

PACEMAKERS

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I. Introduction

About 300,000 patients in the US have permanent pacemakers; they constitute a small but significant portion of the surgical population. Most of us, however, will be exposed to temporary pacing in the post-cardiopulmonary bypass period. Although the variety of temporary pacemakers is limited, there are now a tremendous number of permanent pacemakers since the advent of programmability.

II. Threshold and its Determinants

All pacemakers generate a pulse of current to depolarize a bit of myocardium, the wave then spontaneously spreads to the rest of the myocardium. The pacing threshold or threshold of excitability is determined by (a) the intrinsic excitability of the myocardium, (b) the current density at the electrode-tissue interface and (c) the duration of the electric pulse. Pulse duration is programmable in all currently manufactured permanent pacemakers. The shorter the pulse, the greater the necessary current. Beyond a two millisecond pulse duration, however, the threshold current is little altered.

Current density is determined by output voltage, the resistance to current flow and the surface area of the contact between the electrode to myocardium. With small electrodes less total current is necessary to achieve a given current density but this must be balanced against the higher voltage necessary to drive that current against the high resistance of a small electrode-myocardial surface. Over time, resistance to current flow goes up as scar tissue forms around the electrodes of a permanent pacemaker so a higher voltage is necessary months or years after permanent pacemaker insertion. It is therefore important that the pacemaker output be well above the threshold measured at the time of electrode insertion. Steroid eluting permanent electrodes exhibit very little increase in threshold during the post-implant period. In general, an energy output of 2-3 times threshold is considered an adequate safety margin. "Autocapture" algorithms in some newer pacemaker models permit a lower output setting with reduced chance of loss of capture.

The intrinsic excitability of the myocardium can vary to a small degree (~ 50% of baseline). Usually the pacemaker generates a high enough current density to be unaffected by such small changes in excitability, but in borderline cases capture could be lost or gained depending on external factors. Mild hypoxia, hypercarbia, hypernatremia, an increased ratio of intra- to extracellular potassium, propranolol, procainamide, quinidine, verapamil, amiodarone, flecainide, and the post-prandial state all tend to make electrical stimulation more difficult. Severe hypoxia, a decreased intra- to extracellular potassium ratio, exercise and catecholamines all tend to make electrical stimulation more easy.

III. Basic Pacemaker Operation

All pacemakers have an internal clock that determines when the next pulse should be delivered. All pacemakers have an output circuit that, when triggered by the timer, emit a pulse of fixed duration and voltage (or current if so designed).

Pacemakers that stimulate both atrial and ventricular tissue do so with two output circuits such that the ventricular output occurs after a programmable AV delay. The delay period is programmable in most permanent pacemakers, but can be altered in programmable pacemakers and in the external A-V sequential pacemakers found in the OR.

Inhibitible or demand pacers have circuitry that analyze the ECG (as detected by the pacer's electrodes) and also screen the signal for electromagnetic interference. If a QRS is detected, the internal clock is reset thereby delaying the time until when the next pulse is due (i.e. the escape cycle length). In the event of electromagnetic interference the demand pacemaker may revert to an asynchronous mode and pace continuously until the interference is gone and the intrinsic heart signal is once again detected. Placing a magnet over a non-programmable or a programmable pacemaker will also convert the device to asynchronous mode, but only for as long as the magnet remains. Many demand pacemakers also exhibit hysteresis in that the pulse generator is suppressed until the intrinsic heart rate falls to a rate (e.g., 60 beats/min) that is well below the pacemaking rate (e.g., 72 beats/min). Once the pacemaker begins pacing, however, it will not stop until the intrinsic heart rate climbs above the paced rate, unless the pacemaker is a model with "search hysteresis" which will periodically assess the underlying rhythm.

Some pacemakers are capable of pacing and sensing in both the ventricle and the atrium. Such capability permits the pacemaker to ensure not only an adequate ventricular rate, but to preserve the atrial kick before each ventricular contraction. These pacemakers (DDD classification - see next section) guarantee a minimum atrial rate and also ensure that a ventricular contraction occurs within a specified amount of time after each atrial contraction. The electronics are complicated because it is not always appropriate for ventricular contraction to occur after every atrial contraction. For example, an atrial tachycardia might generate an excessive ventricular rate. Similarly, electromagnetic interference might be detected as a rapid atrial rate. Limitations on ventricular rate are usually built in the circuitry and are programmable.

