TREATMENT WITH PACEMAKERS

KEY POINTS:

1. Pacemakers have been available for over 40 years and are the only effective treatment for bradyarrhythmias.
2. Pacemakers maintain the heart rate, and thereby preserve life, by stimulation of heart muscle with an artificial electric current.
3. They consist of a sealed battery-box with electrical circuits and a radio transceiver, which can communicate with a pacemaker programming computer over short distances, including through the skin after surgery.
4. The battery-box connects to one or more insulated wires that are usually passed through veins and steered into the heart under X-ray control.
5. Mechanisms are built into the tips of these wires or electrodes to keep them anchored to the heart muscle.
6. Very low levels of current are needed to stimulate a heartbeat. Modern pacemakers only stimulate when needed and can detect the heart's own beats, preventing an inappropriate stimulation.
7. Pacemakers speed up with exercise if needed, using built in sensors, or sensing the sinus node of the heart through an atrial electrode.
8. Pacemakers are usually implanted under local anaesthetic in a catheter laboratory within 1-2 hours, and may be safely done as a day-case.
9. There is good evidence that pacing reduces atrial fibrillation, stroke and mortality.
10. Patients who might benefit from a pacemaker may present to general practice, emergency medicine, secondary care and tertiary care.
11. Some patients have an obvious bradyarrhythmia, and all of these patients are believed to get pacemakers in the UK.
12. Other patients have intermittent bradyarrhythmias, and the cause of their symptoms; dizziness, blackout, palpitations and breathlessness, may not be evident on an ECG when they are seen by a doctor. Many of these patients do not receive a pacemaker, even though they require one. This is because of very low levels of cardiology staffing, and inappropriate care-pathways.
13. Failure to unearth the cause of intermittent symptoms in patients with bradyarrhythmias may account for the fact that the UK implants only about 420 pacemakers/million people, and close Western European neighbours implant around 900/million or more.
14. The UK is poorly served for pacing services compared to Western Europe, and these services are patchy, often depending on the local presence of an enthusiastic cardiologist. Such specialists often have a very hard time getting co-operation in establishing services from local commissioners of care in spite of very low implant rates across the UK. Many of these cardiologists are still required to partake in general medical duties, rather than concentrate on cardiology and services such as pacing. This should be addressed and changed nationally, and would result in a rapid increase in local availability of DGH pacing services.
15. A satisfactory pacing service requires at least two implanting specialists per centre, as well as two trained technical staff and good quality facilities for safe, sterile implantation and follow-up. To achieve this across the UK will require a major work-force expansion exercise, and capital investment in facilities.
16. Newer indications for pacing and related device therapies are rapidly increasing the population who can benefit by control of symptoms and prolongation of life. Such new indications include ICDs and biventricular pacing, (see chapter 21). Development of these newer services should be built upon the foundation of an well -established pacemaker service.

Dr Michael Gammage, University of Birmingham

BACKGROUND TO CARDIAC PACING

Development:

Implantable cardiac pacemakers have been available for just over 40 years[1] and are well-established as the treatment of symptomatic slow heart rate or bradyarrhythmia. During the last 20 years there have been very rapid technological advances in pacemaker design and many new proven indications for pacing. As pacing has become more sophisticated, it has become more expensive, and as a result recent studies have also examined cost-effectiveness, and the role of specific pacing therapies is becoming clearer.

Description:

Pacemakers deliver a very short, low voltage electrical pulse via an insulated pacing wire or electrode to the heart muscle. Pacemakers are also designed to detect ("sense") the heart's own electrical impulses. This sensing ability ensures that the pacemaker will only pace when required. Pacemakers may be designed for single chamber use (with only one lead, in the right atrium or ventricle), or dual chamber use, with two leads (one in the right atrium, one in the right ventricle). With newer indications for pacing, some systems now have more than two leads; these devices are biventricular and largely for the management of heart failure. In addition, specific programmes may allow pacing under different circumstances or enable the pacemaker to change the way in which it paces as a result of sensed events such as atrial fibrillation. Many of these functions are now programmable from outside the pacemaker, using a device similar to a laptop computer, via a short-range radio link.

Coding conventions:

With the technical development of pacemakers for different bradycardic indications, it became necessary to develop a code allowing simple description of the function of the device and site of the pacing leads. This coding, developed by the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG)[2] allows description of the cardiac chambers paced, the chambers in which intrinsic cardiac activity can be sensed, and the response to that sensing. It has subsequently been modified to include the presence in the device of a sensor to modulate changes in heart rate and the ability to pace multiple (i.e. more than 2) chambers.

Table 1: Pacemaker coding, modified from NASPE/BPEG by AHA/ACC (3)

Chamber paced	**Chamber(s)sensed**	**Response to sensing**	**Rate modulation**	**Multi-site pacing**
O	O	O	O	O
A	A	T	R	A
V	V	I		V
D (A=V)	D (A=V)	D (T+I)		D (A=V)
S (A or V)	S (A or V			

Key: O = no action
A = atrium
V = ventricle
D = dual (atrium and ventricle)
R = rate modulation
T = triggered
I = inhibited
S = atrium or ventricle (manufacturer's designation only)

It is important that anyone coming into contact with pacemaker patients understands that there is a code that describes the pacing function, because they may see it written or hear it referred to. It is not important to memorise this code, but key facts are that:

- Many modern pacemakers will pace faster than the back up pacing rate programmed into them, under certain conditions, and this may be quite satisfactory pacemaker behaviour.
- The back up rate is there to prevent the heart going any slower.

OBJECTIVES OF PACEMAKER THERAPY

The main objective of pacemaker therapy is to restore the patient to as near normal a lifestyle as possible, with reduction in mortality often as a secondary goal as many of the patients requiring pacemakers are relatively elderly. Evidence exists that pacing reduces the burden and costs of stroke, atrial fibrillation and cardiac events.

Restoration of normal heart rates:

At rest symptoms of bradyarrhythmia are relieved because the heart rate cannot go below the programmed low rate of the pacemaker.

On exercise the heart rate increases with:

- Sensors built-in to the pacemaker that detect and respond to the need to raise the pacing rate.
- Dual chamber pacing where the atrial lead detects activity from the sinus node and uses this to determine the pacing rate in the ventricles after a delay that mimics the normal PR interval.

Restoration of atrioventricular synchrony:

Maintaining optimum cardiac output requires the atria to beat just before the ventricles. This happens in which also minimizes the risk of pacemaker syndrome.[4-6]

Improvement in prognosis:

Historical data suggest that pacemaker implantation in patients with complete heart block (CHB) will improve life expectancy from around 3 to 9 years; no randomized trials of pacing versus no pacing have been undertaken in this group. Evidence for sinus node disease (SND, now the largest indication for pacing across the world) shows that correct pacing mode reduces atrial fibrillation, stroke and mortality. [7-8]

DETAILED EVIDENCE OF BENEFIT IN PACING

Pacemakers have been shown in several controlled clinical trials to be effective in the management of bradyarrhythmia. A comprehensive literature review of the clinical papers was conducted for the period January 1996 to February 2004; the landmark studies reported in this time period include:

- Effects of Physiologic Pacing versus Ventricular Pacing on the Risk of Stroke and Death due to Cardiovascular Causes.[7]

- Canadian Trial of Physiologic Pacing: Effects of Physiological Pacing During Long-Term Follow-Up.[8]

- Relationship Between Pacemaker Dependency and the Effect of Pacing Mode on Cardiovascular Outcomes.[9]

- Complications Arising After Implantation of DDD Pacemakers: The MOST Experience.[10]

- Adverse Effect of Ventricular Pacing on Heart Failure and Atrial Fibrillation among Patients with normal baseline QRS Duration in a Clinical Trial of Pacemaker Therapy for Sinus Node Dysfunction.[11]

Further evidence that might be useful may come from the *United Kingdom Pacing and Cardiovascular Events Trial (UKPACE).* However, at the time of writing, that study is as yet unpublished and unavailable to the wider medical community for review.[12]

Effects of Physiologic Pacing versus Ventricular Pacing on the Risk of Stroke and Death due to Cardiovascular Causes.[7]

This large randomised controlled study, examined the effects of physiologic (dual chamber) pacing versus single ventricular chamber pacing. A total of 2568 patients were enrolled from 32 centres in Canada. Subjects with chronic atrial fibrillation were excluded. Patients were randomly assigned to receive either physiologic (n=1094) or ventricular (n=1474) pacemaker. The primary endpoint was incidence of cardiovascular death or stroke.

