

REVIEW ARTICLE

Temporary epicardial pacing after cardiac surgery: a practical review

Part 2: Selection of epicardial pacing modes and troubleshooting

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Summary

The first part of this two-part review discussed the indications for various types of epicardial pacing systems and an overview of the routine care of a pacemaker-dependent patient. Dual chamber temporary pulse generators now feature many of the refinements developed initially for use in permanent pacemakers. Few of these are utilised in the immediate postoperative period, often solely due to lack of familiarity with all but basic functions. The second part of the review deals with the selection of pacing modes. Troubleshooting real and apparent pacemaker malfunctions, including manual adjustment of parameters such as the AV interval, post atrial refractory period and upper rate limit, to avoid over- and undersensing, cross-talk and pacemaker-mediated tachycardia will also be addressed.

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Epicardial pacing has evolved considerably from simple single chamber systems with very few adjustable parameters, to complex dual chamber systems that incorporate most of the functions of permanent pacemakers. Dual chamber pacing in particular produces many technical challenges that are not immediately apparent. Fortunately, knowledge of the algorithms used by manufacturers to overcome these challenges is seldom required in the context of temporary epicardial pacing. It is tempting to ignore the complexity of modern temporary pulse generator design and fall back on suboptimal modes of pacing should any complication arise. This is an unfortunate approach, as there is often substantial benefit to be gained from adjusting the pulse generator settings correctly. How often temporary epicardial pacemakers require troubleshooting is not known [1]. The only published figure is a 0.4% incidence in 1675 patients over 18 months [2]. Most pacemaker texts deal with permanent pacemakers and only briefly cover much of the knowledge required to operate a temporary epicardial pacing system.

Pacing settings and specific indications: antibradycardia modes

Permanent pacemakers are classified using the North American Society of Pacing and Electrophysiology (now the Heart Rhythm Society)/British Pacing and Electrophysiology Group Generic Code (the NBG code), which (as revised in 2002) [3] consists of five 'positions' (Table 1). Aspects of permanent pacing relevant to anaesthesia were recently reviewed [4]. Only the first three NBG positions are relevant to temporary epicardial pacemakers. A description of pacing modes that ends with a description of the NBG code is clearly incomplete, as only a few of the theoretical possibilities make logical sense or are of any clinical use. The pacing modes applicable to temporary epicardial pacing are described below.

Single chamber pacing modes

If wires leading to only a single chamber (atrium or ventricle) are in place, then the pacemaker can be used in

Table 1 NBG code. Only the first three positions are relevant to temporary epicardial pacemakers.

I	II	III	IV	V
Chamber paced	Chamber sensed	Response to sensing	Rate modulation	Multi site pacing
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	T = triggered	R = rate modulation	A = atrium
V = ventricle	V = ventricle	I = inhibited		V = ventricle
D = dual (A + V)	D = dual (A + V)	D = dual (T + I)		D = dual (A + V)

one of only five modes: AOO, VOO, AAI, VVI, or VVT.

AOO (atrial asynchronous)

Pacing spikes are delivered to the atrium at a set rate, regardless of electrical activity in either chamber of the heart. Stimulation of ventricular contraction in this mode relies on intact conduction through the AV node. There is usually a mechanical advantage in preserving the physiological relationship of atrial to ventricular contraction. There is a risk in asynchronous atrial pacing that a pacing spike might be delivered in the repolarisation phase of an endogenous beat, which may precipitate atrial fibrillation. The refractory period of the AV node should prevent the depolarisation from being conducted to the ventricle, which should prevent VF. Because of this risk of atrial fibrillation, use of AOO is usually restricted to stable bradycardia, where the pacemaker rate reliably exceeds the endogenous rate. If this is the case, the pacemaker spike should always occur before any endogenous impulse would have been generated.

Indications

- Bradycardia with intact AV node conduction, in situations where synchronous modes are contra-indicated. This rarely means anything other than during use of electrocautery, which can interfere with sensing.

Limitations

- Contra-indicated in atrial tachycardia, atrial fibrillation/flutter (due to inability to capture the atrium), and AV node block.

VOO (ventricular asynchronous)

Analogous to AOO, pacing spikes are delivered to the ventricle, regardless of the endogenous electrical activity of the heart. As in the atrium, there is a risk that a ventricular pacing spike might be delivered while the ventricle is in the repolarisation phase of an endogenous beat. This is the classic ‘R-on-T’ phenomenon, known to precipitate ventricular fibrillation (VF).

Indications

- Bradycardia without reliable AV node conduction, in situations where synchronous modes are contra-indicated (e.g. with electrocautery).
- In an emergency, to preserve cardiac output in the case of malfunction of pacing in one of the more sophisticated pacemaker modes, while the cause of the malfunction is rectified. For this reason, some pulse generators have this as a ‘rapid access’ function.

