Prevalence of hypotensive disorders in older patients with a pacemaker in situ who attend the Accident and Emergency Department because of falls or syncope

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Aims To ascertain the proportion of adults with a pacemaker in situ attending the Accident and Emergency Department because of syncope or unexplained falls and the cause of index symptoms in these patients, including the prevalence of hypotensive syndromes.

Methods and results Patients presenting to the Accident and Emergency Department with unexplained syncope or non-accidental falls, who had a pacemaker in situ, were studied. Eligible patients had cardiovascular assessment (morning orthostatic blood pressure measurement, heart rate and BP measurements during carotid sinus stimulation (supine and upright), head-up tilt at 70° for 40 min), assessment of haemodynamics during fixed mode pacing and gait and balance assessment.

Of 5863 patients screened, 13.5% had unexplained syncope or a non-accidental fall; of these only 3% (26 patients) had pacemakers in situ. Of 18 study patients (82 ± 8 years), 10 were female. Sixteen had a hypotensive diagnosis. Seven had more than one attributable hypotensive diagnosis. Five of 13 with vasodepressor carotid sinus syndrome had no previous diagnosis of carotid sinus hypersensitivity. No patients had vasovagal syncope induced during passive head-up tilt testing.

Conclusion It is rare for patients who attend the Accident and Emergency Department because of syncope or unexplained falls to have a pacemaker in situ. In those who do, hypotensive disorders are a common finding.

(Key Words: Pacemaker, Accident and Emergency, syncope, hypotension.)

Introduction

Syncope is defined as a transient loss of consciousness characterized by unresponsiveness and loss of postural tone[1]. It occurs as a result of a temporary reduction in cerebral flow to those parts of the brain that control the level of consciousness[2]. The elderly are prone to syncope[1,2] because of age-related physiological changes which diminish the ability to adapt to a sudden fall in blood pressure. The overall prevalence of syncope in the elderly is not known. Case series have shown a prevalence of up to 23% in institutionalized elderly[3]. Syncope in general accounts for 3% of Accident and Emergency Department (A&E) visits and 1% of medical admissions to a general hospital[4].

At least 20% of cardiovascular syncope in the elderly presents as unexplained falls[2,5]. An accurate history or witness account is required to separate falls and syncope and this is unlikely to be available in over a third of elderly patients[5,6]. For example, 60% of patients with carotid sinus syndrome (CSS) who present with recurrent unexplained falls, demonstrate amnesia for witnessed loss of consciousness during induced asystole[7]. Similarly, over 20% of older patients for whom orthostatic hypotension is an attributable cause of syncope only acknowledge falls and loss of postural balance and deny loss of consciousness[5,6].

The majority of patients who require pacemakers for bradycardia are elderly (over 70% of implants in the U.K. are in patients aged 70 years and over[8]). The commonest aetiologies are sinus node disease and...
atrioventricular block\cite{9}. In Newcastle, since the establishment of a dedicated syncope facility for older adults in 1991, the number of pacemakers implanted for CSS has risen from 2% of new implants in 1991 to over 24% of new implants in 1998\cite{10}.

It is most likely that the principal cause of syncope in older patients who have a pacemaker in situ is hypotension (such as orthostatic hypotension, vasodepressor carotid sinus hypersensitivity, pacemaker syncope or vasovagal syncope). Other less common causes include: pacemaker malfunction or medical conditions such as epilepsy, transient ischaemic episodes, pulmonary emboli, myocardial infarction or ventricular arrhythmias.

As part of an ongoing prospective screening programme of older patients attending the A&E Department with syncope and falls, it was noted that a proportion had a permanent pacemaker (PPM) in situ at presentation. The cause of symptoms in these patients was unclear and there were no previous publications detailing causes in this population.

The objective of this study was to determine, in older adults who attended the A&E Department because of syncope or a fall, what proportion of patients have a pacemaker in situ, and the cause of index symptoms in these patients including the prevalence of hypotensive disorders.

