BASICS OF CARDIAC PACING: SELECTION AND MODE CHOICE

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Heart 2006;**92**:850–854. doi: 10.1136/hrt.2005.076661

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Therapeutic cardiac stimulation has been in clinical practice for many decades.[1] During the evolution of the therapy, pacing generators have shrunk in size, increased in longevity, and increased in device complexity. They offer rhythm and disease state diagnostics, with various algorithms designed to offer timing of delivery of pacing impulse and more recently diagnostics indicative of pathophysiology. Pacing leads have also reduced in size and improved in durability. Pacing electrode configurations have enhanced sensing capability and stability of long term pacing function.

With the potential for a range of biosensors that could be incorporated in pacemaker devices to further the potential for pathophysiological monitoring, cardiac pacing has achieved an extraordinary maturity in the armoury of cardiac treatments. However, it has taken much longer for there to be large scale clinical trials on the therapeutic efficacy of devices, long term patient well being, and the impact of right ventricular pacing on left ventricular function.

INDICATIONS AND MODE CHOICE

These are summarised in published European and North American guidelines.[2 3] There are three electrophysiological conditions (sinus node disease, atrioventricular (AV) node disease, and neurally-mediated (cardio-inhibitory) syncope) which may cause either prognostically or symptomatically significant bradycardia, and this review focuses only on these. These conditions may be treated by single chamber ventricular sensing/pacing, dual chamber sensing/pacing or (if AV nodal function is normal) single chamber atrial sensing/pacing.

The choice of appropriate pacing modality to treat these electrophysiological abnormalities is governed by our understanding of the morbidity that attends the conditions themselves, the influence of pacing on that morbidity, and a further morbidity that may attend the chosen pacing mode. To understand this complex interaction we need to look to the evidence base that reports efficacy and complications of pacing therapy in varying patient populations with differing electrophysiological and cardiac disease.

STUDY BACKGROUNDS

This article focuses on six major studies which have investigated the relative benefits and disadvantages of dual and single chamber pacing[4–8] and on a single major investigation of vasovagal syndrome.[9] Many studies have investigated the effect of different pacing modalities in specific electrophysiological disease conditions. Others have investigated the pacing modality on a generality of bradycardia syndromes. These different approaches complicate the understanding of the relative merits of pacing modality choice.

There are three physiological mechanisms contributing to generation of increased cardiac output with exercise: myocardial contractility, AV synchrony, and heart rate. It is the latter that is the most effective in generating an increased cardiac output. Pacing per se is unable to influence contractility (although it is arguable that the benefits of resynchronisation pacing is a manifestation of improved contractile coordination, and with remodelling, improved contractility). A major consideration is whether the achievement of A-V synchrony (by pace-sense manoeuvres) adds sufficient to paced rate response to justify the implantation of a dual chamber system, which in most instances necessitates the implantation of separate atrial and ventricular leads. While in the presence of sinus node disease a VDD system can operate satisfactorily with a single A sense-V pace lead (which is a well developed technology), this approach has not achieved widespread clinical acceptance.[10] Single atrial lead pacing has all but disappeared from clinical practice,[11] in part due to the perception that is an unsafe clinical strategy because of the increased risk of developing complete heart block in patients with pre-existing sinus node disease. The evidence base suggests that such progression is not very frequent with a series of well performed studies showing that the development of heart block runs at 5–10% over a 3–5 year follow-up period.[12–14] Nevertheless, the risk associated with heart block and perhaps the nuisance of performing single to dual chamber upgrade leads many clinicians to favour dual chamber pacing

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with programming of the device in such a way as to minimise unnecessary ventricular pacing. This strategy needs further evaluation.

The importance of atrial contraction and its timing relative to ventricular diastolic function is well understood. Seminal, comprehensive but small scale studies confirmed (at least) the immediate apparent physiological benefit conferred by this strategy and ushered in the era of dual chamber pacing.[15 16] However, complexity, potential procedural morbidity, and cost are all greater with a more complex pacing approach. Thus, the benefits of dual versus single chamber pacing in a variety of clinical contexts have been revisited.