Limitations

- Competition with intrinsic rhythm; possibility of R-on-T VF.

AAI (atrial demand)

The pulse generator has a sensing ‘timing cycle’, which is determined by the rate set on the pacemaker. If no endogenous depolarisation is sensed by the end of this timing cycle, a pacing spike is delivered to the atrium. However, if an endogenous depolarisation is sensed, no spike is delivered, and the timing cycle begins again. There is one further level of complexity to appreciate. After an atrial depolarisation (either endogenous or a pacing spike), a pacemaker atrial refractory ‘blanking’ period begins, during which there is essentially no sensing at all. A depolarisation occurring during this time does not reset the timing cycle. This is required to prevent atrial after-depolarisations resetting the timing cycle.

Ventricular ectopics are potentially problematic during AAI pacing, as no ventricular depolarisations are sensed. The atrial stimulus can potentially be conducted to the ventricle whilst it is in the repolarisation phase of a ventricular ectopic endogenous beat, precipitating R-on-T VF. Fortunately, this is usually prevented by the AV node, which has entered its refractory period following the ventricular ectopic, and so blocks transmission of the atrial impulse.

Indications

- Bradycardia, with an endogenous atrial rhythm (or frequent ectopics) sufficiently quick to compete with the pacemaker rate.

Limitations

- As for AOO, AAI is contra-indicated in atrial tachycardia, atrial fibrillation/flutter (due to inability to capture the atrium), and AV node block.

VVI (ventricular demand)

VVI is the same as AAI, except the sensing and pacing is in the ventricle. As with VOO, during a paced beat there is no co-ordinated atrial contraction, which can significantly reduce cardiac output.

Indications

- Similar to AAI, but where there is no reliable AV node conduction to the ventricle.
- Bradycardia with AV block, sick sinus syndrome, atrial fibrillation, atrial flutter.
- Overdrive suppression of ectopic beats.

Limitations

- No atrial contribution to ventricular preload.

stopped**Dual chamber pacing modes**

If wires leading to both atrium and ventricle are in place, the pacemaker can be used in one of the single chamber modes listed above, in addition to any of the following: DOO, DVI, DDI, DDD and, in some pulse generators, VDD. DDD is by far the most useful and commonly used, but understanding its timing cycles is facilitated by a preliminary discussion of DOO, DVI and DDI.

DOO (AV sequential asynchronous)

First the atrium and then the ventricle receive a pacing spike, with the spikes separated by a programmed AV delay (simulating the delay in the AV node during an endogenous beat, and improving mechanical efficiency). There is the same risk of R-on-T VF as in the other asynchronous modes (AOO and VOO). While mechanical efficiency is better than in VOO, the ventricular spike spreads throughout the ventricle in an abnormal manner compared to that of an endogenous impulse through an intact conducting system. Mechanical efficiency of the ventricular contraction is usually less. AOO is thus preferred if the conducting system is intact.

Indications

- As for VOO, but in particular in patients who derive substantial haemodynamic benefit from the contribution of atrial contraction to ventricular preload.

DVI (AV sequential, ventricular inhibited)

In the absence of any intrinsic cardiac depolarisation, the pacemaker behaves like a DOO. There is no sensing in

the atrium. When an endogenous ventricular depolarisation is sensed (following either an atrial pacing spike or endogenous atrial depolarisation), the ventricular spike is inhibited. If a ventricular depolarisation is sensed at a time before the delivered atrial spike should have arrived, it is assumed that there has been an endogenous depolarisation in the atrium that has been conducted to the ventricle. As this endogenous rhythm is likely to be mechanically more efficient than pacing, the timing cycle is reset, delaying the next atrial spike and allowing the possibility of ongoing conducted endogenous atrial depolarisations completely inhibiting atrial and ventricular output.

There is a possibility that the atrial spike will not be inhibited when in fact there is an endogenous atrial rate. This may lead to competition if the atrium is beating at a faster rate, which (as in AOO etc.) can precipitate atrial fibrillation. For this reason, DDI or DDD are preferable to DVI in patients with atrial rates high enough to compete with the pacing rate.

There is another potential problem with all ventricular sensing modes. If the ventricular sensitivity is too high, it is possible that the atrial depolarisation might be inappropriately sensed as ventricular activity and the ventricular spike inhibited. If there is no AV conduction, there will be no ventricular contraction. This is called *cross-talk* and is discussed in detail in the next section.

