Optimizing temporary perioperative cardiac pacing

Henry M. Spotnitz, MD

Temporary perioperative cardiac pacing (TPCP) is indicated after cardiac surgery for treatment of heart block or sinus bradycardia.¹ However, TPCP protocols are often arbitrary, lacking routines to maximize cardiac output (CO) and stroke volume (SV). TPCP parameters which can be optimized include heart rate (HR), ventricular pacing site (VPS), and atrioventricular delay (AVD). Also relevant is variable right ventricular–left ventricular delay (RLD) which recently has become available in some permanent pacemakers designed for biventricular pacing (BiVP). Real-time measurement of SV with ultrasonic transit-time aortic flow probes (UFPs)² or arterial pulse contour systems make optimization of CO and SV during changes in TPCP feasible. Dramatic and possibly life-saving benefits of TPCP optimization have been anecdotally reported. Although the theory and practice of TPCP optimization (POPT) will continue to advance, review of current information can lead directly to patient benefits.

Background

The US Food and Drug Administration (FDA) has approved implantable pulse generators for single-chamber atrial or ventricular (VVI) demand pacing, as well as dual-chamber (DDD) generators that can maintain the physiologic synchrony of atria and ventricles.¹ Both endocardial (transvenous) and epicardial permanent leads are available.¹ A typical DDD system uses a single atrial lead and a single ventricular lead. These leads are unipolar or bipolar (1 or 2 conductors). Unipolar systems use the body as the indifferent electrode. Transvenous leads for DDD pacing are usually placed in the right atrium (RA) and right ventricle (RV). Epicardial leads are placed externally on the RA, RV, left atrium, or left ventricle (LV). These leads sense intrinsic electrical activity and pace at low energy levels. Implantable pacemakers are adjusted by using programmers that communicate with generators through telemetry. For TPCP after cardiac surgery, temporary external pacemakers are used with removable epicardial wires. Bipolar systems using 2 wires to each chamber avoid common grounds and reduce sensing and pacing artifacts. TPCP parameters are adjusted manually.

Recently, the FDA approved BiVP for congestive heart failure. BiVP is effective in some patients with left ventricular dysfunction and QRS prolongation beyond 120 ms (intraventricular conduction delay [IVCD]). An IVCD and a left ventricular ejection fraction (EF) of less than 35% often imply loss of synchrony of contraction of the left ventricular free wall and septum.³ Clinical trials confirm that endocardial pacing of the RV and LV (through a second pacing lead in a lateral branch of the coronary sinus) narrows the QRS complex and significantly improves objective and subjective measures of cardiac function in selected patients with dilated cardiomyopathy.^{4,5} Small trials indicate that BiVP can improve function while reducing myocardial oxygen consumption (MVO₂),⁶ that BiVP is most likely to be effective in patients with very low EF and long IVCD of left bundle branch block configuration,⁷ and that the efficacy of BiVP improves when the left ventricular pacing lead is closest to the most delayed site of left ventricular contraction.^{3,8} Additional studies suggest that endocardial BiVP might be effective in atrial fibrillation and sinus rhythm⁹ and that BiVP reduces the severity of mitral regurgitation.¹⁰ The efficacy of BiVP might be increased in some patients by incorporating a small (20–80 ms) delay into the pacing algorithm, so that either the LV or RV is stimulated first (DeLurgio DB, personal communication, 2003). The effect of AVD and RLD

From the Department of Surgery, Columbia University College of Physicians and Surgeons, New York, NY.

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Address for reprints: Henry M. Spotnitz, MD, Department of Surgery, Columbia University College of Physicians and Surgeons, 622 W 168th St, PH 14-103, 14th Floor, New York, NY 10032 (E-mail: hms2@columbia.edu).

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is not consistent in any given patient. RLD optimization more than doubled the improvement in SV obtained with simultaneous RV-LV pacing in an unpublished substudy of the Insync III trial (DeLurgio DB, personal communication).

With epicardial pacing, increasing HR can increase CO, but the energy cost is also increased.¹¹ Compared with VVI, DDD epicardial pacing improves CO at any given HR.¹² AVD has been shown to affect CO; this is patient specific over a range of 100 to 225 ms.¹³ The variable effect of AVD on CO is related in part to variable conduction times across the atria and ventricles,¹⁴ as well as to intra-atrial conduction delays (IADS). CO is affected by the epicardial pacing site.^{12,15,16} BiVP is effective with epicardial leads.^{17,18} BiVP might be effective in either right bundle branch block or left bundle branch block, requiring protocols specific to the conduction block.¹⁹ Epicardial BiVP might increase arterial systolic pressure and reduce mitral regurgitation.¹⁸ BiVP might facilitate weaning of critically ill patients from cardiopulmonary bypass.²⁰ POPT and BiVP can improve hemodynamics after operations for congenital heart disease.²¹⁻²³ Benefits of BiVP after coronary artery bypass grafting in patients with poor left ventricular function are uncertain.²⁴ In experimental animals epicardial DDD pacing reduces MVO_2 compared with VVI pacing.¹¹ In human subjects endocardial BiVP can improve ventricular function and reduce MVO_2 , whereas inotropic agents increase MVO_2 .⁶

Pacing Protocols

TPCP at physiologic atrial rates typically involves temporary bipolar right atrial and right ventricular wires and a DDD temporary pacemaker. VPS is the anterior or diaphragmatic RV. HR is usually 25% higher than physiologic rate, and AVD is physiologic. Representative values in adults are HR of 90 beats/min and AVD of 150 ms.

