WORKING PARTY REPORT

Recommendations for pacemaker prescription for symptomatic bradycardia

Report of a working party of the British Pacing and Electrophysiology Group

After the first permanent pacemaker implantation in 1958,¹ the initial developments in pacemaker technology were aimed towards more efficient pacemaker performance. The standard mode of pacing in these early years was single chamber ventricular stimulation and this became established as effective life-saving therapy.² Over the past 10 years, however, there has been a wealth of different features incorporated into pacemaker design, including programmability, telemetry, and different "modes" of pacing.³⁻⁶

The British Pacing and Electrophysiology Group (BPEG) was formed as a specialist subgroup of the British Cardiac Society in 1976 and consists of cardiologists, technicians, and representatives from the pacemaker manufacturing industry. BPEG is organised for the purposes of scientific communication, research, and education specifically related to cardiac pacing and electrophysiology. The group organises an active scientific and educational programme and has more than 200 members.

A working party was formed by BPEG to establish guidelines for the use of the different modes of cardiac pacing. The nomenclature for pacemaker modes accepted for international use is that produced by a combined working party of the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG) known as the NBG (NASPE/BPEG generic) code.⁷ This is a code of five letters of which the first three are most often used (table 1). In addition, a fourth letter R is used if adaptive rate pacing is programmed. In adaptive rate (or rate responsive) pacing there is an additional sensor in the pacemaker system that detects a physiological or semi-physiological result of exercise or emotion and increases the pacemaker rate on the basis of a programmable algorithm. Sensors that are presently successfully used in pacemakers include activity,⁸ respiratory rate⁹ and minute ventilation,¹⁰ right ventricular pressure (dP/dt),¹¹ central venous temperature,¹² evoked QT interval,⁵ and oxygen saturation.¹³ Whatever the sensor effective adaptive rate pacing can be used independently of atrial activity. Recently, activity sensing has been combined with dual chamber pacing.⁶

The NBG code also uses a fifth letter relating to antitachycardia function which is not

Table 1 NBG pacemaker code

FIRST letter signifies the chamber(s) of the heart being PACED	
A	(atrium)
V	(ventricle)
D	(atrium and ventricle)
SECOND letter indicates the chamber(s) being SENSED	
A	(atrium)
V	(ventricle)
D	(atrium and ventricle)
THIRD letter indicates the MODE of sensing	
I	(inhibited)
T	(triggered)
D	(inhibited and triggered)

discussed in this document. Each of the pacing modes under discussion is described in the Appendix.

AHA/ACC Guidelines

Over the past 30 years the clinical indications for pacing have increased to include a large number of different cardiac arrhythmias. To clarify the situation a combined working group of the American Heart Association and the American College of Cardiology has published guidelines on the indications for pacing.¹⁴ There are three main classes of patients, which may be summarised as follows:

Class I—Conditions in which there is general agreement that permanent pacemakers should be implanted. This includes syncope patients with complete heart block or prolonged sinus arrest.

Class II—Conditions in which permanent pacemakers are frequently used but there is some divergence of opinion about whether they are needed. Included within this group are the sick sinus syndrome patients without syncope but with other symptoms and also patients with symptomatic incomplete atrioventricular block. Asymptomatic complete heart block is also in this category.

Class III—Conditions in which there is general agreement that pacemakers are not necessary. This includes asymptomatic patients with sinus node disease or first or second degree atrioventricular block and patients with unexplained syncope without obvious cardiac arrhythmia.

BPEG Guidelines

The AHA/ACC recommendations have

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provided a logical foundation to guide physicians considering cardiac pacing as a treatment for their patients. There are, however, some areas where published data indicate that pacing should be more widely used as a first-line therapy.