There is one particular use for DVI pacing. If a ventricular paced beat is conducted retrogradely up the AV node, an atrial sensing wire might interpret this as endogenous atrial activity, which in DDI and DDD modes would precipitate a release of a ventricular spike. This is pacemaker-mediated tachycardia (discussed below), which has a number of solutions. The solution requiring the least understanding of timing cycles is to switch the pacemaker to DVI, ideally while consideration is given to a better remedy.

Indications

- Seldom used. Immediate treatment of pacemaker-mediated tachycardia.

Limitations

- Contra-indicated in atrial tachyarrhythmias.
- Risk of precipitating atrial fibrillation.

DDI (AV sequential, non-P-synchronous, with dual chamber sensing)

DDI improves on DVI by adding atrial sensing. This prevents the possibility of the atrial pacing spike competing with an endogenous atrial rhythm. The maximum rate of delivery of pacing spikes is the same as the minimum rate set on the pulse generator. (NB: the 'minimum rate' is commonly just referred to as the 'rate')

of the pacemaker, but this must be distinguished from the upper rate limit, discussed later). This is the difference between DDI and DDD (below). The maximal rate in DDD is not the set lower rate limit; instead the ventricular pacing spikes can be delivered at a higher rate so as to 'track' atrial activity. DDI is thus better than DDD in the context of rapid atrial arrhythmias, as in DDD the ventricle will potentially be paced too rapidly. Indeed, most permanent pacemakers set to DDD will automatically change to DDI if a too-rapid atrial rate is detected. Temporary pulse generators will not do this (but do have an automatic defence against tracking of a too-rapid atrial rhythm (see below)). Postoperatively, patients so commonly develop atrial tachyarrhythmias (many of whom will have been paced in DDD) that at least one pulse generator (the Medtronic 5388: Medtronic, Minneapolis, MN) identifies DDI with a unique indicator to facilitate rapid changeover to this mode.

Indications

- As for DDD pacing, but in patients with paroxysmal atrial tachyarrhythmias.

Limitations

- Compared to DDD, with no atrial tracking there may be no increase in pacemaker rate in the context of physiologically appropriate sinus tachycardia.

DDD (AV universal)

This is the most commonly used mode in patients with both atrial and ventricular wires. The pacemaker waits for an endogenous atrial depolarisation. If none is sensed, an atrial spike is delivered. The pacemaker then waits for an endogenous ventricular depolarisation, in response to either the atrial pacing spike or endogenous atrial depolarisation, should this have occurred. If there is no endogenous ventricular depolarisation, a ventricular pacing spike is delivered.

As explained above, there is a risk of ventricular tracking of atrial tachyarrhythmias in DDD. Most temporary pulse generators address this risk by allowing the setting of a 'maximum tracking rate' or 'upper rate limit', as explained in detail below.

Indications

- All indications for pacing, with the exception of atrial tachyarrhythmias.

VDD (P wave synchronous)

This mode is unusual amongst the dual chamber modes in that only the ventricle is paced. The pulse generator inhibits its ventricular spike in response to a sensed ventricular depolarisation. A sensed atrial depolarisation, however,

triggers a ventricular spike if an endogenous ventricular depolarisation is not sensed. If there is no endogenous atrial depolarisation, a ventricular pacing spike is delivered.

Indications

- The specific indication for VDD is AV node block with an intact sinus node. The pacemaker acts as a conduit around the AV node. There is the same risk of tracking an atrial tachy-arrhythmia as in DDD, and the same safeguards must be in place to prevent this.

A summary of the various antibradycardia pacing modes and their indications is presented in a decision-tree approach in Figure 1.

Triggered modes

Triggered modes (VAT, AAT, DAT) are more commonly employed in permanent pacemakers in special circumstances, but are available in some temporary external pulse generators (e.g. the Ocor PACE 203H, Oscar Inc, Palm Harbor, FL, USA). Triggered modes prevent inappropriate inhibition from oversensing (such as with electrocautery) [5], but in practice asynchronous modes are more commonly used for this indication.

Whenever contemplating overdrive pacing, it is essential to confirm that the atrial output of the pulse generator is indeed connected to the atrial wires, as such rapid pacing of the ventricle is likely to precipitate ventricular tachycardia. Even if the wires are correctly attached, the current may be high enough to stimulate the ventricle directly: this must be checked by setting the pacemaker 10–15 beats above the ventricular rate and ensuring the ventricle does not track [7].

Pacing settings and specific indications: antitachycardia modes

Tachyarrhythmias are common following cardiac surgery. The presence of epicardial pacing wires allows many of these to be effectively treated by means other than pharmacotherapy or DC cardioversion. The exceptions are ventricular and atrial fibrillation and sinus tachycardia, which cannot be controlled by pacing. When attempting overdrive pacing, ventricular tachycardia or fibrillation may result and so DC cardioversion must be immediately available.