Another phenomenon, pacemaker mediated tachycardia, may occur in DDD pacemakers as the result of a PVC, or any depolarization of ventricular origin which is conducted in a retrograde fashion through the AV node to the atrium. A PVC may cause retrograde atrial depolarization that is in turn detected by the pacemaker. A pacer-induced ventricular depolarization therefore occurs after the pre-set A-V interval and the ventricular depolarization is conducted back to the atrium and the cycle self-perpetuates. Pacemaker mediated tachycardia can be prevented by the addition of a post-ventricular atrial refractory (blanking) period to the circuit. The blanking period begins with any ventricular depolarization and typically lasts 300-400 milliseconds and is programmable. During the PVARP no triggered ventricular response to atrial sensing is permitted; thus, a retrograde P wave or any other early P wave would not lead to another ventricular depolarization.

Most pacemakers now have a the capability of varying the pacing rate. Once upon a time, only patients with intact SA nodes and pacemakers that sense the atrium and pace the ventricle could adjust their heart rate as dictated by body demands. Now pacemakers have been devised that in some fashion sense the patient's level of activity and accordingly adjust the (demand) pacing rate (variable rate pacing). One such device uses a piezoelectric crystal in the unit to detect body motion transmitted from underlying muscles. The rate of pacing is determined by the sensed level of activity. The anesthesiologist should be aware that activity of muscle overlying the generator (e.g., fasciculation from succinylcholine) or that pressure on the generator (e.g., surgeon leaning on the generator) may be interpreted by the unit as activity and lead to an increase in heart rate, though this would be unlikely due to "filters" which screen out high (rigors, tremor, motor vehicle) and low frequency (respiration, heartbeat) vibrations. Another method of determining the presence of physical activity utilizes detection of the respiratory rate. Using electronics similar to how our anesthetic monitors sense a breath (impedance measurement), these pacemakers adjust the

minimum pacing rate according to the respiratory rate, on the logic that rapid breathing indicates exertion. This method of respiratory rate detection involves application of a small electrical current to the patient. When the respiratory rate detection system of a patient monitor is used in a patient with a variable rate pacemaker that also uses respiratory rate detection, the systems may interact. The pacemaker may interpret the patient monitor's current as rapid breathing, and consequently pace the patient at a very high rate. You should try to determine before surgery if a pacemaker provides variable rate pacing, find out what method the pacemaker uses to determine physical exertion, have a heightened awareness of the interactions with our drugs and equipment and be prepared to deal with the tachycardia (eg. quit pushing on the pacemaker, wait for the fasciculations to end, turn off the respiratory rate detection on the monitor depending on the circumstances).

IV. Pacemaker Classification

Currently, only multiprogrammable pacemakers are inserted into patients. To a large extent, the function of both non-programmable (fixed function) and programmable pacemakers can be characterized by a three letter coding system. The first letter tells which chamber is paced (A=atrial, V=ventricle, D=both). The second letter describes which chambers are sensed (A, V, D as above, plus 0=no sensing). The third letter tells the response to the sensed signal (0=no sensing, I=inhibit, i.e., the pulse is suppressed and the internal clock reset; T=trigger, i.e., a pulse is administered; D=both triggering and inhibiting functions exist). The simplest pacemaker provides asynchronous ventricular pacing (no sensing, no inhibition) and is designated VOO. No pacemaker is manufactured with this mode alone, but most can be programmed to it. A demand ventricular pacemaker would be VVI. A patient with complete heart block, but a normal SA node would be a candidate for a permanent VAT pacemaker in order to ensure a ventricular contraction after each atrial depolarization.