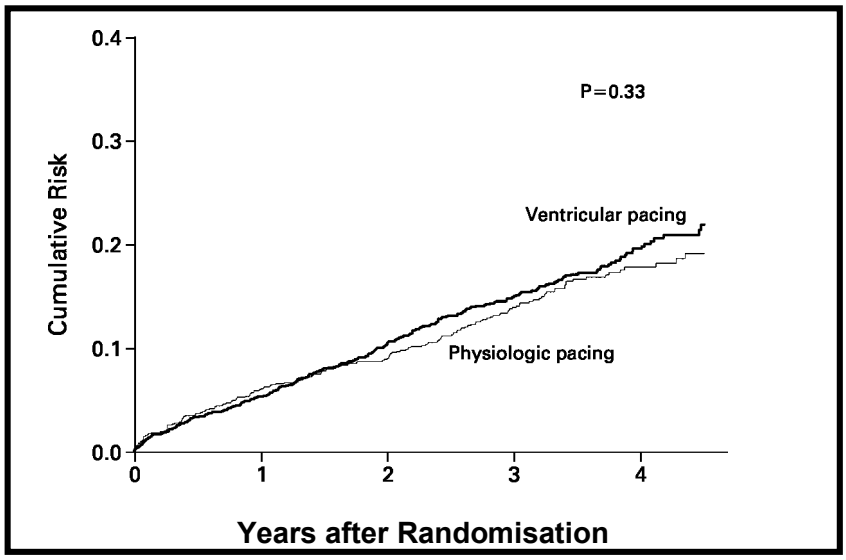


Figure 1: The cumulative risk of stroke or death due to cardiovascular causes according to pacing mode

The annual rate of stroke or death due to cardiovascular causes was 5.5% in ventricular group compared with 4.9% in physiological group. The incidence of atrial fibrillation was significantly lower in the physiologically paced arm. The relative risk reduction (RRR) was 18%. This observation was only made after two years post implantation which lead the investigators to recommend a longer period of follow-up than had originally been intended. In addition to the RRR of AF in the dual chamber arm, there was an insignificant reduction in the number of patients hospitalised for heart failure with physiologic pacing at annual rates of 3.5% v 3.1%.

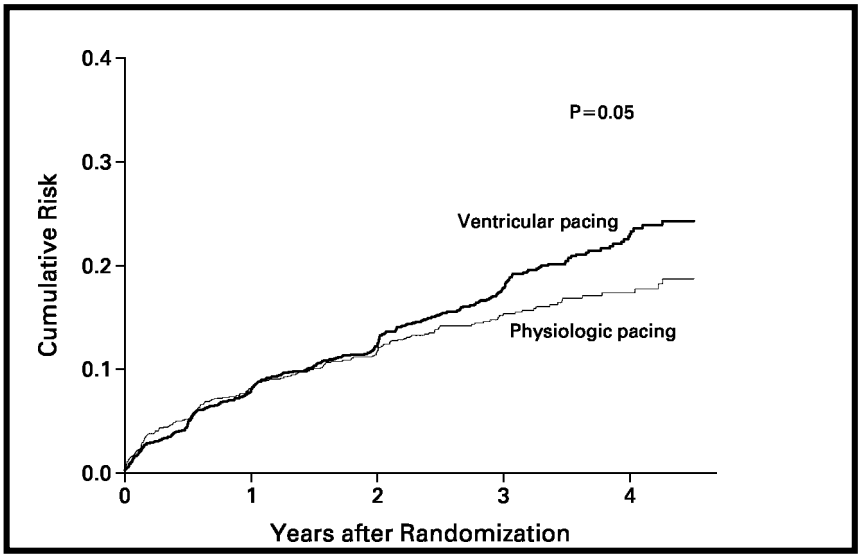


Figure 2: The cumulative risk of atrial fibrillation according to pacing mode

The authors theorised that the effects of dyssynchrony between the atria and ventricles could lead to stress on the atria leading to AF in the long term.

Thus, the CTOPP trial showed further benefits of physiologic pacing. Furthermore, there was a significant difference in the incidence of AF.

Canadian Trial of Physiologic Pacing: Effects of Physiological Pacing During Long-Term Follow-Up. [8]

Further follow-up of the CTOPP patients was published in Jan 2004, because in other trials such as the Danish Study, long-term follow-ups demonstrated significant reductions in death and the onset of atrial fibrillation in the physiologically paced group.

The original 32 centres were retained and 2002 patients remained eligible for the extension study. Although the number of patients being paced physiologically after 8 years had dropped to 75% (single chamber 93%), the observation that AF burden was reduced remained. The relative risk reduction for AF improved from 18% to just over 20%. The investigators could not confirm the earlier suggestion from CTOPP that younger patients may demonstrate the greatest long term benefits from dual chamber pacing. However, although not significant, there was a tendency for patients with slow unpaced heart rate of ≤60bpm.

The CTOPP investigators conclude that there is a persistent significant reduction in the development of AF with physiological pacing.

Relationship Between Pacemaker Dependency and the Effect of Pacing Mode on Cardiovascular Outcomes, 2001 [9]

This 'spin-off' study from CTOPP, examined the relationship between unpaced heart rate (UHR) and different pacing modes. Of the 2568 patients recruited for CTOPP, 2244 had pacemaker dependency test performed. A total of 1302 patients were assigned to ventricular only pacing, VVI(R) and 942 to physiologic pacing, DDD(R). The results did not demonstrate a statistically significant difference in the incidence of cardiovascular disease. However, a significant difference was observed in patients with poor UHR. Thus, the investigators conclude that unpaced heart rate below 60 bpm at the time of implant may be an indicator for patients who may derive the most beneficial impact from physiologic pacing modes.

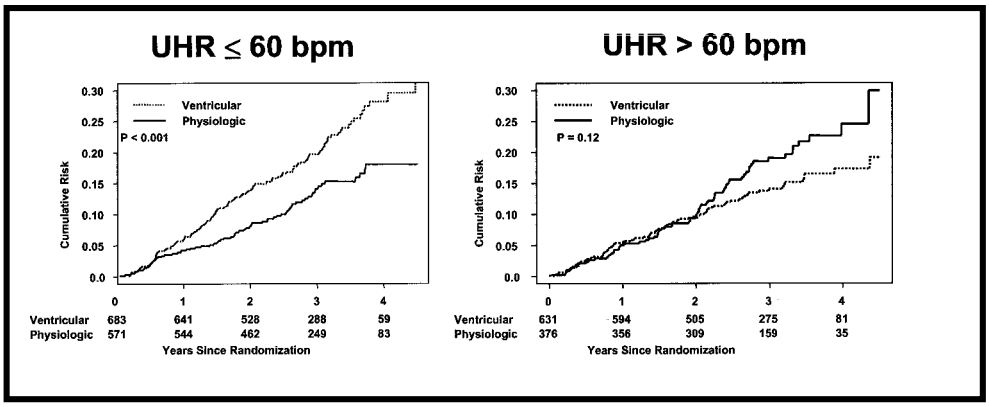


Figure 3: Kaplan-Meier estimates of cumulative risk of cardiovascular death or stroke for the 2 UHR subgroups. There was a significant treatment effect of physiological pacing in the UHR≤60bpm group but not in the UHR>60 group.

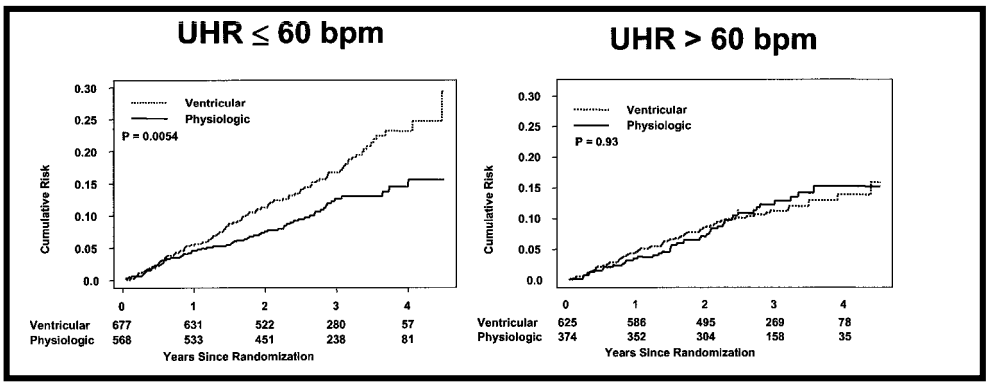


Figure 4: Kaplan-Meier estimates of cumulative risk of all-cause mortality for the UHR subgroups. There was a significant treatment effect of physiological pacing in the UHR≤60 group but not in the UHR>60 group.

This study demonstrated that UHR at first follow-up has an important influence on how pacing mode selection affects cardiovascular death and total mortality. Pacemaker-dependent patients with low UHR will probably be paced frequently and will likely benefit from physiological pacing.

Progression to Chronic Atrial Fibrillation After Pacing: The Canadian Trial of Physiologic Pacing, 2001 [13]

This study examined the effect of physiologic pacing on the development of chronic atrial fibrillation in the CTOPP trial. Patients who were prospectively found to have persistent atrial fibrillation lasting greater than or equal to one week were defined as having chronic atrial fibrillation (CAF).

