Refractory hypertensive emergency associated with complete heart block resolved after permanent pacemaker implantation: A case report

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Abstract
Severe hypertension sometimes improves with treatment of bradycardia but this phenomenon is under-reported. Here, an elderly gentleman with complete heart block and a hypertensive emergency was refractory to medical therapies and blood pressure only improved following pacemaker implantation. We discuss the possible mechanisms relating to heart rate and artificial pacing.

KEYWORDS
blood pressure, bradycardia, case report, complete heart block, hypertension, permanent pacing

1 | INTRODUCTION

Significant hypertension and bradycardia are often seen together in clinical practice. Anecdotally, blood pressure (BP) often improves following the treatment of bradycardia when caused by atrioventricular (AV) nodal conduction disturbance; however, this is under-reported in the literature. Permanent pacemaker implantation is a class I indication for complete heart block (CHB).[1]

We report on a case of a hypertensive emergency that was refractory to medical management and was only controlled following the treatment of co-existing CHB with permanent pacemaker implantation.

2 | CASE REPORT

An 88-year-old man was admitted to hospital on the acute medical ward with gradually worsening breathlessness and peripheral edema. His comorbidities included myocardial infarction and coronary stenting 9 years ago, essential hypertension, hypercholesterolemia, and stage III chronic kidney disease. On initial assessment, his BP was noted to be 232/92 with a heart rate (HR) of 52 bpm. There was clinical evidence of congestive cardiac failure with bilateral basal lung crepitations and pitting lower limb edema. There was no papilledema on ophthalmoscopy examination and no evidence of neurological compromise. An ECG revealed CHB with a narrow complex escape rhythm at 46 bpm (Figure 1). A chest X-ray showed mild pulmonary edema. Admission blood tests were satisfactory including normal thyroid function and electrolytes. There was no evidence of kidney injury. His regular medication included lacidipine 6 mg, candesartan 28 mg, bisoprolol 10 mg, furosemide 20 mg, aspirin 75 mg, and atorvastatin 40 mg all taken once daily.