If both the atrium and ventricle are paced and the pacing is done asynchronously, the designation would be DOO. The physician sets the rate. In addition, an A-V delay interval must be selected. For example, an A-V delay of 170 msec would place the atrial pulse 170 msec before the ventricular pulse. When sensing is added to the pacemaker function, things get more complicated. The simplest sensing system would only sense ventricular electrical events. Detection of ventricular depolarization would be used to inhibit electrical stimulation of the heart, i.e., DVI mode. What can be confusing is what the pacemaker does after a ventricular depolarization is sensed. In the case of a DVI pacemaker, a ventricular sense causes inhibition of any pending ventricular pulse and also resets the timer. In other words, the next ventricular pulse cannot come for another full R-R interval and therefore the next atrial pulse cannot occur until the R-R interval less the A-V delay interval has passed. Unfortunately, in the heart room A-V demand pacing can be difficult to achieve because the epicardial pacing wires used by the surgeons provide a weak and often unstable signal to the pacemaker. As such, reliable sensing is often impossible.

If a DDD pacemaking configuration is used, then the pacemaker first looks for a spontaneous atrial depolarization. If an atrial depolarization is not sensed within the allotted time defined by the programmed heart rate, the pacemaker then electrically depolarizes the atrium. After either a spontaneous atrial depolarization or a pacemaker electrical spike to the atrium, the pacemaker then waits for up to a specified time (the A-V interval) to sense a ventricular depolarization. If no ventricular depolarization is sensed an electrical shock is sent to the ventricle. After a ventricular sense or a ventricular shock, the internal clock is reset and the cycle starts over.

Selection of the pacemaking configuration in programmable pacemakers is made by a hand-held control unit that is placed over the pacemaker. The control unit is set for the desired program and then a button is pushed whereupon a pulse is emitted that carries the code for the program to the

pacemaker. Sometimes the control unit contains a magnet. The magnet serves a dual purpose; it converts the pacemaker to asynchronous mode and it closes a switch in the pacemaker that makes the pacemaker receptive to programming signals. Modern pacemakers are designed with much redundancy in their signal receiving electronics in order to prevent stray electromagnetic signals from programming the pacemaker in an arbitrary fashion. Nevertheless, arbitrary re-programming can occur during the use of electrocautery as discussed later.

V. Indications for Pacing

Pacing can be instituted on a temporary or a permanent basis and the indications for each are somewhat different. Most temporary pacing requirements occur in association with acute myocardial infarction. The development of new bundle branch block plus 1° block, bifascicular block, symptomatic bradycardia in anterior MI or Mobitz II block in anterior MI are all indications for temporary pacing in acute infarction.

Permanent pacemaker insertion is warranted for complete heart block (though congenital 3° block may not need a pacemaker until young adulthood), sick sinus syndrome, symptomatic Mobitz II block or symptomatic bradycardia. Bifascicular block, if asymptomatic, is a controversial indication. Electrophysiologic measurement of H-V conduction times may be of some value in this situation. In addition to maintaining a minimal heart rate, pacemakers may be placed to suppress ventricular ectopy or to suppress atrial tachyarrhythmias via timing a pulse to interrupt an atrial re-entrant pathway.

Additional indications for placement of a pacemaker wire prior to anesthesia are not clearly defined; however, some suggestions can be made. Placement of a PA catheter in a patient with left bundle branch block may warrant temporary pacing capability for fear that additional transient right bundle branch block during catheter insertion would lead to complete heart block. Inhalational anesthetics can worsen conduction. Patients with left bundle branch block or bifascicular blocks with or without 1° block rarely progress to life-threatening block, although symptomatic bradycardia is not uncommon. Even Mobitz II block rarely progresses to complete heart block during anesthesia. Consequently, placement of a transvenous pacing catheter prophylactically is not recommended for these pre-existing arrhythmias. It would be wise, however, to have a transcutaneous pacemaker close at hand and have a predetermined plan ready to implement if severe bradycardia or 3° block develops.