The results demonstrated physiologic pacing reduced the development of CAF by 27.1%, from 3.84% per year to 2.8% per year (p=0.016). Three clinical factors predicted the development of CAF: age >74 yrs (p=0.057), sinus node dysfunction (p<0.001) and prior AF (p<0.001).

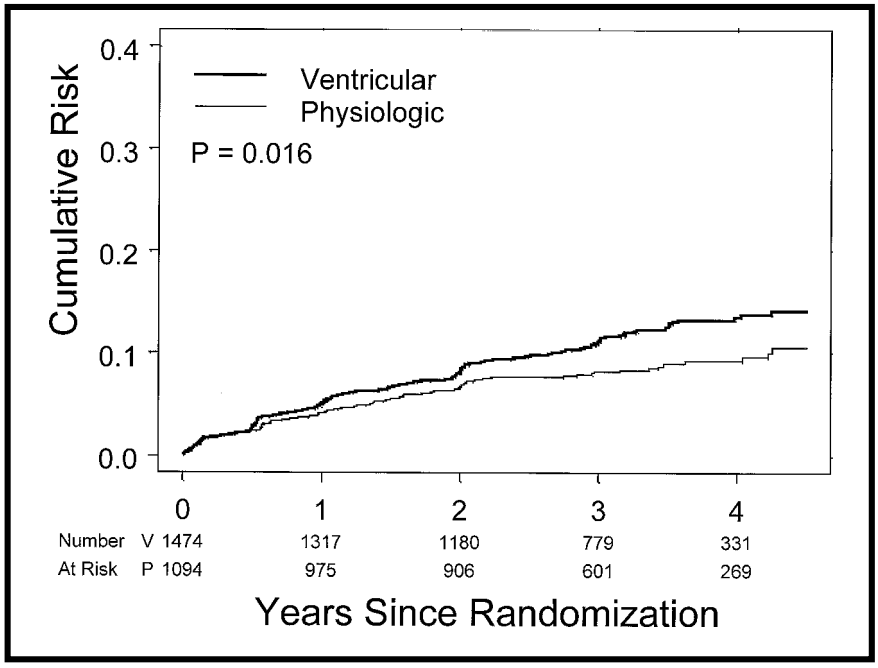


Figure 5: Cumulative risk of developing AF for both ventricular and physiologic (dual-chamber) pacing.

The authors conclude physiologic pacing reduces the annual rate of development of chronic AF in patients undergoing first pacemaker implant and that patients with structurally normal hearts appear to derive greatest benefit.

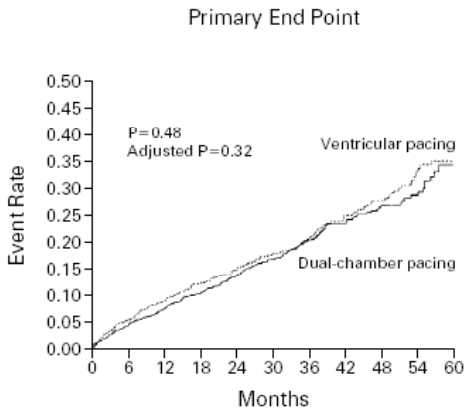
Ventricular Pacing Or Dual-Chamber Pacing For Sinus-Node Dysfunction, (MOST), 2002 [14]

The MOST Selection Trial was a five-year study designed to compare ventricular pacing and dual chamber pacing specifically in those patients with sinus-node dysfunction. The primary endpoints were death from any cause and non-fatal stroke. The secondary endpoints consisted of incidence of AF, heart failure, HF and the pacemaker syndrome. Quality of life was also a secondary endpoint.

Ninety-one centres recruited 2010 patients with a median age of 74 years. Further, 21% also had AV block. The split between dual chamber and ventricular-only was 1014 and 996 respectively.

No significant differences were reported between the two modes in respect of the primary endpoint. The incidence of AF development in the dual chamber group was significantly lower. 24.2% of the study population suffered with AF and almost 5% developed AF following implantation. Of these chronic AF ran at 15.2% in the dual chamber arm and 26.7% in the ventricular arm. This evidence then supports the fact that patients who receive a dual chamber pacemaker are 50% less likely to develop AF afterwards if they had no previous experience of such an arrhythmia. For those with prior AF, this percentage reduction was less at 14%.

There was a modest reduction in heart failure symptoms amongst the dual-chamber patients. Longer-term follow-up might have increased benefit.



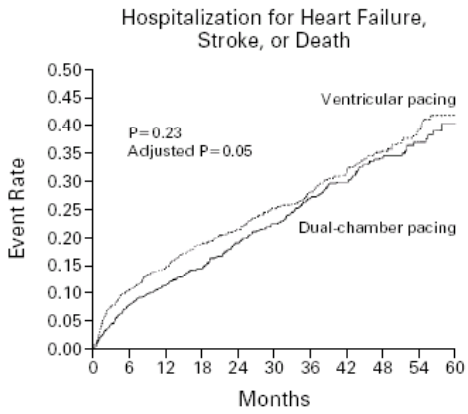
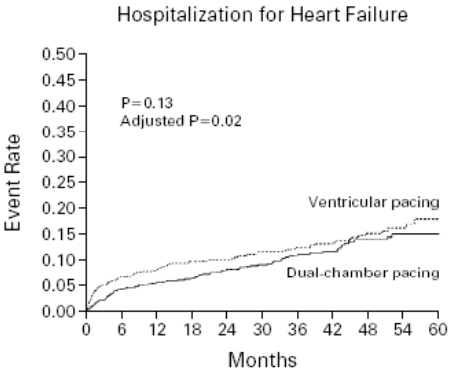


Figure 6: Rates of clinical events, (i)



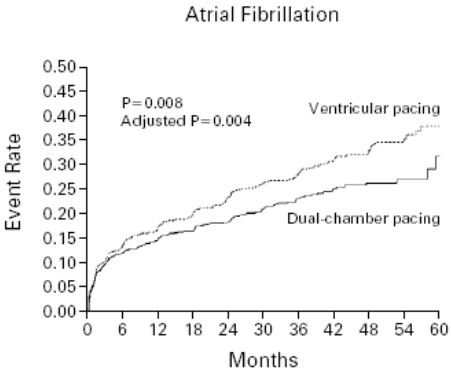


Figure 7: Rates of clinical events, (ii)

Quality of Life (QoL), was significantly better for dual chamber patients, with benefit seen at three months post implant in six out of the eight categories.

The MOST investigators concluded dual-chamber pacing reduced pacemaker syndrome, improved quality of life and reduced AF.

<u>Complications Arising After Implantation of DDD Pacemakers: The MOST Experience, 2003</u>[10]

There were no deaths within 24 hours of pacemaker implantation. Fourteen deaths occurred within 30 days of implantation. The causes of death were ischaemia (n=2), infection (n=1), VF (n=6), stroke (n=1), heart failure (n=1) and unknown (n=1). The complication rate during the median follow-up period of 33 months was 7.5% (n=146).

The three key contributors to complication as a direct result were atrial leads becoming displaced 1.7% (n=34), collapsed lung 1.5%(n=32) and ventricular lead becoming displaced 0.7%(n=15). No particular lead model or fixation method contributed disproportionately to failure and the risk of complication fell from 4.8% within 30days post implant to 2.7% later.

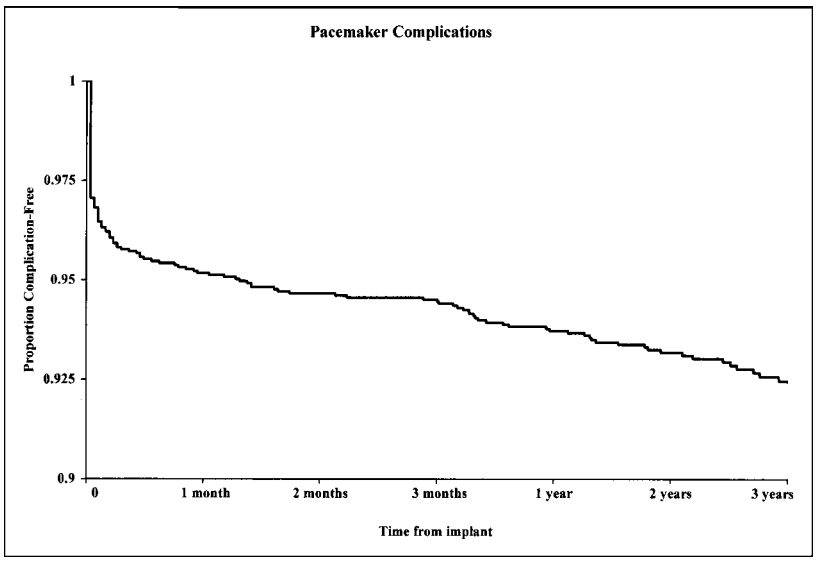


Figure 8: Kaplan-Meier plot of time course of complication resulting from pacemaker implantation

Key Points:

1. Dual-chamber pacing has an impact on the likelihood of death from cardiovascular causes, but this has been poorly quantified. Such benefits might be clearer if longer follow-up were available in published work.