Carotid sinus syndrome¹⁵ and, more recently, malignant vasovagal syncope¹⁶ would now qualify as firm indications for pacemaker implantation when appropriate investigation shows a major cardioinhibitory effect.^{16,17} Furthermore, untreated persistent second degree atrioventricular block, even in asymptomatic patients, has been shown to be associated with a reduced expectation of life. In this group of patients prophylactic pacing restores life expectation to normal.¹⁸ The Wenckebach phenomenon can occur during sleep in normal individuals with high vagal tone and these people do not need to be considered for treatment. However, those asymptomatic patients with either Wenckebach or Mobitz II second degree atrioventricular block occurring during much of the day and night would qualify for pacemaker implantation,¹⁹ as would patients with asymptomatic complete heart block. With these modifications, the AHA/ACC guidelines can be used both as a clinical aid and a basis for medical audit of the indications for pacemaker implantation. However, recommendations about the choice of pacemaker prescription are limited.

Choice of pacemaker mode

When the choice of pacemaker mode for an individual patient is considered, the ideal is production of a paced cardiac rhythm with as many features of normal sinus rhythm as possible (table 2). The guidelines proposed in this paper are based on an attempt to allow the pacemaker response to behave as physiologically as possible. This includes restoration of atrioventricular synchrony and/or rate adaptive pacing wherever needed. To achieve this ideal, detailed information of the underlying cardiac rhythm should be obtained before or during pacemaker implantation. The following features should be considered.

ATRIOVENTRICULAR SYNCHRONY

Pacemaker systems that maintain atrioventricular synchrony with a physiological atrioventricular interval have been shown to increase cardiac output both at rest and on exercise in patients with normal and impaired left ventricular function.²⁰ Even in the absence of an exercise induced rate response, atrioventricular synchrony reduces the number of minor symptoms (dizziness, lethargy) that are associated particularly with sinus node disease.²¹

Table 2 General principles of pacemaker choice

- (1) The ventricle should be paced if there is actual or threatened atrioventricular block.
- (2) The atrium should be paced/sensed unless contraindicated.
- (3) Rate response is not essential if the patient is inactive or has a normal chronotropic response.
- (4) Rate hysteresis may be valuable if the bradycardia is intermittent.

RATE MODULATION

Adaptive rate (or rate responsive) pacing significantly increases cardiac output on exercise in patients with atrioventricular block⁸ and sinus node disease.²² Patients treated with rate responsive pacemakers achieve higher workloads and greater exercise tolerances.^{8,22}

ATRIAL PACING

Evidence is accumulating of a considerable reduction in the high spontaneous incidence of the development of atrial fibrillation²³ and of systemic emboli^{24,25} in patients with sinus node disease treated by a pacing system that maintains atrioventricular synchrony.

LEFT VENTRICULAR PERFORMANCE

Spontaneous (normal) ventricular contraction may allow better ventricular function than a paced ventricular beat.²⁶ Typically, the ventricular pacing lead is inserted into the right ventricle and delivery of the pacing stimulus at this site results in the equivalent of left bundle branch block. This causes measurable impairment of left ventricular contraction which may be of haemodynamic relevance in both normal²⁷ and compromised left ventricles.²⁸

RATE HYSTERESIS

Hysteresis is a programmed feature whereby the pacemaker paces at a faster rate than the sensing rate. For example, pacing at 80 pulses a minute with a hysteresis rate of 55 means that the pacemaker will be inhibited at all rates down to 55 beats per minute. Having been activated at a rate below 55, the pacemaker then switches on and paces at 80 pulses a minute. Unless careful record is made in the patient's notes that hysteresis has been programmed, pacemaker malfunction could be incorrectly inferred.

ATRIAL ARRHYTHMIAS

The presence of atrial activity other than chronic or frequently repetitive atrial flutter or fibrillation generally requires an atrial pacemaker electrode. Paroxysmal atrial arrhythmias are not a contraindication to atrial pacing²⁹ or sensing in most cases, because atrial pacing may stabilise the atrial rhythm³⁰; in others concomitant drug treatment may be beneficial. In those patients with atrial bradycardia and intact atrioventricular conduction, a single chamber atrial pacemaker can be considered to be the treatment of choice.²⁹ However, the presence of minor abnormalities of atrioventricular conduction—for example first degree atrioventricular block (PR interval prolongation) or bundle branch disease with normal atrioventricular conduction generally suggests a need for placement of a ventricular lead in patients with symptomatic sinoatrial disorders.^{31,32}