AV junctional tachycardia

AV junctional tachycardia (with rates around 100–120 beats.min⁻¹) is common following cardiac surgery [5], and is effectively managed using atrial (AOO or AAI) or AV sequential overdrive pacing (DOO or DDD). The pacing rate is increased to around 120% of the endo-

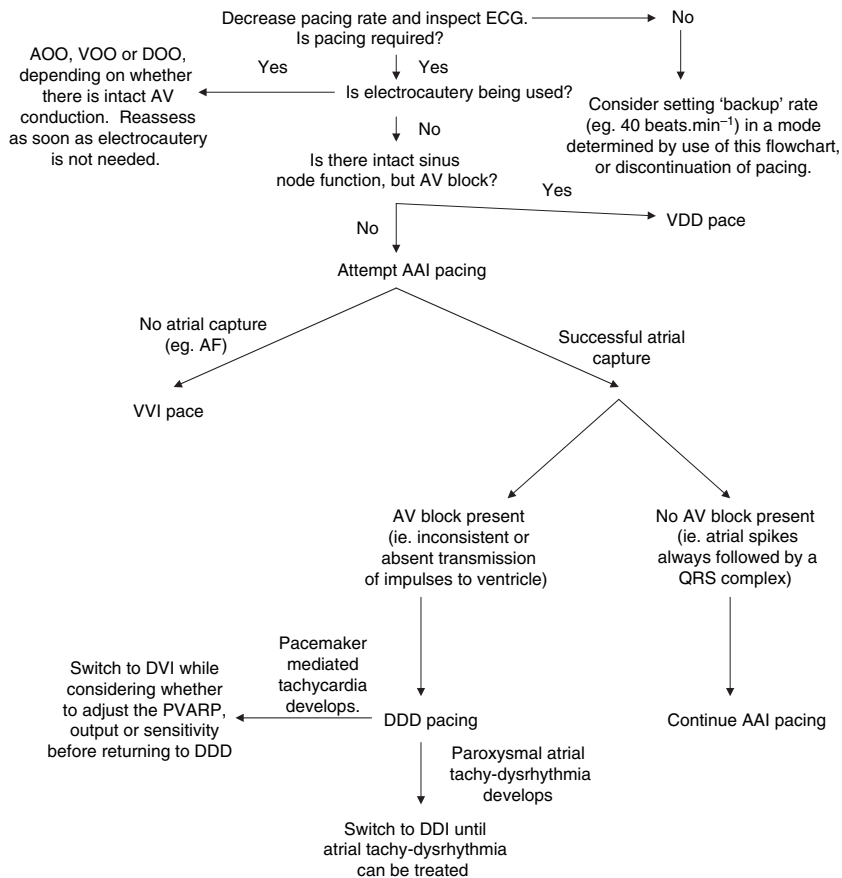


Figure 1 Decision tree approach to setting the dual chamber epicardial pacing mode.

genous rate. Once 1 : 1 capture of the myocardium is achieved, the pacemaker rate is gradually reduced. As the pacemaker rate falls below the endogenous sinus rate, a stable sinus rhythm is often established.

Paroxysmal re-entrant SVT

Paroxysmal re-entrant supraventricular tachycardia can also be terminated by atrial pacing: either 'underdrive' pacing (at less than the SVT rate) if the pacing spike induces a refractory period in the segment of the myocardium forming the re-entrant loop; or 'overdrive', where the atrial pacemaker is set above the SVT rate in a manner similar to that described for AV junctional tachycardia. The myocardium in the re-entrant limb is depolarised by an anterograde pacing spike before re-entrant depolarisation of the preceding beat arrives; when it does arrive, the myocardium is in its refractory state, so the re-entrant pathway is effectively blocked. After capture, the rate can be gradually reduced to the desired target [6].

Atrial flutter

Overdrive pacing is effective in type I atrial flutter (< 320–340 beats.min⁻¹), but not in type II with rates in

excess of this [5]. The pacemaker is set to just above the flutter rate and then gradually increased until the atrial complexes on the surface ECG change morphology. Typically, this will be 10–20 beats.min⁻¹ faster than the flutter rate [6] and indicates the flutter has been terminated. The pacemaker is then slowed to an acceptable rate, or below the endogenous sinus rate. Failure of this technique is usually attributable to insufficiently rapid pacing rates, insufficient duration of atrial pacing, or insufficient stimulus strength.