2. There is a higher incidence of atrial fibrillation and pacemaker syndrome in patients paced in a ventricular mode compared to those paced in an atrial or dual mode.

3. Further studies demonstrate a non-significant trend towards benefits for dual chamber therapy in the incidence of stroke, heart failure and all cause mortality.

4. No studies reported a statistically significant benefit for single chamber pacing in the ventricles.

ECONOMIC EVIDENCE

There is scarce literature on the economics of pacemakers specifically for the UK. The Birmingham Technology Assessment Report (2001)[15] identified four studies having any aspects of costs reported and only one, Sutton & Bourgeois[16] is relevant to the UK.

Sutton & Bourgeois' [16] study concludes that the overall costs of dual chamber pacemakers to be lower than single chamber pacemakers however two older studies [17,18] concluded that the overall costs of dual chamber pacemakers were greater than the costs of single chamber pacemakers.

Clarke *et al*[19] evaluated the safety and cost-effectiveness of single chamber pacing in patients with sinus node disease in the UK. This study was a retrospective review of patients with sinus node disease (no evidence of atrioventricular block) who were implanted with a single chamber atrial pacemaker during the period 1992-1996. Patients were identified from a database of patient records at the Cardiothoracic Centre, Liverpool. Case notes were examined to establish the number of patients who required a further procedure to upgrade their pacemaker system to a dual chamber system.

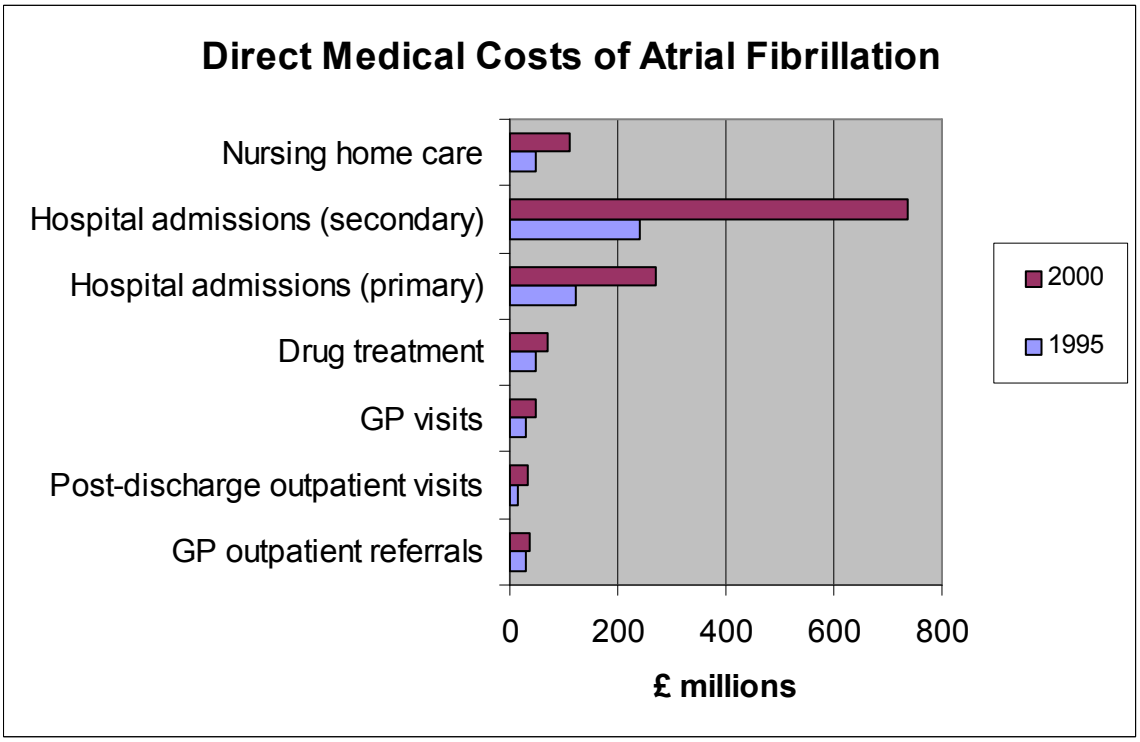
A total of 343 procedures were conducted of which 19 (5.5%) were atrial pacemakers, 271 (79%) were dual chamber and 53 (15.5%) were ventricular. The authors estimate savings of **£10,3000** per year by assuming that all implanted patients should have received atrial pacemakers and added to the cost of upgrading to dual chamber for 5.8% of patients (the rate observed in the database).

However, these analyses are not an economic but a purely financial. It does not take into consideration the total direct and indirect costs associated with the management of this group of patients. Hospitalisation costs were excluded. There were no estimates of quality of life.

Sutton & Bourgeois[16] estimated costs based on a review of the literature. This study developed a model to evaluate the cost effectiveness of dual versus single chamber therapy. Dual chamber pacing was cost effective in patients with sick sinus syndrome and in patients with AV block. In addition, the 10-year survival rate with DDD versus VVI pacing was 71% versus 57% in SSS and 61/5 vs. 51% respectively in AV block. The prevalence of heart failure in the 10-year survivors was reported as 60% lower with DDD pacing, AF is the most common sustained arrhythmia encountered in clinical practice and is associated with significant morbidity and mortality, and treatment is often complex and costly. [20,21]

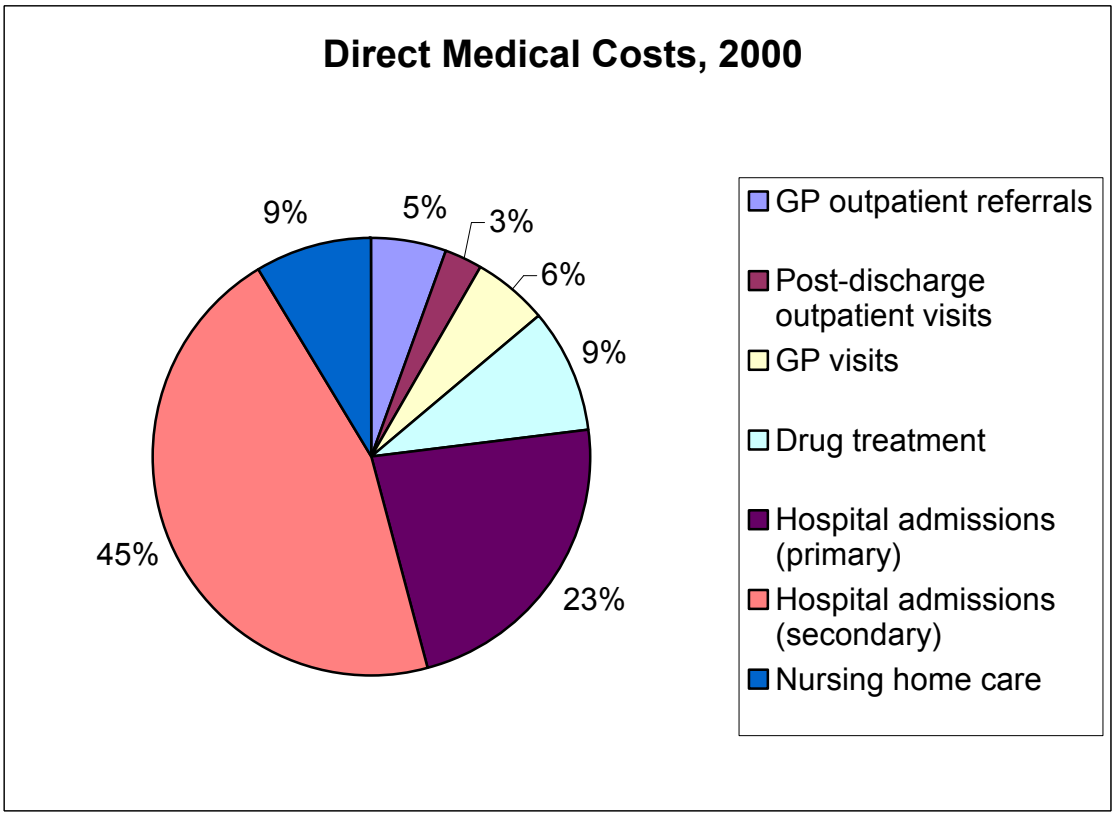
Atrial fibrillation represents a significant economic burden to the NHS. Stewart *et al*[22] conducted an economic analysis of AF in the UK that illustrates the cost of AF is approximately £459million. This study was based on a prevalence-based approach to calculating total costs to the NHS and social services. Prevalence estimates of AF were taken from UK specific cross sectional studies. The model evaluated only direct costs to the NHS and social services, these included hospitalisation, GP consultations, outpatient clinic visits and nursing home care. Over the period 1995-2000, the direct costs of AF to the NHS almost doubled from £531.2 million to £1307.4 million (Figures 9 & 10). Excluding nursing home care costs and secondary hospital admissions, the economic impact is estimated to range from £243.9m in 1995 to £459m in 2000, (see also chapter 16).

