Indications for Temporary and Permanent Pacemakers

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Introduction

Every year, over 200,000 pacemakers are implanted in the United States alone, accounting for a significant proportion of electrophysiology procedures [1]. Despite being considered trivial in the current era, pacemakers represent a major development in the twentieth century. Besides treating life-threatening bradycardias, they allowed significant evolutions in cardiac surgery since acquired heart block was a major cause of postoperative mortality. From the early days of asynchronous pacemakers to the newest developments of leadless pacemakers and techniques to directly pace the conduction system for cardiac resynchronization, the indications for pacing have been expanded over the years beyond their original objective to treat bradyarrhythmias.

A brief review of the history of pacemakers gives insight into the evolution of pacing modalities still used in current clinical practice. The problem at hand was a clinical entity known as Stokes-Adams syndrome, described as sudden transient loss of consciousness with slow pulse and named at the end of the nineteenth century after two of the early physicians who recognized this association [2]. Given the clear relationship between profound sudden bradycardia with decrease in cardiac output and syncope, treatment attempts consisted of intracardiac injections of stimulants such as epinephrine, which were also aimed at treating sudden cardiac death and gained popularity in the 1920s. In the 1930s, however, New York

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cardiologist Albert Hyman noted that the simple mechanical irritation of the heart muscle by probing it with a metallic needle caused electrical activation of the heart [3]. This observation led to the subsequent development of the first artificial pacemaker, which delivered small electrical current through a percutaneous needle inserted directly into the heart to cause myocardial "irritation" and ventricular contractions [4, 5]. Despite representing a revolutionary invention to resuscitate patients who developed asystolic cardiac arrest, Hyman's artificial pacemaker did not reach much popularity given the need for direct myocardial contact with the pacing electrode, making it an impractical invasive procedure in a pre-cardiac surgery era.

In the early 1950s, Boston cardiologist Paul Zoll reported his experience pacing the heart indirectly, without the need for direct myocardial contact. This was first achieved in dogs using transesophageal electrodes, followed by transcutaneous pacing with intradermal electrodes placed on the chest wall to successfully treat two patients with syncope due to transient asystole in the setting of complete heart block [6]. The principles employed then not only remain valid to contemporary transcutaneous pacing but also led to the development of cardiac defibrillators. In the same decade, the advent of cardiac surgery posed the issue of acquired complete heart block, which would complicate about 10% of the patients who would undergo surgical repair for atrial septal defect. Given surgical access to the heart, Hyman's idea of directly pacing the myocardium seemed much more trivial. Walt Lillehei and Earl Bakken, respectively a cardiac surgeon and an engineer from Minnesota, employed transistorized pacemakers using tunneled epicardial pacing leads—similarly to what is still used today—to painlessly pace the heart [7]. This allowed miniaturization of the artificial pacemaker compared to the vacuum tubes employed by Hyman a few decades earlier, making these battery-powered devices wearable for prolonged periods of time in patients who underwent cardiac surgery, as well as in non-surgical patients with Stokes-Adams syndrome for whom pacing leads were implanted percutaneously [8].

Inspired by Lillehei and Bakken's work, Stockholm cardiac surgeon Åke Senning replicated externalized percutaneous pacing in conjunction with physician-engineer Rune Elmqvist. However, they were concerned about the risks of infection from the externalized device, as well as the potential for damage to the externalized lead and the inconvenience of carrying the pacemaker. Later that decade, they reported the first implantable pacemaker experience in humans, implanting the self-contained pulse generator subcutaneously in the epigastrium [9].

Following these groundbreaking inventions, pacing technology advanced exponentially to include transvenous leads, synchronous pacing with underlying rhythm sensing, multi-chamber pacing, conduction-system pacing, and more recently leadless intracardiac pacing, all of which still rely on the same concepts first described almost a hundred years ago. In the following sections, different pacing techniques will be reviewed, some of which directly resemble some of the pioneering work described above. Different pacing modalities will be reviewed, as well as current indications for temporary and permanent pacemaker utilization.