PACING FOR NEURALLY-MEDIATED SYNCOPE

There is clear evidence that single chamber ventricular pacing is disadvantageous in the management of neurally-mediated syncope.[17–19] There is also evidence that neurally-mediated syncope is a much greater clinical problem than is generally recognised.[20 21] A recent major European study of dual chamber pacing in patients with "tilt positive" cardio-inhibitory syncope demonstrated that this treatment approach offers long term protection from recurrent syncope, but patient selection for pacing therapy remains challenging as even in untreated patients the burden of recurrence is low. The population of elderly "fallers" is potentially much larger and in them dual chamber pacing is very effective at reducing the risk of recurrent falls.[22]

DUAL CHAMBER PACING FOR SINUS NODE DISEASE

The "Danish study" was a multicentre investigation of dual chamber pacing.[4] Patients received a pacemaker implant if they suffered from symptomatic bradycardia which was defined as a heart rate less than 50 beats/min or symptomatic pauses greater than 2 seconds. To be enrolled patients were older than 50 years, with no history of chronic or paroxysmal atrial fibrillation and with no evidence to suggest significant underlying A-V conduction disease.

The study's primary end points were all cause mortality or cardiovascular death, secondary end points being onset of atrial fibrillation, thromboembolic events, or development of heart failure or AV block. Patients were randomised to either VVI or AAI pacing. Two hundred and twenty five patients were enrolled, with a mean age of 75.5 years and 75% being female. Patients were followed for up to eight years. Patient survival curves began to separate early in the course of the study. There was a survival improvement of about 30% in patients with AAI pacing compared to VVI pacing, and AAI pacing also reduced the risk of either paroxysmal or permanent atrial fibrillation. New York Heart Association (NYHA) defined heart failure status worsened in the VVI paced group who also exhibited increased uptake of heart failure treatments, perhaps suggesting a deleterious effect on ventricular function of ventricular pacing. However, all these parameters were studied in an open fashion allowing possible observer bias. Pacemaker event counters were not a part of the study database so that there was no correlation possible between cumulative time spent in ventricular or atrial pacing and parameters characterising left ventricular dysfunction.

In contrast the Canadian Trial of Physiological Pacing (CTOPP) tested the broader hypothesis that physiologic (that is, dual-chamber or atrial) pacing is superior to single-chamber (ventricular) pacing because it is associated with lower risks of atrial fibrillation, stroke, and death.[5] Thirty two

Cardiac output changes with:

- heart rate
- myocardial contractility
- synchrony of atrioventricular (AV) and interventricular contraction sequences

Canadian centres participated in a prospective randomised study, the primary end points of which were stroke or death from cardiovascular causes. Secondary end points were death from any cause, atrial fibrillation, or hospitalisation for heart failure. For enrolment, patients had to have any pacing-indicated cause of bradycardia, be older than 18 years, and not be in permanent atrial fibrillation. A total of 2568 patients (all receiving first pacemaker implants) were recruited and randomised to either ventricular-based pacing (1474 patients) or physiologic pacing (1094 patients). At follow-up (three years) there was no significant difference in stroke or cardiovascular death, but physiologic pacing slightly reduced the risk of paroxysmal or permanent atrial fibrillation. The pacemaker devices used in this study did not have algorithms for minimisation of ventricular pacing. The possible consequence of this could have been a high cumulative percentage of ventricular pacing which may have offset any benefits of atrial-based pacing. The follow-up period of three years was relatively short and may not have allowed enough time for detection of a true treatment effect, particularly if there is a delay before such an effect becomes evident. Overall this study provides little evidence that physiologic pacing (dual-chamber or atrial) offers significant benefit over ventricular pacing.

The MOST (Mode Selection Trial) study tested the hypothesis that dual-chamber pacing improves survival and quality of life when compared with single-chamber ventricular pacing in patients with sinus node disease.[6] Its primary end point was death from any cause or non-fatal stroke. The secondary end point was a composite of death from any cause, first occurrence of stroke, or first hospitalisation for heart failure. Heart failure status was assessed by the Minnesota Living with Heart Failure score, pacemaker syndrome with a need for permanent reprogramming to dual-chamber, and a health-related quality of life assessment. Enrolment criteria were age $>$ 21 years, pacemaker implantation indicated by symptomatic sinus node disease, and presence of sinus rhythm at implant. A total of 2010 patients were recruited in 91 US sites with 1014 randomised to physiologic pacing and 996 to ventricular pacing. Follow up was for a median of 33 months. During follow up there was no difference in mortality or stroke or heart failure hospitalisation. Neither was there any difference in the combined end point of stroke, heart failure or death. Physiologically paced patients had a statistically significant reduction in episodes of new onset atrial fibrillation although the extent of the change was small and its clinical relevance questionable. There was no convincing evidence of advantage from physiologic pacing with subgroup analysis. The study did show reduction in newly diagnosed paroxysmal atrial fibrillation and chronic atrial fibrillation, reduction in signs and symptoms of heart failure, and slightly improved quality of life in patients with sinus node disease receiving dual-chamber pacing compared to single-chamber ventricular pacing. Patients who received single chamber ventricular