**Methods**

**Study population**

The study took place at inner-city A&E Departments in Newcastle upon Tyne during a 6 month period. All patients aged over 50 years were screened by interview between 09:00 and 19:00 daily. Those presenting out of hours (14%) were screened by telephone communication on the following morning. Patients eligible for study inclusion were those who presented with unexplained falls and had previously undergone PPM implantation. An unexplained fall was defined as ‘coming to rest at a lower level, with or without loss of consciousness’\cite{11} with no clear explanation for the episode (such as trip or slip) or apparent medical cause (such as epilepsy, dysrhythmia, etc). Patients were excluded if they: (1) presented with an accidental fall (simple slip or trip); (2) had a readily or subsequently diagnosed medical cause of syncope or falls within 10 days of presentation (e.g. myocardial infarction, stroke, gastrointestinal haemorrhage, alcohol intoxication, dysrhythmia, etc); (3) had cognitive impairment (Mini-Mental State Examination <24/30); patients with cognitive impairment would be unable, accurately to describe a fall; (4) were unable to speak English or were illiterate; (5) were immobile; (6) were registered blind or (7) had contraindications to carotid sinus massage. Details of this screening process are published elsewhere\cite{13}.

Eligible patients were invited to attend the cardiovascular investigation unit for further assessment. A full history, examination and a semi-structured interview took place, details of symptoms (syncope, falls, dizziness, injuries) and current medication were recorded. Patients were asked if symptoms of dizziness, syncope or falls had improved, remained the same, or deteriorated since PPM implantation. Aetiological indications for pacemaker insertion documented at the time of implant were obtained from the regional pacing database.

Patients had a cardiovascular and neurological assessment. Cardiovascular assessment included blood pressure and heart rate changes during active standing (when the patient was asked to stand unsupported for 3 min after 5 min supine rest) and carotid sinus massage (CSM). CSM was carried out when supine and then upright (70° head-up tilt). Longitudinal massage was performed over the site of maximal carotid impulse (superior to the upper border of the thyroid cartilage and approximately 2 cms inferior to the angle of the jaw). CSM was carried out for 5 s on the right and then on the left sides after an interval of 1 min and following stabilization of heart rate and blood pressure. All patients also had a prolonged head-up tilt at 70°C for 40 min. Heart rate and phasic blood pressure were recorded non-invasively during these tests using surface electrocardiogram (paper speed 25 mm/s) and digital photoplethysmography (Finapres, Ohmeda\cite{8}). Pacemaker function was assessed using standard methods to monitor programmed parameters\cite{14}.

Contraindications to carotid sinus massage were myocardial infarction within the previous 3 months, cerebrovascular event within the previous 3 months, a recent history of ventricular dysrhythmia or the presence of a carotid bruit\cite{15}.

All patients also had a full neurological assessment including gait and balance. Patients walked a fixed distance (12 m) and the number of steps and the time taken to complete these was recorded. Gait speed (m . s\(^{-1}\)) and stride length (m) were calculated from this. Mobility with or without the use of walking aids was also calculated.

**Definition of abnormal responses**

**Orthostatic hypotension** was defined as a fall in systolic blood pressure on 20 mmHg or more which was sustained after 3 min of standing\cite{16}.

**Vasodepressor CSH** was defined as a 50 mmHg or more fall in systolic blood pressure during CSM either supine or when tilted upright\cite{15}.

**Pacemaker syndrome** was defined as reproduction of adverse haemodynamic effects (hypotension), signs (cannon waves) or symptoms (presyncope, syncope, shortness of breath) during induced ventricular pacing, with surface ECG documentation of retrograde atrioventricular conduction (paper speed 50 mm . s\(^{-1}\))\cite{14}.

**Abnormal gait** was defined as gait speed and stride length outside published values for healthy age matched controls. Normal gait is 0.8–1.5 m . s\(^{-1}\) and stride length 0.94–1.71 m\cite{17}.
Vasovagal syncope was a fall in systolic blood pressure in excess of 50 mmHg with symptom reproduction during head up tilt at 70° for 40 min.

An abnormal haemodynamic response or gait response was considered an attributable cause of symptoms, if symptoms were reproduced during the response; if symptoms were reproduced during more than one manoeuvre each was considered a possible attributable cause.

The study was approved by the Local Ethics Committee and informed consent was obtained from all subjects.

Results

During the 6 month screening period, 5863 patients aged 50 years and over presented to the accident and emergency departments with syncope or falls. Seven hundred and ninety-four had syncope or unexplained falls (13·5%). Twenty-six of the latter had permanent pacemakers in situ (3%). Of these, five declined further study and three had contraindications to repeat carotid sinus massage (Fig. 1).

Of the remaining 18 patients, mean age 82 ± 8 years (range 62–94 years), 10 were female. Cardiovascular comorbidity (in 11 patients) included ischaemic heart disease (7), transient ischaemic cerebral episodes or stroke disease (2), chronic cardiac failure (1) and systolic or diastolic hypertension (3).

Previous symptoms necessitating pacemaker implantation were syncope in 14 and presyncope in four. Aetiological diagnoses pre-implant were carotid sinus syndrome (CSS) (10), conduction tissue fibrosis (5), post-ablation (1), postmyocardial infarction (1) and sick sinus syndrome (1).

Twelve patients had dual chamber systems (10 with CSS, one postmyocardial infarction and one sick sinus syndrome). The remaining six had single chamber ventricular systems: two were in atrial fibrillation. The average time since pacemaker implantation was 3 ± 2 years (range 1–6 years).

The commonest baseline rhythm pre-implant was sinus rhythm in eight patients. Two patients had sinoatrial arrest, one patient had bradycardia, two patients were in atrial fibrillation, four patients had complete heart block and one patient had right bundle branch block.

Eighty per cent reported fewer episodes of syncope since implant, 54% reported fewer falls and 45% noted dizziness was less marked. The mean time since implant was 3 years (range 1–6 years).

Seventeen patients had a hypotensive diagnosis — orthostatic hypotension in 10, vasodepressor carotid sinus hypersensitivity in 13, and pacemaker syndrome in one (Fig. 2). Hypotensive diagnoses overlapped in seven.

Symptomatic hypotension during testing was present in 11 patients: orthostatic hypotension in two, vasodepressor carotid sinus hypersensitivity in 10 and pacemaker syndrome in one, overlap occurred in two (orthostatic hypotension and vasodepressor carotid sinus hypersensitivity in one and pacemaker syndrome and vasodepressor carotid sinus hypersensitivity in the other).

Of 13 patients with a significant vasodepressor response during carotid sinus massage, 10 had symptom...
reproduction (loss of consciousness in two, pre-syncope in three, and severe dizziness in five). Five of 13 patients with vasodepressor carotid sinus hypersensitivity did not have a previous diagnosis of carotid sinus syndrome. Previous diagnoses in these were chronic tissue fibrosis (2), post-ablation (1), post-myocardial infarction (1) and sick sinus syndrome (1). Of these five patients, four had dizzy symptoms during CSM and one lost consciousness (this patient also had symptomatic pacemaker syndrome).

The mean vasodepressor response was $66 \pm 13$ mmHg for all 13 patients. In 10 patients who experienced reproduction of symptoms — either dizziness or syncope — during carotid sinus stimulation the vasodepressor response was $70 \pm 12$ mmHg (ns). The response was predominantly right sided (66%), and elicited only when upright in seven patients (58%).

Of 10 patients who had orthostatic hypotension, the fall in blood pressure was transient in eight. The remaining two patients had symptom reproduction during orthostatic hypotension (one had syncope and one presyncope). Both of these patients also had carotid sinus syndrome. Average fall in blood pressure in these ten patients was $47 \pm 25$ mmHg.

No-one had symptom reproduction or haemodynamic changes diagnostic of vasovagal syncope during prolonged passive head-up tilt testing.

Five patients had abnormal gait and stride length. All also had a hypotensive diagnosis — orthostatic hypotension in one and vasodepressor carotid sinus hypersensitivity (CSH) in four.