Temporary Pacemakers

Although permanent pacemaker implantation can be done safely and with minimal complication risks, in certain clinical settings, pacing is desired but necessary only transiently, without long-term need for a permanent pacing. In other circumstances, pacing may be needed immediately, and permanent pacemaker implantation would impose an unacceptably long delay in stabilizing a clinical urgency or emergency. Finally, there are situations in which permanent pacing is desired, but the clinical setting is not favorable for permanent device placement. In such situations, temporary pacing can be established for clinical stabilization until a more definitive device, if warranted, can be implanted. In this section, different temporary pacing modalities will be reviewed, from least invasive to most invasive. Their unique characteristics will be described, along with scenarios in which they may be preferred. Complications specifically related to each of these pacing modalities will also be described.

Temporary Pacing Modalities

Transcutaneous Pacing

The most immediately available temporary pacing modality is transcutaneous pacing, when electrical current is delivered through the chest wall using external pads from cardioverter/defibrillators to capture and pace the heart. Commercially available cardioverter/defibrillators allow transcutaneous pacing with manual rate and current output adjustments. The pads are placed in standard defibrillation position (anterior pads or anterior/posterior pads), and usually, pacing starts at maximum output that is titrated down until capture is still observed with minimal or no chest discomfort.

The main advantage from this pacing modality is the ability to establish pacing within seconds, without the need for any invasive procedure that might delay appropriate rhythm stabilization. This is typically employed in the most emergent situations, when patients suddenly become unstable from bradyarrhythmias and pacing must be established immediately. Another scenario in which it is employed clinically is when patients are at risk of needing temporary pacing but there are no imminent pacing needs. In such cases, patients are planned for permanent pacemaker implantation within hours or the next day, so transcutaneous pads are placed on their chest with pacing in standby to avoid invasive procedures that may lead to transient periprocedural bacteremia.

Despite its advantages, transcutaneous pacing poses significant issues when implemented. Although the goal is myocardial pacing, a side effect from this modality is chest wall muscle capture, which can be painful and uncomfortable in awake patients. Sedation can be used to minimize this issue, but transcutaneous pacing is usually not tolerated for prolonged periods of time. In addition, chest wall capture explains the most important complication from this pacing modality, which is

unrecognized lack of myocardial capture. Although some providers unfamiliar with transcutaneous pacing may rely on telemetry to assess for myocardial capture, chest wall muscle capture creates large electrical signals that can be easily mistaken for myocardial capture. More importantly, most telemetry monitors auto-adjust their tracings for signal amplitude, rendering the native bradycardic escape (if present) rather small compared to the very prominent chest wall myopotentials. These misleading tracings make it harder to recognize lack of capture and should not be used to assess for appropriate capture in transcutaneous pacing. Instead, an astute clinician should always look for electromechanical association during transcutaneous pacing, such that arterial pulses can be palpated (typically on the femoral artery) during transcutaneous pacing to ensure the patient has been properly stabilized.

Finally, there are situations in which current transmission from the chest wall to the myocardium is impaired, making this a less desirable technique. This is particularly evident in the immediate postoperative period following chest surgery, when some degree of pneumothorax and pneumomediastinum can lead to very inefficient current delivery from transcutaneous pads to the heart.

Transesophageal Pacing

Although transesophageal pacing has more historical than clinical relevance in current clinical practice, it remains an important method of temporary pacing in patients who exhibit bradyarrhythmias, especially when atrioventricular conduction is preserved. This pacing method relies on the close anatomic proximity between the esophagus and the posterior left atrial wall, so that electrical impulses generated from a source in the esophagus can capture tissue for atrial pacing. Technically, this can be easily achieved by advancing a flexible bipolar electrode through the nares or oropharynx into the esophagus until atrial electrogram recording and capture can be observed. Although ventricular capture can also be achieved with this system, this requires advancing the lead more distally toward the stomach, which often results in unreliable capture due to high pacing thresholds.

Since these devices are not in direct contact with myocardium, their strength-duration curve is significantly different from transvenous leads. Transesophageal pacing usually requires much longer pulse duration (up to 10 ms) and much higher current (at least 10 mA), with optimal threshold at around 5 ms and 15 mA. Ideally, they require different pulse generators than those typically used for transvenous pacing, which exhibit much shorter pulse duration (usually 2 ms) [10]. Standard pacing catheters can be placed in the esophagus for this purpose, such as those used in electrophysiologic studies, but usually they would have worse pacing threshold due to shorter interelectrode distance compared to pacing leads specifically designed for esophageal pacing.