TYPES OF ARRHYTHMIAS REQUIRING PACING

Table 3 shows the percentage occurrence of arrhythmias in a typical population of patients with bradycardia requiring pacing.³³ For the purposes of table 3, sinus node disease includes

Table 3 Percentage incidence of various bradyarrhythmias in a typical pacemaker population

Arrhythmia	%
Sinus node disease	25
AV block	42
Sinus node disease + AV block	10
Atrial fibrillation + AV block	13
Carotid and vasovagal syndromes	10

AV, atrioventricular.

sinus bradycardia, sinus arrest, sinoatrial block, and the tachycardia-bradycardia syndrome, but does not include atrial bradyarrhythmias associated with carotid sinus syndrome or malignant vasovagal syndrome.

CAROTID SINUS AND MALIGNANT VASOVAGAL SYNDROMES

Carotid sinus syndrome can be diagnosed in a patient with syncope by the presence of a positive carotid sinus massage test. The development of either a period of asystole in excess of three seconds, or the appearance of atrioventricular block, during five to six seconds of firm carotid sinus massage (not compression) is regarded as diagnostic.¹⁷ The test should not be performed in patients with carotid artery bruits or documented stenosis, after recent myocardial infarction, or if the patient is digitalised. Variations of carotid sinus syndrome include micturition syncope,³⁴ cough syncope,³⁵ and deglutition syncope.³⁶ Malignant vasovagal syncope can be demonstrated by performing a 60° head-up tilt test. Syncope associated with bradycardia or hypotension or both occurs within 40 minutes.¹⁶

In patients with carotid sinus and malignant vasovagal syndromes there are two components to the syncope—bradycardia (cardioinhibition) and peripheral and splanchnic vasodilation (vasodepression). In patients with a pronounced vasodepressor effect, pacing is less successful than in patients with a dominant cardioinhibitory effect.¹⁵ If patients are suitable

Table 4 Recommended pacemaker modes

Diagnosis	Optimal	Alternative	Inappropriate
SND	AAIR	AAI	VVI VDD
AVB	DDD	VDD	AAI DDI
SND and AVB	DDDR DDIR	DDD DDI	AAI VVI
Chronic AF with AVB	VVIR	VVI	AAI DDD VDD
CSS	DDI	DDD VVI*	AAI VDD
MVVS	DDI	DDD	AAI VVI† VDD

The optimal mode of pacing should be considered for most patients. The alternative mode should be regarded as being less satisfactory, but acceptable in some groups of patients—for example those who are disabled by another disease, those with very intermittent symptoms, or those who have a short life expectancy because of another disease. When a patient with, for example, a previous hemiplegia or with terminal neoplasia has atrioventricular block, VVI may suffice to reduce symptoms.

*If VVI is ever chosen for the management of carotid sinus syndrome rate hysteresis is recommended.³⁸ Patient selection should follow the guidelines suggested by Brignole *et al.*³⁹

†See reference.⁴⁰

AVB, atrioventricular block; AF, atrial fibrillation or flutter; MVVS, malignant vasovagal syndrome; SND, sinoatrial node disease; CSS, carotid sinus syndrome.

for pacing the mode of choice for carotid sinus syndrome and malignant vasovagal syndrome is DDI,³⁷ frequently with hysteresis as an added feature.³⁸

ATRIAL TRACKING PACING SYSTEMS

It is recognised that the pacing system known universally as DDD behaves for most of the time as a VDD system. The only difference is the behaviour at the lower programmed rate, where the DDD paces into the atrium and the VDD has a back-up VVI mode. For the patient with bradycardia that is slower than the lower programmed rate, this may have significant disadvantages including pacemaker syndrome. In sinus node disease the potential benefit of pacing the atria is not available in VDD mode.

Table 4 shows the recommended modes, based on these principles, for patients with all varieties of bradyarrhythmias requiring pacing.

Complications of inappropriate mode selection

Inappropriate pacemaker modes (table 4) should be avoided to prevent the following complications:

(a) Pacemaker syndrome,⁴¹ which is caused either by retrograde conduction from the ventricle to the atrium or by lack of synchronous atrioventricular activity that results in loss of atrial transport function. Pacemaker syndrome may cause symptoms of syncope or pre-syncope as a result of retrograde conduction.⁴¹ Dyspnoea may occur where there is high pulmonary venous pressure and low cardiac output and associated mitral regurgitation (demonstrable by Doppler echocardiography) caused by a lack of atrioventricular synchrony.⁴² Neck pulsation may be seen when retrograde atrioventricular conduction causes tricuspid regurgitation or right atrial contraction against a closed tricuspid valve. The true incidence of pacemaker syndrome is not well defined but it is thought that 10% of patients paced VVI have quite severe symptoms and another 15% have a reduced quality of life as a result.⁴¹ All these symptoms can be relieved by a more appropriate pacing mode.

(b) Inappropriate atrial tracking of atrial tachyarrhythmias by a DDD or VDD system.⁴³ Atrial tachycardia, flutter, or fibrillation produce an electrogram which can be easily detected by the atrial sensing amplifier of the pacemaker, which will then track this rhythm at its upper pacing rate. Newer dual chamber rate adaptive systems incorporate algorithms that prevent or minimise inappropriate ventricular pacing in response to atrial fibrillation.

(c) Additional atrioventricular block in a patient with sinus node dysfunction may not be easily detected initially.⁴⁴ Normal 1:1 atrioventricular conduction at atrial pacing at rates of 140 usually indicates adequate atrioventricular nodal function. The appearance of the atrioventricular Wenckebach block at slower rates may be considered indicative of important impairment of atrioventricular node conduction which warrants placement of a ventricular

lead. There may be other evidence of inadequate atrioventricular conduction from Holter monitoring, exercise stress testing, or His bundle electrography.

(d) Lack of physiological heart rate response on exercise. Unrecognised sinus node dysfunction in a patient with atrioventricular block would result in inadequate performance of an atrial tracking (DDD) pacemaker. In addition, there are some patients with sinus node disease treated by atrial rate modulated pacing systems who may develop abnormal atrioventricular conduction on exercise⁴⁵ resulting in an unusual variety of pacemaker syndrome.

Additional guidelines

The following additional guidelines are recommended to enable accurate assessment of a patient requiring pacemaker implantation:

(a) In selected patients atrioventricular conduction should be assessed by Holter, carotid sinus massage, and atrial pacing to assess the atrioventricular Wenckebach point at the time of implant if atrial pacing (AAI) is being considered.

(b) Retrograde atrioventricular conduction should be assessed by ventricular pacing if VVI or VVIR pacing is considered. Head-up tilting during ventricular pacing may reveal latent symptoms.

(c) If VDD or DDD pacing is being considered in active patients the chronotropic response of the sinus node should be assessed by Holter or exercise testing. If the response is inadequate an appropriate sensor driven system may be needed.

Consequences of increased use of dual chamber and rate responsive pacing

COST

A VVIR unit costs about 40–50% more and a dual chamber unit costs 40–100% more than a multiprogrammable single chamber pacemaker. In addition, a dual chamber system requires two pacing leads which further increase the cost. Rate-responsive pacing systems have a shorter battery lifetime than non-rate responsive modes because of their overall faster pacing rate and the inherent current drain of the sensor.

However, pacing is cost effective in terms of relief of symptoms, prolongation of life, and improvement of the quality of life.^{20 46–50} Patients with pacemakers rarely need to be readmitted to hospital because of problems. So a tailored pacemaker prescription that follows the guidelines in this report will offer good value for money.

FOLLOW UP

Patient follow up with VVIR and dual chamber systems is initially more complex and time consuming. Additional programming is necessary together with Holter taping and exercise testing where required. Support staff need to be trained. When the pacing system has been “fine tuned” to the individual patient’s requirements, however, subsequent follow up is less time consuming.

COMPLICATIONS

Complex pacing systems are more susceptible to problems. Dual chamber systems require two endocardial leads and lead related complications are therefore slightly increased.⁵¹ Inappropriate programming may also give rise to difficulties. A badly programmed sensor may give excessive or inadequate rate responses. It is possible to select an inappropriate sensor system for an individual patient—for example, an activity sensor in a bedbound patient or a minute-ventilation sensor in a patient with chronic lung disease are as inappropriate as a DDD system would be in a patient with atrial flutter.

Which hospitals should implant and follow up pacemakers?

Over recent years, it has been the policy of some regional health authorities to encourage pacemaker implantation in district general hospitals. This is acceptable but it demands at least one cardiologist fully trained in the complex disciplines of dual chamber pacing and follow up, together with at least one similarly trained physiological measurement technician. Hospitals with only one cardiologist would need to make arrangements with similarly trained staff in a neighbouring district to cover periods of absence.

Choice of pacing system

When choosing a pacemaker system for a patient, a physician usually adopts one of three policies:

(a) A basic VVI system for everyone, irrespective of need, symptoms, or electrocardiographic findings. This policy is clearly inappropriate.

(b) The routine use of the most sophisticated dual chamber rate adaptive pacemaker (DDDR) for every patient, programmed to match the patients’ needs. This is medically acceptable, but inappropriate on grounds of cost.

(c) A device that is carefully prescribed according to the guidelines in this report. This again is medically sound, but this policy requires thorough assessment of the patient before pacemaker implantation and meticulous follow up.

This working party of BPEG recommends that selection of the appropriate pacemaker mode should follow the basic principles and guidelines discussed above. This will require a fairly radical change of practice in some pacemaker centres in the United Kingdom and additional funding for the more complex pacemaker systems and their subsequent follow up.

- 1 Elmqvist R, Senning A. An implantable pacemaker for the heart. Smyth CN, ed. In: *Medical electronics. Proceedings of the second international conference on medical electronics*. London: Illife, 1959:253.
- 2 Ginks W, Leatham A, Siddons H. Prognosis of patients paced for chronic atrioventricular block. *Br Heart J* 1979;41:633–6.
- 3 Sutton R, Citron P, Perrins J. Physiological cardiac pacing. *PACE* 1980;3:207–17.