Supraventricular tachycardias with rapid ventricular response: failure to revert to sinus rhythm

Occasionally after attaining 1 : 1 capture with overdrive pacing, sinus rhythm is not re-established on turning down the rate and the SVT with rapid ventricular response persists. In this situation it may be preferable to induce atrial fibrillation by rapid atrial pacing (up to 800 beats.min⁻¹, depending on the pulse generator). Termination of rapid atrial pacing will sometimes lead to sinus rhythm where other techniques have not [6]. If not, remaining in electrically induced rapid atrial fibrillation

may be preferable if the AV block is sufficiently high to make the ventricular rate slower than that when in SVT.

Ventricular tachycardia

There is some suggestion that either underdrive or overdrive ventricular pacing can terminate ventricular tachycardia [6], but there is a risk of precipitating VF by doing this. DC cardioversion remains the accepted standard of care for VT.

Common pacing system faults

Failure to pace

'Failure to pace' occurs when there is no electrical output at the pacing wire tips when the set pacing mode calls for such an output. This is distinguished from 'failure to capture' (below) by the absence of pacing spikes in the surface ECG, and a heart rate less than that set on the pacemaker as the minimum rate. Failure to pace can be due to:

- lead malfunction or an unstable connection between the lead and the pulse generator;
- insufficient power in the pulse generator (which should be apparent from the battery indicator).
- Cross-talk inhibition (see below).
- Oversensing (as distinct from cross-talk). Any electrical potential across the sensing wires can be misinterpreted as endogenous depolarisation, with resulting inhibition of the pacing spike. Such potentials can be caused by electromagnetic interference (from electrocautery, or even mobile telephones [8]), skeletal muscle activity (including fasciculations caused by suxamethonium), or intermittent contact between the pacing wires, which can generate small 'make and break' potentials [9].
- (Apparent failure to pace) Detection of endogenous extrasystoles, which are of insufficient amplitude to register on the surface electrode, but which inhibit pacemaker output.

An appropriate first response to failure to pace is to switch to an asynchronous mode. The first two cases above can be distinguished by noting that no pacemaker spikes are delivered after making this change.

Failure to capture

'Failure to capture' is when there is electrical output at the pacemaker wire tips (confirmed by visible pacing spikes on the ECG), but this does not cause a cardiac contraction, as shown by the absence of a mechanical cardiac impulse on the arterial pressure or pulse oximeter waveform. The cause is an increase in the resistance at the wire/myocardium interface, most commonly due to fibrosis around the pacemaker lead. Additional factors contributing to increased threshold may be:

- myocardial ischaemia;
- electrolyte imbalance, particularly hyperkalaemia, acidosis and alkalosis;
- following defibrillation;
- medications, including flecainide, moricizine, propafenone, sotalol, and possibly beta blockers, lidocaine, procainamide, quinidine and verapamil [9].

Failure to capture (or a progressively increasing pacing energy requirement) is the commonest problem encountered with temporary epicardial pacing. Correction of any of the exacerbating causes listed above should be attempted. Reversing the polarity of both bipolar and unipolar lead systems may help. In a bipolar lead system, the distal (negative) electrode usually develops fibrosis first. If this occurs, the proximal electrode may remain adequate to use as a unipolar electrode (now connected to the negative terminal), with a return electrode inserted into the subcutaneous tissues. Clearly these techniques are at best a temporary solution to impending irrecoverable failure to capture. If the threshold is progressively increasing and the patient is dependent on the pacemaker, it is wise to place an alternative means of stimulus delivery (such as a temporary transvenous wire) before capture is entirely lost.

Failure to sense

In essence the same mechanisms of failure to capture can cause failure to sense [9]. True failure to sense must be distinguished from normal pacemaker function with inappropriate settings, such as over-long refractory periods.

Uncommon pacemaker faults

Cross-talk

In a dual chamber system with atrial and ventricular pacing and ventricular sensing (DVI, DDD, DDI), it is possible that the atrial pacemaker spike will be sensed by the ventricular wire, misinterpreted as a ventricular depolarisation and thereby inhibit the ventricular pacemaker output. In the absence of AV conduction, this will lead to ventricular standstill. In atrial sensing systems, the ventricular spike can be similarly misinterpreted, leading to inhibition of the atrial spike; however, ventricular pacing persists, making this less serious. Of more concern is the system that allows such atrial sensing 'cross-talk' to trigger a ventricular pacemaker spike. This will cause a form of pacemaker-mediated tachycardia, which is discussed in detail in the next section.

The simplest approaches to eliminating cross-talk are to:

- reduce the sensitivity (increase the lowest power that is sensed) in the atrial or ventricular channel; or
- reduce the power delivered to the ventricular or atrial pacing wire.

However, given the possibility of ventricular standstill, manufacturers have incorporated a number of features to guard against inappropriate inhibition of output in the ventricular wires.

A modification of dual chamber pacing – ‘committed pacing’ – whereby an atrial spike is always followed by a ventricular spike, regardless of endogenous ventricular activity. This mode was developed for use in unipolar systems, where the large atrial spike is commonly misinterpreted as endogenous ventricular activity. However, as unipolar dual chamber systems are now rarely used, most temporary and permanent pulse generators do not function in this manner.

A ventricular blanking period immediately after atrial depolarisation in which ventricular depolarisation is ignored by the pacemaker. Only after this period passes does the pacemaker become alert for a ventricular depolarisation. If one occurs, it is assumed to be a conducted beat; if not, a ventricular spike is delivered. A problem might occur if a premature ventricular complex occurred within the ventricular blanking period. The pacemaker spike (having not been inhibited) could theoretically fall within the T wave of the premature beat, causing R-on-T fibrillation. In practice, the spike is almost always delivered sooner than the repolarisation phase of a premature complex that has fallen within the blanking period.

Ventricular safety pacing. If the ventricular lead senses depolarisation after the blanking period, but before the period in which it looks for ventricular depolarisation normally transmitted through the AV node (i.e. the ‘cross-talk sensing window’) the pacemaker assumes that there is either cross-talk or a PVC. Not knowing which, it emits a ventricular spike a little earlier than the usual AV delay. If the problem is cross-talk, this leads to mild AV dyssynchrony rather than ventricular standstill; if the problem was a PVC, the spike is emitted sufficiently early to arrive before the repolarisation phase of the PVC.

Most temporary pacemakers employ a fixed ventricular blanking period and default to set parameters for ventricular safety pacing. As such, cross-talk is rarely if ever problematic as long as reasonable sensitivity and output is set.

Pacemaker-mediated tachycardia

This is a potential problem in only VDD or DDD pacing. The simplest form is far-field atrial sensing of a ventricular pacing spike, which is interpreted as an endogenous atrial depolarisation, leading to another ventricular impulse. This is overcome by use of an atrial blanking period, during which the atrial channel will not sense any depolarisation. Temporary pace generators have a preset atrial blanking period that should be sufficient to guard against this.

A more difficult problem exists when there is retrograde conduction between the ventricle and atrium, through either the AV node or an accessory pathway. More than 50% of patients receiving permanent dual chamber pacemakers are susceptible to such conduction [9] and there is no reason to suspect patients with temporary epicardial wires will be any less prone. The conduction may be intermittent and so may not be appreciated when the pulse generator is first set. In addition, it is often only a premature ventricular contraction that is initially conducted back up into the atrium. This may be sensed in the atrial wire as an endogenous atrial depolarisation, which (after the AV delay) triggers another ventricular depolarisation. This ‘endless loop’ continues with a periodicity that is the sum of the programmed AV delay and the time taken for retrograde conduction.

This problem is overcome by having an adjustable post ventricular (pacing spike) atrial refractory period, the PVARP. The atrial sensing channel must be refractory when the retrograde depolarisation arrives. The PVARP is set to a default value in all pacing generators. However, as the speed of conduction in the retrograde pathway is variable between individuals, *it is not uncommon to need to adjust the PVARP.* The disadvantage of setting a very long PVARP is that it limits the maximum rate of atrial tracking. This limitation would rarely be clinically significant in the ICU patient, and is more a concern for physiological rate response in permanent pacemakers.

As briefly touched on in the previous section, there is a simpler, but in some circumstances less ideal, solution to pacemaker-mediated tachycardia. The re-entrant pathway will be terminated if the mode is switched to VVI or DVI, but this may incur the penalty of losing AV synchrony.

Other causes of inappropriate tachycardia

Tracking of atrial tachyarrhythmias

In dual chamber modes with atrial tracking (DDD or VDD) the pacemaker emits a ventricular spike for every atrial impulse detected. In the absence of a protection mechanism, a tracked rapid atrial arrhythmia would rapidly lead to VF. All pacemakers must incorporate a means of protection against this possibility. Permanent pacemakers may be programmed to switch automatically to DDI mode when a set upper atrial rate is exceeded. DDI can be manually set on the temporary pulse generator, but as there will be an inevitable delay before this can be done, temporary pulse generators must incorporate an upper rate limit as well. This is described in detail below.

Oversensing (as distinct from cross-talk)

Oversensing can cause failure to pace, as already described. In DDD, external electrical impulses can also

be misinterpreted as atrial activity, causing pacemaker-mediated tachycardia. If the electrical interference is likely to continue, it may be necessary to reduce the sensitivity of the pacemaker (i.e. increase the sensitivity threshold) or switch to an asynchronous mode.