Interestingly, the fact that atrial recordings and pacing maneuvers can be performed from transesophageal leads makes this an attractive strategy to perform electrophysiology studies non-invasively. This becomes particularly useful in children with supraventricular tachycardias, so that radiation exposure can be avoided in this very young age group of patients. In adult patients with complex congenital heart disease who have limited transvenous access to the cardiac chambers (e.g.,

such as those who underwent Fontan palliation), transesophageal leads can also be employed to minimize the number of intracardiac diagnostic catheters, making the procedure safer and faster and minimizing the potential for residual shunts from multiple transseptal punctures [11].

Transvenous Temporary Pacing

Temporary pacing with transvenous leads is the most commonly utilized pacing technique in patients who require urgent or emergent pacing. This modality is preferred because of its efficacy; since the pacing electrode is in direct contact with myocardium, it has lower pacing threshold and more reliable capture compared to transcutaneous or transesophageal pacing.

Similar to permanent pacemakers, transvenous temporary pacemakers consist of two components: an intracardiac pacing lead and a pulse generator. The main difference to permanent pacemakers is the pulse generator, which is externalized and manually controlled instead of directly implanted in the patient's body. Most commonly, externalized pacing leads have no fixation mechanism and have a balloon at their tip to facilitate placement at the bedside, without the need for fluoroscopy to guide positioning. Conversely, temporary leads without a balloon tip require fluoroscopy for placement, as these stiffer leads impose a significant risk of perforation if performed blindly compared to flotation leads. They may be preferred in patients for whom bedside pacemaker placement is difficult, such as patients with severe tricuspid regurgitation precluding advancement of a balloon-tipped catheter into the right ventricle. There are also pacing Swan-Ganz catheters, which are positioned with their tip in the pulmonary arterial circulation but have pacing electrodes proximally for passive contact with the myocardium. These are typically used in critical care setting when hemodynamically unstable patients are at risk of bradyarrhythmias. Finally, active fixation externalized leads can also be used in cases of high-risk for dislodgement. In patients who require prolonged temporary pacing, an active fixation lead can be connected to an external standard pacemaker generator until definitive implantation can be performed.

Bedside Transvenous Pacing Technique

Unlike non-balloon tipped temporary pacemakers, which rely on fluoroscopy for appropriate placement, and pacing Swan-Ganz catheters, which rely on either fluoroscopy or pressure tracings to guide placement, balloon-tipped temporary pacing leads rely on electrograms for emergency placement at the bedside. A very clear understanding of the technical skills and expected observed tracings during transvenous pacemaker insertion is critical for fast and effective stabilization in these situations.

After vascular access is established and the patient is under electrocardiographic monitoring, the pacing lead is positioned in a sterile sleeve and connected proximally to the generator pacing unit. This is set to asynchronous pacing at a rate above the patient's native rhythm and at high output, so that capture can be observed whenever there is contact with myocardium. The catheter is inserted in the venous sheath until its tip is free in the intravascular space, beyond the sheath introducer.

The balloon is inflated with 1–1.5 mL of air, facilitating catheter floatation with the venous blood flow toward the heart. Once the catheter is set to pace in the intravascular space, ECG recordings will show pacing artifact without capture.