Bisoprolol was stopped on Day 1 in view of CHB, and he was referred to the cardiology team to consider permanent pacemaker implantation in case CHB persisted off bisoprolol. An intravenous frusemide infusion of 240 mg over 24 h was commenced. Despite a good diuresis in the first 24 h, his BP remained 202/69; therefore, an intravenous glyceryl trinitrate (GTN) (50 mg/50 ml at 1–10 ml/h)
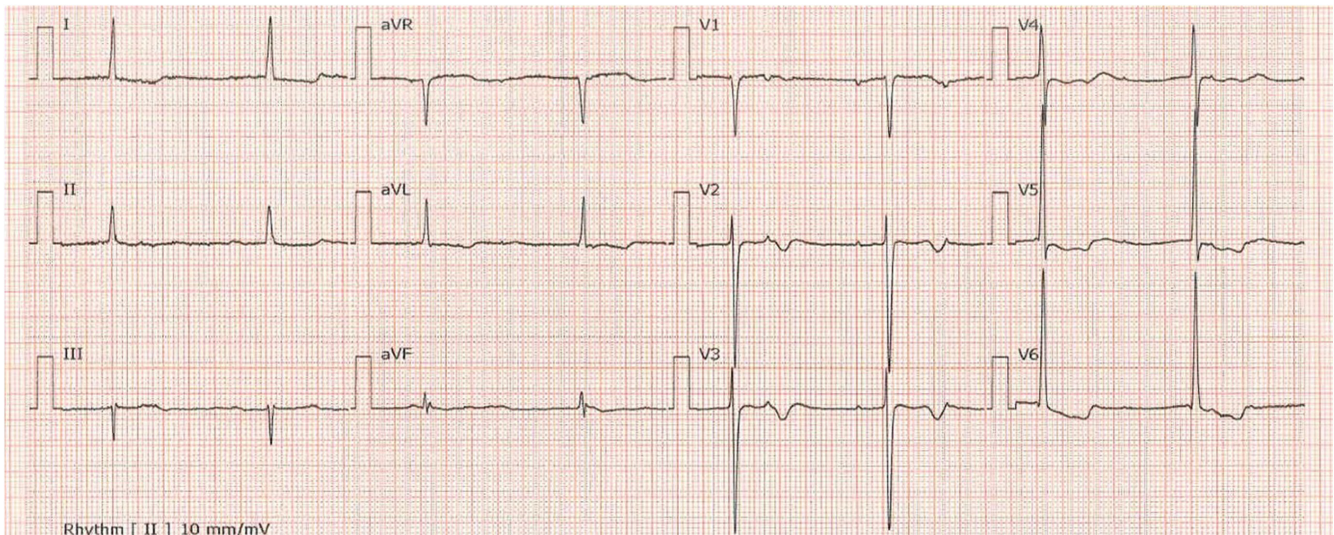
FIGURE 1 ECG before pacemaker implantation showing complete atrioventricular dissociation with a narrow complex escape rhythm at 42 bpm
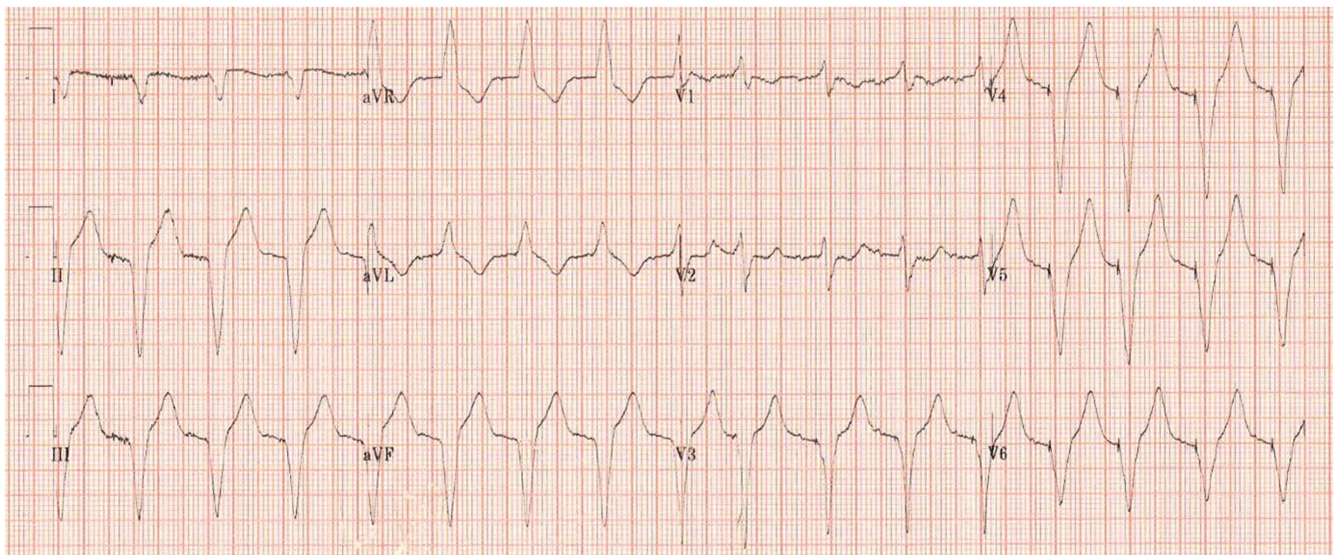

FIGURE 2 ECG following pacemaker implantation showing an atrial-tracked, ventricular-paced rhythm at 90 bpm

infusion was started. BP remained 190/90 with 8 ml/h of GTN.

Secondary causes of hypertension were investigated with normal 24-h urinary catecholamine and serum angiotensin-converting enzyme levels, and normal sized kidneys on renal ultrasound. There was heavy proteinuria with 24-h urinary collection measuring 6.1 g. An echocardiogram showed normal left ventricular (LV) size and function, moderate concentric LV hypertrophy, and mild aortic stenosis. There was no LV dilatation or regional wall motion abnormality seen.

He achieved a 5.3 kg diuresis over 5 days, and his symptoms of breathlessness and peripheral edema had improved. Attempts were made to wean off GTN; however, this only led to BP rising again to 215/89. Indapamide

2.5 mg once daily had a limited effect. Doxazosin was started and gradually increased to 4 mg twice daily. Methyldopa was initiated and uptitrated to 250 mg twice daily.

On Day 7, pacemaker implantation was due to go ahead. The patient remained in CHB. On arrival to the cardiac catheter laboratory, BP was 180/70 with GTN running at 10 ml/h. GTN was discontinued shortly before the procedure anticipating that sedation and analgesia might conversely lead to acute hypotension. A dual-chamber pacemaker was implanted successfully with no immediate complications. The right ventricular (RV) pacing lead was positioned at the RV apex. BP during the procedure was 169/51. With the device set for atrial sensing and ventricular pacing, HR was maintained around 90 bpm. BP

immediately after pacing was 160/80 and remained controlled at this level without GTN. The post-implant ECG showed an atrial-sensed, ventricular-paced rhythm at 90 bpm (Figure 2), and post-implant chest X-ray showed satisfactory lead positioning with no acute complications. Bisoprolol was restarted at 10 mg. The final documented BP prior to discharge was 100/52 with a HR of 82.

3 | DISCUSSION

This case interested us due to the combination of bradycardia and a hypertensive emergency that was refractory to medical treatments. The first treatment to be initiated was a frusemide infusion. Although this improved symptoms related to pulmonary and peripheral edema, it had very little effect on BP suggesting that blood volume was not a significant factor in the mechanism of hypertension. Some effect on BP was achieved by the GTN infusion which suggests that peripheral vascular resistance was a factor. Other medications including the diuretic and vasodilator indapamide, the alpha1-adrenergic receptor blocker doxazosin, and the centrally acting antihypertensive methyldopa did not seem to provide any additional benefit, suggesting little further role in modulating

volume overload, peripheral vasoconstriction, or sympathetic tone.

BP markedly improved following the treatment of CHB with permanent pacemaker implantation. A summary of BP readings during the clinical course is shown in Figure 3. It was interesting to note that the lowest diastolic BP before pacing was 51 mmHg and immediately rose after initiation of pacing to 80 mmHg. Pulse pressure before pacing was between 100 and 140 mmHg and after pacing fell to around 50 mmHg. We suspected this was a direct effect of increasing HR, shortening the diastolic period, and limiting the fall in arterial pressure before the next cardiac cycle. We confirmed before pacing that the large pulse pressure did not have an alternative cause such as aortic regurgitation.

Systolic BP also decreased by 20 mmHg following device implantation without the need to restart the GTN infusion and even once the effects of sedation had worn off. A combination of factors may have played a part; however, the contrast was striking. While it proved impossible to wean off GTN prior to pacing, it was no longer required at all afterward. The reduction in diastolic filling time could have reduced ventricular preload and stroke volume (SV) to some extent, but it is difficult to say how much this contributed to the reduction in systolic BP.
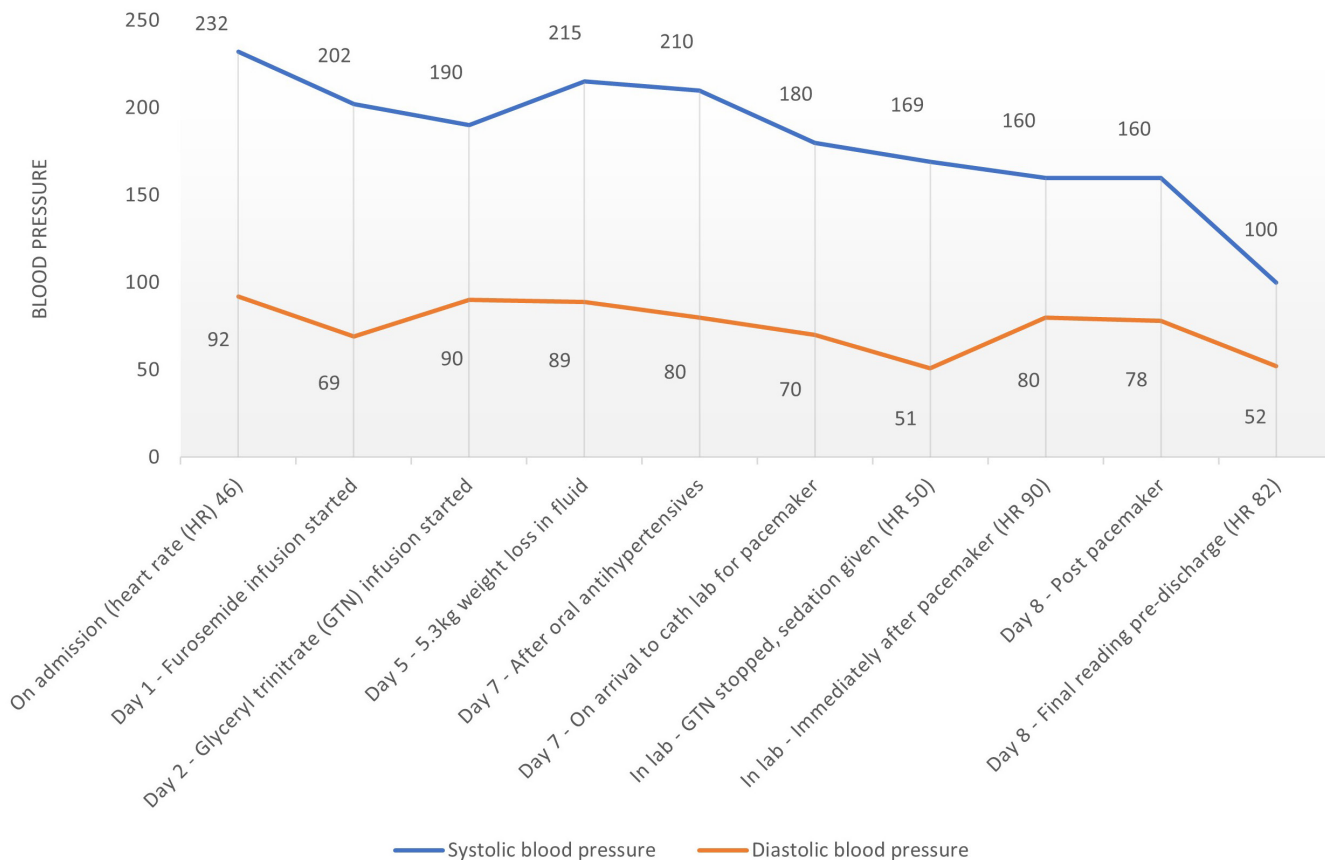

FIGURE 3 Blood pressure recordings taken during the clinical course influenced by events during this episode

BP is a function of cardiac output (CO) which can either be calculated using Fick's equation utilizing markers of oxygen consumption, or by the formula $CO = HR \times SV$. A lower HR reduces CO, but this is partly mitigated by a rise in SV since lower HRs also increase diastolic filling time and ventricular preload. Historically, it has been described that as HR slows, pulse pressure widens, that is, diastolic BP drops and systolic BP increases.[2,3] The hemodynamic effects of artificial pacing in CHB have also been described, suggesting that increasing HR lowers SV.[4] Conversely, SV has been shown to increase on the induction of CHB in the animal model.[5]

RV pacing may directly affect SV by induction of dyssynchronous myocardial contraction. Prior to pacing in this case, the escape rhythm was junctional in nature; hence, LV activation will have been via the normal His-Purkinje system. Change in the pattern of regional myocardial strain by pacing has been demonstrated in dogs by MRI imaging. SV was significantly reduced by RV apical pacing due to dyssynchronous LV myocardial contraction.[6] Another study looking at lengthening AV delay during pacing showed an immediate increase in both SV and BP. However, BP quickly declined after a few seconds while SV was maintained, suggesting compensatory peripheral vasodilatation.[7] Artificial pacing algorithms to shorten AV delay have been proposed as a treatment for hypertension; however, this has so far remained restricted to clinical studies.[8,9]

BP is also dependent on systemic vascular resistance (SVR) under autonomic control via baroreceptors. Cardiogenic shock induces a sympathetic response to compensate with peripheral vasoconstriction and increases SVR acutely.[10] Chronically raised SVR may have played a role in this case with evidence of longstanding hypertension, with echocardiography revealing moderate concentric LV hypertrophy, and there was heavy proteinuria. SVR is also known to increase with advanced age associated with atherosclerosis.[11,12] Higher SVR may have exaggerated the BP response to increased SV.

We noted only one previous case report of bradycardia and malignant hypertension. It reports on 65-year-old man presenting with left upper limb weakness, bilateral foot paresthesia, and headache with a BP of 240/90 with an ECG showing CHB at 39 bpm. Brain imaging was normal, and symptoms resolved after BP control with intravenous GTN. After permanent pacing, BP improved to 140/80. The authors attributed better BP control to reduced diastolic filling time and SV.[13]

In hindsight, pacemaker implantation was unduly delayed due to a combination of inter-speciality logistics and laboratory availability. Greater efforts to enable earlier pacemaker implantation may have led to sooner control of BP and shorter hospital stay, and this is a learning point. We should also make mention of alternative pacing sites such as RV outflow tract, RV septum, or His bundle pacing, which may have preserved more physiological ventricular contraction. Although the hemodynamic effects of these methods have been studied with relation to intracardiac function, the effect in this context on systemic BP needs further study.[14]

The key message highlighted by this case is that artificial pacing may contribute to BP reduction by altering diastolic filling, myocardial contractility, and atrioventricular synchrony. If pacing is otherwise indicated in a refractory hypertensive patient, these physiological effects may need to be considered before the addition of multiple antihypertensive medications.

4 | CONCLUSION

Severe hypertension often improves following the treatment of bradycardia, but this phenomenon is underreported. In this case, bradycardia as a result of CHB occurred in the context of a hypertensive emergency refractory to multiple medical therapies. BP was only controlled following permanent pacing with an immediate rise in diastolic BP likely related to a faster HR. A reduction in diastolic filling time and SV may have contributed to better control of systolic BP, although other factors such as pacing-induced dyssynchrony, atrioventricular delay, and SVR could have also played a role. Pacing may therefore benefit patients in similar situations early in their clinical course.

CONSENT
Published with the written consent of the patient.

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