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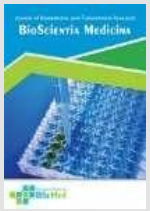

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Emergency Bedside Transvenous Pacing Without Fluoroscopic Guidance for Refractory Symptomatic Bradycardia Following Cardiac Arrest in Acute Inferior ST-Elevation Myocardial Infarction: A Case Report

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A B S T R A C T

Background: Symptomatic bradycardia is a recognized complication of acute inferior myocardial infarction due to atrioventricular node ischemia. Temporary transvenous pacemaker insertion traditionally requires fluoroscopic guidance, which may be unavailable in emergency departments. Alternative non-fluoroscopic approaches are essential in time-critical, resource-limited settings. **Case presentation:** A 62-year-old male with uncontrolled diabetes mellitus presented to the emergency department with respiratory and cardiac arrest. Following cardiopulmonary resuscitation, return of spontaneous circulation was achieved. Electrocardiography revealed inferior ST-elevation myocardial infarction with second-degree atrioventricular block type II. Despite administration of atropine sulfate (cumulative dose 1.25 mg intravenously) and vasopressor support, the patient developed refractory symptomatic bradycardia with heart rate declining to 25-30 beats per minute, culminating in a second cardiac arrest. After achieving return of spontaneous circulation for the second time, a temporary transvenous pacemaker was inserted at the bedside in the emergency department via the femoral vein using a blind technique with electrocardiographic monitoring as the sole guide for successful placement. The pacemaker was set to VVI mode at 70 beats per minute. Subsequent fluoroscopic confirmation in the catheterization laboratory revealed the catheter tip in the right ventricular outflow tract, which was repositioned to the right ventricular apex. Percutaneous coronary intervention was subsequently performed. **Conclusion:** Bedside blind temporary transvenous pacemaker insertion using electrocardiographic monitoring is a feasible and effective approach in emergency settings where fluoroscopic guidance is unavailable.

1. Introduction

Cardiac pacing is a fundamental therapeutic intervention in the management of bradyarrhythmias and conduction abnormalities. Permanent pacemakers are implanted for long-term management of chronic conditions, while temporary pacemakers serve as a critical bridge therapy in acute settings.[1] These devices provide mechanical and electrical support when the heart's intrinsic conduction system fails or becomes insufficient to maintain adequate cardiac output. In the emergency department setting, temporary pacing devices are essential lifesaving interventions that can stabilize critically ill patients until more definitive therapy can be administered.

Bradyarrhythmia represents a significant complication in acute myocardial infarction, occurring in approximately 12-25 percent of patients with inferior infarction.[2] The pathophysiology underlying this complication is intimately related to the vascular supply of the atrioventricular node. In the majority of

individuals, the right coronary artery provides the primary blood supply to the atrioventricular node, making an inferior wall myocardial infarction a particularly high-risk situation for conducting system abnormalities. The occurrence of bradyarrhythmia in this clinical context reflects direct ischemic injury to the specialized tissues responsible for impulse conduction and heartbeat initiation.

Several methods of temporary cardiac pacing exist, each with distinct advantages and limitations. Transcutaneous pacing offers the advantage of rapid deployment without venous access; however, this approach is frequently associated with patient discomfort, unreliable electrical and mechanical capture, and limited duration of efficacy, making it suitable primarily as a bridge to more definitive therapy.[3] Transcutaneous pacing can be initiated within minutes and requires only skin electrodes and a pacing device. However, patient tolerance is limited, and sustained therapy beyond 20 to 30 minutes becomes increasingly difficult due to progressive skin irritation, muscle contraction, and pain requiring escalating doses of sedation.

In contrast, transvenous pacing provides superior reliability, stable pacing thresholds, improved patient tolerance, and the capacity for prolonged support. Traditional transvenous pacemaker insertion has relied upon fluoroscopic guidance, which offers real-time visualization of catheter advancement and positioning within the right ventricle.

However, fluoroscopic guidance presents significant limitations in the acute emergency setting. Most emergency departments lack immediate access to fluoroscopy or interventional facilities, necessitating patient transfer to a distant catheterization laboratory, a process that introduces substantial delay and carries inherent risks for unstable patients.[4] Furthermore, fluoroscopic imaging entails radiation exposure to both patient and operator, adds considerable expense, and requires specialized infrastructure and expertise often unavailable in resource-limited healthcare settings. The distance between the emergency department and the cath laboratory can be substantial in many hospitals, creating prolonged periods of vulnerability for patients with life-threatening bradyarrhythmias.

Recognition of these limitations has prompted investigation of alternative guidance techniques for temporary transvenous pacemaker insertion. Transthoracic echocardiography has emerged as a viable approach, offering real-time visualization of catheter advancement and reducing procedural time compared to fluoroscopy-guided techniques.[5] Three-dimensional transesophageal echocardiography and intracavitary electrocardiography have also demonstrated utility in improving visualization and confirming catheter positioning.[6,7] Additionally, the blind technique, relying solely upon electrocardiographic monitoring and clinical assessment, has been employed successfully in various clinical settings, particularly when imaging modalities are unavailable.

The emergency department environment presents unique challenges for the management of life-threatening arrhythmias. Time-critical clinical decisions must be made with limited resources, and delays in definitive therapy can precipitate irreversible deterioration. In our patient, the convergence of multiple adverse factors, recurrent cardiac arrest, refractory bradycardia, and unavailable fluoroscopy, created an urgent clinical dilemma requiring immediate intervention at the bedside.