Figure 9: Cost of atrial fibrillation as a potential complication



Source: adapted from Stewart, S[22]

Figure 10: Relative costs incurred by atrial fibrillation



Source: adapted from Stewart (22)

Key Points:

1. Little good data exists to make a convincing case for the cost-effectiveness of cardiac pacing, but this reflects lack of quality research, as benefits are clear, especially in obvious bradyarrhythmias.
2. All the co-morbidities that are seen in inappropriate pacing prescription, such as AF, heart failure and hospitalisation, are known to be a huge drain on healthcare resources.
3. Pacing is very cheap when amortised over the life of a pacemaker of 8-12 years.
4. Pacing for intermittent bradyarrhythmias is likely to be a very effective antidote to repeated blackouts and hospital admissions, but these patients are difficult to manage and may get very poor care in the UK.

IDENTIFYING PATIENTS REQUIRING PACEMAKER IMPLANTATION

Presenting symptoms/signs:

Patients with bradyarrhythmias suffer syncope, pre-syncope, falls, breathlessness and exercise intolerance. Many patients may accept such symptoms as part of the ageing process, delaying diagnosis.

This is especially likely in the patient with *intermittent* bradyarrhythmia.

Patients with bradyarrhythmia due to abnormalities of the cardiac conduction system will benefit from pacemakers; some patients with bradyarrhythymia due to excessive vagus nerve activity (i.e. reflex syncope) may be better treated with drugs as first line. In adults the

drug with strongest clinical evidence for benefit is midodrine; in children atropine or similar drugs are used in such situations.

Investigations:

Where a bradyarrhythmia is not obvious ambulatory ECG monitoring, external loop and implantable loop recorders along with echocardiography, tilt testing and, in rare case requiring referral to tertiary centers, electrophysiological testing. These investigations are underprovided in the UK. There are staff shortages at medical, technical and nursing levels, and it is likely that many patients in the UK with intermittent bradyarrhythmias are not getting pacemakers when they should be. Patients with obvious bradyarrhythmias are, because they are easily recognised.

Getting the pacemaker prescription right:

Recommendations for pacemaker prescription in the UK were published 1991;[23] although these have been reviewed by other organisations (e.g. ACC/AHA)(3) they have not been significantly revised in the light of studies during the last decade. AAI is not frequently used in the USA and the UK due to concern over progression to AV block in sinus node dysfunction. The rate of this progression is thought to be around 2-6% per year.[19,24]

Table 6: Recommended pacing modes

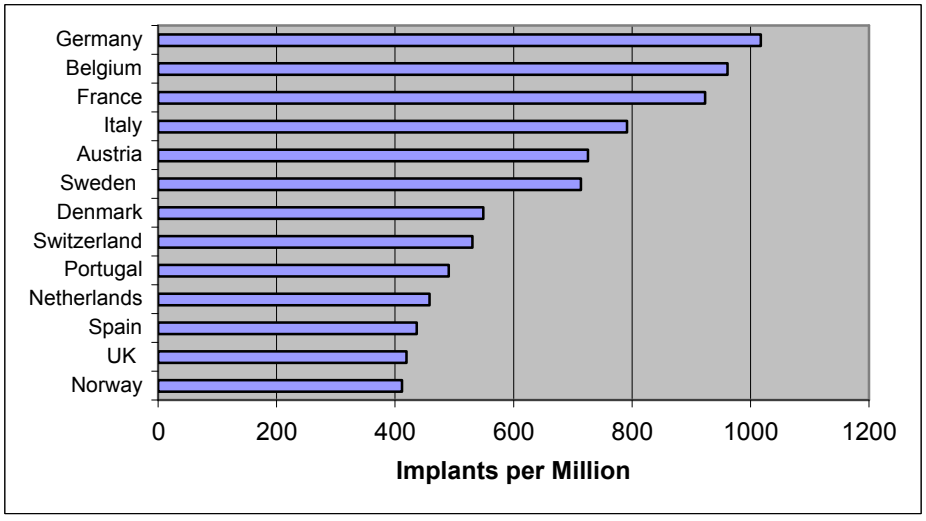
Diagnosis	Optimal	Alternative	Inappropriate
SND	AAIR	AAI	VVI VDD
AV block	DDD	VDD	AAI DDI
SND& AV block	DDDR DDIR	DDD DDI	AAI DDD VDD

Source: BPEG recommended pacing modes (1991) for selected diagnoses[i]

SHORTFALL IN PACEMAKER IMPLANTATION COMPARED WITH EUROPE

The UK has very low rates of pacemaker implantation compared with Western European countries. [25]

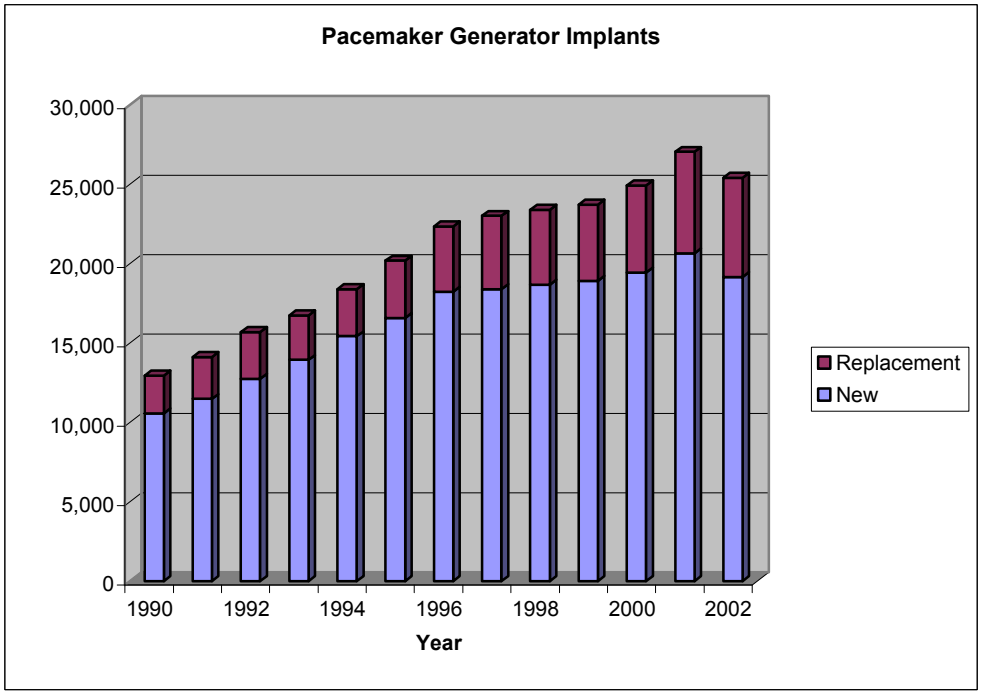
Figure 11: Pacemaker implantation in the major Western European countries during the period April 2002-March 2003. (New and replacements).



There is no evidence that the UK has a lesser incidence of bradyarrhythmia then Western Europe. The UK clearly has an implantation rate of 420 per million. Germany has an implantation rate of 1018 per million population.

