] inhibitors, beta (blockers and disretics) and may also have had several revascularisation procedures with no further scope for improvement in cardiac function. Once patients are intubated, ventilated and on inotropes, wearing from such invasive therapy is often difficult. The case above describes a relatively young patient, with end-stage cardiac disease, who despite revascularisation and maximal pharmacological therapy could not be weaned from mechanical ventilation because of recurrent pulmonary oedema. One would assume that 12 days is sufficient time for any acute myocardial stunning to have reversed. This is the first report of biventricular pacing being employed to aid weaning from mechanical ventilation.

Approximately 30% of patients with chronic heart failure have evidence of abnormal interventricular conduction, demonstrable on a 12-lead ECG, with a QRS duration of greater than 120 ms.¹ This is most commonly seen as left bundle branch block. This implies that there is a significant delay in conduction to, and hence contraction of, the left ventricle compared to the right. The resulting interventricular dyssynchrony causes delayed contraction of the lateral and inferior walls of the left ventricle, which in its severest form may even contract during early diastole.² There is also a delay in opening and clos-



Key messages

- Left bundle branch block on an ECG is suggestive of interventricular dyssynchrony
- This dyssynchrony results in reduced left ventricular ejection fraction and symptoms of heart failure
- Cardiac resynchronisation with a biventricular pacing system has been shown to improve cardiac function and heart failure symptoms
- Intensive care patients with interventricular dyssynchrony and cardiac failure may benefit from biventricular pacing to assist weaning from ventilation

ing of the acitic and mitral valves compared to right-sided events, resulting in a relative decrease in the duration of left ventricular filling. Left ventricular ejection fraction is reduced, along with cardiac cutput and mean arterial blood pressure. In parients with heart failure, the presence of a wide QRS complex is associated with an in reased mortality.³

Ventricular dyssyrichrony causing a deterioration in left ventricular function has been demonstrated when right ventricular bacing (which effectively induces a left bundle branch pattern) has been used in the treatment of heart failure.⁴

The detrimental mechanical effects of ventricular dyssynchrony can be corrected by cardiac resynchronisation by way of piventricular pacing. In addition to the conventional dual chamber pacing system with endocardial leads placed in the right atrium and right ventricle, a third lead is placed in the left ventricle. The current technique was described by Daubert in 1998.⁵ Specially designed catheters are placed via the right atrium and coronary sinus into a tributary that drains the left ventricle. The right atrium, right ventricle and the left ventricle can then be paced in a synchronous manner.

Several clinical trials have demonstrated benefit in patients with heart failure but none on ventilated patients. The Multicenter InSync Randomized Clinical Evaluation (MIRACLE) study demonstrated significant improvements in exercise capacity, New York Heart Association (NYHA) class, quality of life, cardiac function (by echocardiography) and a reduction in hospital admissions for cardiac failure, in those patients with biventricular pacing.⁶ The Comparison of medical therapy, pacing and defibrillation in chronic heart failure (COMPANION) study was halted prematurely after showing a significant reduction in all-cause mortality and all-cause hospitalisation in patients with dilated cardiomyopathy who were randomised to cardiac resynchronisation.⁷ There was even greater benefit in those who had biventricular pacing with an implantable cardiac defibrillator.⁸

There is considerable evidence that cardiac resynchronisation improves symptoms and markers of functional status in patients who have ventricular dyssynchrony and moderate to

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severe heart failure.9 Implantation of a biventricular pacemaker should be considered in the intensive care management of patients with left ventricular failure.

Conflict of interest

None declared.

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