As the catheter is advanced, the operator should pay close attention to the catheter markings showing how far it is inserted. Once the catheter tip enters the right atrium, the operator will notice that the pacing artifact will be followed by a P-wave, demonstrating atrial tissue capture. This may or may not be followed by a QRS, depending on whether the patient has preserved atrioventricular conduction. At this point, the catheter is carefully advanced more distally until the pacing artifact is followed by a paced QRS morphology, indicating ventricular tissue capture. The catheter natural curve will have a tendency to orient its tip toward the interventricular septum, while the balloon will have a tendency to bring the catheter anteriorly to the right ventricular outflow tract. As such, gentle counterclockwise torque is applied to the catheter as it is advanced a few more centimeters, such that septal orientation is maintained while the catheter is inserted in the right ventricle away from the tricuspid annulus. It is important to not overdo this torque to prevent directing the catheter tip toward the right ventricular free wall, which would increase the risk of perforation. The pacing output is then decreased gradually until capture is lost to document appropriate pacing threshold, which should ideally be below 1–2 mA. The balloon is deflated to stabilize the catheter and prevent dislodgement to the pulmonary artery, and if pacing threshold remains appropriate, it is locked in place at the sheath. If the catheter needs repositioning, it is critical to withdraw the catheter back to the right atrium before reinflating the balloon to prevent inadvertent inflation inside the pulmonary vasculature or in the apex against ventricular myocardium. In addition, the catheter should never be withdrawn while inflated to avoid disruption of the pulmonic or tricuspid valves.

This technique can be employed for atrial pacing, although this is rarely done in clinical practice because most hemodynamically unstable bradyarrhythmias warrant ventricular pacing for stabilization. In addition, even though transvenous lead placement during high output pacing is preferred in emergencies, bedside insertion can also be done by recording local electrograms if the underlying rhythm is stable. Instead of connecting the pacing catheter to the generator, the negative port is connected to an electrocardiogram lead (typically V1) so the pacing lead records local unipolar electrograms. As the catheter is advanced from the venous system to the right atrium, it will record low amplitude electrograms that are timed to the P-waves. When the catheter is at the tricuspid valve annulus, it will simultaneously record atrial and ventricular electrograms, and once it reaches the ventricle with good tissue contact, it will record high amplitude electrograms timed to the QRS complexes with local injury current. The balloon is deflated and the catheter is confirmed to be in stable position with good pacing threshold.

Once the lead is locked in the sheath, its proximal end is looped and secured to the patient's skin, such that enough slack is left to compensate for neck movement without causing traction with lead dislodgement. The distance from the catheter tip to the sheath is recorded (usually between 30 and 40 cm) and the pacing output is set to a safety margin of 2–3 times the pacing threshold. The operator should avoid

pacing at an output too much higher than threshold because, in this scenario, any loss of capture would potentially happen when the catheter is virtually outside of the right ventricular cavity, without any chance for output adjustment or minor repositioning. Conversely, if the set output is two to three times above threshold, loss of capture would more likely reflect poor tissue contact that can be circumvented with higher output until lead repositioning can be performed. Finally, if the patient exhibits an underlying rhythm, sensitivity should be adjusted to prevent underpacing or overpacing if asynchronous pacing will not be employed. At the end of the procedure, a chest X-ray is indicated to document lead positioning, so the operator knows how proximal or distal the tip is in relationship with the tricuspid and pulmonic valves in case repositioning is required.

Externalized Semipermanent Pacing

As will be reviewed later in this section, temporary transvenous pacing is indicated in clinical scenarios that require stabilization until permanent pacing can be performed. However, it is unsafe to rely on a pacing lead that is not secured to the myocardium in cases that require prolonged delay in permanent pacemaker implantation. In these scenarios, the preferred modality is a permanent, active fixation screw-in lead that is externalized through the vascular access site (typically the internal jugular vein or the axillary/subclavian vein) and connected to a pacing generator, usually an automatic pacing unit like the implantable ones. An externalized pacing lead is implanted in the electrophysiology laboratory under fluoroscopy guidance, similar to the way permanent pacemaker leads are inserted. Active fixation leads are employed instead of passive fixation to allow easy removal when they are no longer needed. The lead is connected to a pulse generator, which is externalized and taped to the patient's body instead of being implanted in the chest. This is important because unipolar pacing cannot be used in this setting due to high impedance between the pulse generator and the patient's skin. When pacing is no longer indicated, or when permanent pacemaker can be implanted, the lead is unscrewed and removed from the patient's body.

