Optimal pacing strategies for coming off CPB

Optimal pacing strategies employed after CPB enhance ventricular function with improvements in cardiac output and tissue perfusion. Pacing is optimized for each patient by taking into consideration the underlying electrophysiological abnormalities, as well as co-existing structural cardiac diseases. The primary objective of rhythm stability and improved cardiac function in the post-CPB period is obtained by -

1. Optimizing pacemaker lead configuration and positioning as well as capture and sensing properties
2. Appropriate programming of pacemaker timing and intervals and use of correct pacing modes
   a. Adjusting pacing rate to obtain balance between cardiac output and myocardial oxygen demand
   b. Maintaining optimal atrio-ventricular (AV) synchrony and AV conduction delay
   c. Maximizing ventricular ejection through coordinated ventricular activation

Pacemaker lead configuration and placement

Epicardial wires (or leads) can be placed in a unipolar or bipolar configuration. In a unipolar system a single wire (the negative anode) is connected to the epicardium, while the positive electrode is attached at a distance in the subcutaneous tissues. The bipolar system, on the other hand, requires both the wires (cathode and anode) to be connected to the epicardium. As the current travels a much shorter distance between electrode tips, less electrical energy is required to bring the myocardium to threshold in a bipolar system as compared to a unipolar system. Pacemakers also use the same epicardial wires to sense intrinsic electrical activity; the smaller current path of bipolar electrodes makes them less susceptible to electrical interference when performing the sensing function. For these reasons, placing bipolar wires is most desirable before coming off-CPB.

Site of placement of pacing wires is important both for the obtaining appropriate sensing and pacing thresholds, and for achieving the desired mechanical activity (ejection) in the cardiac chambers. Pacing electrode (cathode) on the cephalic atrial wall between the left and right atrial appendages, where the Bachmann bundle indwells, provides good atrial pacing and sensing thresholds. Right atrial appendage is another frequently employed site for atrial pacing. For ventricular pacing, epicardial electrodes in the RV outflow tract provide superior hemodynamics when compared to RV apical electrodes. For patients with dilated LV who have severely reduced EF or conduction delays, BiV pacing can be effective. In addition to the RV lead, another ventricular lead is placed on the LV basal posterior-lateral wall, allowing both the ventricles to be stimulated simultaneously. Unfortunately, unlike permanent pacemakers, no current temporary pacemaker generator allows programming of timed activation delays between the two ventricles.

Pacemaker/Programmer timings and settings

Temporary pacing generators are typically constant-current output devices (as compared to the permanent pacemakers that are constant-voltage devices), and are designed to function against resistances of 300-1000 ohms. The stimulus pulse width is usually 1-2 msec. They typically allow adjustable pacing rates (30-180/min), sensitivity settings (0.1mV-asynchronous), and current outputs (0.1 mA-25 mA).

Commonly used temporary programmers in US are - (a) Medtronic model 5348 and 5388 (Medtronic, Minneapolis, MN, USA). (b) St Jude models 3085 and 3077, (St Jude Medical, Sylmar, CA, USA)

Rate: Pacing rate should ideally be adjusted to optimize both cardiac output and myocardial oxygen demand. In practice, the optimal heart rate is infrequently accurately titrated to cardiac output and is usually left at 80–90 beats/min. As the patient transitions to complete reliance on intrinsic rhythm, a period of 'backup' pacing (with the pacemaker set ~ 60-70 beats/min) can be employed. This approach allows the patient to remain in an intrinsic rhythm until the point of significant hemodynamic compromise, while providing the ability to continuously monitor sensing thresholds.
AV Interval: Each patient will have an optimal AV delay. Though the default value is usually sufficient in pt with normal systolic or diastolic function, in the hemodynamically compromised patients, adjustment of the AV delay to a real-time measure of cardiac output (such as Doppler time-velocity integral at the aortic valve, pulse contour analysis CO, or the mixed venous oxygen saturation) may be required. Optimal AV delay varies between 100 msec and 225 msec.

Refractory intervals and blanking periods: All demand pacing modes incorporate various blanking and refractory intervals that follow intrinsic or paced depolarizations during which there is essentially no sensing. These intervals are important in preventing inappropriate sensing of after-depolarizations, far-field potentials, and ectopic beats. These intervals can be adjusted to optimize the pacing rhythms.

Pacing or Capture thresholds: The capture threshold is the minimum pacemaker output required to stimulate a propagated action potential in the myocardium. Pacing threshold is confirmed by setting the pacemaker rate above the patient's intrinsic rate such that the respective chamber is being consistently paced. The pacemaker energy output is then slowly reduced until loss of capture occurs. This current value is the capture threshold. Typically, the output is left at twice the threshold to allow a margin of safety for pacing. However, if the threshold is > 10 mA, the margin of safety is set to a lesser value, so as not to accelerate fibrosis at the lead/myocardium interface. Failure to capture (or increasing pacing output needs) is the most common problem encountered with temporary epicardial pacing. Reversing the polarity of both bipolar and unipolar lead systems may improve capture. Conditions that might worsen capture threshold during the weaning phase of CPB are- Myocardial ischaemia or fibrosis; Electrolyte imbalance, particularly hyperkalaemia, acidosis and alkalosis; Post-defibrillation period; and Medications- flecainide, propafenone, sotalol, and possibly beta blockers, lidocaine, procainamide, and verapamil.