pacing suffered significantly greater adverse effect from ''pacemaker syndrome''.[23] The study had several limitations. There was random assignment only of pacing mode and not type of pacemaker generator, so easing patient crossover and reducing the number of clinical events in the ventricular arm. Also the pacemakers implanted did not have algorithms for minimisation of ventricular pacing which again may have been high. A high cumulative percentage of ventricular pacing could have reduced the physiological advantage of atrial-based pacing. The study did not demonstrate whether atrial-based pacing prevents atrial fibrillation or whether ventricular pacing is arrhythmogenic and actually promotes atrial fibrillation.

The UK Pace study was designed to investigate the mismatch between clinical practice and published guidelines for pacemaker implantation in the UK where there is evidence that there is an ''ageist'' pattern to pacemaker implantation.[7] The study aimed to evaluate the long term clinical impact and cost-utility of dual chamber pacing, compared with single-rate and rate-adaptive single chamber ventricular pacing, in patients aged 70 years or over with high-grade atrioventricular block. Patients eligible for enrolment were older than 70 years of age and with high-grade AV block necessitating first pacemaker implant. Patients were excluded if they had established atrial fibrillation of $>$ 3 months' duration, advanced malignancy, NYHA class IV heart failure, total immobility, or advanced cognitive dysfunction. A total of 2021 patients were recruited and randomised to VVI (25%), VVIR (25%), and DDD (50%). The study was powered at 90% to detect a 25% mortality reduction, assuming a mortality rate at 8% per annum. The primary end point was all-cause mortality and secondary outcomes were a series of specified ccardiovascular events (atrial fibrillation (lasting $>$ 15 mins), heart failure, stroke/transient ischaemic attack (TIA)/thromboembolic event, revision of pacing system, new onset angina or myocardial infarction). Four hundred and ninety five (99.0%) of 504 patients randomised to VVI mode received that mode, and 484 (96.6%) remained in it at final follow-up, while 496 (99.2%) of 505 patients received their randomised VVIR mode, and 483 (96.4%) were in that mode at final follow-up. For DDI pacing, 949 (94.9%) of 1012 patients received that randomised mode and 880(87.7%) patients were DDI paced at final follow-up. Mortality rates were analysed at five years. Comparing VVI and VVIR modes, grouped together and individually, with DDI pacing modes showed no significant differences in all cause mortality. At three years heart failure, myocardial infarction, new onset angina, and pacing system revision were similar in all groups. Likewise at three years follow up there were no differences in atrial fibrillation occurrence. Although there was no difference in cerebrovascular or other thromboembolic events at three years comparing DDI and VVIR pacing modes, there was a significant reduction in these events when VVI and DDI paced groups were compared (hazard ratio 1.58; $p < 0.035$). However, the clear message is that this study failed to demonstrate any significant influence of pacing mode on all-cause mortality in the first five years after pacemaker implantation in elderly patients with high-grade atrioventricular block. While fixed-rate single chamber ventricular pacing was associated with an increased risk of stroke, TIA or thromboembolism when compared with dual chamber pacing, pacing mode did not otherwise significantly affect cardiovascular events in the first three years after pacemaker implantation. Longer follow-up is being undertaken and is required to exclude the possibility of a delayed effect of pacing mode on atrial fibrillation, heart failure and other outcomes.

Studies of benefits of dual chamber pacing using right ventricular pacing suggest:

- little or no impact on mortality reduction
- a small impact on heart failure related hospitalisation
- at best a small reduction in burden of atrial fibrillation