Heart Rate

The determinants of left ventricular SV are preload (left ventricular end-diastolic volume), afterload (reflecting outflow resistance and ventricular dimensions), and contractile state. Under physiologic conditions, considerable cardiac reserve exists. During exercise, increased venous return can increase preload, CO, and SV. However, cardiac reserve is often diminished after cardiac surgery. Treatment of postoperative low output states includes optimization of preload (with volume administration), afterload (with vasodilators), and contractility (with inotrope administration). Under these circumstances, HR affects systolic or mean arterial pressure (MAP) through several mechanisms. Theoretically, $\text{CO} = \text{MAP}/\text{R}$, where R is arterial resistance. Thus increases in CO and MAP are linearly related until R is affected by reflex changes (Prasso and colleagues, manuscript in preparation). On the other hand, systolic pressure reflects both MAP and pulse pressure, and pulse pressure is affected by SV. If HR

is cut in half and CO remains constant, SV must double, and systolic pressure will increase. For this reason, MAP is more reliable than systolic blood pressure in POPT.

Atrioventricular Delay

DDD protocols optimize ventricular filling by synchronizing the onset of ventricular systole with completion of the atrial kick. For patients with regular atrial rhythm, AVD begins with right atrial depolarization or stimulation and ends when the RV depolarizes or is stimulated. The AVD in a permanent pacemaker is typically 150 ms during atrial sensing and 200 ms during atrial pacing. The differential of 50 ms allows for IAD, the interval between the atrial pacing stimulus and the P wave. IADs are prolonged to 150 to 200 ms in some patients. A prolonged IAD of 200 ms combined with an AVD of 200 ms will result in simultaneous right atrial and right ventricular contraction, negating the atrial kick. Variable IAD and variable conduction time from the RA to the left atrium and atrioventricular node to the RV and the LV require AVD optimization.

Recommendations

POPT begins with CO measurement. Many techniques are available for this, but real-time SV by UFP best facilitates POPT. On the downside, application and removal of UFPs requires some manipulation of the ascending aorta. Fluid immersion of the UFP and aorta maximizes ultrasound transmission and data reliability. CO varies with respiration; averaging data over one complete respiratory cycle is most meaningful. Alternative methods for measuring changes in CO include arterial pulse contour systems (Berberian and coworkers, manuscript in preparation), thermal or indicator dilution, ventricular conductance, echo-Doppler, Fick determinations,^{22,23} or mixed venous oxygen saturation in the RA. Changes in arterial blood pressure can be useful; MAP is most closely related to CO (Prasso and colleagues, manuscript in preparation).

During TPCP for sinus bradycardia or heart block, POPT involves simply maximizing CO. HR is usually determined by means of clinical assessment of benefits of HR-dependent increases in CO versus detrimental effects of decreased diastolic filling time and possible subendocardial ischemia. Converting VPS from the RV to a biventricular state can increase CO in this setting. Temporary epicardial wires can be added to the obtuse margin of the LV. After identifying which lead of the pair has the lowest threshold, BiVP is implemented by bringing the best right ventricular and left ventricular leads to the negative terminal of a temporary pacemaker; the other pair is combined at the positive terminal. If the patient's body is used as the ground, the myocardial wires should be combined at the negative terminal, and the patient's body should be connected to the positive terminal. The output of the temporary pacemaker should be at least twice the value of the right ventricular or

left ventricular threshold, whichever is higher. Paired temporary atrial wires should be connected as usual. We find these maneuvers consistently increase CO by 10% or more in patients with physiologic atrial rates after valve replacement surgery. In some patients, the improvement is 20% to 30%. BiVP effects on CO can be quickly tested and a clinical decision reached on whether continuation is warranted, considering a small risk of bleeding on removal of the left ventricular wires. If the benefit does not warrant the risk, the left ventricular wires can be removed. The improvement in CO with BiVP is so consistent that it might reflect reversal of the detrimental effects of single-site right ventricular pacing in most instances.

The relationship between AVD and CO is curvilinear, with CO decreasing at AVDs shorter than 100 ms or longer than 200 ms. Exceptions are common in advanced heart failure, and objective testing can overcome errors of conventional recommendations. With a long IAD, very long AVDs might be needed for POPT.

RLD is not currently available in temporary pacemakers but is available in some FDA-approved BiVP generators. If permanent BiVP is clinically indicated, it can be implemented with epicardial leads at the conclusion of cardiopulmonary bypass. If this is done, the optimization sequence should be AVD first and then RLD at the optimum AVD. An RLD that results in right ventricular stimulation first is most likely to benefit patients with primary right ventricular dysfunction,²⁵⁻²⁷ whereas the LV first should be favored in left ventricular dysfunction. At present, no reliable guidelines for POPT after cardiac surgery have been developed. The stability of POPT over time is uncertain and could require frequent cycles of measurement and adjustment.

Pacing for Heart Failure

BiVP might prove valuable in patients undergoing cardiac surgery with advanced preoperative ventricular dysfunction and an IVCD. Little is known about the risks and benefits of pacing for heart failure after cardiac surgery in patients with no other indication. The importance of low EF and IVCD in this setting are largely unproved. Early studies demonstrate beneficial effects of multisite pacing in congenital heart disease with right bundle branch block.^{22,23} Until more information and FDA-approved devices are available, studies of BiVP are best conducted with institutional review board protocols and informed consent.

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