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Compromising bradycardia: Management in the emergency department[☆]

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KEYWORDS

Bradycardia;
Cardiac pacing;
Outcome

Summary

Aim of the study: Bradycardia may represent a serious emergency. The need for temporary and permanent pacing is unknown.

Methods: We analysed a registry for the incidence, symptoms, presenting rhythm, underlying mechanism, management and outcome of patients presenting with compromising bradycardia to the emergency department of a university hospital retrospectively during a 10-year period.

Results: We identified 277 patients, 173 male (62%), median age 68 (IQR 58–78), median ventricular rate 33 min⁻¹ (IQR 30–40). The leading symptoms were syncope [94 (33%)], dizziness [61 (22%)], collapse [46 (17%)], angina [46 (17%)] and dyspnoea/heart failure [30 (11%)]. The initial ECG showed high grade AV block [134 (48%)], sinus bradycardia/AV block [46 (17%)], sinu-atrial arrest [42 (15%)], bradycardic atrial fibrillation [39 (14%)] and pacemaker-failure [16 (6%)]. The underlying mechanisms were primary disturbance of cardiac automaticity and/or conduction [135 (49%)], adverse drug effect [58 (21%)], acute myocardial infarction [40 (14%)], pacemaker failure [16 (6%)], intoxication [16 (6%)] and electrolyte disorder [12 patients (4%)]. In 107 (39%) patients bed rest resolved the symptoms. Intravenous drugs to increase ventricular rate were given to 170 (61%) patients, 54 (20%) required additional temporary transvenous/transcutaneous pacing. Two severely intoxicated patients could be stabilised only by cardiopulmonary bypass. A permanent pacemaker was implanted in 137 patients (50%). Mortality was 5% at 30 days.

Conclusion: In our cohort, about 20% of the patients presenting with compromising bradycardia required temporary emergency pacing for initial stabilisation, in 50% permanent pacing had to be established.

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Introduction

The need to treat bradycardias is dictated primarily by the clinical presentation of the patient.¹ Bradycardia may be an incidental finding in asymptomatic patients.² On the other hand, bradycardia may present with haemodynamic instability and loss of consciousness.^{3–6} Out of the bradycardic rhythms to be encountered in these patients, high grade atrioventricular-block represents a significant portion.⁷ Compromising bradycardia requires an effective diagnostic and therapeutic approach.^{1–7}

The goal of initial management is stabilisation by increase of the ventricular rate by both pharmacological and non-pharmacological interventions, sometimes even cardiopulmonary resuscitation is required.^{3–10} A careful clinical workup for potential causes, including ischaemic and non-ischaemic aetiologies, is crucial for successful management.¹¹

There are only a few reports available on the management and outcome of patients presenting with compromising bradycardia to the emergency department (ED).^{1,9,12} Therefore, the primary goal of this analysis was to investigate the need for temporary and permanent pacing. Further, we report the incidence, symptoms, presenting rhythm, underlying mechanism, management, and outcome of patients presenting with compromising bradycardia to the ED.

Methods

Study design

The study was designed as a retrospective analysis from a bradycardia registry. Consecutive patients, (age >18 years), admitted to the ED of a tertiary care 2000 bed university hospital,¹³ within a 10-year period (from March 1994 to March 2004) with compromising bradycardia, represented the study population. This ED is part of a tertiary care university hospital in the capital of a western European country, and provides treatment for life- and non-life-threatening emergencies of all specialties in adults except for trauma patients. Patients were either referred by the local emergency system, by primary care physicians or were self-referred.

Patients with ECG documented bradycardia less than 60 min⁻¹ compromised due to low ventricular rate were eligible. Asymptomatic patients with ventricular rates less than 60 min⁻¹ and patients presenting with bradycardia as a sign of terminal illness were excluded from the analysis.

Leading symptoms

The following symptoms were accepted as leading: syncope, collapse, dizziness, dyspnoea/heart failure and angina.

Cardiac rhythm at presentation

Five rhythm categories were predefined:

- (1) Sinu-atrial arrest without adequate escape rhythm.
- (2) Sinus bradycardia/atrioventricular-block (first degree AV block, second degree AV block-Mobitz I).
- (3) Bradycardic atrial fibrillation.
- (4) High grade AV-block (second degree AV-block-Mobitz II and third degree AV-block).
- (5) Failure of a previously implanted pacemaker device.