THE DANGERS OF VENTRICULAR PACING AS A ''SIDE EFFECT'' OF DUAL CHAMBER PACING

An implantable cardioverter-defibrillator (ICD) study, the DAVID (Dual Chamber and VVI Implantable Defibrillator) trial, tested the hypothesis that aggressive management of LV dysfunction with optimised drug treatment and with dual chamber pacing could improve the combined end point of total mortality and hospitalisation for heart failure, compared to similarly optimised drug treatment supported by ventricular backup pacing alone.[8] The study was designed as a single blinded, multicentre, parallel group, randomised trial which compared DDDR (70 beats/min lower rate) with VVI (40 beats/min lower rate) pacing. The primary end point was freedom from death and heart failure hospitalisation. Patients enrolled had an ICD but no pacemaker indication, an ejection fraction less than 40%, and no persistent, frequent or uncontrolled atrial fibrillation. Five hundred and six patients were randomised to VVI (back up pacing rate of 40 beats/min) and 256 patients to DDR pacing at 70 beats/min. All patients received optimised medical treatment for heart failure. Although this is a study of pacing modality in an ICD population (which by definition must be at high risk of sudden cardiac death), in DDDR paced patients there was an increased risk of hospitalisation or death from heart failure (a primary end point, hazard ratio 1.61; $p < 0.03$) compared with back up VVI pacing. This study's results are actually consistent with the pacing literature. It has been shown that AAI pacing mode is associated with slightly better survival and lower rate of severe heart failure compared to VVI pacing mode in patients with sick sinus syndrome.[24] If ventricular pacing occurs more than 40% of the time it is associated with increased number of heart failure hospitalisations.[25] However, the benefit of DDDR pacing is most evident in patients who needed continuous pacing.[5]

Considering these results it becomes clear that bradycardia pacing options in patients who have received dual-chamber ICDs should be optimised on an individual basis. Right ventricular pacing may be harmful in patients with left ventricular dysfunction who have no bradycardia indication for pacing. In this population, programming of dual chamber devices to backup ventricular pacing is justified. However, in considering this evidence it is wise to remember that the specific programming choices made by investigators (choice of DDDR pacing rate, choice of AV interval) could have affected the trial results as could pacemaker choice—DDDR devices did not have algorithms to promote intrinsic conduction and therefore reduce ventricular pacing. Furthermore, these studies' results may not be extrapolated to patients with a normal ejection fraction or with standard pacing indications.

Dangers of right ventricular pacing

- Right ventricular pacing is associated with increased morbidity and mortality in patients with impaired ventricular function
- Pacer programming should be optimised to minimise unnecessary right ventricular pacing
- Most patients with sinus node disease have AV conduction sufficient for the avoidance of right ventricular pacing

CONCLUSION

Major clinical trials have been unable to demonstrate a clear benefit of DDDR over VVIR pacing for the clinical end points of total mortality, cardiovascular mortality and stroke. However, it seems that right ventricular pacing when the ventricular mass is capable of activation via the specialised conduction system "robs Peter to pay Paul". Although AV synchrony is better than no AV synchrony, right ventricular apical pacing is worse than normal ventricular activation via the conduction system. The benefit of the former may be reduced by the impact of the latter at least in the longer term. Thus it is possible that the higher level of ventricular pacing associated with conventional DDDR pacing systems has adverse long-term effects on ventricular performance that mitigate the benefit of AV synchrony. There is evidence in the literature to support that supposition. Most patients with sinus node dysfunction have reliable AV conduction and normal ventricular activation. These patients would benefit from pacing systems that promote intrinsic AV conduction. The optimal pacing strategy probably should minimise inappropriate ventricular pacing but a secure strategy to achieve that has yet to be identified. These observations confound strategic interpretation of the dual chamber versus single chamber pacing literature. Stroke prevention by dual chamber pacing presents a mixed picture. DDDR pacing probably does reduce the risk of developing atrial fibrillation and may reduce signs and symptoms of heart failure and hospitalisations for heart failure in some, but not all, patients.

To attempt to reduce unnecessary ventricular pacing, AAI/DDI mode selections and programming long AV delays are limited solutions. Novel pacing algorithms may optimise the level of ventricular pacing while maintaining AV synchrony and offering rate response.

Ventricular remodelling as a function of activation mechanism and a complex set of interactions which may be highly variable between patients depending on age and disease state is an area of interest.

Basics of cardiac pacing: key points

- Dual chamber pacing is not always the optimal pacing mode, but has significant advantage (in respect of quality of life, heart failure occurrence, atrial fibrillation, stroke risk and mortality) in specific clinical scenarios
- Dual chamber pacing is best employed with manoeuvres which minimise unnecessary ventricular pacing
- There is a trade off between the advantages of A-V synchrony and the disadvantages of right ventricular apical pacing
- Novel pacing algorithms are becoming available which will allow optimisation of ventricular pacing to reduce adverse effects of unnecessary ventricular pacing

Perhaps pacemaker type, pacing site and precise mode prescription will need to be very carefully adjusted to individual patient requirements. The concept of "physiological pacing" must evolve to include optimisation of ventricular systolic and diastolic electrical function, not just AV synchronous contraction.

In compliance with EBAC/EACCME guidelines, all authors participating in Education in *Heart* have disclosed potential conflicts of interest that might cause a bias in the article

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