Portions of pacemaker timing cycles that can be manually adjusted

All modern temporary pace generators will default to timing settings that are adequate in the vast majority of patients. Most of these will be automatically adjusted when the rate is altered. For this reason, it is possible to remain ignorant of much of the following and still adequately manage a pacemaker-dependent patient. However, there will be circumstances in which manual adjustment of one of the following will be the only alternative to pacing in a haemodynamically suboptimal mode. Some of the concepts below have been touched on in the above sections.

AV delay

This is the interval following an atrial depolarisation before a ventricular spike is delivered. It allows the atrial contraction sufficient time to empty blood into the ventricle before ventricular contraction. As such, it allows the pacemaker to perform the function of the AV node. The patient's AV node may be able to conduct an impulse. If the set pacemaker AV interval is longer than the time to conduct through the patient's AV node, the ventricular pacing spike is inhibited; if it is shorter, the spike will be delivered, regardless of whether the atrial impulse would have eventually made it through the node.

Many permanent pacemakers, but no temporary pulse generators, use a 'differential' AV interval for atrial paced and endogenous depolarisations. The spread of depolarisation is more rapid following an endogenous depolarisation, and so the AV interval that follows an endogenous depolarisation is shorter than that following an atrial pacing spike to achieve the same mechanical delay between atrial and ventricular contraction.

The AV delay is shortened in ventricular safety pacing as described above. Cross-talk is detected by noticing that the AV delay is consistently shorter than that which is set on the pacemaker. The sensitivity and output of the pacemaker should be adjusted, as ventricular safety pacing usually produces a suboptimal cardiac output.

Many permanent pacemakers feature 'AV interval hysteresis'. Whenever set to a mode such that both atrial and ventricular spikes are being delivered, the system periodically extends the AV delay to look for a conducted R wave; if one is detected, the AV delay is automatically

reset to a longer interval to allow endogenous conduction. However, if an R wave is missed, the system reverts to the original AV interval, allowing pacing to continue. As this automatic feature is not available on temporary pulse generators, it may be worth occasionally manually checking for AV node function in this fashion, especially if the patient remains pacemaker dependent and a permanent pacemaker is being considered.

There will be an optimal AV delay for each patient. Whereas the default value is usually sufficient, in the borderline patient, titration of the AV delay to a near-instantaneous measure of cardiac output (such as the time-velocity integral at the aortic valve (by echo), the value provided by pulse contour analysis, or the mixed venous oxygen saturation) may be warranted. In a series of only 13 patients undergoing cardiac surgery, the optimal AV delay varied between 0.100 and 0.225 s [10].

Post ventricular atrial refractory period

This has been discussed in detail above (under 'pacemaker-mediated tachycardia'). It sets how early after a ventricular impulse (paced or sensed) that a sensed atrial depolarisation can trigger a ventricular spike, and is the timing parameter that most often needs adjustment.

VA interval (atrial escape interval)

This is the interval from a ventricular sensed or paced event to an atrial paced event. The lower rate limit (i.e. the set pacemaker rate) is the sum of the AV interval plus the VA interval. In DDD and VDD, an endogenous atrial depolarisation after the PVARP, but before the end of the atrial escape interval, will be sensed, inhibiting atrial output and starting the AV interval timer. The VA interval is determined by the AV interval and the lower rate limit settings and (as it is not independently adjustable) is mentioned here only to facilitate understanding.

Duration of pulse

The ability of the pacemaker to bring the myocardium to action potential is determined by both current intensity and duration of pacing stimulus. The relationship between these two is expressed as a strength-duration curve (Fig. 2). In many temporary pulse generators, the pulse duration is fixed at around 1.5 ms, which is on the flat part of the curve. Further increases in pulse duration would not reduce the current required for depolarisation. Reducing the pulse duration may reduce fibrosis (and delay failure to capture), and in permanent pacemakers is an even more important consideration as this will also conserve battery life. In practice, the pulse duration in temporary pacemakers is rarely altered, even if this option is present.

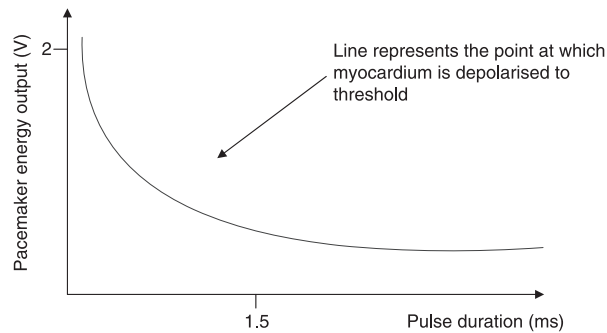


Figure 2 Pacemaker strength-duration curve [11]. The default pulse duration in temporary pacemakers is typically around 1.5 ms, beyond which further increases do not reduce the required pacemaker energy output.