- 4 Clarke M, Allen A. Use of telemetered electrograms in the assessment of normal pacemaker function. *J Electrophysiol* 1987;1:388-95.
- 5 Rickards AF, Norman J. Relation between QT interval and heart rate. New design of a physiologically adaptive cardiac pacemaker. *Br Heart J* 1981;45:56-61.
- 6 Kappenberger L, Hepers L. Rate responsive dual chamber pacing. *PACE* 1986;9:987-91.
- 7 Bernstein A, Camm J, Fletcher R, et al. The NASPE/BPEG generic pacing code for antibradyarrhythmia and adaptive rate pacing and antitachycardia devices. *PACE* 1987;10:794-9.
- 8 Humen D, Anderson K, Brumwell D, Huntley S, Klein G. A pacemaker which automatically increases its rate with physical activity. In: Steinbach K, Glogard A, Laszkovicsa D, Schneibelhofer W, Weber H, eds. *Cardiac pacing*. Darmstadt: Steinkopff Verlag, 1983:252-64.
- 9 Rossi P, Plicchi G, Canducci G, Rognoni G, Aina F. Respiration as a reliable physiological sensor for controlling cardiac pacing rate. *Br Heart J* 1984;51:7-14.
- 10 Lau C, Antoniou A, Ward D, Camm AJ. Reliability of minute ventilation as a parameter for rate responsive pacing. *PACE* 1989;12:321-30.
- 11 Sutton R, Sharma A, Ingram A, Camm J, Lindemans F, Bennett T. First derivative of right ventricular pressure as a sensor for an implantable rate responsive VVI pacemaker [abstract]. *PACE* 1988;11:487.
- 12 Alt E, Völker R, Högl B, MacCarter D. First clinical results with a new temperature-controlled rate responsive pacemaker. *Circulation* 1988;78 (suppl III):116-24.
- 13 Stangl K, Wirtzfeld A, Heinze R, Laule M. First clinical experience with an oxygen saturation controlled pacemaker in man. *PACE* 1988;11:1882-7.
- 14 Frye RL, Collins JJ, DeSanctis RW, et al. Guidelines for permanent pacemaker implantation—May 1984. A report of the joint American College of Cardiology/American Heart Association Task Force on assessment of Cardiovascular Procedures (Subcommittee on Pacemaker Implantation). *J Am Coll Cardiol* 1984;4:434-42.
- 15 Morley CA, Perrins EJ, Grant P, Chan SL, McBrien DJ, Sutton R. Carotid sinus syncope treated by pacing. Analysis of persistent symptoms and role of atrioventricular sequential pacing. *Br Heart J* 1982;47:411-8.
- 16 Fitzpatrick A, Sutton R. Tilting towards a diagnosis in recurrent unexplained syncope. *Lancet* 1988;i:658-60.
- 17 Morley C, Sutton R. Carotid sinus syndrome—editorial review. *Int J Cardiol* 1984;6:287-93.
- 18 Shaw D, Kekwick C, Veale D, Gowers J, Whistance T. Survival in second degree atrioventricular block. *Br Heart J* 1985;53:587-93.
- 19 Campbell RWF. Chronic Mobitz type I second degree atrioventricular block. Has its importance been underestimated? *Br Heart J* 1985;53:585-6.
- 20 Kruse I, Arnman K, Conradson T, Ryden L. A comparison of the acute and long-term haemodynamic effects of ventricular inhibited and atrial synchronous ventricular inhibited pacing. *Circulation* 1981;65:846-55.
- 21 Mitsuoka T, Kenny RA, Yeung TA, Chan SL, Perrins JE, Sutton R. Benefits of dual chamber pacing in sick sinus syndrome. *Br Heart J* 1988;60:338-47.
- 22 Rognoni G, Bolognese L, Aina F, Occhetta E, Magnani A, Rossi P. Respiratory dependant atrial pacing, management of sinus node disease. *PACE* 1988;11:1853-9.
- 23 Rosenqvist M, Brandt J, Schuller H. Long-term pacing in sinus node disease; effects of stimulation mode on cardiovascular morbidity and mortality. *Am Heart J* 1988;116:16-22.
- 24 Santini M, Alexidou G, Porto M, Santini A, Ammirati F, Ansalone G. The sick sinus syndrome: prognosis as function of age, conduction defects and pacing mode [abstract]. *PACE* 1989;12:1237.
- 25 Bianconi L, Boccadamo R, Di Florio A, et al. Atrial versus ventricular stimulation in sick sinus syndrome; effects on morbidity and mortality [abstract]. *PACE* 1989;12:1236.
- 26 Theodorakis G, Fitzpatrick A, Ahmed R, Travill C, Sutton R. Doppler estimation of cardiac output during AAI and DDD pacing (with varying AV delay) at different pacing rates [abstract]. *PACE* 1989;12:1185.
- 27 DeNardo D, Antolini M, Pitucco O, et al. Effects of left bundle branch block on left ventricular function in apparently normal subjects. *Cardiology* 1988;75:365-71.