VI. Pre-operative Assessment

In addition to the standard pre-anesthetic evaluation, certain information specific to the patient's pacemaker must be obtained. The type of pacemaker, why it was implanted and whether or not it is functioning correctly are probably the most important issues. The patient may provide some of the information, especially with regards to pre-pacemaker symptoms (dizziness, syncope) and whether or not those symptoms have recurred. Sometimes muscle activity (myopotentials) may suppress pacer stimulus generation. If pacer dependent, the patient may then note symptoms with certain activities. This may suggest a contraindication for succinylcholine, because the fasciculations may suppress the pacemaker. Information about the pacemaker unit may also be provided by old records or a pacemaker identification card. Manufacturer and model are usually stamped on the generator and visible via an x-ray. Books exist in which the code can be looked up and information about operation and testing can be obtained. Breaks in the wires as well as unipolar versus bipolar lead systems may be seen, too. When the leads attach to the endocardium, the generator is usually placed over the pectoral muscle; if the leads are epicardial, the generator is

in the abdominal wall. Current medications and electrolyte levels (especially K⁺) should be reviewed in order to search for effects that may impair pacemaker capture or enhance arrhythmias.

Complete evaluation of pacemaker function is not easy, but a good starting point is the electrocardiogram. The presence of pacemaker spikes followed by one-to-one capture suggests a normally functioning generator unit provided that the spikes occur as frequently as programmed. One of the earliest signs of battery failure is a slowing of the impulse rate (rarely the rate speeds up). In many patients with demand pacemakers, the intrinsic heart rate will be high enough to suppress the pacemaker and no spikes will appear on the ECG. In that case placement of the magnet over the unit will convert the unit to asynchronous mode and the spike frequency can be determined. One-to-one capture may not be observed if some of the pacer stimuli fall in the refractory period. The sensing portion of the unit is the hardest to test, because it is difficult to change the patient's heart rate to observe alternatively suppression and generation of stimuli. However, removal of the magnet should restore sensing and suppress the unit once again. If the patient is pacer dependent, magnet placement might not seem to do anything. Most pacemakers, however, pace at a different rate when the magnet is placed, even if only for a few beats. Careful observation of the EKG on placement of the magnet will often provide some evidence that the magnet has had an effect on the pacemaker. Sensing may not be testable in a pacemaker dependent patient, however, unless an extrasystole fortuitously occurs to reset the timer and alter the period before the next pacer stimulus. Ideally, every patient with a pacemaker who is anticipating surgery should have a complete pacemaker interrogation before and after the surgery. The interrogation should include a check of the battery condition. Batteries near their end-of-life are more susceptible to failure in the OR due to electrocautery interference or the increased power drain if a magnet is over the pacemaker (see next section). If specific recommendations are required, such as temporarily reprogramming of the pacemaker, these can be performed preoperatively. As not all pacemakers respond to a magnet in an identical fashion, it is **IMPERATIVE** that the effect of a magnet be known before surgery, even if this means placing the magnet over the pacemaker ahead of time to see what happens.

VII. Permanent Pacemaker Function in the Operating Room

The stress to the body imposed by anesthesia and surgery makes it imperative that permanent pacemakers function normally throughout an operation. The most common cause of intraoperative pacemaker dysfunction is electromagnetic (EM) interference from an electrocautery. The lines of EM flux emanating from the cautery wires cut across the pacemaker leads and induce a current flow in them. Bipolar cautery tips emit far less flux than unipolar units, because the current path of a bipolar unit forms a much smaller loop (less of an antenna). Similarly, unipolar pacemaker leads create a large receiving antenna, because their leads are so much further separated than are bipolar leads. Pacemaker units with unipolar leads are therefore more likely to experience electrocautery interference than are bipolar units. Unipolar cautery units are highly dependent on a good ground pad connection to the patient. Poor ground pad contact raises resistance to current flow from the cautery tip to the pad. In addition to generating more heat at the pad and the potential for a burn, the high resistance encourages the current to seek other paths back to the cautery machine. The new path will likely involve the patient, the OR table and possibly other equipment attached to the patient including pacemaker leads. Should that current travel through a lead, heart damage in the form of a burn or heart stimulation including fibrillation could occur.