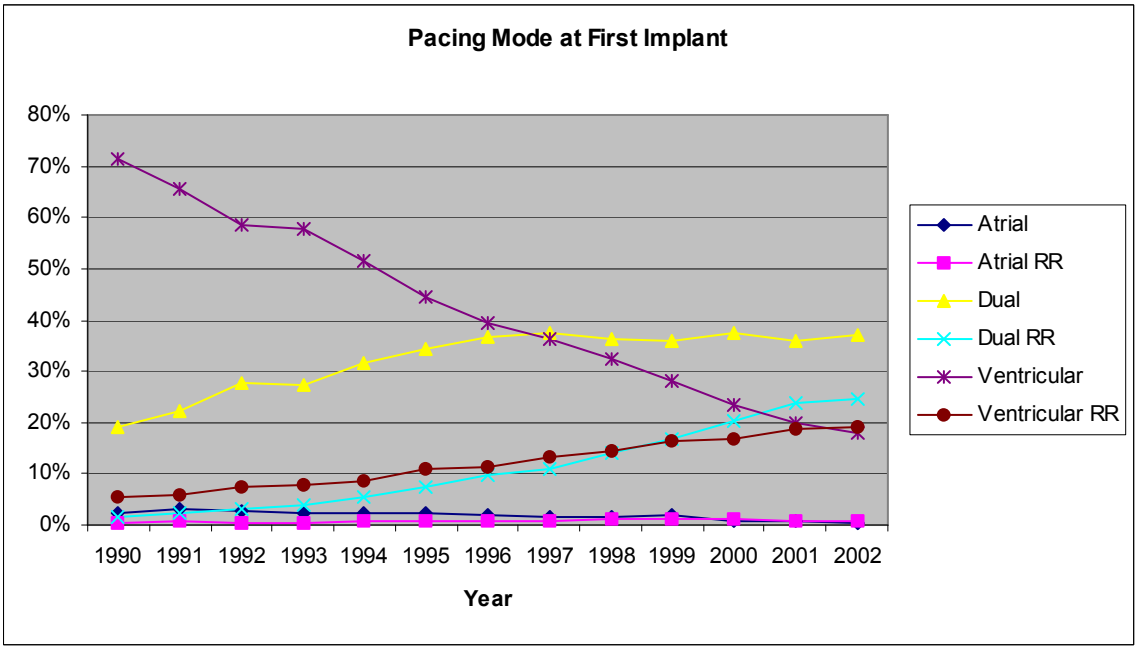
Activity in the UK

Figure 2: The growth of total pacemaker implants (new and replacement) over the period 1990-2002. There is very little growth recent growth, with a decrease in the total number of implants in 2002 (25,397) compared to 2001 (27,028).



Improvements in technology have led to the use of a greater number of dual chamber pacemakers over the period 1990-2002, (see Figure 3).

Figure 3: Pacing mode at first implant in the UK between 1990 and 2002



According to the National Pacemaker and ICD database report (2002)[25] the majority of dual chamber pacemakers were implanted for sick sinus syndrome and complete heart block.

In summary, although, the annual implantation rate in the UK has been increasing during the past decade, there has been little growth in the past 4 years and the implant rate remains 29% behind that of other European countries (Birmingham report, 2001)[15] The British Pacing and Electrophysiology Group (BPEG) guidelines recommend dual chamber or single chamber rate-responsive pacemakers as optimal therapy;[23] however it is clear from the literature that there has been limited uptake of these guidelines.

Key Points:

1. The UK has very low levels of pacing compared to Western European countries.
2. The reasons for this are multi-factorial but include; very low levels of cardiology staffing, patients under the care of the wrong doctors, strain on investigative facilities, and failure to correctly investigate and diagnose intermittent bradyarrhythmias.
3. UK pacemaker implant rates have increased steadily until 2002, but now appear to have stopped increasing.
4. Many cardiologists working in DGHs have to undertake unselected acute medical duties, and cannot concentrate on delivering cardiology services.
5. DGH cardiologists should be relieved of acute medical duties as soon as staffing levels allow, should concentrate on delivering cardiology services, and a high priority amongst these is the establishment of a local, DGH based pacemaker service in all Districts.
6. Pacemakers are very effective treatment, completely resolving severe symptoms in many patients. They are very cheap when costs are considered over the life of the pacemaker, and pacemakers are very reliable. Cost-skimping on pacemaker prescription is therefore not a valid strategy, and may lead to new symptoms and an increased risk of costly side-effects, e.g. more AF.

LEVELS OF PACEMAKER IMPLANTATION WITHIN THE UK ARE VERY VARIABLE

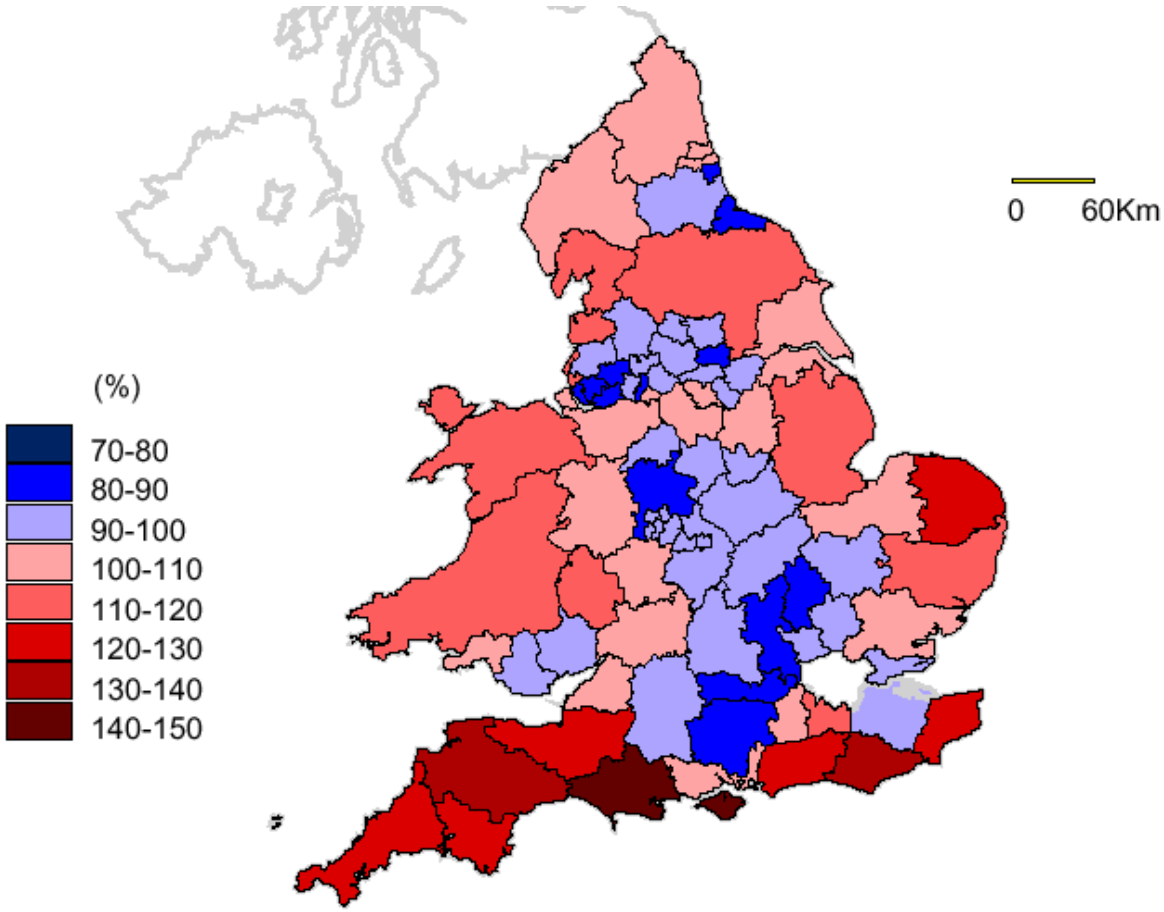
Cardiac pacing is performed mainly in older patients (average age in UK = 74 years). Variation in pacing rates are affected by, among other factors, population age.

Dorset has high pacing rates as a result of an elderly population (*data kindly supplied by Dr A Rozkovec, Bournemouth*); as a result of this high pacing rate a recent investigation examined the reasons for high pacing rates and concluded that the rate was entirely appropriate for the population served. This highlights the need for pacing services to be linked to population served rather than national averages.

Population data were obtained from the Office for National Statistics (ONS) as mid-1998 estimates by District Health Authority (DHA). Data were available for 216 district health authorities in England and Wales.

A relative need for pacing was calculated for each DHA in England and Wales by combining known pacing rates for different age groups with the age distribution in each DHA. Relative need for pacing provision ranged by DHA from 147% (i.e. 47% *higher* need than average) to 70% (30% *lower* than average requirement).

Figure 12: Relative need for pacing in England and Wales



Relative Need for Pacing by District Health Authority

It is immediately clear that Dorset and the Isle of Wight have the highest relative need for pacing in England and Wales.

Data are from O.P.C.S. mid-1992 estimates for England and 1997 actual for Dorset

	Males		Females	
	Dorset	**England**	**Dorset**	**England**
% 65 and over	**18.4%**	**13.1%**	**25.3%**	**18.6%**
% 75 and over	**8.4%**	**4.9%**	**13.9%**	**9.1%**
% 85 and over	**1.8%**	**0.8%**	**4.4%**	**2.4%**

Service Delivery

Pacing is undertaken in some DGHs in the UK, but many patients are still transferred to other hospitals for pacing.

Many patients presenting with symptomatic bradyarrhythmia do so as an emergency admission, and they are nursed on a general medical or geriatric ward. Often there are long delays before transfer for pacing. In older patients these delays may contribute to delayed discharge after pacemaker implantation.

Provision of pacing in **every DGH** would greatly reduce the expense and impact of these delays.