To our knowledge, this is one of the few reported cases describing bedside insertion of a temporary transvenous pacemaker without any radiological guidance in the emergency department for a post-cardiac arrest patient with acute inferior ST-elevation myocardial infarction complicated by refractory symptomatic bradycardia. While previous studies have reported on fluoroscopy-guided, echocardiography-guided, and even zero-fluoroscopy techniques using three-dimensional electroanatomic mapping systems for temporary transvenous pacemaker insertion, reports of purely blind bedside insertion guided solely by surface electrocardiographic monitoring in the emergency department setting for

patients with recurrent cardiac arrest remain exceedingly rare in the published literature.[4]

The novelty of this case lies in the unique convergence of three critical factors that distinguish it from existing reports: first, the patient had experienced two episodes of cardiac arrest with successful resuscitation prior to the procedure, representing an exceptionally high-risk clinical scenario rarely described in the context of blind transvenous pacemaker insertion; second, the procedure was performed entirely without any imaging guidance, relying exclusively on surface electrocardiographic monitoring as the sole confirmatory tool; and third, the successful hybrid approach of emergency blind insertion followed by subsequent fluoroscopic optimization in the catheterization laboratory demonstrates a practical stepwise strategy applicable to resource-limited emergency departments worldwide. This case report aims to describe the clinical decision-making process, technical execution, and outcome of emergency bedside blind temporary transvenous pacemaker insertion in this critically ill patient, and to discuss the implications of this approach for emergency medicine practice in settings where fluoroscopic guidance is unavailable.

2. Case Presentation

The demographic and clinical characteristics of the patient at initial presentation to the emergency department are summarized in Table 1. Notable findings included profound hemodynamic instability with unmeasurable blood pressure, absent pulse, and severely depressed consciousness.

Table 1. Demographic and clinical characteristics of the patient.

Parameter	Finding
Age (years)	62
Gender	Male
Past medical history	Uncontrolled diabetes mellitus
Presenting complaint	Respiratory and cardiac arrest
Duration of symptoms before arrest	Headache and epigastric pain since the evening of June 3rd, 2023
Glasgow Coma Scale at arrival	3 (E1V1M1)
Blood pressure at arrival	Unmeasurable
Heart rate at arrival	Absent (cardiac arrest)
Temperature	36.0°C
Peripheral oxygen saturation	80% (gasping respirations)
General appearance	Unconscious, no signs of anemia, icterus, cyanosis, or dyspnea

Mr. M., a 62-year-old male, was brought to the emergency department on June 4th, 2023, at 05:15 AM by his family in a state of unconsciousness with absent spontaneous respirations and palpable pulse. His wife reported that on the evening of June 3rd, 2023, he had complained of a headache and epigastric pain, but the family did not seek medical attention at that time. His past medical history was significant for diabetes mellitus, which remained uncontrolled without regular pharmacological treatment. He had no history of myocardial infarction, cerebrovascular disease, or prior cardiac arrhythmias. He was not known to have hypertension, chronic kidney disease, or malignancy.

Upon arrival at the emergency department, physical examination revealed an unconscious male with a Glasgow Coma Scale score of 3 (E1V1M1), indicating profound altered consciousness. Blood pressure was unmeasurable, and no palpable pulse was detected, consistent with cardiac arrest.

Respiration was gasping in character, and peripheral oxygen saturation measured 80 percent. Body temperature was 36.0 degrees Celsius. General inspection revealed no obvious signs of anemia, jaundice, cyanosis, or acute respiratory distress. Examination of the head and neck was unremarkable. Thoracic examination was limited due to the critical condition and the need for immediate resuscitation. Cardiac auscultation could not be adequately performed. Abdominal examination revealed mild distension with audible bowel sounds and soft consistency on palpation. The extremities were cold and notably acrocyanotic, indicating severe peripheral vasoconstriction and hypoperfusion.

Immediate resuscitative efforts were initiated in accordance with Advanced Cardiovascular Life Support protocols. A bedside blood glucose test revealed 589 mg/dL, indicating severe hyperglycemia and reflecting markedly uncontrolled diabetes mellitus, a finding that portended a poorer prognosis. Electrocardiographic monitor showed pulseless electrical activity. Standard cardiopulmonary resuscitation was performed, with one intravenous bolus of epinephrine 1 mg administered. Ventilation was provided using a Jackson-Rees system with 100 percent oxygen at a flow rate of 12 liters per minute. Return of spontaneous circulation was achieved at 05:35 AM, approximately 20 minutes after arrival.

Following initial resuscitation, a comprehensive panel of laboratory and diagnostic investigations was performed. The results, as presented in Table 2, revealed several critical abnormalities that guided subsequent management decisions.

Table 2. Laboratory and diagnostic findings.

Parameter	Result	Reference range	Interpretation
Blood sugar (stick test)	589 mg/dL	70-100 mg/dL	Markedly elevated
ECG rhythm (arrival)	Pulseless electrical activity	Normal sinus rhythm	Cardiac arrest
ECG rhythm (post-ROSC)	Inferior STEMI, 2nd degree AV block type II	Normal sinus rhythm	Acute ischemia with conduction abnormality
ST elevation	Leads II, III, aVF	No ST elevation	Inferior STEMI
Chest X-ray	Cardiomegaly	Normal heart size	Cardiac enlargement
GCS post-ROSC	3 (E1V1M1)	15 (fully alert)	Profound coma
Heart rate post-ROSC	76 bpm	60-100 bpm	Acceptable
Systolic BP post-ROSC	<90 mmHg	>100 mmHg	Hypotension

Following return of spontaneous circulation, the patient remained profoundly comatose with a Glasgow Coma Scale score of 3 (E1V1M1). Twelve-lead electrocardiography demonstrated ST-segment elevation in leads II, III, and aVF, consistent with acute inferior myocardial infarction. Notably, the electrocardiogram also revealed a second-degree atrioventricular block type II, characterized by progressive PR interval prolongation followed by dropped atrial impulse, a finding indicating significant atrioventricular nodal ischemia. Chest radiography revealed cardiomegaly. Heart rate was 76 beats per minute immediately post-resuscitation, but systolic blood pressure was markedly reduced at less than 90 mmHg.