Potential voltage generated on a pacemaker lead (such as generated by the radio frequency emission of electrocautery) can also affect the pacemaker itself. The effects vary with the type of pacemaker. Any pacemaker with a sensing unit may pick up electromagnetic noise from an

electrocautery. Most pacemakers are extremely resistant to that noise and will continue to sense and pace appropriately. Nevertheless, any pacemaker may pace erratically and this could lead to bradycardia or even asystole. After several seconds of a noisy signal such that the QRS cannot be reliably detected, some units will spontaneously revert to an asynchronous mode (VOO or DOO) and remain that way until the QRS is once again detected. Once the interference goes away, it takes a few seconds for such a pacemaker to resume normal demand pacing. These two time lags can lead to pacemaker shut-down in the presence of frequent, short bursts of electrocautery. If the cautery burst is short the pacemaker will not have time to revert to asynchronous mode. If the next burst comes before the pacemaker has had enough time to resume normal function, that next burst will further suppress the pacemaker. It should be apparent that the patient must be continuously monitored by a device that is not affected by electrocautery noise. Modern EKG monitors often suppress the electrocautery noise sufficiently to permit QRS detection during electrocautery use. If so, monitoring the EKG is sufficient because electrocautery does not cause electromechanical dissociation. In other words, if you can see the QRSs during electrocautery use, you can assume there is a pulse. If the EKG is too distorted to be useful during electrocautery, then you must use some other device to detect the pulse (eg. oximeter, arterial line).

In most cases, placement of a magnet (ring magnet, never use a horseshoe magnet) over the unit will convert a demand pacemaker (fixed or programmable) to an asynchronous pacemaker and therefore prevent asystole. In general, whether the asynchronous mode will be atrial pacing only, ventricular pacing only, or A-V sequential pacing depends on what mode of demand pacing was in existence at the time of conversion to asynchronous mode. For example, AAI would go to AOO, VAT or VVI would go to VOO an DVI or DDD would become DOO. Having the pacemaker in asynchronous mode will create a rhythm that will compete with the heart's intrinsic rhythm (if any), so the use of a magnet will likely be only necessary in pacemaker dependent patients who exhibit bradycardia or asystole during electrocautery use. These facts illustrate the importance of monitoring the pulse during electrocautery, and knowing ahead of time what a magnet will do to the pacemaker.

Any pacemaker can be damaged to the point of malfunction or to cease function by high density electromagnetic flux such as from electrocautery. This occurs most often if the cautery is used too close to or touches the pacemaker or wires (usually six inches or more away is safe) or if the ground pad is improperly positioned or malfunctions. In addition, current surges into programmable pacers can cause a transient decrease in battery power that causes the pacer to revert to default settings. Programmable pacemakers may also be arbitrarily re-programmed by electrocautery. If cautery will take place near the generator or leads, it is wise to have easy access to a programming box and know how to use it. As mentioned earlier, magnet placement may or may not make a programmable pacemaker more susceptible to accidental reprogramming. As a general rule, programmable pacemakers installed in 1995 or later are unlikely to be susceptible to random reprogramming because of improved electronics. In older, more susceptible pacemakers, it may be wise to convert the pacemaker to asynchronous mode (if tolerated by the patient) just prior to surgery, then convert back after surgery. Use of a magnet has one other possible adverse effect – it causes the pacemaker to start emitting its telemetry signal, which requires power and drains the battery. A weak battery may then worsen enough to affect pacemaker function.

There are other situations in which pacemakers may malfunction. Electroconvulsive therapy often generates strong myopotentials (muscle electrical activity) that may interfere with sensing just as does electrocautery. Radiation therapy can destroy some of the electronic components of pacemakers. The limit is on the order of 500 rads delivered to the pacemaker unit itself. Nuclear magnetic resonance employs very strong permanent magnets. Patients with pacemakers cannot go near these devices.

VIII. Monitoring in the OR of Patients with Pacemakers

As the preceding section makes clear, the potential for pacemaker malfunction in the operating room is very real indeed. In consequence, it is mandatory that some means of pulse detection be employed that is not affected by electrocautery. Many options are available and include pulse oximetry, doppler, arterial line and even a finger on the pulse. Modern electrocardiogram devices are more resistant to Bovie noise than ever before so it may also be possible to monitor the rhythm during cautery.

Much can be done to minimize cautery interference. The ground pad should be farther from the pacemaker than the site of cautery. The ground pad should make excellent contact with the patient. If these principles are applied to the ECG cables, interference to that monitor will also be reduced. So long as electrocautery is not performed near the pacemaker generator, damage to or re-programming of the pacemaker is unlikely, and the need for the programming box and/or a backup temporary pacemaker will be small.