Short stay/Day case:

The majority of routine permanent pacing can be done with a short stay (e.g. 23 hour) or as a day case. This is much easier if no hospital-to-hospital transfer is required. Pacemaker replacements will usually be undertaken day case procedures unless complicated by other factors e.g. need for anticoagulation.

Permanent pacing in every DGH and good liaison with primary care should extend to day case implantation in the future.

Treatment Centres:

With expansion of cardiology consultant posts all DGHs should prioritise a permanent pacemaker service. Training in cardiac pacing for bradyarrhythmia must be emphasised so that all new consultants appointed in the UK can provide a pacing service given appropriate support.

Current recommendations are:

- A minimum of 2 cardiologists with appropriate training and experience.
- A minimum implant rate of 50 pacemakers per unit per year, with at least 40% of these being dual chamber.
- Each cardiologist should implant a minimum of 20 pacemakers per year
- If an implanter falls below 12 implants per year for 2 consecutive years, re-training is necessary.
- Provision for implants should be made on the basis of 52 weeks per year.
- Facilities for pacemaker implantation should be as given in the recent Fifth Report from the British Cardiac Society.

Secondary versus tertiary interface:

Most routine pacemaker implantation will take place in a secondary care setting but good links to a tertiary centre will be necessary for a limited number of patients.

These include:

- Patients who have failed implant at a secondary centre
- Complex anatomical problems in patients with congenital heart disease (paediatric or adult)
- Patients with pacemaker infection requiring lead extraction – this should be undertaken in experienced centres with cardiac surgical facilities
- Patients requiring biventricular pacing (resynchronization therapy) or combined ICD/pacemaker therapy
- Patients requiring investigation not always available in secondary centres e.g. those with syncope

Key Points:

1. Post-code provision of pacemaker services is very much a feature of the UK, and pacing services rely on local enthusiasm too often.
2. Local commissioners of care need to be educated and motivated to provide an adequate pacemaker service locally in their DGH setting.
3. Resources need to be identified, possibly by provision of "Acute Medical Physicians" to allow cardiologists in DGHs to give up acute medical responsibilities and concentrate on acute cardiology and the provision of local pacing services.
4. Many patients in DGHs wait far too long for permanent pacing. Often this involves a temporary pacing electrode in situ which is an infective risk, and competition with other patients for transfer into a "Regional Centre".
5. Pacing services need to be urgently devolved into the DGHs, making better use of DGH cardiologists skills, providing rapid access at the point of need, and freeing up time in "Regional Centres" for tertiary cardiac care.

DEVELOPMENT NEEDS AND GOALS

The Fifth Report [26] recommends implant rates of 450/million population per year (new pacemakers) or 550/million including pacemaker replacements. This target is well above current UK levels and well below the Western European average [25]

Developments in cardiac pacing need therefore to recognise:

- Geographical variation in needs according to population age
- An overall need to increase pacemaker implant rates across the UK
- A need to plan for future technological improvements in pacemaker therapy which are likely to be associated with increased cost
- Further evidence-based changes in practice will have additional resource implications; these may include new indications for pacing (e.g. anti-tachycardia, multi-chamber pacing) and changes in current pacemaker prescription
- These developments need supporting infrastructure in primary, secondary and tertiary care, including education, training, staffing and diagnostic facilities at all levels
- As the population continues to age, more pacemakers will be required; patients will also live longer with their devices and need more replacement systems

TRAINING

Technical:

Technical staff in pacing centres shoulder much of the work, but they are a very scarce resource.

Their responsibilities are rarely reflected in grading/salary; training is often limited by service activities and staffing levels. The development of a Consultant technical grade will offer chance to correct this situation, improve the standard and consistency of service, and improve career opportunities and staff retention.

Many of the skills learned through the pacemaker service are transferable to other areas, e.g. ICD management; staff training should reflect this in order to assist with development of services in these other areas.

Nursing:

In the current pacemaker care models, nursing staff tend to have lesser input than technical or clinical/medical staff. Involvement is largely through implant laboratories, coronary/cardiac care units and brief ward-based care; recent concentration on ischaemic heart disease means that many nursing staff managing pacemaker patients have little understanding of the devices or their role in arrhythmia management. Education and training could extend the role of nurses in screening patients for bradyarrhythmia in the primary care/admission unit environments, in managing concurrent problems in pacemaker clinics (many elderly patients have multiple complaints (some cardiac) not related to their pacemaker; these often present problems for technical staff in the clinic) and also perhaps in the implant process as has occurred in cardiac catheterisation.

Clinicians:

Many cardiologists see pacemaker implantation as a chore rather than because of a specific interest. This is certainly the result of poor training by other disinterested cardiologists combined with the strong training emphasis on ischaemic heart disease and coronary intervention.

All SpRs must be evaluated in competency for recognising patients with symptoms and signs of bradyarrhythmia, interpreting appropriate investigation results and implanting permanent pacemakers. This will staff the local pacing services that are needed. Many patients present to primary care physicians, emergency physicians, general physicians and geriatricians; these clinicians may be less familiar with the diagnosis and management of bradyarrhythmia and are often unaware or unconvinced of the appropriateness of pacemaker implantation in the elderly or very elderly. Education and training should, therefore, be extended to these groups in order to ensure prompt and appropriate identification and referral of patients with bradyarrhythmia.

INTERFACE WITH BIVENTRICULAR PACING/ANTITACHYCARDIA PACING/ICDS

Although these therapies are the subject of a separate report, there is considerable overlap in patient population and skills of technical, nursing and clinical staff managing patients with pacemakers and these other implantable devices. In addition, many patients starting out with pacemakers may subsequently need revision of their pacemaker system (possibly through the detrimental effects of cardiac pacing which are just becoming apparent or through deterioration in cardiac function from ageing or other cardiac disease) to a biventricular pacemaker, or ICD. The patient population requiring implantation of these newer devices is still being defined; as a result, this area of crossover is likely to increase in the next 3-5 years.

Strategic planning

Immediate:

The immediate need is to plan to meet the targets for pacemaker implantation set out in the Fifth Report. This requires recognition of need from PCTs in addition to extension of service provision, predominantly within secondary referral centres. The average increase in activity will be from 420 to 550 implants per million per year; assuming this is spread relatively evenly across the country this equates to an increase in activity of approximately 30%, or rates between 385 and 809 per million (from 294-617 total implants per year).

Cardiologists in DGHs should be freed from non-cardiology duties in order to increase the time available to set up and run a DGH pacing service.

Dr Michael Gammage, University of Birmingham

Medium term:

There is little to suggest that the prevalence of bradyarrhythmia should be significantly different in the UK in comparison with other Northern European populations (eg France, Germany, Belgium); if anything, bradyarrhythmia might be anticipated to be more common as the UK population is older. This would suggest that implant rates of 860-1018/million might be anticipated if adequate screening and diagnostic facilities were in place in the UK. This would represent an increase in activity of 40-50% on top of that required to come in line with the Fifth Report recommendations. It may be that implant rates are excessive in those countries (driven, perhaps, by reimbursement rather than clinical issues) but it seems unlikely that that such a great difference is entirely due to differences in practice. It would seem reasonable, therefore, to plan for further increases to bring total implant rates up to the region of 800/million over the next 5 years.

Cardiologists in training should be encouraged to take a keen interest in the indications for pacemaker and other device therapy, implantation of devices and device functions and follow-up, since this will likely become a core duty of provision in DGHs.

Long term:

Pacing therapy has been driven for four decades by technological development but is now being influenced by new considerations, for example the functional effects of pacing site; this led to the development of biventricular pacing as a new therapy for heart failure rather than bradyarrhythmia. Similar technological developments are likely to extend pacemaker/device therapy into new clinical areas; this will require the skills of technical/nursing and clinical staff to be utilised in a wider patient population and hence will require further expansion of staffing and facilities. Traditional pacemaker therapy may be replaced in the next 20 years by bio-therapy but the skills to deliver and monitor such therapy, along with the mechanisms to identify and follow-up appropriate patients, will remain a limiting step in the management of bradyarrhythmias.

With an aging population more patients will have bradyarrhythmias. Regular review of service requirements will be needed.

Key Points:

1. A National Pacing Strategy must work to free up DGH cardiologist's time.
2. A National Pacing Strategy must recognise and tackle "post-code" prescribing of pacemakers.
3. Local commissioners must recognise the health and economic benefits of pacing over the full life of a pacemaker, and the very real benefits to quality-of-life, free of hospital, brings to patients who are successfully paced.
4. Training for SpRs in cardiology must correct the notion that pacing is less valuable than other activities, e.g. coronary intervention, and SpRs must have a very thorough grounding in pacing and arrhythmia management to enable them to provide local services in DGHs where appropriate.
5. Technical staff to support pacing services are in short-supply, and may limit attempts to establish a National Pacing Programme in DGHs, thus s concerted effort to grow the workforce is urgently needed.
6. Pacing services provide huge benefits to patients, and may provide a skills-base for more complex device activity, such as ICD and biventricular pacing. Such activities should be established on the back of a proven track-record of provision of a safe, effective and successful pacing service.