Hemodynamic management was immediately initiated. The patient was administered a fluid challenge with rapid crystalloid infusion of 250 milliliters over 10 minutes with a favorable response, followed by continued infusion of 500 milliliters over the subsequent hour. At 06:00 AM, after confirmation of the acute inferior STEMI diagnosis, the patient was expedited for urgent percutaneous coronary intervention. Loading doses of dual antiplatelet therapy were administered via nasogastric tube: clopidogrel 300 mg and aspirin 160 mg. Atropine sulfate 0.25 mg was given intravenously, and enoxaparin sodium 6000 anti-Xa international units

was administered via subcutaneous injection.

By 06:30 AM, the patient's heart rate had declined precipitously to 32-35 beats per minute, a markedly symptomatic bradycardia accompanied by hemodynamic compromise. A second intravenous dose of atropine sulfate 0.25 mg was administered. Simultaneously, norepinephrine infusion was initiated at 100 nanograms per kilogram per minute (titrated to a maximum of 200 nanograms per kilogram per minute) with a target systolic blood pressure greater than 100 mmHg. Five minutes later, despite vasopressor support, blood pressure deteriorated to 79/39 mmHg. Norepinephrine dose was escalated to the maximum of 200 nanograms per kilogram per minute, and intravenous crystalloid infusion was adjusted to maintain a rate of 500 milliliters per 24 hours.

At 06:40 AM, the patient's heart rate had further deteriorated to 25-30 beats per minute, representing profound bradycardia refractory to pharmacological intervention. A third dose of atropine sulfate 0.5 mg was administered intravenously, bringing the cumulative atropine dose to 1.25 mg, exceeding the standard upper limit of 1.0 mg but justified by the clinical urgency. Five minutes later, at 06:45 AM, the patient experienced a second episode of cardiac arrest. Immediate cardiopulmonary resuscitation was commenced per Advanced Cardiovascular Life Support protocols, with one intravenous bolus of epinephrine 1 mg given. Return of spontaneous circulation was re-established at 07:00 AM.

The complete chronological sequence of interventions, pharmacological doses, and corresponding clinical responses throughout the entire resuscitation period is detailed in Table 3. This timeline illustrates the escalating severity of the clinical situation and the progressive failure of pharmacological interventions that ultimately necessitated mechanical pacing.

Table 3. Treatment timeline and clinical response.

Time	Intervention	Dose/Details	Clinical response
05:15 AM	Patient arrival	Cardiac arrest; pulseless electrical activity	Unconscious, GCS 3, apneic
05:15-05:35	CPR per ACLS	Epinephrine 1 mg IV, ventilation 12 L/min	ROSC achieved at 05:35 AM
05:35 AM	12-lead ECG	Inferior STEMI, 2nd degree AV block type II	ST elevation leads II, III, aVF
06:00 AM	Antiplatelet loading	Clopidogrel 300 mg, aspirin 160 mg via NG tube	Adequate absorption anticipated
06:00 AM	Initial atropine and anticoagulation	Atropine 0.25 mg IV, enoxaparin 6000 IU SC	HR 76 bpm, transient stability
06:30 AM	Bradycardia management	Atropine 0.25 mg IV, norepinephrine initiated at 100 ng/kg/min	HR declined to 32-35 bpm; hypotensive
06:35 AM	Vasopressor escalation	Norepinephrine increased to 200 ng/kg/min, crystalloid 500 cc/24h	BP dropped to 79/39 mmHg
06:40 AM	Refractory bradycardia	Atropine 0.5 mg IV (cumulative dose 1.25 mg)	HR 25-30 bpm, severe bradycardia
06:45 AM	Second cardiac arrest	CPR per ACLS, epinephrine 1 mg IV	Cardiac arrest recurrence
07:00 AM	ROSC achieved	Resuscitation successful	ROSC re-established
07:15 AM	Intubation and pre-TTPM atropine	Endotracheal intubation, atropine 0.5 mg IV	Airway secured, HR initially 32 bpm
07:20 AM	Temporary transvenous pacemaker insertion	Femoral vein access, VVI mode 70 bpm, blind technique with ECG guidance	Pacing capture achieved, HR 70 bpm
Post-TTPM	Transfer to the catheterization laboratory	Fluoroscopy performed	Catheter tip repositioned from RVOT to RV apex
Post-TTPM	PCI performed	Culprit vessel identified and treated	Reperfusion achieved

To facilitate a comprehensive understanding of the clinical progression, the entire clinical course of this patient from initial presentation through definitive intervention is illustrated graphically in Figure 1. This figure provides a visual overview of the five distinct clinical phases, highlighting the temporal relationships between interventions, hemodynamic responses, and the critical decision points that culminated in the bedside blind temporary transvenous pacemaker insertion.

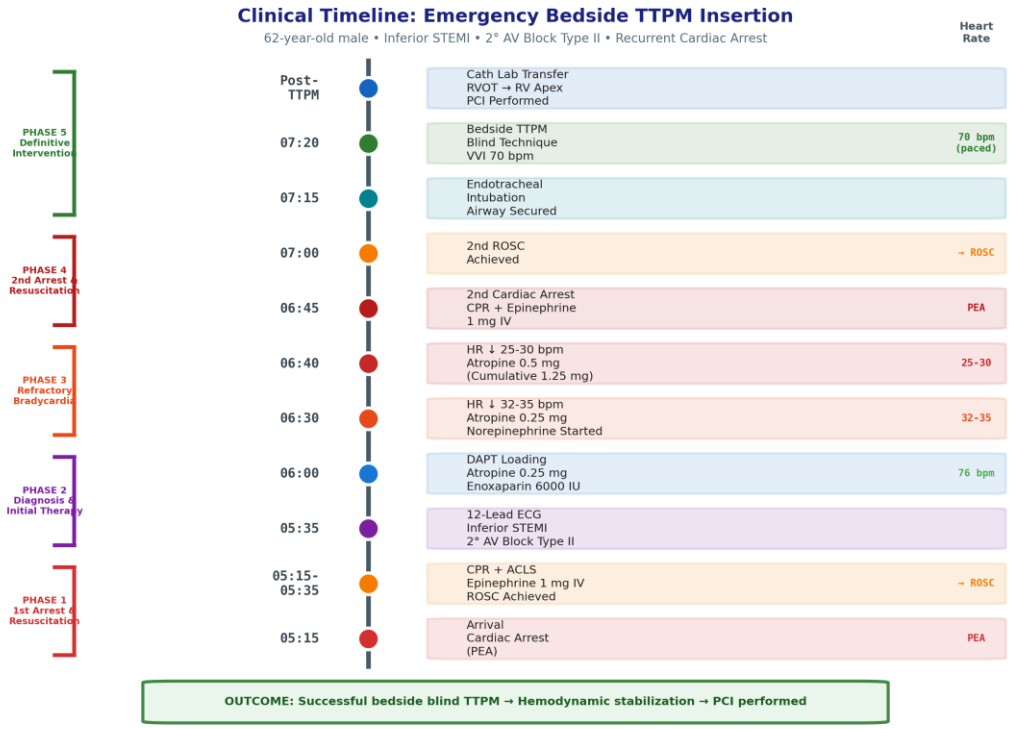

Figure 1. Clinical timeline of emergency bedside temporary transvenous pacemaker (TTPM) insertion. The figure illustrates the sequential clinical events in a 62-year-old male presenting with cardiac arrest secondary to an inferior ST-elevation myocardial infarction complicated by refractory symptomatic bradycardia. Five distinct clinical phases are depicted, from initial arrest and resuscitation through definitive intervention with bedside blind TTPM insertion and subsequent percutaneous coronary intervention. PEA = pulseless electrical activity; ROSC = return of spontaneous circulation; STEMI = ST-elevation myocardial infarction; AV = atrioventricular; DAPT = dual antiplatelet therapy; HR = heart rate; TTPM = temporary transvenous pacemaker; VVI = ventricular pacing, ventricular sensing, inhibited mode; RVOT = right ventricular outflow tract; RV = right ventricular; PCI = percutaneous coronary intervention.

At 07:15 AM, following the second resuscitation and achievement of return of spontaneous circulation, the decision was made to secure the airway with endotracheal intubation to protect against aspiration and facilitate mechanical ventilation. Immediately prior to the transvenous pacemaker insertion procedure, an additional dose of atropine sulfate 0.5 mg was administered intravenously.

At this critical juncture, a momentous clinical decision was made. The patient had now experienced two cardiac arrests within 90 minutes, with the immediate precipitant being profound refractory bradycardia unresponsive to maximum pharmacological therapy. The distance between the

emergency department and the interventional catheterization laboratory was substantial, and transfer of an unstable post-resuscitation patient carried considerable risk of a third cardiac arrest en route. Fluoroscopic guidance was not available in the emergency department. Under these extraordinary circumstances, it was determined that immediate bedside insertion of a temporary transvenous pacemaker using blind technique with electrocardiographic monitoring was the most prudent course of action.

The temporary transvenous pacemaker insertion procedure was performed at the bedside in the emergency department at 07:20 AM. The femoral vein was selected as the site of venous access because it permitted rapid cannulation, involved minimal time, carried a lower risk of pneumothorax compared to subclavian or internal jugular vein approaches, and could be managed with direct manual compression for hemostasis, an important consideration given that the patient had already received anticoagulation with enoxaparin. The femoral vein anatomy is relatively constant and straightforward, making successful access achievable even in critically ill, hypotensive patients.

A 6-French introducer sheath was inserted into the femoral vein using the Seldinger technique. The electrode catheter was marked externally to estimate the appropriate insertion depth to achieve right ventricular positioning. The catheter was then attached to the pulse generator unit set to VVI mode (ventricular pacing, ventricular sensing, inhibited mode) at a rate of 70 beats per minute, sensitivity set to 7 millivolts, and amplitude (output) set to 2 volts. VVI mode was selected because it is simpler than other modes, provides adequate rate support, and requires only single-chamber sensing without the added complexity of dual-chamber atrial and ventricular sensing.

The electrode catheter was advanced systematically through the introducer sheath into the venous system. As the catheter tip approached and entered the right ventricle, the surface electrocardiogram, displayed continuously on the monitor, showed a characteristic pattern of right ventricular capture, represented by a left bundle branch block morphology on the monitor lead. This electrocardiographic finding confirmed successful contact between the catheter tip and the right ventricular myocardium with achievement of electrical capture. The procedure was completed successfully without any perioperative complications. Following successful pacemaker insertion, the patient's heart rate increased to 70 beats per minute, and blood pressure stabilized.