Sensing thresholds: The sensitivity setting on the temporary pacing generator is the least amount of current that the pacemaker is able to sense. Lower value corresponds to a greater sensitivity. To confirm the sensitivity, the pacemaker is programmed in VVI, AAI or DDD modes and the pacing rate is set below the intrinsic rate. The sensitivity value is slowly increased (making the pacemaker less sensitive) until the sense indicator stops blinking and the pacing occurs asynchronously. The sensitivity value is then turned down (making the pacemaker more sensitive) until the sense indicator blinks with each intrinsic depolarization (P or QRS); this value defines the pacing threshold. I recommend leaving the pacing generator programmed at half the pacing threshold to allow for detection of abnormally small signals. In the absence of an intrinsic rhythm, it is impossible to determine the pacemaker sensitivity, in which case the sensitivity is typically programmed to 2 mV. If the sensitivity value is too low (i.e. the pacemaker is too sensitive), there may be inappropriate sensing of far field signals such as R or T waves, and these may inappropriately inhibit pacing. Threshold testing can usually be performed after coming off CPB.

Typical temporary pacemaker settings

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Setting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower rate limit (pacing rate):</td>
<td>50-60 (backup); 75–95 (pacing)</td>
</tr>
<tr>
<td>PR interval/AV delay:</td>
<td>150 ms (50–300 ms)</td>
</tr>
<tr>
<td>PVARP:</td>
<td>~250 ms; or Auto-determined by rate</td>
</tr>
<tr>
<td>Atrial overdrive stimulation:</td>
<td>up to 800 ppm</td>
</tr>
<tr>
<td>Atrial and ventricular output:</td>
<td>10 mA (5–25 mA)</td>
</tr>
<tr>
<td>Atrial sensitivity:</td>
<td>5 mV (0.4–10 mV)</td>
</tr>
<tr>
<td>Ventricular sensitivity:</td>
<td>5 mV (0.8–20 mV)</td>
</tr>
</tbody>
</table>

Understanding the various available pacing modes is important for their effective utilization in improving cardiac function. The NASPE/BPEG nomenclature for pacing modes is described -
Only the first three letters are relevant for the predominantly used temporary pacemaker generators.

Single chamber pacing modes

**AOO (atrial asynchronous), VOO (ventricular asynchronous)**

In AOO mode, pacing stimuli to the atrium are delivered at a fixed rate, regardless of the underlying electrical rhythm in either chamber of the heart. Ventricular activation and contraction relies on intact AV node conduction. In majority of patients, ability to recruit atrial contraction provides an improvement in cardiac function (SV, EF%) after CPB. This mode can be reliably employed to wean patients from CPB if the underlying rhythm is sinus bradycardia or junctional with intact AV nodal conduction. However, underlying competing rhythms frequently lead to uncoordinated electrical and mechanical activity with all asynchronous pacing modes. Analogous to AOO, VOO pacing stimuli are delivered to the ventricle at a fixed rate. Atrial contribution to ventricular filling and ejection is largely compromised in VOO. Although it does not provide the most favorable hemodynamics for weaning from CPB, it is sometimes employed because of convenience.

**AAI (atrial demand), VVI (ventricular demand)**

In AAI mode, if no intrinsic depolarization (atrial activity) is sensed by the end of a programmed timing cycle a pacing stimulus is delivered to the atrium. However, an intrinsic atrial depolarization that is sensed resets the timing cycle and no stimulus is delivered. AAI mode is used during weaning from CPB if the underlying rhythm is bradycardia, but with an intrinsic atrial rate (or frequent ectopics) sufficiently fast to compete with the programmed pacemaker rate. VVI is similar to AAI, except the sensing and pacing is in the ventricle. As with VOO, lack of coordinated atrial contraction with ventricular paced beats can significantly reduce cardiac output.

Dual chamber pacing modes

**DOO (AV sequential asynchronous)**

Both the atria and ventricles are asynchronously paced at a fixed rate with a programmed AV delay (simulating the delay in the AV node during an endogenous beat). While mechanical efficiency is better than in VOO due to recruitment of atria, the ventricular depolarization from V-pacing spreads throughout the ventricle in an abnormal manner. Mechanical efficiency of the ventricular contraction in a DOO mode is usually less when compared to an impulse conducted through AV node and conduction system. AOO is thus preferred for weaning from CPB if the conducting system is intact. DOO should be used in place of atrial pacing if the patient has higher degree heart blocks and abnormalities of the conduction system. DOO has a similar risk of R-on-T VF as in the other asynchronous modes (AOO and VOO).