Management of bradycardia

Local standards of care at the ED widely conformed with the treatment algorithms published in the guidelines for bradycardia by the ILCOR/ERC.^{3–6} Final implantation of a permanent pacemaker in our hospital is performed by a cardiothoracic surgeon according to current guidelines issued by AHA/ACC/ESC/NASPE¹¹ after consultation with a cardiologist.

Causes and mechanisms of bradycardia

Six categories of underlying mechanism for the bradycardia were predefined:

- (1) Primary abnormality of cardiac automaticity and/or conduction (if no other specific cause could be found).
- (2) Unintended side-effects of therapeutic levels of cardio-active drugs (β -blockers, calcium channel blockers, digitalis glycosides).
- (3) Bradycardia during the course of an acute myocardial infarction.
- (4) Failure of a previously implanted pacemaker device.
- (5) Toxicity due to intended misuse or overdose of cardio-active drugs.
- (6) Severe electrolyte imbalance (serum potassium level >6 mmol/l).

Outcome assessment

A 30-day mortality of patients with compromising bradycardia was determined in all 277 patients by review of medical charts and after discharge by

telephone contact to the patients, their relatives or the treating physician.

Statistical methods

Continuous data are presented as the median and interquartile range (range from the 25th to the 75th percentile). Discrete data are given as counts and percentages. Calculations were performed with Excel (release 10.0) and SPSS for Windows (Version 10.0).

Results

Patients, incidence, symptoms, presenting rhythm

During the 10-year study period 277 patients with compromising bradycardia were managed in the ED. Demographic data and cardiovascular risk factors of the patients are given in Table 1. None of the female patients was pregnant. Thirty patients per year (IQR 16–40) with compromising bradycardia were admitted to the department, which are 6 out of 10,000 patients seeking medical care at the ED for any reason (incidence 0.006%). However, in the subgroup of patients admitted for intermediate and/or intensive care treatment, the incidence is 1%. Nine of the 277 admitted patients were seen primarily by the local trauma unit due to injuries acquired during compromising bradycardia, and then transferred to our department. The leading clinical symptom in patients was syncope [93 (34%)], followed by dizziness [61 (22%)], collapse [46 (17%)], angina [46 (17%)] and dyspnoea/heart failure [30 (11%)].

Median ventricular rate at presentation was 33 min^{-1} (IQR 30–40). Analysis of pre-hospital and in-hospital ECG-recordings showed high grade AV-block in 134 (48%) patients as the presenting

Table 1 Demographics of 277 patients with compromising bradycardia at the ED

Demographics	n = 277
Age (IQR)	68 (58–78)
Male sex (%)	173 (62)
Hypertension (%)	227 (83)
Diabetes (%)	65 (24)
Hyperlipidemia (%)	137 (50)
COPD/cigarette abuse (%)	68 (25)
Coronary artery disease (%)	128 (47)
Arterial occlusive disease (%)	42 (15)
Chronic renal failure (%)	44 (16)

Table 2 Underlying mechanisms of bradycardia in 277 patients

Causative mechanism of bradycardia	n = 277
Disturbance of automaticity/conduction (%)	135 (49)
Adverse drug effect (%)	58 (21)
Acute myocardial infarction (%)	40 (14)
Pacemaker dysfunction (%)	16 (6)
Intoxication (%)	16 (6)
Electrolyte disorder (%)	12 (4)

rhythm. Other rhythms were sinus bradycardia/AV-block [46 (17%)], sinu-atrial arrest [42 (15%)], bradycardic atrial fibrillation [39 (14%)] and pacemaker-failure [16 (6%)].

Causes and underlying mechanisms of bradycardia

A treatable specific cause for compromising bradycardia was identified in 140 (51%) patients including 16 patients with failure of a previously implanted pacemaker. A primary disturbance of cardiac automaticity and/or conduction was assumed in 135 (49%) patients (Table 2).

Emergency management of compromising bradycardia

Flat positioning and bed rest resolved compromising symptoms initially in 107 (39%) patients (Figure 1).

Intravenous drug treatment to increase ventricular rate was started in 170 (61%) patients. Atropine up to 3 mg (mean 1 mg) was administered in 141 patients. Titrated doses of catecholamines were used in 92 patients: orciprenaline in 62, adrenaline in 24 and dopamine/dobutamine in 6, respectively.