The patient was subsequently transferred to the interventional cardiac catheterization laboratory for definitive imaging and intervention. Fluoroscopy revealed that the electrode catheter tip was located in the right ventricular outflow tract, a known but suboptimal position that can occur with blind catheter advancement. The catheter was repositioned under fluoroscopic guidance to achieve a position at the right ventricular apex, which represents the optimal site for long-term pacing stability. Repeat fluoroscopy confirmed proper positioning of the catheter tip at the right ventricular apex. Electrocardiographic monitoring demonstrated persistent pacing capture at the set rate of 70 beats per minute.

Coronary angiography was performed to identify the culprit vessel in this acute inferior myocardial infarction. The right coronary artery was identified as the vessel responsible for the inferior wall infarction and atrioventricular node ischemia. Percutaneous coronary intervention was performed on the right coronary artery with successful revascularization and restoration of antegrade flow. Following the intervention, a chest radiograph confirmed stable positioning of the temporary transvenous pacemaker electrode catheter at the right ventricular apex. The patient's subsequent course and long-term outcome were not documented in the available clinical records.

3. Discussion

This case exemplifies a profound and life-threatening presentation of acute myocardial

infarction in which cardiac arrest represented the initial manifestation of acute coronary disease. The prodromal symptomatology, headache and epigastric pain, occurred on the evening prior to arrest but was not recognized by the family as potentially indicative of a cardiovascular emergency, resulting in a critical delay in seeking medical attention. Such delayed presentation is characteristic of acute coronary syndrome in diabetic patients, in whom anginal chest pain is often absent or atypical.[8]

This pattern of atypical symptom presentation is particularly common in diabetic patients, in whom autonomic neuropathy impairs the usual protective warning signals of ischemic disease. Diabetic patients frequently experience myocardial ischemia without the characteristic chest discomfort that typically prompts earlier medical evaluation.[9] The absence of typical anginal symptoms allows progression of ischemia to infarction and even to the point of hemodynamic collapse and cardiac arrest without earlier recognition and treatment. This phenomenon represents a substantial clinical challenge in risk stratification and early identification of at-risk patients.

The laboratory finding of blood glucose of 589 mg/dL revealed markedly uncontrolled diabetes mellitus, representing a glucose level substantially above the normal range and indicative of inadequate glycemic management. Severe hyperglycemia in the acute setting of myocardial infarction is independently associated with a substantially poorer prognosis, including increased mortality, increased infarct size, and greater propensity for complication development.[10] The combination of acute myocardial infarction superimposed upon a background of uncontrolled diabetes mellitus created a particularly high-risk clinical scenario. Hyperglycemia worsens myocardial damage through multiple mechanisms, including increased oxidative stress, impaired mitochondrial function, and augmented inflammatory response. The acute stress response to myocardial infarction itself causes further elevation of blood glucose through catecholamine-induced insulin resistance.

The temporal sequence of clinical events in this case was striking: cardiac arrest at presentation, followed by successful resuscitation to return of spontaneous circulation, followed by progressive bradycardia refractory to pharmacological therapy, followed by recurrent cardiac arrest, and again followed by successful resuscitation. This oscillating pattern, deterioration requiring repeated interventions, reflects the severe myocardial injury and conduction system dysfunction resulting from extensive acute inferior myocardial infarction. Each cycle of arrest and resuscitation exposed the patient to ischemic insult from reduced perfusion and carried the risk that a subsequent arrest might not be reversible. Post-cardiac arrest syndrome, characterized by myocardial dysfunction, systemic ischemia-reperfusion response, and brain injury, further compounds the clinical complexity in such presentations.[11]

The pathophysiological foundation underlying bradyarrhythmia and atrioventricular conduction abnormalities in acute inferior myocardial infarction is rooted in the anatomy of the coronary blood supply to the specialized conduction tissues. The atrioventricular node receives its primary arterial blood supply from the atrioventricular nodal artery in approximately 90 percent of individuals.[12] In the vast majority of these cases, the atrioventricular nodal artery arises as a branch from the right coronary artery. When an acute inferior myocardial infarction results from right coronary artery occlusion, as occurred in our patient, the atrioventricular node is deprived of its primary blood supply, resulting in direct ischemia of the nodal tissue. The degree of hemodynamic impact depends on the timing of the ischemia, the presence of collateral circulation, and the extent of the infarction.

Beyond the immediate effects of ischemia, acute inferior myocardial infarction is accompanied by a marked increase in parasympathetic (vagal) tone, mediated through a phenomenon known as the Bezold-Jarisch reflex. This reflex, triggered by

ischemia in the inferior left ventricular wall, results in sudden augmentation of vagal efferent activity that further depresses atrioventricular nodal function and heart rate. The reflex arc involves mechanoreceptors in the left ventricle sensing the ischemic injury and signaling through vagal afferent fibers to the nucleus ambiguus in the medulla, which then increases parasympathetic efferent activity. The combination of direct ischemic injury to nodal tissue and heightened parasympathetic tone creates a potent depressant effect on atrioventricular conduction.[13] The association between inferior myocardial infarction and atrioventricular conduction disturbances has been well documented, with various underlying etiologies, including ischemia, medication effects, and systemic illness, contributing to the pathophysiology.[14]

The electrocardiographic finding in our patient of second-degree atrioventricular block type II carries important pathophysiological and prognostic implications. Second-degree atrioventricular block type II is characterized electrocardiographically by a relatively stable PR interval followed by an abrupt failure of atrioventricular conduction, resulting in a nonconducted atrial impulse and a dropped QRS complex. This pattern indicates that the site of conduction block lies at or below the level of the His bundle, in the infraHisian system, rather than at the more proximal atrioventricular node itself.[15] Block at this level is more ominous than the block at the atrioventricular nodal level, as it reflects more extensive conduction system pathology and carries significantly greater risk of progression to complete atrioventricular block.[16] Various clinical parameters, including electrocardiographic variables and biomarkers such as NT-proBNP, have been investigated as predictors of complete atrioventricular block risk in the setting of inferior myocardial infarction.[17]