Epicardial Temporary Pacing

When access to the heart is obtained during cardiac surgery, it is prudent to place temporary epicardial pacing leads, especially when the surgery involves high risk of bradyarrhythmias and atrioventricular conduction block, like surgery for valve repair or replacement. These leads can have different configurations for myocardial contact, such as a helical fixation tip or a sutured bipolar lead, and often are connected to the right atrial appendage and right ventricular diaphragmatic surface to allow sequential pacing with atrioventricular synchrony (Fig. 22.1). They are externalized to the patient's skin below the sternotomy site and connected to a pacemaker pulse generator, the same used for transvenous pacemakers and with similar strength-duration curve. This allows real-time adjustment in the immediate postoperative period to treat bradyarrhythmias, to pace-terminate atrial and ventricular tachyarrhythmias, and to increase cardiac output by employing higher heart rates in case of hemodynamic instability [12, 13].

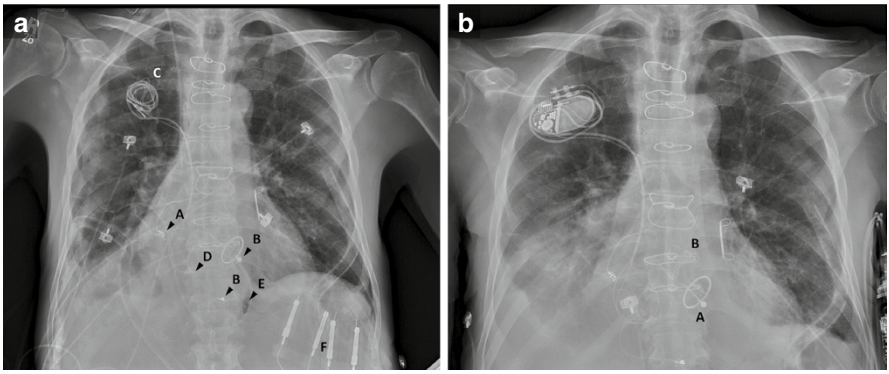

Fig. 22.1 (**a**) Chest X-ray showing permanent epicardial pacing leads in the right atrium (A, screwed helix) and right ventricle (B, two pacing poles) tunneled to the right chest (C) in a pacer-dependent patient who underwent aortic valve replacement due to endocarditis and required system extraction intraoperatively. Temporary epicardial pacing leads were also placed in the right atrium (D) and right ventricle (E), and externalized to be connected to a portable pacing unit (F). (**b**) Once cultures cleared, permanent pacemaker generator was implanted in the right chest. Note that this patient had prior mechanical mitral valve replacement (A) in addition to bioprosthetic aortic valve replacement (B)

Temporary epicardial pacing wires deteriorate on a daily basis, so careful monitoring for underlying rhythm and pacing threshold should be done in the immediate postoperative period. Once the patient has recovered from surgery and temporary pacing is no longer needed, these leads are removed with gentle traction. These patients are then observed to ensure they do not develop pericardial bleeding with tamponade.

Indications for Temporary Pacing

In broad terms, the usual indications for temporary pacing are the same as those for permanent pacemaker implantation, except that temporary pacing is employed whenever any delay could compromise hemodynamic stability, when permanent pacing is not immediately available, or when the arrhythmia is transient and might not require long-term pacing. The current indications for temporary pacing are summarized in the 2018 guideline on evaluation and management of patients with bradycardia and cardiac conduction delay, published jointly by the American College of Cardiology, the American Heart Association, and the Herat Rhythm Society [14]. These categories are sinus node dysfunction, atrioventricular block, bradycardia in the context of acute myocardial infarction, cardiac surgery (including coronary artery bypass grafting and valve repair or replacement), and transcatheter aortic valve replacement. The indications for temporary pacing are summarized in Table 22.1 and will be reviewed here.