Dr Michael Gammage, University of Birmingham

LAY SUMMARY

Pacemakers were first put in over 40 years ago. There is no other effective treatment for symptoms related to a slow heart beat. Pacemakers maintain the heart beat by triggering heart muscle with an artificial electric current. They consist of a sealed battery-box that also contains electrical circuits and a radio-link which can talk to a pacemaker programming computer over short distances, including through the skin after surgery. The battery-box is connected to one or more plastic-coated wires that are usually passed through veins and steered into the heart under X-ray control. Coils or fins are built into the tips of these wires or electrodes to keep them anchored to the heart muscle. Very low levels of current are needed to stimulate a heartbeat. Modern pacemakers only stimulate when needed and can detect the heart's own beats, preventing a pulse from the pacemaker battery. Tiny crystals inside the pacemaker box respond to vibration and muscle movement by speeding the pacemaker up. Pacemakers are usually implanted under local anaesthetic to freeze the skin, in a catheter laboratory within 1-2 hours, and may be safely done on the day of discharge home.

There is evidence that pacing may reduce atrial fibrillation, stroke and mortality.

Patients who might benefit from a pacemaker may present to family doctors, casualty departments, local hospitals or teaching centres. Some patients who present may have symptoms and a very obvious slow pulse, and all of these patients are believed to get pacemakers in the UK, although they may not be available in the hospital where a patient is admitted, and patients may have a long wait. Other patients only have a slow or unreliable heart beat part of the time, and this may cause dizziness, blackout, palpitation and breathlessness, but may not be evident on an ECG when a patient is admitted, puzzling doctors about the cause of the symptoms. Many of these patients do not receive a pacemaker, even though they deserve one.

The failure to recognise this may explain why the UK implants only about 420 pacemakers/million people, and Western Europe implants around 900/million. The UK is poorly served for pacing services compared to Western Europe, and services are patchy, often depending on the presence locally of an enthusiastic specialist. Such specialists often have a very hard time getting co-operation in establishing services from local commissioners of care in spite of very low implant rates across the UK. This is because Government has not yet made a priority of these services, cost-effective and simple though they are. Many of these cardiologists are still required to partake in general medical duties, rather than concentrate on cardiology and services such as pacing. This is because Hospital Trusts have not made a priority of pacing services cost-effective and simple though they are.

A good local pacing service requires at least two fully trained doctors (Consultant Cardiologists) skilled in fitting pacemakers, as well as two trained technical staff and good quality facilities for safe implantation and follow-up. To achieve this across the UK will require a major expansion in trained doctors, and money for good facilities.

Newer types of pacing are rapidly increasing the population who can benefit by control of symptoms and prolongation of life. Development of these newer services should be built upon the foundation of an established local pacemaker service.

Dr Michael Gammage, University of Birmingham

REFERENCES:

1. Furman S, Schwedel JB. An intracardiac pacemaker for Stokes-Adams seizures. N.Engl.J.Med. 1959;261:943-8.
2. Bernstein AD, Camm AJ, Fletcher RD, Gold RD, Rickards AF, Smyth NP et al. The NASPE/BPEG generic pacemaker code for antibradyarrhythmia and adaptive-rate pacing and antitachyarrhythmia devices. Pacing Clin.Electrophysiol. 1987;10(4 Pt 1):794-9.
3. Gregoratos G, Cheitlin MD, Conill A, Epstein AE, Fellows C, Ferguson TB, Jr. et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J.Am.Coll.Cardiol. 1998;31(5):1175-209.
4. Ausubel K, Boal BH, Furman S. Pacemaker syndrome: definition and evaluation. Cardiol.Clin. 1985;3(4):587-94.
5. Ellenbogen KA, Gilligan DM, Wood MA, Morillo C, Barold SS. The pacemaker syndrome -- a matter of definition. Am.J.Cardiol. 1997;79(9):1226-9.
6. Furman S. Pacemaker syndrome. Pacing Clin.Electrophysiol. 1994;17(1):1-5.
7. Connolly SJ, Kerr CR, Gent M, Roberts RS, Yusuf S, Gillis AM et al. Effects of physiologic pacing versus ventricular pacing on the risk of stroke and death due to cardiovascular causes. Canadian Trial of Physiologic Pacing Investigators. N.Engl.J.Med. 2000;342(19):1385-91.
8. Kerr CR, Connolly SJ, Abdollah H, Roberts RS, Gent M, Yusuf S et al. Canadian Trial of Physiological Pacing: Effects of physiological pacing during long-term follow-up. Circulation 2004;109(3):357-62.
9. Tang AS, Roberts RS, Kerr C, Gillis AM, Green MS, Talajic M et al. Relationship between pacemaker dependency and the effect of pacing mode on cardiovascular outcomes. Circulation 2001;103(25):3081-5.
10. Ellenbogen KA, Hellkamp AS, Wilkoff BL, Camunas JL, Love JC, Hadjis TA et al. Complications arising after implantation of DDD pacemakers: the MOST experience. Am.J.Cardiol. 2003;92(6):740-1.
11. Sweeney MO, Hellkamp AS, Ellenbogen KA, Greenspon AJ, Freedman RA, Lee KL et al. Adverse effect of ventricular pacing on heart failure and atrial fibrillation among patients with normal baseline QRS duration in a clinical trial of pacemaker therapy for sinus node dysfunction. Circulation 2003;107(23):2932-7.
12. Toff WD, Skehan JD, De Bono DP, Camm AJ. The United Kingdom pacing and cardiovascular events (UKPACE) trial. United Kingdom Pacing and Cardiovascular Events. Heart 1997;78(3):221-3.
13. Skanes AC, Krahn AD, Yee R, Klein GJ, Connolly SJ, Kerr CR et al. Progression to chronic atrial fibrillation after pacing: the Canadian Trial of Physiologic Pacing. CTOPP Investigators. J.Am.Coll.Cardiol. 2001;38(1):167-72.
14. Lamas GA, Lee KL, Sweeney MO, Silverman R, Leon A, Yee R et al. Ventricular pacing or dual-chamber pacing for sinus-node dysfunction. N.Engl.J.Med. 2002;346(24):1854-62.
15. Dretzke J, Lip G Raftery J Toff W Fry-Smith A Taylor R. Dual chamber versus single chamber pacemaker therapy for atrioventricular block and sick sinus syndrome. West Midlands Health Technology Assessment Group. 1-106. 2001. University of Birmingham. Birmingham Technology Assessment Group Report. Department of Public Health and Epidemiology.
    Ref Type: Serial (Book,Monograph)
16. Sutton R, Bourgeois I. Cost benefit analysis of single and dual chamber pacing for sick sinus syndrome and atrioventricular block. An economic sensitivity analysis of the literature. Eur.Heart J. 1996;17(4):574-82.
17. Eagle KA, Mulley AG, Singer DE, Schoenfeld D, Harthorne JW, Thibault GE. Single-chamber and dual-chamber cardiac pacemakers. A formal cost comparison. Ann.Intern.Med. 1986;105(2):264-71.

18. Hughes PD. The use of poorly validated and expensive technology. Med.J.Aust. 1994;161(11-12):726.
19. Clarke KW, Connelly DT, Charles RG. Single chamber atrial pacing: an underused and cost-effective pacing modality in sinus node disease. Heart 1998;80(4):387-9.
20. Dell'Orfano JT, Patel H, Wolbrette DL, Luck JC, Naccarelli GV. Acute treatment of atrial fibrillation: spontaneous conversion rates and cost of care. Am.J.Cardiol. 1999;83(5):788-90, A10.
21. Dell'Orfano JT, Kramer RK, Naccarelli GV. Cost-effective strategies in the acute management of atrial fibrillation. Curr.Opin.Cardiol. 2000;15(1):23-8.
22. Stewart S. Epidemiology and economic impact of atrial fibrillation. J.Cardiovasc.Nurs. 2004;19(2):94-102.
23. Clarke M, Sutton R, Ward D, Camm AJ, Rickards A, Ingram A et al. Recommendations for pacemaker prescription for symptomatic bradycardia. British Heart Journal 1991;66(2):185-91.
24. Kristensen L, Nielsen JC, Pedersen AK, Mortensen PT, Andersen HR. AV block and changes in pacing mode during long-term follow-up of 399 consecutive patients with sick sinus syndrome treated with an AAI/AAIR pacemaker. Pacing Clin.Electrophysiol. 2001;24(3):358-65.
25. Cunningham, D, Rickards, A, and Cunningham, M. National Pacemaker Database. 2002. Ref Type: Internet Communication
26. Royal College of Physicians, British Cardiac Society. Fifth report on the provision of services for patients with heart disease. Heart 2004;88(Suppl III):iii1-iii59.