The presence of cardiomegaly on chest radiography in our patient provided additional evidence of right ventricular involvement. When an acute inferior myocardial infarction extends to involve the right ventricle, which occurs in approximately 30-50 percent of inferior infarctions, the hemodynamic consequences are substantially more severe. Right ventricular infarction compromises the contractile function of the right ventricle, impairs right ventricular filling and output, and, through preload-dependent mechanisms, severely reduces left ventricular filling and cardiac output.[16] This hemodynamic deterioration is compounded by the concomitant atrioventricular conduction disturbance, as loss of the atrial kick that normally contributes approximately 15-25 percent of ventricular filling becomes particularly consequential when right ventricular function is compromised. In right ventricular infarction, the ventricle becomes exquisitely preload-dependent, and any loss of atrial contribution can result in catastrophic hemodynamic collapse.

In our patient, the administration of atropine sulfate, a muscarinic cholinergic antagonist that blocks the parasympathomimetic effects mediated through the atrioventricular node, failed to reverse the bradycardia and conduction abnormality. The cumulative atropine dose of 1.25 mg administered intravenously considerably exceeded the standard upper limit of 1.0 mg. The failure of such aggressive atropine therapy to ameliorate the bradycardia indicates that the underlying pathophysiology was not merely reversible parasympathetic overdrive but rather represented intrinsic conduction system dysfunction from direct ischemic injury to infraHisian tissue. This finding underscored the critical need for mechanical pacing.[18]

The management of symptomatic bradycardia in acute myocardial infarction follows a well-established algorithmic approach. The American College of Cardiology and American Heart Association guidelines recommend atropine sulfate as the initial pharmacological intervention for symptomatic bradycardia accompanied by hemodynamic compromise.[2] When bradycardia persists despite adequate atropine therapy, or when atropine is contraindicated or ineffective, transcutaneous pacing is recommended as a temporizing bridge therapy.

Transcutaneous pacing can be rapidly deployed without venous access and may restore hemodynamic stability sufficiently to allow transport to a facility capable of providing more definitive therapy.[1] Ultrasound has been proposed as a method to confirm mechanical capture during external transcutaneous pacing, although this technique requires additional equipment and training.[19]

However, transcutaneous pacing, despite its advantages in the prehospital and early emergency setting, carries significant limitations that restrict its utility in the acute hospital setting. Transcutaneous pacing is frequently associated with inadequate electrical capture in 10-20 percent of attempts, variable mechanical capture despite electrical capture, patient discomfort severe enough to require sedation and analgesia (which are themselves hemodynamically compromising in a critically ill patient), and limited duration of effective support typically restricted to 20-30 minutes before electrode site irritation and muscle fatigue render further pacing ineffective.[3]

In contrast, transvenous pacemaker insertion offers substantial advantages when facing a patient with an anticipated prolonged need for pacing support. Transvenous pacing provides highly reliable electrical and mechanical capture with capture thresholds that remain stable over extended periods. Once properly positioned, the electrode catheter achieves consistent and reliable capture even at low output settings. Patient discomfort is minimal or absent. Most importantly, transvenous pacing can sustain effective support for weeks to months, allowing time for resolution of the underlying ischemic and inflammatory processes that have caused the acute conduction abnormality.[1] Recent innovations in pacing catheter design, including novel atrioventricular sequential pacing catheters, have further expanded the options available for temporary cardiac pacing.[20]

In our patient, the clinical constellation of findings compelled consideration of transvenous pacing: refractory symptomatic bradycardia unresponsive to pharmacological therapy with a cumulative atropine dose of 1.25 mg, recurrent cardiac arrest in the setting of bradycardia indicating an unstable rhythm situation with high likelihood of further deterioration, and the patient's critically ill post-cardiac arrest state with profound coma and hemodynamic instability. The patient had already experienced two cardiac arrests within 90 minutes; the risk that a third arrest would occur was substantial and ever-present. Under these circumstances, the risk of expedited transport to a distant catheterization laboratory, a journey during which the patient might experience another cardiopulmonary arrest with potentially fatal consequences, seemed to outweigh the risks of immediate bedside pacemaker insertion.[4]

The choice of the femoral vein as the site of vascular access reflected careful consideration of the clinical context. The femoral vein provides rapid, reliable access with minimal time expenditure in an emergency setting. The femoral approach avoids the risk of pneumothorax or hemothorax, which represents a serious complication of subclavian or internal jugular vein approaches and would be particularly catastrophic in an already critically ill post-arrest patient.[21] Furthermore, since the patient had already received anticoagulation with enoxaparin, the femoral approach permitted effective hemostasis through direct manual compression, whereas other venous sites would be more difficult to achieve reliable hemostasis.

