

Stroke rehabilitation therapy in a patient with a cardiac pacemaker for chronic atrial fibrillation

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A 65-year-old man was implanted with an artificial pacemaker for chronic bradycardic atrial fibrillation associated with hypertensive heart disease. Five years after the pacemaker implantation, he suffered from a cerebral embolism. Approximately 4.5 months after the ictus, he was transferred to the rehabilitation ward. He had flaccid left hemiplegia and severe disuse syndrome. He could not sit and could tilt his head up for only two minutes because of severe orthostatic hypotension. By modulating the rate-responsive mode of the pacemaker every 2–4 weeks, we were able to rehabilitate the patient. Thus, the patient could sit in a wheelchair for more than three hours. This case emphasizes the importance of examining the mode and function of a previously implanted artificial pacemaker. In accord with varying rehabilitation programs and gradual

improvement in a patient's physical activities, periodic modulation of a programmable pacemaker can lead to a better functional outcome during rehabilitation therapy. *International Journal of Rehabilitation Research* 26:317–321 © 2003 Lippincott Williams & Wilkins.

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Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered in clinical practice and accounts for 35% of arrhythmia diagnoses (Kottkamp *et al.*, 1998). Non-valvular (NV) AF has an overall prevalence of 0.4% in adults (Kannel *et al.*, 1982). The prevalence of NVAF shows a strong age-dependency with a substantial increase to 2–4% after the age of 60 (Kannel *et al.*, 1983). Between 20 and 26% of patients with NVAF have congestive heart failure (CHF) associated with underlying diseases and/or loss of booster pump action, i.e., asynchronous atrioventricular (AV) sequential contraction, which contributes 10–30% of the cardiac ejection fraction at rest or during exercise (Benjamin *et al.*, 1994, Atwood *et al.*, 1997).

Chronic NVAF is not an entirely benign arrhythmia because it is associated with significant morbidity, including an increased susceptibility to cerebral embolism (Wolf *et al.*, 1998). The annual incidence of cerebral embolism among elderly patients with chronic NVAF reaches 3–9% (6% on average) (Cerebral Embolism Task Force, 1986; Hinckley *et al.*, 1998). Ischaemic stroke lesions associated with AF are mostly large in the total area of occluded arteries ('territorial infarct') because of their sudden onset and little time to develop sufficient collateral circulation (Hinckley *et al.*, 1998, Warlow *et al.*, 2001). Functional disability from the ictus is usually severe (Karataş *et al.*, 2000). In addition, chronic AF restricts the endurance of exercise and (Atwood *et al.*, 1997), therefore, can limit or even prevent

the progression of routine rehabilitation programs for stroke patients.

In the clinical practice of stroke rehabilitation therapy, patients with an artificial pacemaker for chronic AF are not uncommon. With these patients, we are inclined to recognize the importance of pacing failure, but not necessarily the functional mechanism of the pacemaker. This case report demonstrates the valuable experience of successful stroke rehabilitation therapy in a patient with bradycardic AF and whose programmable pacemaker was regularly and appropriately modulated to resolve orthostatic intolerance/hypotension in the course of his rehabilitation therapy.

Case report

A 65-year-old, right-handed man suffered from CHF (New York Heart Association: NYHA functional class II) (Harvey *et al.*, 1974) due to chronic NVAF and sick sinus syndrome associated with hypertensive heart disease. Although he had no episodes of syncope attack associated with low cardiac output, i.e., Adams-Stokes syndrome, examinations by ambulance dynamic electrocardiogram (ECG) revealed frequent episodes of heart rate lower than 40 beats/min. A single chamber pacemaker (Guidant VIGOR SR, Model 1130, Indianapolis, IN, USA) in VVI-R (ventricular pacing, ventricular sensing, inhibition response, rate adaptation) mode was implanted to treat the bradycardia and CHF (Gregoratos *et al.*, 1998). The minimum pacing rate had been set at 70 pulses per minute, and the rate-adaptive pacing function had not

been turned on. After implantation of the pacemaker, he functioned well as an office worker and could play a round of golf (CHF in NYHA class I).

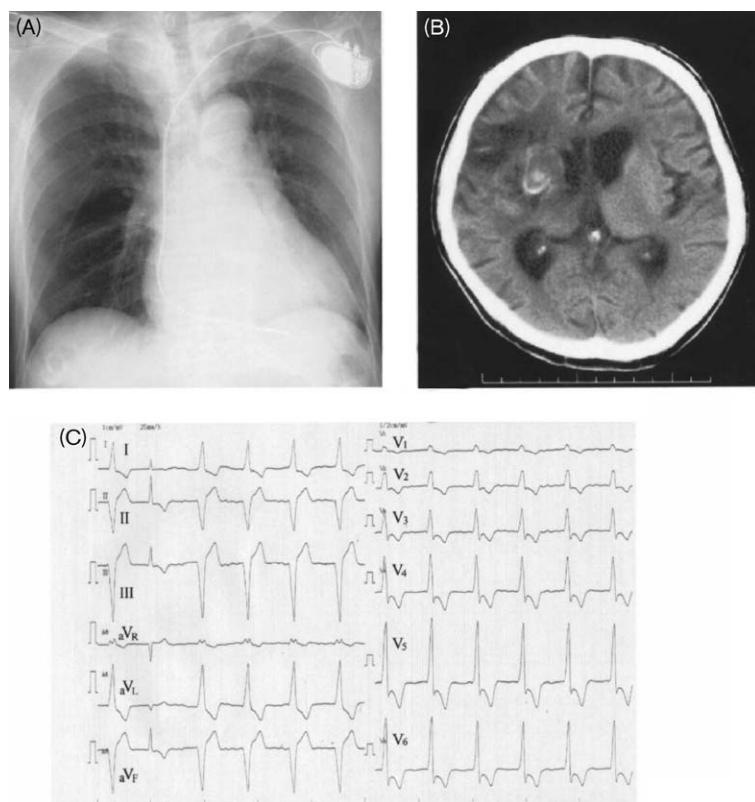
On November 15, 2000, approximately five years after the pacemaker implantation, he had a sudden stroke with left hemiplegia and unconsciousness, and was admitted to an emergency care unit. A computed tomography (CT) scan revealed hemorrhagic infarction in a large area of the brain perfused by the right middle cerebral artery. Because an angiographical examination denoted complete occlusion of the right internal cerebral artery, he was diagnosed as having a cardiogenic cerebral embolism associated with chronic AF. Although he was treated conservatively, he had serious complications of pneumonia and CHF, which caused severe disuse syndrome.

Approximately 4.5 months after the ictus, he was transferred to our rehabilitation ward for intensive therapy. On admission, his chest X-ray showed CHF with a cardiothoracic ratio (CTR) of 62% (Figure 1A). A head CT scan denoted large, low-density areas with partial high-density areas in the right hemisphere of the brain (Figure 1B). An ECG revealed chronic AF, poor AV conduction, and a heart rate of 70 beats/min, which was

imposed almost all of the time by the artificial pacemaker beats (Figure 1C). His activities of daily living (ADL) were totally dependent (Barthel index = 0). Clinical problems at the start of rehabilitation therapy were: (1) severe disuse syndrome after prolonged immobilization on a bed; (2) flaccid severe left hemiplegia (Brunnstrom's stage: all I for arm, hand and leg); (3) higher cortical dysfunction; (4) totally dependent status in ADL only on a bed; and (5) complication of CHF (NYHA class IV).

The patient could not sit or even tilt his head up for two minutes, because of severe orthostatic hypotension with dizziness and presyncope, and flaccid left hemiplegia. The pulse rate was 70 beats/min, and the blood pressure was 122/76 mmHg in the supine position and it fell to 92/54 mmHg in the sitting position with chronotropic incompetence, i.e., heart rate of 70 beats/min. At the start of intensive rehabilitation therapy, he was treated not only by continuous medications, including digoxin (0.125 mg/day), torasemide (8 mg/day) and denopamine (40 mg/day), but also by fine modulation of the programmable pacemaker. Its function was reset every 2–4 weeks, depending on the various rehabilitation programs that were initiated and the levels of physical activity that were required. The functions that were modulated were: (1)

Fig. 1



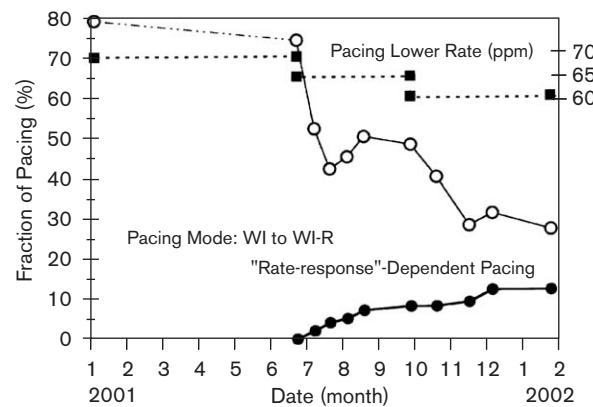
Chest X-ray (A), head CT scan (B) and electrocardiogram (C) on admission.

minimum (optimal) pacing lower rate; (2) rate-adaptive pacing rate in response to motion/activity, including upper sensor rate, activity threshold, activity acceleration time, response factor and recovery time. Firstly, we adopted physiological rate-responsive function to attain chronotropic competence by paced heart beats, which could successfully treat the orthostatic hypotension upon orthostatic stress. The fall in blood pressure was not significant; his blood pressure was 124/78 mmHg in the supine position and 120/80 mmHg in the sitting position for 10 minutes with chronotropic competence (heart rate of 80 beat/min).

Figure 2 shows typical histograms of paced or sensed (intrinsic) beats during each periodic check-up. The physiological rate-responsive pacing in response to physical activity, i.e., exposure to orthostatic stature, gradually and substantially increased during the course of hospitalisation for rehabilitation. The fraction of rate-responsive pacing to the total pacing in every interval of pacemaker check (Fig. 2B, C and D) obviously and gradually increased as compared to that by the initial check (Fig. 2A) in the rehabilitation ward. The time in which the heart was paced decreased to approximately 30%. Meanwhile, the fraction of rate-response-dependent pacing increased from zero to as much as 12% of the total number of heart beats (Figure 3). Especially

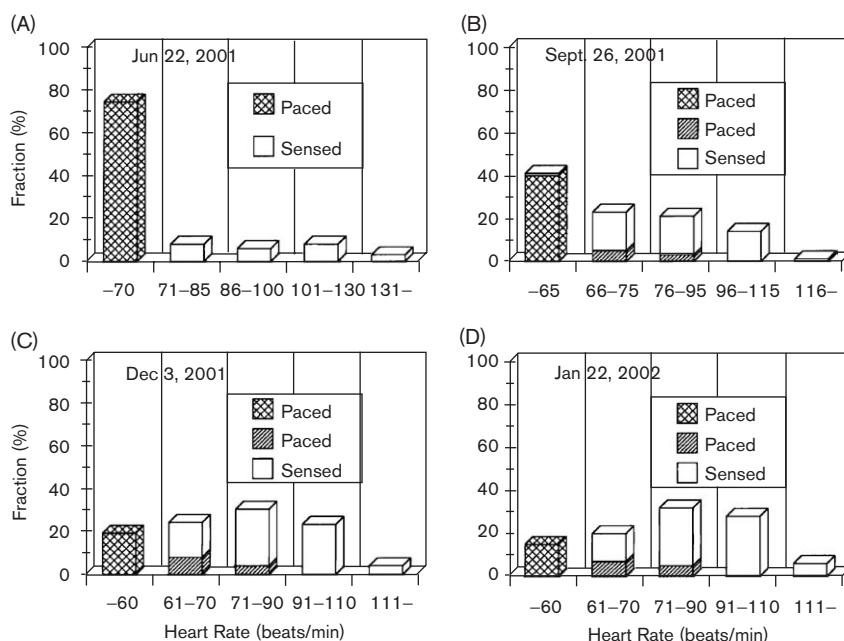
with restoration of chronotropic competence by sensed (intrinsic) as well as paced heart beats, CHF and poor AV conduction were improved (CTR = 54%, NYHA class III). After months of rehabilitation and pace-

Fig. 3



Fraction of pacing during the course of rehabilitation. After the 'rate-response' mode of pacemaker function was enabled (VVI to VVI-R) and regulated, the total fraction of paced heart beats (empty circles) by the implanted pacemaker gradually decreased. Meanwhile, the fraction of rate-responsive paced heart beats (filled circles) substantially increased. Filled squares denote the programmed pacing lower rates, i.e., initially 70 pulses/min, next 65 and finally 60.

Fig. 2



Histograms of paced and sensed (intrinsic) QRS complexes in each periodic check of pacemaker function in VVI-R mode. The pacemaker had a readable memory. Fractions of paced rhythm (meshed or shaded columns) or sensed rhythm (blank columns) are shown in each range of heart rate. The paced rhythms (shaded columns) above the programmed pacing lower rate, i.e., >70 pulses/min in A, >65 in B, >60 in C and D, indicate 'rate-responsive' pacing to increased physical motion/activity. Chi-square analysis revealed that the fraction of rate-responsive pacing to the total pacing in every interval (B, C, D) significantly ($p < 0.001$) increased as compared to that in the initial check (A).

maker modulation, the patient could sit in a wheelchair for more than three hours without significant orthostatic hypotension, and his ADL had improved from bed rest to the wheelchair level with moderate assistance.

Discussion

In developed countries where rheumatic fever is rare, the prevalence of NVAF increases as the patient population continues to age: it is approximately 1% of the general population and doubles with each advancing decade of age, from 0.5% at 50–59 years of age to almost 9% at 80–89 years (Feinberg *et al.*, 1995, Kannel *et al.*, 1998). In addition, sinus node and AV node functions are reduced with age, accompanying atrophy of the nodal cells and fatty infiltration (Chida *et al.*, 1999). This phenomenon is related to the increased prevalence of sick sinus syndrome, AV block and chronic AF with aging (Sutton *et al.*, 1986). Therefore, the number of elderly patients with chronic bradycardic AF, in which a permanent pacemaker is implanted, is reasonably large.

A VVI(-R) mode pacemaker is generally employed for the treatment of bradyarrhythmias, especially permanent bradycardic AF (Gregoratos *et al.*, 1998). Because its implantation is technically simple, with a single wire and established procedures, it is the most popular pacemaker in clinical treatments at present. However, because the VVI mode fails to produce sequential and synchronous contractions of the atria and ventricles, this mode has disadvantages when one tries to improve cardiac stroke volume and exercise endurance in patients with low cardiac reserve (Rosenqvist *et al.*, 1991). Physiological rate-adaptive (-R) pacemaker function is designated to provide a change in the pacing rate proportional to the level of metabolic demand ('chronotropic competence'), and therefore to increase the cardiac output in response to increased physical activity. The most widely used are vibration- or motion-sensing devices, either a piezoelectric crystal activity sensor fixed to the inside of the pacemaker case or an accelerometer. The crystal detects vibration generated by body motion, and the accelerometer senses motion directly. Other rate-responsive pacemakers use the right ventricular blood temperature, minute ventilation, the QT interval in ECG, mixed venous oxygen saturation, and other physiological parameters as variables to modulate the pacing rate (Schweikert *et al.*, 1999). The rate-response to body motion could be most effective to the orthostatic intolerance associated with bradycardic chronic AF and the disuse syndrome in the present patient, especially at the beginning of the rehabilitation therapy. In other words, if we had not adopted the rate-responsive pacing function, we could not start to rehabilitate the patient and to develop the program of the rehabilitation therapy.

Chronic AF and CHF patients implanted with VVI mode pacemakers belong to a group at high-risk for cerebral embolism (Andersen *et al.*, 1994). Once the ictus occurs, these patients exhibit critically severe hemiplegia and/or higher cortical dysfunction, disability, and a serious disuse syndrome associated with the prolonged initial therapy in acute care units. Serious orthostatic intolerance/hypotension is incidental to those patients with flaccid hemiplegia and marked disuse syndrome. In the present case, the orthostatic intolerance could have been due to: (1) severe disuse syndrome and the plegic lower extremity, as well as deteriorated autonomic regulation of the peripheral circulation and increased venous compliance; (2) the absence of a physiological Bainbridge reflex and chronotropic incompetence associated with the underlying sick sinus syndrome; (3) poor AV node conduction; and (4) almost all (> 70%) of the heart beats dependent on the VVI pacing without the booster pump effect.

Orthostatic intolerance after prolonged bed rest can be attributed, at least in part, to the depletion of intravascular volume, which might have been compounded by an increase in venous pooling in the lower extremities because of increased venous compliance after bed rest (Downey *et al.*, 2001). The veins of the lower extremities are part of the post-capillary blood pool, which makes up approximately 70% of the overall intravascular volume, and approximately 80% of the blood pooled in the lower limbs is contained in the upper legs (Smit *et al.*, 1999). One half to one litre of thoracic blood is transferred to the lower body upon orthostatic stress. The bulk of venous pooling occurs within the first 10 seconds, and the total transfer is almost complete within 3–5 minutes of the orthostatic stress (Smit *et al.*, 1999). This time course and the hemodynamic changes are well correlated with the appearance of serious orthostatic hypotension in the present case that had severe disuse syndrome and the severe plegic left lower extremity.

In summary, modulation of a VVI-R programmable pacemaker, especially its rate-responsive pacing that enabled chronotropic competence, was an effective countermeasure to serious orthostatic intolerance in a patient with bradycardic chronic NVAF. We argue that manipulations of the physiological rate-responsive function of the pacemaker were, indeed, primarily responsible for the initial start and subsequent advancement of the rehabilitation programs. Periodic adjustments of the pacemaker, especially increased rate-responsive function to physical activities, significantly improved his physical endurance, ADL and quality of life.

Conclusions

This case reminds us of the necessity to examine the mode, type, function and characteristics of previously implanted cardiac pacemakers in stroke patients with

arrhythmias, and confirms the merits of regularly modulating pacemaker function during routine stroke rehabilitation therapy to obtain a better functional outcome. In bradycardic chronic AF and stroke patients who have been implanted with an artificial pacemaker, the VVI mode is most common but might not improve cardiac output and physical activity/tolerance because of the AV asynchrony. Thus, an additional rate-adaptive pacing mode, i.e., VVI-R, is essential for effective stroke rehabilitation therapy by chronotropic competence to increase cardiac output and to treat orthostatic hypotension.

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