In 18 patients with acute myocardial infarction, aminophylline was given to increase ventricular rate. Specific digitalis antidote therapy was administered in three cases, glucagon in one patient with calcium-channel antagonist related intoxication.

As neither drug therapy to increase ventricular rate nor cause-specific treatment could stabilise 54 of the 170 patients, temporary transvenous pacing was attempted. Only in seven of these 54 patients (13%), emergency transcutaneous pacing was attempted as bridging therapy to transvenous pacing. It was successfully established in four. In three patients transcutaneous pacing was ineffective: Either the electrical stimulation documented in the ECG was not followed by QRS complexes, or no palpable pulses were achieved. However, in all seven patients temporary transvenous pacing was

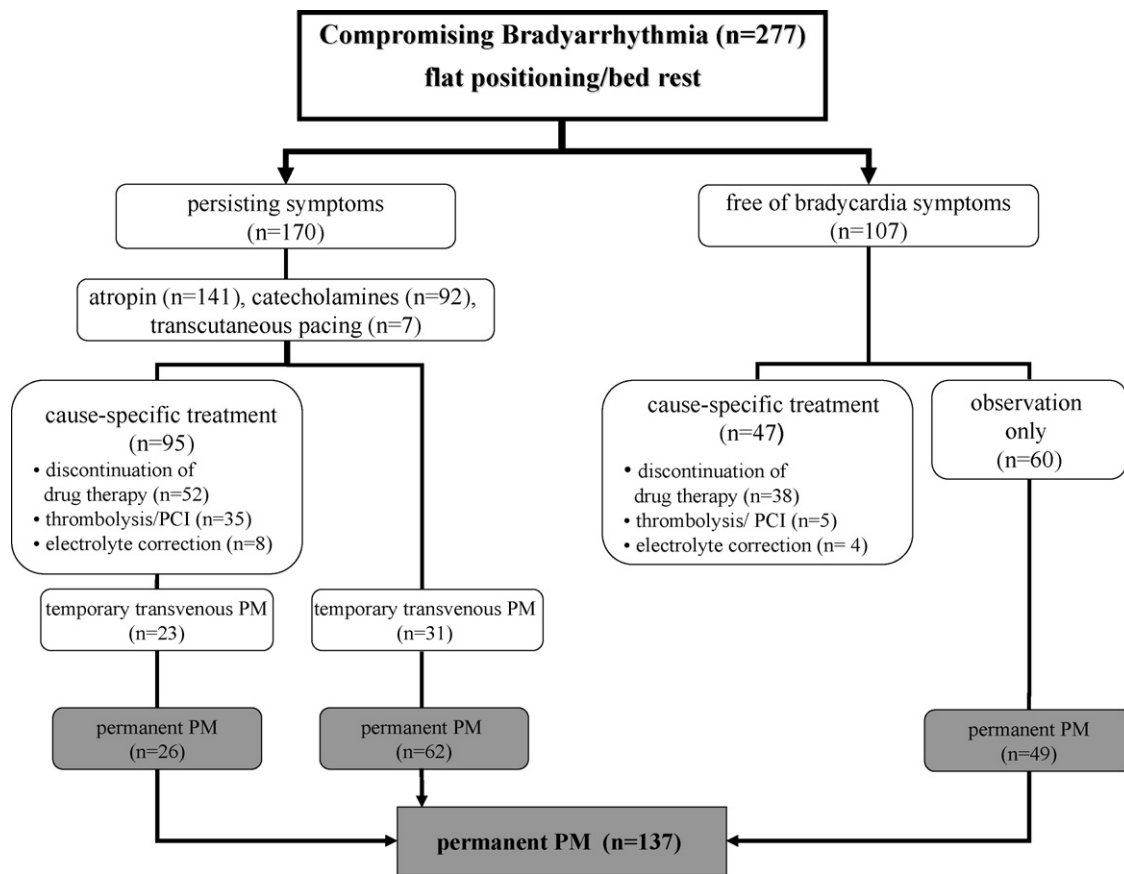


Figure 1 Acute and definitive management of 277 patients with compromising bradycardia at the ED.

established successfully eventually. The underlying mechanisms in the 54 patients requiring temporary transvenous pacing were automaticity/conduction abnormality in 29, acute myocardial infarction in 17, intoxication in five, pacemaker dysfunction in two and severe electrolyte imbalance in one patient, respectively. In three patients (6%) minor complications of temporary transvenous pacemaker insertion (haematoma in two patients, malpositioning of the electrode in one patient) occurred. Out of the 54 patients needing temporary transvenous pacing, 39 were discharged after implantation of a permanent pacemaker.