Fluoroscopy has long been considered the gold standard for guiding transvenous pacemaker electrode catheter placement. Real-time fluoroscopic visualization permits identification of major anatomical landmarks, the superior vena cava, right atrium, tricuspid valve, and right ventricular apex, and allows immediate recognition of catheter malposition or complications. However, fluoroscopy-guided pacemaker insertion is not universally available, particularly in most emergency departments in resource-limited healthcare settings. Recent advances in fluoroscopic technique, including caudal angulation approaches, have aimed to improve lead

placement accuracy when fluoroscopy is available.[22]

In response to these limitations, several alternative guidance techniques have been investigated and successfully employed in clinical practice. Transthoracic echocardiography provides real-time ultrasonic visualization of the moving catheter tip within the cardiac chambers as it is advanced. A prospective study by Ferri and colleagues compared echocardiography-guided transvenous pacemaker insertion with fluoroscopy-guided insertion and found that echocardiography achieved a mean procedural time of 22 minutes compared with 43 minutes for fluoroscopy and was associated with fewer vascular complications.[5] Transesophageal echocardiography offers even superior real-time visualization of the catheter as it traverses the right atrium and right ventricle. A study by Cao and colleagues demonstrated that three-dimensional transesophageal echocardiography-guided transvenous pacemaker insertion achieved a mean needle-to-pacing time of 5.2 minutes compared with 8.5 minutes for standard fluoroscopic guidance.[7]

Intracavitary electrocardiography represents another alternative approach. This technique involves recording the unipolar electrocardiogram between the advancing catheter tip and a reference electrode, making the catheter position visible through characteristic electrocardiographic morphology changes as it traverses different cardiac structures. As the catheter tip contacts the endocardium of the right ventricle, a distinctive pattern of right ventricular cavity potentials becomes apparent.[6] This approach requires no imaging technology beyond standard electrocardiographic equipment and has proven effective in multiple clinical studies.

The blind technique, which relies upon surface electrocardiographic monitoring, clinical assessment of mechanical capture (such as palpation of a pulse), and sometimes measurement of pacing impedance, represents another approach that eliminates dependence upon imaging guidance entirely. Sofi and colleagues reported a series of 1093 patients who underwent unguided transvenous pacing insertion, achieving an impressive success rate of 99.9 percent with a complication rate of 10.7 percent (predominantly minor complications such as transient arrhythmias), and a mean procedural time of 11.5 minutes.[23] This large-scale experience provides compelling evidence that the blind technique is a viable and effective approach for transvenous pacemaker insertion, particularly in emergency settings. The speed of the procedure is notable, 11.5 minutes from venous access to pacing capture, a timeframe considerably faster than fluoroscopic approaches. More recently, zero-fluoroscopy approaches using advanced three-dimensional mapping systems such as the EnSite system have demonstrated feasibility for bedside temporary pacemaker placement with precise lead positioning.[24]

In our case, we employed the blind technique using electrocardiographic monitoring as the sole guide for confirming successful electrode catheter positioning. As the catheter was advanced through the femoral vein, right atrium, and toward the right ventricle, the surface electrocardiogram, continuously displayed on the bedside monitor, provided real-time feedback regarding catheter position. When the catheter tip contacted the right ventricular endocardium, the monitor lead demonstrated a characteristic left bundle branch block morphology (an expected finding when pacing from the right ventricle), indicating successful contact with the right ventricular myocardium. The monitor also demonstrated electrical capture, a characteristic wide QRS complex following each pacemaker stimulus artifact, confirming that the electrode catheter had achieved adequate cathode-myocardium contact to depolarize the ventricle.

A known limitation of the blind technique is that the electrode catheter may lodge in the right ventricular outflow tract rather than achieving the more optimal position at the right ventricular apex. This less-than-ideal position can occur because the catheter naturally follows the path of venous return along the lateral right atrial wall, and when advanced beyond the tricuspid valve, may preferentially enter the right ventricular outflow tract rather than passing

to the apex.[25] Such was the case in our patient. Initial blind insertion resulted in catheter positioning in the right ventricular outflow tract. However, electrical capture was still achieved, indicating adequate contact between the catheter and myocardium despite the suboptimal position. Following transfer to the catheterization laboratory, fluoroscopic confirmation was obtained, the suboptimal position was recognized, and the catheter was carefully repositioned under fluoroscopic guidance to the right ventricular apex, the preferred long-term position. This sequence of events demonstrates a practical hybrid approach: blind insertion in the emergency department to achieve rapid, life-saving pacing, followed by subsequent optimization under fluoroscopic guidance when such imaging becomes available.

To contextualize the present case within the existing body of evidence, we compared the clinical characteristics, procedural details, and outcomes of our patient with those of similar cases reported in the recent literature. These comparisons are presented in Table 4, which highlights the distinctive features of our case relative to previously published reports.

Table 4. Comparison with similar cases from the literature.

Feature	Senturk et al. 2021	John et al. 2020	Wang et al. 2024	Present case
Study type	Retrospective single-center	Case report	Case report	Case report
Sample size/patient age	234 patients, mean age 75 years	Single patient, 58 years	Single patient, 67 years	Single patient, 62 years
Primary diagnosis	Symptomatic bradyarrhythmia	Inferior STEMI with AV block	Bradyarrhythmia requiring pacing	Inferior STEMI with 2nd degree AV block type II
Prior cardiac arrest	Not specified	Yes	Not mentioned	Yes, twice
Pacing method	Transvenous, multiple access sites	Transvenous	Transvenous	Transvenous (blind technique)
Venous access site	Multiple (subclavian, jugular)	Not specified	Femoral	Femoral
Guidance method	Fluoroscopy	Fluoroscopy	EnSite 3D system (zero-fluoroscopy)	Electrocardiography alone (blind)
Initial lead position	RV apex (intended)	Not specified	RV apex (with 3D guidance)	RVOT (blind), repositioned to RV apex
Complications	Bleeding (5 patients)	None reported	None	None
Outcome	Favorable; stable pacing	Favorable; alive and stable	Favorable; successful revascularization	Stabilization achieved; PCI performed

Our case demonstrates several distinctive features when compared with previously published reports. First, our patient had experienced two documented episodes of cardiac arrest with successful resuscitation prior to pacemaker insertion, reflecting particularly severe hemodynamic compromise and demonstrating the extreme fragility of the patient's clinical status. Second, we employed a purely blind technique with electrocardiographic monitoring as the sole guide, without echocardiography, intracavitary electrocardiography, or any imaging modality other than the surface electrocardiogram. Third, despite initial malposition in the right ventricular outflow tract, adequate pacing was achieved, and the patient was stabilized, after which the catheter was optimized under fluoroscopic guidance in the catheterization laboratory. This hybrid approach, blind insertion in the emergency department for life-saving purposes, followed by optimization when resources become available, represents a practical and clinically effective strategy.

This case report offers several important clinical lessons applicable to emergency medicine practice and the management of life-threatening bradyarrhythmias. First, it demonstrates that emergency physicians operating in resource-limited settings can successfully perform temporary transvenous pacemaker insertion using basic principles and a simple bedside technique. The equipment required is minimal: a femoral venous catheter, a pacemaker generator unit, and continuous electrocardiographic monitoring. No specialized imaging technology is necessary, though ultrasonography to confirm venous access may be helpful if available.

Second, the case demonstrates that electrocardiographic monitoring alone can reliably confirm successful electrode catheter positioning. The characteristic morphology changes of the surface electrocardiogram, specifically the transition from atrial bipolar signals to right ventricular endocardial signals, are distinctive and easily recognized by any clinician experienced in electrocardiographic interpretation. The appearance of left bundle branch block morphology during catheter advancement is virtually pathognomonic of right ventricular pacing and confirms appropriate catheter-myocardium contact.

Third, the case illustrates that initial catheter malposition in the right ventricular outflow tract, while not optimal, does not preclude successful pacing. Adequate electrical capture can be achieved from the outflow tract, and the patient can be successfully stabilized. Subsequent optimization, either in the emergency department using additional maneuvers or in the catheterization laboratory under fluoroscopic guidance, can then be pursued once the immediate life threat has been averted.

Fourth, the femoral vein approach offers particular advantages in the emergency setting, including ease of access, avoidance of pneumothorax risk, and the ability to achieve hemostasis through manual compression, important considerations in an anticoagulated patient such as ours. The femoral approach represents an appropriate first choice for bedside transvenous pacemaker insertion in the emergency department.

Fifth, the case reinforces the critical importance of clinical judgment and risk-benefit analysis in emergency medicine. The standard approach to an unstable patient requiring specialty intervention (such as percutaneous coronary intervention) is to transfer the patient to a facility equipped to provide that intervention. However, in this case, the risk of transfer, specifically the high likelihood of a third cardiac arrest during transport, outweighed the risks of attempting definitive intervention (pacemaker insertion) at the bedside despite limitations in available resources. Such clinical decision-making requires careful consideration of the individual patient's circumstances, the nature and severity of the underlying condition, and the risks inherent in any chosen course of action.

This case report has inherent limitations. First, as a single case report, the findings cannot be generalized to broader populations or used to draw conclusions about the reliability or safety of blind transvenous pacemaker insertion techniques. Larger prospective studies would be required to establish the frequency of success, complication rates, and long-term outcomes with this approach. Second, no echocardiographic assessment was performed during the transvenous pacemaker insertion procedure, which might have provided additional information regarding catheter positioning and cardiac function. Third, hemodynamic parameters such as cardiac output or pulmonary wedge pressure were not measured post-pacemaker insertion, limiting assessment of the physiological impact of achieving mechanical pacing. Fourth, the long-term outcome of the patient was not documented in the available clinical records, so we cannot comment on the patient's ultimate survival or functional recovery. Fifth, no formal comparison of procedural time, success rates, or complication frequencies between blind technique and fluoroscopy-guided approaches was performed in this case. Sixth, follow-up imaging

to assess the integrity and stability of pacemaker placement over time was not documented.

4. Conclusion

Symptomatic bradycardia complicated by second-degree atrioventricular block type II represents a recognized and serious complication of acute inferior myocardial infarction due to atrioventricular nodal ischemia and heightened parasympathetic tone. This bradyarrhythmia frequently proves refractory to pharmacological management, including aggressive atropine therapy, necessitating mechanical pacing as a life-saving intervention.

When patients experience recurrent cardiac arrests precipitated by profound bradycardia, the immediate clinical priority becomes prevention of a potentially fatal subsequent arrest. In circumstances where fluoroscopic guidance is unavailable, and the distance to a remote catheterization laboratory creates a substantial risk of deterioration during transport, bedside temporary transvenous pacemaker insertion using a blind technique with electrocardiographic monitoring represents a feasible, effective, and potentially life-saving approach.

Our case demonstrates that emergency physicians can successfully perform this procedure using basic equipment and straightforward bedside technique. Femoral venous access is appropriate for emergency pacemaker insertion, offering rapid access and reliable hemostasis. The VVI pacing mode provides adequate ventricular rate support. Initial catheter positioning in the right ventricular outflow tract, while not optimal, does not preclude successful pacing, and subsequent optimization can be performed when fluoroscopic guidance becomes available.

This case encourages further investigation and study of non-fluoroscopic approaches to temporary transvenous pacemaker insertion in the emergency setting. In resource-limited healthcare systems and in circumstances where rapid access to fluoroscopic guidance is unavailable, the blind technique with electrocardiographic monitoring may prove to be an invaluable life-saving approach. Future prospective studies examining the safety, effectiveness, and long-term outcomes of this technique would enhance understanding of its appropriate role in emergency cardiac pacing.

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