**VDD (P wave synchronous)**

In this unique dual chamber mode, only the ventricle is paced. The pulse generator inhibits its ventricular pacing in response to a sensed ventricular depolarization. A sensed atrial depolarization, however, triggers ventricular pacing if no intrinsic ventricular depolarization is sensed. If the absence of intrinsic atrial depolarization, ventricular pacing is initiated. The specific indication for VDD is AV node block with an intact sinus node with the pacemaker serving as a circuit around the AV node.

**DVI (AV sequential, ventricular inhibited)**
In DVI mode, sensing of an intrinsic ventricular depolarization (following either an atrial pacing spike or intrinsic atrial depolarization), inhibits ventricular pacing. In the absence of any intrinsic cardiac depolarization, the pacemaker behaves like a DOO. Since there is no atrial sensing, there is a possibility that the atrial pacing stimulus will not be inhibited even though there is an intrinsic atrial rhythm. This may lead to competition if the atrium is beating at a faster rate, and can precipitate atrial arrhythmias. For this reason, DDI or DDD are preferable to DVI in patients with atrial rates high enough to compete with the pacing rate. DVI is primarily used for immediate treatment of pacemaker-mediated tachycardia. If a ventricular paced beat is conducted retrograde to the atria, atrial sensing might interpret this as intrinsic atrial activity, which in DDI and DDD modes would precipitate a release of a ventricular stimulus creating a ‘loop’ pacemaker-mediated tachycardia.

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

DDI is an improvement over DVI as it includes atrial sensing, which prevents the possibility of the atrial pacing stimulus competing with an intrinsic atrial rhythm. The maximum rate of paced stimuli is the same as the minimum rate set on the pulse generator, a feature that distinguishes DDI from DDD mode. In DDD, the maximal pacing rate is not the set lower rate limit; instead the ventricular pacing stimuli can be delivered at a higher rate in order to 'track' atrial activity. DDI is thus better than DDD in the presence of rapid atrial rates (arrhythmias), as since it avoids pacing the ventricle 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 automatically, but can be manually programmed to DDI in the setting of the not-so-infrequent atrial tachycardias.

DDD (AV sequential, P-synchronous, dual chamber sensing, universal)

DDD is commonly used mode in patients with both atrial and ventricular pacing. Both the chambers can be paced, sensed, triggered or inhibited depending upon the underlying electrical activity. DDD is probably the most versatile pacing mode. However, there is a risk of ventricular tracking of atrial tachycardias in DDD; most temporary pulse generators address this risk by allowing the setting of a 'maximum tracking rate' or 'upper rate limit' or adjusting the PVARP.

VAT, AAT, DAT (Triggered modes)- These modes are rarely used in the immediate post-CPB period.

BiV pacing

Typically, single ventricle or AV sequential pacing is employed to treat the underlying electrophysiologic or mechanical cardiac dysfunction after cardiac surgery. However, hemodynamic benefits observed with single ventricle or AV sequential pacing can be limited, especially in patients with substantial interventricular or intraventricular dyssynchrony. Patients with severely impaired left ventricular (LV) function often demonstrate prolonged inter- and intraventricular conduction. Ventricular dyssynchrony can lead to decreased cardiac output, increased filling pressures, and diastolic mitral regurgitation

Biventricular (BiV) pacing and cardiac resynchronization therapy (CRT) have been shown to improve systolic and diastolic function in non-surgical patients with congestive heart failure (CHF) and conduction block. These benefits have been quantified by the use of various hemodynamic and echocardiographic modalities including 2-D, 3D echocardiography as well as tissue Doppler and strain Doppler imaging. Several reports have also shown improvements in ventricular function in cardiac surgical patients following BiV pacing (pacing leads on the RV and LV). In the majority of patients with reduced LV function, temporary biventricular pacing improved CO and arterial blood pressure after surgery, especially when LV dilatation was present. BiV pacing may reduce the need for excessive inotropes and their side effects, intra-aortic balloon pumps, or ventricular assist devices in weaning from cardiopulmonary bypass.

Intra-aortic balloon pump (IABP) timing and Pacing

Pacing stimuli may be misinterpreted by the IABP as QRS complexes. For isolated ventricular pacing, this small timing discrepancy can be easily overcome by manual adjustment of the IABP timing.
parameters. However, if both atrial and ventricular stimuli are misinterpreted as two separate QRS complexes, timing adjustment may not be feasible. To avoid this problem, either the IABP should be timed according to the arterial pulse, or high frequency filters should be applied to the EKG signal in the monitor. This error is minimized with use of bipolar leads that have a smaller EKG signals as compared to unipolar leads.

References: