Pacemaker syndrome in older people

RUTH A. ROSS, ROSE ANNE KENNY

Cardiovascular Investigation Unit, Department of Medicine, School of Clinical Science and Institute of the Health in the Elderly, Newcastle upon Tyne, UK

Address correspondence to: R. A. Kenny, Cardiovascular Investigation Unit, Victoria Wing, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP, UK. Fax: (+44) 191 222 5638 Email: r.a.kenny@ncl.ac.uk

Keywords: pacemaker syndrome, pacing, elderly people

Introduction

Pacemaker syndrome arises from adverse electrophysiological and haemodynamic consequences of pacing—in particular ventricular pacing—despite normal pacemaker function [1, 2]. The symptoms are diverse, ranging from breathlessness, syncope, palpitations, neck pressure, dizziness and flushing to more vague symptoms of lethargy and impaired exercise tolerance. Clinical signs include cannon ‘a’ waves in the jugular venous pressure waveform, intermittent or sustained hypotension and congestive cardiac failure [3].

In the normal cardiac cycle there is a sequential electrical depolarization from atria to ventricles. Loss of this co-ordinated sequence has profound consequences on cardiac output [4]. When ventricular function is normal, the atrial contribution to cardiac output can be up to 25% [5]. With advancing years the ventricles thicken and become stiffer, and consequently the atrial contribution to cardiac output becomes even more important [6]. Thus, older adults are more vulnerable to the consequences of loss of atrioventricular synchrony. Loss of atrioventricular synchrony occurs with single chamber ventricular pacing.

Adverse effects of pacing

During ventriculo-atrial conduction cardiac conduction spreads back towards the atria from the paced right ventricle. The potential for this retrograde conduction is preserved unless the atroventricular node is damaged, as 90% of ventriculo-atrial conduction is via the atroventricular node. If ventriculo-atrial conduction occurs, atrial contraction can coincide with ventricular systole (during ventricular pacing) against closed atrioventricular valves. This results in raised mean pressures in both atria, a fall in cardiac output [1, 7, 8] and altered neurohumeral factors such as catecholamines and atrial natriuretic peptide. This sequence is responsible for the syndrome.

Autonomic effects

There are changes in vascular tone, the sympathetic nervous system and atrial natriuretic peptide (triggered by atrial distension) during pacing [9–12]. The autonomic nervous system may adapt to pacing by enhancing sympathetic activity (modulated by arterial baroceptors which are triggered by low blood pressure) during ventricular pacing. Atrial distension and elevated arterial and pulmonary pressures result in inhibitory reflexes mediated by the vagal nerve to oppose sympathetic vasoconstriction. In some patients, these opposing reflexes result in an inadequate vasoconstrictor response and decreased vascular tone.

Diversity in these autonomic reflexes explains the variability of symptom severity of the syndrome [2]. Congestive cardiac failure results from altered autonomic tone and elevated atrial natriuretic peptide levels as a consequence of high atrial pressures.

Types of pacemakers

Pacemaker function is coded by five letters [13]. The first letter refers to the chamber being paced [A (atrium), V (ventricle) or D (dual)]; the second to the chamber being sensed (A, V, D) and the third to the mode of sensing [T (triggered), I (inhibited), D (both) or O (magnet function)]. When evaluating pacing function, placing a magnet over the pacemaker generator results in asynchronous pacing i.e. no electrical signals are sensed and the pacemaker generator delivers output pulses without regard to spontaneous cardiac electrical activity. The fourth letter denotes whether the pacemaker is rate-responsive. This means that the pacemaker paces at a pre-set rate at rest but will increase the pacing rate according to a programmable algorithm in response to a sensed physiological or semiphysiological trigger. The fifth letter relates to anti-tachycardia devices—P [pacing (anti-tachyarrhythmia)], S (shock) or D (dual).
Pacemaker syndrome predominantly occurs with VVI pacing and provoked ventriculo-atrial conduction. However, it has been described in atrial and in dual chamber pacing modes if atrioventricular synchrony is lost. For example, in DVI mode (in which the atria are not sensed), a fast atrial rate may compete with generated atrial activity. In DDI mode, atrial depolarization may occur at the end of the atrial refractory period, prolonging the atrioventricular interval excessively. In DDD mode, a pseudo-Wenckebach or higher-degree atrioventricular block may occur [1, 14].

Current guidelines recommend DDD or DDI pacing for patients predominantly in sinus rhythm [15, 16]. VVI or VVIR systems are recommended in atrial fibrillation. In sinus node disease, DDD systems are often implanted in preference to AAI systems because of possible development of atrioventricular block [17].

Reports of the incidence of pacemaker syndrome vary between 7% [18] and 83% [19, 20]. The variation depends on the criteria used to make the diagnosis, the age of the patients studied and comorbidity. In patients with carotid sinus syndrome, clinical characteristics before pacing cannot predict the risks of developing the syndrome [21]. In a randomized controlled trial of single ventricular versus dual chamber pacing in 407 patients over 65 with either atrioventricular conduction disease or sinus node disease, 26% of those initially randomized to VVIR pacing were upgraded to dual chamber systems because of pacemaker syndrome [22].

If pacemaker syndrome is suspected, a diagnosis can be confirmed by placing a magnet over the pacemaker, thus converting the system to VVO mode—fixed pacing at a pre-determined rate. In most patients with pacemaker syndrome this will induce ventriculo-atrial conduction [23]. The P waves (leads II or V2) occur with a fixed relationship to paced QRS complexes—generally interval lengths of less than 400 ms from the pacing spike. This electrocardiogram pattern correlates with reduced stroke volume and low systolic blood pressures. Patients may experience symptoms if they are tested upright in VVO mode.

Treatment

Intervention strategies include changing from single to dual chamber pacing and re-programming to restore a more physiological conduction pattern.

Traditionally, pacing leads are positioned in the right atrial appendage and right ventricular apex. Newer alternative sites include the left atrium, intratrial septum and four chamber pacing [24, 25]. Preliminary hemodynamic data from alternative pacing sites are encouraging, but experience is limited, surgical access is more difficult and the feasibility in older patients has not been studied.

In the UK over two-thirds of pacemaker implants are in people over 70. Single chamber systems are usually implanted in elderly patients [26]. Because of age-related cardiovascular and cerebrovascular physiological changes (including reduced cardiac output, blunted autonomic compensatory responses and altered cerebral autoregulation), the adverse consequences of ventricular pacing should be more marked and more common in older patients [6, 27, 28].

Few researchers have measured self-perceived health or mental status, health-related quality of life or cognitive function for different pacing modes [19, 20, 29, 30]. Some domains from the SF-36 instrument are better for dual chamber systems than single ventricular chamber systems (in sinus node dysfunction). There are no significant differences in patients with atrioventricular conduction disease [22]. Interpretation of these data is difficult as many patients changed from single to dual chamber systems because of pacemaker syndrome.

The cost of pacemakers varies according to the complexity of the device implanted. In our centre, a single chamber system may cost from £600 to £1500 (before tax). Dual systems cost twice as much. All dual systems incur additional costs for the second lead of approximately £200.

UK studies of single versus dual chamber pacing in older patients with atrioventricular conduction disease (UKPACE) [31] and sinus node disease (STOP-AF) [32] are under way. Both studies are evaluating health status, mental status, cognitive function and symptoms of pacemaker syndrome. These prospective randomized trials may resolve the issues of appropriateness and cost-effectiveness of the different pacing modes, and determine appropriate evidence-based practice.

Key points

- Pacemaker syndrome results from adverse electrophysiological and haemodynamic consequences of pacing, and predominantly occurs with VVI pacing and provoked ventriculo-atrial conduction.
- Studies comparing health status, mental status, cognitive function and symptoms of pacemaker syndrome as a result of single and dual chamber pacing in older patients with atrioventricular conduction disease and sinus node disease are under way.

References


Pacemaker syndrome in older people