Table 22.1 Summary of indications for temporary pacemakers according to current bradycardia and conduction delay guidelines

Temporary pacing recommendation	COR	LOE
Unstable SND refractory to medical therapy until a permanent pacemaker is placed or the bradycardia resolves	IIa	C
Transcutaneous pacing in unstable SND refractory to medical therapy until transvenous pacing (temporary or permanent) is placed	IIb	C
Second- or third-degree AV block with symptoms or hemodynamic compromise refractory to medical therapy	IIa	B
Transient or reversible AV block (Lyme, drug toxicity), before determination of need for permanent pacing	I	B
Acute MI with symptomatic or hemodynamically significant bradycardia due to SND or AV block	I	B
Routine placement of temporary epicardial pacing wire in isolated CABG	IIa	B
Routine placement of temporary epicardial pacing wire in surgery for AF	I	B
Routine placement of temporary epicardial pacing wire in mitral valve surgery	IIa	C
Routine placement of temporary epicardial pacing wire in tricuspid valve surgery	I	C
Routine placement of temporary epicardial pacing wire in aortic valve replacement or repair	I	C

Based on data in Kusumoto et al. [14]
ACC American College of Cardiology, *AHA* American Heart Association, *AF* atrial fibrillation, *AV* atrioventricular, *CABG* coronary artery bypass grafting, *COR* class of recommendation, *HRS* Heart Rhythm Society, *LOE* level of evidence, *MI* myocardial infarction, *SND* sinus node dysfunction

Sinus Node Dysfunction

Sinus node dysfunction can manifest as symptomatic bradycardia or syncope due to pauses. In the inpatient setting, it can cause hemodynamically significant bradycardias that may require temporary pacing for stabilization. In patients who are symptomatic despite medical therapy, temporary transvenous pacing is usually employed for stabilization until it resolves (if a reversible cause is present) or permanent pacemaker can be pursued. Transcutaneous pacing can also be employed in this setting for stabilization until a transvenous wire can be placed. Some of the most dramatic presentations are prolonged hemodynamically significant pauses after cardioversion (electrical, chemical, or spontaneous) requiring chest compression until resumption of sinus rhythm, or even pause-dependent polymorphic ventricular tachycardia or ventricular fibrillation, when prolonged pauses lead to dispersion in repolarization allowing an escape rhythm to trigger reentry (Fig. 22.2).

Atrioventricular Block

In patients who develop hemodynamic instability or symptoms in the setting of second- or third-degree AV block, temporary pacemaker is indicated for clinical stabilization until a permanent device can be implanted or the AV block resolves. When the AV block is expected to be transient or reversible, the guideline document gives its strongest recommendation for temporary pacing (class of recommendation [COR] I). Usual examples of reversible AV block are myocarditis (including Lyme disease), drug toxicity (such as digoxin or other AV nodal blockers), and increased vagal tone (such as in transient increased intracranial pressure) (Fig. 22.3). Although

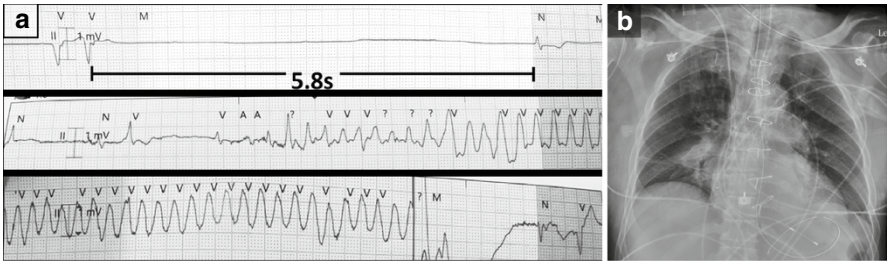

Fig. 22.2 Telemetry tracing showing significant sinus node dysfunction, with sinus arrest and junctional escape with over 5-s pause (panel **a**, top) in a transplanted patient hospitalized with acute rejection. This led to pause-dependent polymorphic ventricular tachycardia (panel **b**, middle) that required external cardioversion (panel **a**, bottom). This patient underwent emergency transvenous pacemaker to prevent further pauses (panel **b**)