**Medication**

The mean number of tablets per patient was $4 \pm 2$ (range 1–8). Probable culprit medication (i.e. cardiovascular medications known to contribute to abnormal blood pressure or heart rate responses) was prescribed in seven patients. Cardiovascular medications were prescribed for one patient with orthostatic hypotension who did not have symptom reproduction during orthostatic stress and in six patients with vasodepressor CSH (diuretics (3), calcium channel blockers (2), nitrates (1), ACE inhibitors (1)). Two of these had symptom reproduction during vasodepressor CSH.

**Discussion**

Because of the overlap between syncope and unexplained falls in the elderly, both symptoms were included in this series. Previous work has shown that up to 30% of patients with carotid sinus syndrome present with unexplained falls and deny syncope. Up to 20% of patients with orthostatic hypotension similarly present with falls and deny syncope. The incidence of unexplained falls in patients with sinus node disease, atrioventricular conduction disease or atrial fibrillation has not been reported.

Patients with cognitive impairment were excluded, thus patients should have been able to recall both the circumstances and characteristics of the fall and whether or not testing reproduced symptoms. Most patients had symptoms of both syncope and falls prior to pacing, which improved in the majority after pacemaker implant. Further data on the frequency of these symptoms, comparing pre- and post-implant data, are not detailed because of poor reliability of such retrospective information.

This was a carefully screened population of consecutive adult attendances at the A&E Department. From this large series, a fall was the commonest cause of attendance — 37% of all attendees over 50 years, 45% of those over 65 years and 52% of over 75 years old. Eighty-six per cent of all fallers were screened at the time of presentation and the remainder were screened by telephone communication the following day. The implant rate for the same catchment area is 416 million per year, or 257 pacemaker systems per year. Although the objective of the study was to determine the prevalence of hypotensive disorders in paced patients attending the A&E Department, the number of such patients was small, i.e. 3% of in-scope attendees.

Comorbidity is common in older patients, as is polypharmacy. Attributing a cause for symptoms is difficult. The finding of an exaggerated vasodepressor response to carotid sinus massage does not necessarily imply that this caused the index presentation. Symptoms were attributed to a hypotensive disorder if blood pressure changes during either carotid sinus stimulation, or standing, or retrograde atrioventricular conduction reproduced dizziness, presyncope, syncope or falls. In this context, hypotensive disorders were present in 17 patients and attributable causes of symptoms in 11. The commonest hypotensive disorder was vasodepressor carotid sinus hypersensitivity — which was present in eight of 10 patients who had a pacemaker implanted for carotid sinus syndrome and in a further five patients who had systems implanted for other indications. This may be because carotid sinus hypersensitivity co-existed with the original indications for pacing or because the original diagnosis was incorrect, or carotid sinus hypersensitivity evolved in the intervening period since implant.

Most patients were on cardiovascular medications known to exaggerate either orthostatic hypotension or vasodepressor carotid sinus responses. Intervention strategies for hypotensive disorders in patients with pacemakers include manipulation of culprit medications, non-pharmacological and pharmacological treatment of orthostatic hypotension and reprogramming of pacing modes to modify vasodepressor responses.

In only two patients were cardiovascular medications considered to be an attributable cause of symptoms. Symptoms due to pacemaker syndrome are alleviated by upgrading pacing systems. Only one patient in this series had pacemaker syndrome as a possible attributable cause of symptoms. In our practice, 90% of mobile patients who are in sinus rhythm, receive dual chamber
systems and this may explain the low incidence of pacemaker syndrome. Five patients had an abnormal gait which may have contributed to the index fall, all of these also had an underlying hypotensive disorder. We have previously hypothesized that a combination of moderate haemodynamic changes with background gait and balance instability may cause falls in older adults\(^{20}\). Overlap for hypotensive disorders and gait and balance disturbances is not uncommon in this population\(^{21}\).

In conclusion, it is rare for patients who attend the A&E Department because of unexplained syncope or falls to have a pacemaker in situ. In the small number of cases who do, hypotensive disorders are a common finding and common attributable cause of symptoms.

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References