Potassium balance should be maintained, hypokalemia may lead to an inability to capture and hyperkalemia can cause arrhythmias and decreased contractility.

Succinylcholine induced fasciculations and post-op shivering may interfere with sensing.

The external leads of temporary pacers are a prime site for microshock fibrillation. Current applied directly to the heart need only be 100 microamps in magnitude to cause fibrillation. Static electricity built up on one's fingers may provide sufficient current. Although not always done, external leads should never be touched without gloves and should always be covered by some insulating material when not connected to a pacemaker.

IX. Implantable Cardioverter-Defibrillators

These units have become quite complicated. For example, most units now have the ability to pace a patient as bradycardia is commonplace after a shock. No longer will the sole effect of magnet placement be to turn these units off or on.

Automatic implantable cardioverter-defibrillators (ICDs) typically sense V-tach or V-fib by determining the presence of a wide complex tachycardia. Electrocautery noise may mimic this signal and be detected as a shockable rhythm. While the unit charges, an audible alarm may sound. If so, and the electrocautery is the cause of the problem, instantly stop the electrocautery. The unit may sense normal rhythm in time to prevent delivering a shock. In general, any patient with an ICD should have its tachyarrhythmia detection ability disabled, either by re-programming it off, or by placement of a magnet over the ICD.

For almost any unit, placement of magnet will disable tachyarrhythmia detection and prevent shock delivery. There may be no way to tell, however, if the unit is actually sensing the presence of the magnet. Furthermore, in general, the magnet will not turn off the demand pacing component of the unit. This is not a problem, so long as the patient doesn't normally use the demand pacemaker function of the ICD which could be suppressed by the electrocautery. Knowing what a magnet will do to a given unit is problematic, as it varies not only among manufacturers, but within models as well. It can therefore be argued that the best plan is to have the ICD turned completely off by the programming box prior to surgery, and turned back on as soon as the patient leaves the OR. On the other hand, if a magnet is used to disable tachyarrhythmia detection, and the patient has V-fib in the OR, just taking off the magnet should quickly result in the ICD defibrillating the patient.

The use of external paddles or defib pads should not damage the ICD, provided the ICD is not directly beneath the paddle/pad. So plan ahead as to where you will put the paddles or pads on the patient.

Rooke's Rules for Pacemakers and Implantable Cardioverter-Defibrillators

1. Determine as much as you can about the function of the unit before surgery. Ask the patient, contact the manufacturer or rep or get a Cardiology Consult if necessary. Be sure to know if the pacemaker has variable rate pacing and if so, how the unit senses exercise. Ideally, know how a magnet affects the unit. For programmable pacemakers, will magnet use increase the risk of electrocautery reprogramming? (probably not). For ICDs, does it turn off the shock capability? (probably) For ICDs, does it turn off demand pacing? (probably not).
2. Ideally, all units should be tested (interrogated) before surgery (eg. to check battery status, sensing and pacing thresholds).
3. If the unit is programmable (and almost all modern pacemakers and ICDs are), determine if a box and technical support is available to program the pacemaker to asynchronous mode or turn off the shock function of an ICD. If it is decided that a programmable pacemaker should be converted to asynchronous mode, make sure it is tolerated by the patient.
4. For programmable and non-programmable pacemakers, does placement of a magnet convert the pacer to asynchronous mode? (Try it while monitoring the ECG and look for a rhythm change.)
5. Place the electrocautery ground pad such that the current flow from the surgical site to the ground pad will not cross the pacemaker/ICD or its leads. In addition, place the ground pad as close to the surgical site as feasible.
6. Use some sort of pulse monitor in addition to the ECG.
7. For pacemakers, have a plan for alternative pacing (eg. transcutaneous pacemaker). For all units, think about what could go wrong and how you will handle it.
8. If electrocautery interference becomes intolerable, consider bipolar cautery. The surgeons won't be happy (less power), but it may help prevent patient harm.
9. If a pacemaker does become re-programmed, the magnet may still put it into asynchronous mode at a reasonable rate.
10. ICD tachyarrhythmia detection must be turned off during electrocautery. At a minimum, have a magnet on the unit during electrocautery.