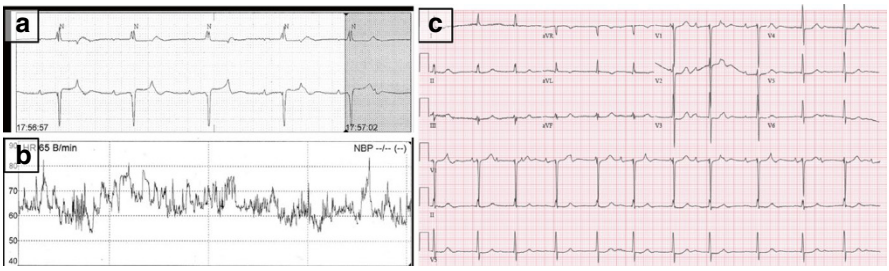

Fig. 22.3 Telemetry showing 2:1 AV block in a patient who suffered large middle cerebral artery stroke with intracranial hypertension (**a**). Heart rate trend in 24 h shows no evidence of significant pauses or bradycardia (**b**). ECG shows evidence of 2° AV block with variable PR interval on conducted beats, with robust junctional escape during periods of high-grade AV block and no evidence of infranodal conduction disease (**c**). These findings are consistent with AV nodal block from increased parasympathetic tone that did not warrant temporary pacing

not explicitly contemplated in the guidelines, temporary transvenous pacemaker is also usually employed in patients presenting with acute myocarditis and ominous signs for risk of His-Purkinje system injury, such as syncope with electrocardiographic evidence of infranodal conduction disease (Fig. 22.4).

In patients with myocardial infarction who experience sinus node dysfunction or atrioventricular block that is hemodynamically significant or refractory to medical therapy, temporary pacing also receives the strongest recommendation (COR I), with the observation that waiting for resolution should be the norm instead of permanent implantation in this setting. This is exemplified in Fig. 22.5, which shows transient 2° AV block Mobitz I in the setting of ST-elevation myocardial infarction that completely resolves after 2 weeks.

If temporary pacing is required for prolonged periods of time, externalized semipermanent pacemakers are recommended over transvenous wires without active fixation given their long-term stability (COR IIa). In clinical practice, the most common scenario in which this strategy is employed is when pacing is needed with