In two patients who went into cardiac arrest due to intoxication, percutaneous cardiopulmonary bypass was needed to provide adequate tissue perfusion and sufficient cerebral oxygen supply until the drug levels were reduced and restoration of spontaneous circulation could be successfully achieved.

Definite management of bradycardia and outcome

An implantation of a permanent pacemaker was necessary in 137 out of the 277 patients (50%)

(Figure 1). In the majority of these patients (114; 83%) a dual chamber pacemaker system, and in 23 (17%) patients a single lead device, was implanted. Coronary reperfusion, correction of electrolyte disorder, discontinuation of the offending agent as cause-specific therapy could resolve bradycardia successfully in 49 (18%) patients. We referred 24 out of the 277 (9%) patients with transient sinus arrest or sinus-bradycardia as the presenting rhythm for further cardiologic outpatient evaluation. None of them was judged to require a permanent pacemaker later on. Successful adjustment/revision of a previously implanted pacemaker was performed in 16 patients presenting with acute failure of the system (median heart rate 37; IQR 32–40). In 4 patients ‘‘end of life’’ of the pacemaker generator was the malfunction, whereas in 12 other patients a lead dysfunction was discovered.

With exception of the two severely intoxicated patients treated by cardiopulmonary bypass all patients ventricular heart rates were stabilised initially. During a 30-day follow up period, 15 of the 277 patients (5%) died, median 8 days after presentation (IQR 3–16). Nine patients died due to pump failure, six out of the nine suffering

acute myocardial infarction; four patients died in septic shock and two due to severe intracranial bleeding.

Discussion

The main findings of our retrospective analysis are that of the 277 patients presenting with compromising bradycardia, about 20% required temporary emergency pacing for initial stabilisation, and about 50% had to undergo implantation of a permanent pacemaker system. In the majority of patients though, initial stabilisation was achieved by simple interventions including bed rest and the administration of intravenous atropine and/or catecholamines. However, for maintenance haemodynamic stabilisation in about 50% (140 out of 277) of the patients, some kind of cause-specific treatment was necessary.

We would like to emphasize that even if initial stabilisation of a patient by simple measures like bedrest and administration of intravenous drugs is effective, this does not mean that permanent pacing will be avoidable. More importantly, even if the patient did not require temporary transvenous pacing initially does not imply that he will not need insertion of a permanent pacemaker.

We found the incidence of bradycardia in our ED to be 6/10,000 patients/year. According to the specific responsibilities and settings of emergency departments, in units serving different populations, the necessities for specific therapy may be different due to different underlying causes and mechanisms of bradycardia. This has to be taken into account when trying to extrapolate our results to other centres.

Bradycardia is a common finding in general practice, and the clinical presentation may range from an incidental finding in asymptomatic patients to life-threatening heart block.^{1,2,7} In our observation, syncope, dizziness and collapse were the leading symptoms and were present in more than two thirds of the patients. It is crucial for the management of patients with bradycardia that the symptoms have to be recognised as related to a low ventricular rate. We found no publication addressing the frequency of symptoms in patients with compromising bradycardia.

At the out-of-hospital setting, haemodynamically unstable bradycardia may represent a serious life threatening emergency with 30 day mortality rates up to 16%.^{8,9,12} In the ED setting a better outcome may be expected. In our cohort 5% of patients died during the 30 days following the index event, none of them due to bradycardia. Initial

management in our setting included pharmacological interventions, non-pharmacological interventions including transcutaneous and transvenous pacing and circulatory support by cardiopulmonary bypass.

The suspicion of bradycardia in the differential diagnosis of haemodynamically compromised patients will usually be followed by immediate ECG recording and interpretation. We found a high grade AV-block in about half of the patients with compromising bradycardia as the underlying rhythm, which is in line with previous reports.⁷⁻⁹

It is common practice to bring a fainting or collapsing person into a reclining position at first. This manoeuvre initially resolved compromising symptoms in more than 50% of the patients in our cohort. Identification of treatable, reversible causes of the bradycardia is the key for successful management, although pharmacological intervention to increase ventricular heart rate will be the next step. Atropine is the drug of choice.^{8,9,14,15} If, after atropine, there is not an adequate increase in ventricular heart rate, adrenaline or isoprenaline is recommended.³⁻⁶ Encouraging reports have supported aminophylline as a potential drug in the treatment of bradycardia during the course of acute myocardial infarction and/or cardiac arrest.¹⁴⁻¹⁹ In two prospective controlled non-randomised cohort studies in hospitalised adults the administration of aminophylline improved heart rate, symptoms and signs associated with bradycardia that did not respond to atropine.^{16,18} Current guidelines therefore recommend the use of this drug in compromising bradycardia, when other measures are not successful or available.^{20,21}

Glucagon may be an option, particularly in pharmacological therapy of β -blocker and calcium channel antagonist derived bradycardia.²² In our cohort, only one patient received this therapy, therefore no recommendations can be given. Future studies are needed before wider use may be recommended. In summary, in our cohort, intravenous drug treatment to increase ventricular rate was initiated in 61% of the patients: Atropine up to 3 mg and titrated doses of catecholamines were used in a majority of these patients.

Compromising bradycardia in acute myocardial infarction may represent a serious problem when complicating extended anterior wall myocardial infarction with cardiogenic shock. In inferior/posterior myocardial infarction, bradycardia will usually be transient and benign.^{20,21} In our observation, 6 of the 40 patients with compromising bradycardia during the course of acute myocardial infarction died during their hospital stay due to heart failure.

In our series, intoxication was the cause of bradycardia in 16 cases. Two of these cases, patients with severe prolonged but reversible cardiotoxicity, may have benefited from the use of cardiac assist devices. Antidote treatment in compromising bradycardia as part of intoxication is commonly described in literature.²³ However, if mechanical capture of electric heart activity is hindered by toxic effects, CPR and cardiac assist devices may be necessary during the time of elimination of the toxin. At present, however, there is insufficient evidence concerning the use of cardiac assist devices as a treatment for severe cardiac impairment due to poisoning.²⁴

Both percussion/fist pacing and transcutaneous pacing represent techniques may help to bridge to the next therapeutic step. Percussion pacing is not established in our ED. Transcutaneous pacemaker therapy is a safe, effective non-invasive ventricular stimulation that is tolerated also in conscious patients and allows clear recognition of the electrocardiographic response.^{25–29} In our study cohort, this was used infrequently as the insertion of a temporary transvenous system is well established. This can be a lifesaving procedure in the emergency department, but will not be available in the prehospital setting. Birkhahn et al. recently reported a 97% success rate in transvenous pacemaker insertion at the ED.³⁰ In our analysis, temporary transvenous pacing was attempted in 54 patients and established successfully in all of them.

After initial stabilization a careful clinical workup is mandatory for all patients. This should include history taking, physical examination and laboratory studies including tests of thyroid function as well as continuous 24- to 48-h electrocardiographic recording.^{2,11} Thus, a wide range of possible intrinsic or extrinsic causes of sinus-node dysfunction or atrioventricular-block may be discovered. Consecutively, in our cohort, a very high percentage of patients had to be provided with a permanent pacemaker. This may be explained by the great number of patients with high grade AV-block, sick-sinus syndrome and pacemaker dysfunction, which may be different under other conditions and settings.

Limitations

As this is a retrospective observational study, patients were not randomly assigned to various treatments. Therefore we cannot exclude a selection bias. This study is, however, a reflection of real-life conditions, and treatment was based on internationally acknowledged guidelines which

were followed to a very high degree. Further, this is a consecutive series of patients, which further reduces the effect of selection bias. The observational period was long (10 years), but there was only a negligible change in the recommended procedures.

Conclusions

Temporary emergency transcutaneous/transvenous pacing was necessary in 20% of the cases. Therefore training in temporary emergency pacing techniques seems to be warranted and mandatory for emergency physicians. Recognition of bradycardia as a cause for the presenting symptoms and awareness for the underlying mechanism are both crucial for timely and effective treatment. Patients might benefit further if expert help from toxicologists, cardiologists or surgeons is sought early.

Conflict of interest statement

Hereby, I state that there are no conflicts of interest of my coauthors and myself regarding the proposed manuscript "Compromising bradyarrhythmias: Management in the emergency department".

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