- 28 Gibson DG, Chamberlain DA, Coltart DJ, Mercer J. Effect of changes in ventricular activation on cardiac haemodynamics in man. Comparison of right ventricular, left ventricular, and simultaneous pacing of both ventricles. *Br Heart J* 1971;33:397-400.
- 29 Ryden L. Atrial inhibited pacing—an underused mode of cardiac stimulation. *PACE* 1988;11:1375-9.
- 30 Egobasti A, Gueunoun M, Sasdjian A, et al. Long-term follow-up of patients treated with VVI pacing and sequential pacing with special reference to VA retrograde conduction. *PACE* 1988;11:1929-34.
- 31 Sutton R, Kenny R. Natural history of sick sinus syndrome. *PACE* 1986;9:1110-4.
- 32 Rosenqvist M, Obel I. Atrial pacing and the risk for AV block: Is there a time for change of attitude? *PACE* 1989;12:97-101.
- 33 British Pacing and Electrophysiology Group Database—statistics for the City General Hospital, Stoke-on-Trent, 1986-1989.
- 34 Haldane JH. Micturition syncope. *Can Med Assoc J* 1969;101:712-4.
- 35 Hart G, Oldershaw P, Cull R, Humphrey P, Ward D. Syncope caused by cough-induced complete atrioventricular block. *PACE* 1982;5:564-66.
- 36 Igaluer S, Swartz B. Heart block periodically induced by the swallowing of food in a patient with cardiospasm (vasovagal syncope). *Ann Otol Rhinol Laryngol* 1936;45:875-81.
- 37 Sutton R, Ingram A, Clarke M. DDI pacing in the treatment of sick sinus, carotid sinus and vasovagal syndromes [abstract]. *PACE* 1988;11:827.
- 38 Morley C, Perrins EJ, Chan SL, Sutton R. The role of rate hysteresis pacing in the hypersensitive carotid sinus syndrome. *PACE* 1983;6:1224-8.
- 39 Brignole M, Menozzi C, Lolli G, Oddone D, Roti S. When is DDD pacing necessary and when is VVI pacing sufficient in carotid sinus syndrome? [abstract]. *PACE* 1989;12:1169.
- 40 Fitzpatrick AP, Travill CM, Vardas PE, et al. Recurrent symptoms after ventricular pacing in unexplained syncope. *PACE* 1990;13:619-24.
- 41 Ausubel K, Furman S. The pacemaker syndrome. *Ann Intern Med* 1985;103:420-9.
- 42 Naito M, Dreyfus L, David D, Nicholson E, Mardela T, Kmetz J. Re-evaluation of the role of atrial systole in cardiac hemodynamics: evidence for pulmonary venous regurgitation during abnormal atrioventricular sequencing. *Am Heart J* 1983;105:295-302.
- 43 Castellanos A, Lemberg L, Rodriguez-Tocker L, et al. Atrial synchronised pacemaker arrhythmias, revisited. *Am Heart J* 1968;76:199-205.
- 44 Steinbach K, Forohner K, Meisl F, et al. Atrial stimulation. In: Pérez Gómez F, ed. *Cardiac pacing*. Madrid: Grouz, 1985:629.
- 45 Clarke M, Allen A. Rate responsive atrial pacing resulting in pacemaker syndrome [abstract]. *PACE* 1987;10:1209.
- 46 Perrins JE, Morley CA, Chan SL, Sutton R. Randomised controlled trial of physiological and ventricular pacing. *Br Heart J* 1983;50:112-7.
- 47 Lau CP, Rushby J, Leigh-Jones M, et al. A double blind cross-over study on the symptomatology and quality of life in patients with rate responsive pacemakers [abstract]. *J Am Coll Cardiol* 1989;13:113.
- 48 Fitzgerald WR, Graham IM, Cole T, Evans DW. Age, sex, and ischaemic heart disease as prognostic indicators in long-term cardiac pacing. *Br Heart J* 1979;42:57-60.
- 49 Edhag O, Swann A. Prognosis of patients with complete heart block or arrhythmic syncope who were not treated by cardiac pacemakers. *Acta Med Scand* 1976;200:457-63.
- 50 Williams A. Economics of coronary artery bypass grafting. *BMJ* 1985;291:326-9.
- 51 Sutton R, Ingram A, Briers L, et al. Ten years of physiological cardiac pacing. In: Belhassen B, Feldman S, Copperman Y, eds. *Cardiac pacing and electrophysiology*. Jerusalem: R & L Creative Communications, 1987:141-2.
- 52 Cunningham T. Pacemaker syndrome due to retrograde conduction in DDI pacemaker. *Am Heart J* 1988;115:478-9.
- 53 Ricci D, Rider A, Mason J. Recurrent tachyarrhythmia associated with a bifocal demand pacemaker. *Chest* 1977;72:120-3.
- 54 Den Dulk K, Lindemans FW, Banf W, et al. Pacemaker related tachycardia. *PACE* 1982;5:476-85.
- 55 Torresani J, Egobasti A, Allard-Latour G. Pacemaker syndrome with DDD pacing. *PACE* 1984;7:1148-51.