Upper rate limit (or ‘maximum tracking rate’)

The upper rate limit is the fastest the pacemaker will pace the ventricle in response to a sensed atrial event. This is the one ‘automatic’ defence against ventricular overpacing in the event of a rapid atrial tachycardia in temporary pacing systems.

When the upper rate limit is reached, one solution might be to ignore every second atrial depolarisation: in essence a 1 : 2 block. However, this would have the effect of abruptly reducing the ventricular rate from the upper rate limit to a value half, one third or one quarter of this. This might not be problematic in the case of atrial tachyarrhythmias, but in the patient with sinus tachycardia that gradually approaches the upper rate limit, a sudden halving of the ventricular rate would at least transiently cause a substantial fall in cardiac output. To avoid this, rather than introduce a 1 : 2 block, an electronic ‘type II Wenkebach block’ is introduced. This involves gradually lengthening the AV interval until it is so long that the next atrial depolarisation falls within the PVARP of the preceding ventricular spike. It is thus ignored and the rate of ventricular tracking reduced.

Most pace generators will set the upper rate limit automatically, determined by the programmed AV interval and PVARP ($AVI + PVARP = \text{total atrial refractory period, TARP}$). When the endogenous beats are separated by less than the TARP, every second beat will simply not be sensed by the pacemaker. To be effective at preventing abrupt 2 : 1 block, the upper rate limit (which instead introduces Wenkebach) must be set less than this rate.

The appearance of Wenkebach in a paced patient should alert the clinician that the upper rate limit is being employed to prevent rapid atrial tracking. Ideally, the atrial tachycardia should be treated or the pacing mode changed (perhaps to DDI), rather than allow this to continue.

Blanking periods

The atrial or ventricular blanking period begins immediately after a pacing spike is delivered in the *other* cardiac chamber. During this period there is no sensing and so no timing intervals are reset. This is designed to prevent cross-talk and is usually not adjusted.

Conclusion

It is tempting to ignore much of the complexity available in modern pacing generators, and rely on haemodynamically suboptimal pacing modes when commonly used settings fail. As patients with increasingly borderline cardiovascular function undergo surgery, the need to fully optimise pacing parameters will increase. This two-part review contains all the information required to competently manage single and dual chamber pacing systems and alludes to emerging pacing techniques (such as bi-atrial and biventricular pacing) that are likely to become increasingly employed in cardiac anaesthesia and intensive care.

Acknowledgement

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References

- 1 Wasiak J. *What Is the Incidence of Temporary Epicardial Pacemakers Requiring Troubleshooting?* Clayton, Victoria, Australia: Center for Clinical Effectiveness, Monash University, 2000.
- 2 Del Nido P, Goldman BS. Temporary epicardial pacing after open heart surgery: complications and prevention. *Journal of Cardiac Surgery* 1989; **4**: 99–103.
- 3 Bernstein AD, Daubert JC, Fletcher RD, et al. The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. *Pacing and Clinical Electrophysiology* 2002; **25**: 260–4.
- 4 Allen M. Pacemakers and implantable cardioverter defibrillators. *Anaesthesia* 2006; **61**: 883–90.
- 5 Rozner MA, Trankina M. Cardiac pacing and defibrillation. In: Kaplan JA, Reich DL, Lake CL, Konstadt SN, eds. *Kaplan’s Cardiac Anesthesia*. Philadelphia: W. B. Saunders, 2006; 827–43.
- 6 Donovan KD. Cardiac pacing in intensive care. *Anaesthesia and Intensive Care* 1985; **13**: 41–62.
- 7 Bojar RM. *Manual of Perioperative Care in Adult Cardiac Surgery*, 4th edn. Malden, MA: Blackwell Publishing, 2004.

- 8 Trigano AJ, Azoulay A, Rochdi M, Campillo A. Electromagnetic interference of external pacemakers by walkie-talkies and digital cellular phones: experimental study. *Pacing and Clinical Electrophysiology* 1999; **22**: 588–93.
- 9 Atlee JL, Bernstein AD. Cardiac rhythm management devices (Part II): perioperative management. *Anesthesiology* 2001; **95**: 1492–506.
- 10 Durbin CG Jr, Kopel RF. Optimal atrioventricular (AV) pacing interval during temporary AV sequential pacing after cardiac surgery. *Journal of Cardiothoracic and Vascular Anesthesia* 1993; **7**: 316–20.
- 11 Coates S, Thwaites B. The strength-duration curve and its importance in pacing efficiency: a study of 325 pacing leads in 229 patients. *Pacing and Clinical Electrophysiology* 2000; **23**: 1273–7.