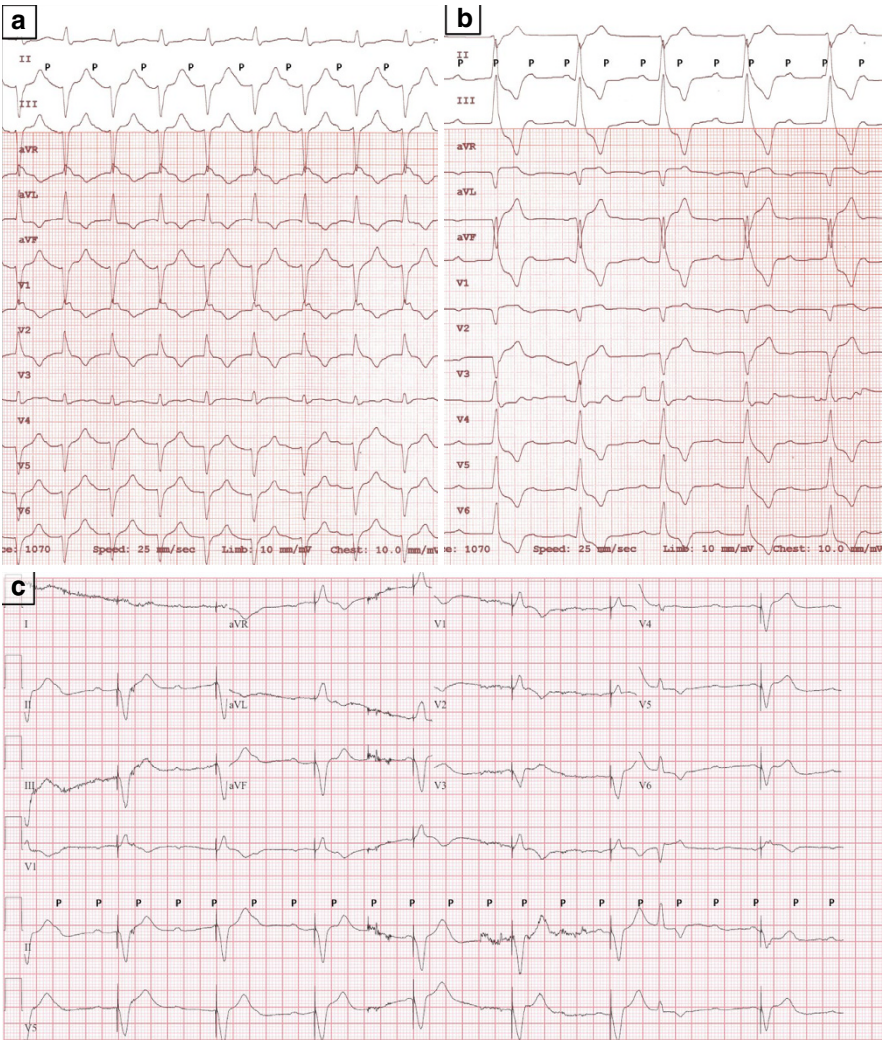

Fig. 22.4 Electrocardiographic progression to complete heart block in a young patient who presented with syncope and flu-like symptoms, later diagnosed with lymphocytic myocarditis. Presenting rhythm with sinus tachycardia with first AV block, RBBB and left anterior fascicular block (**a**), which progressed to complete heart block with junctional escape and LBBB pattern 30 min later (**b**). A transvenous pacemaker was placed, with no reliable escape when paced at 50 bpm (**c**), ECG figures courtesy of Dr. Gan-Xin Yan

ongoing infection that precludes permanent pacemaker implantation. Figure 22.6 shows 2:1 AV block due to prosthetic valve endocarditis with abscess formation in a patient who was not eligible for urgent valve surgery due to intracranial hemorrhage complicating embolic strokes. From an electrical standpoint, at least, the patient was stabilized with an externalized pacemaker due to inability to perform source control in the near term.

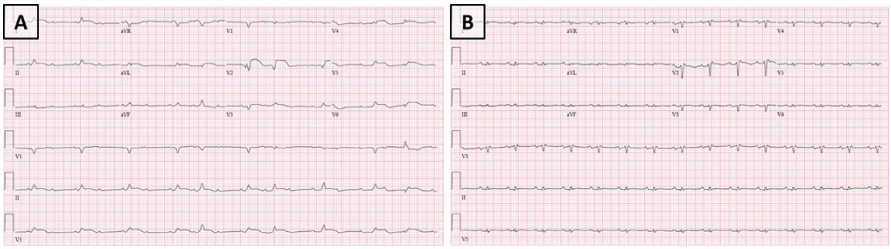

Fig. 22.5 Electrocardiogram showing acquired 2° AV block Mobitz I in the setting of anterolateral ST-elevation myocardial infarction (left), which was complicated with cardiogenic shock requiring transvenous pacing in addition to mechanical circulatory support for stabilization. Note that the AV block completely resolves 2 weeks later (right). Although the AV node arterial blood is usually supplied by a branch of the right coronary artery, the left circumflex can originate this branch in a minority of patients, which explains transient AV nodal level block in the absence of right coronary artery involvement in this case

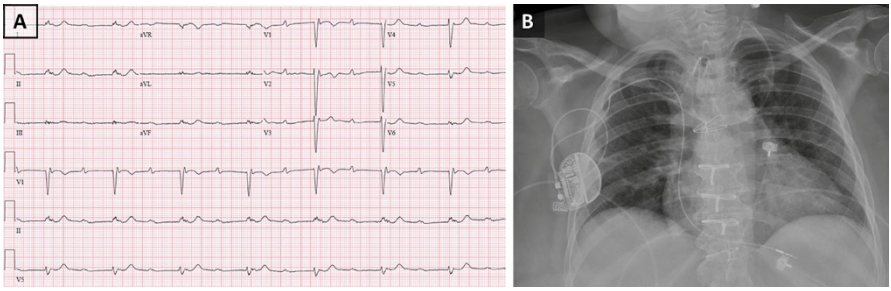

Fig. 22.6 Electrocardiogram showing 2:1 AV block (panel A) in the setting of prosthetic mitral and aortic valve endocarditis complicated with abscess. Although this is at very high risk of evolving to complete heart block, this patient was complicated with embolic strokes with hemorrhagic transformation, precluding immediate definitive surgical treatment. As such, this patient underwent an externalized semipermanent pacemaker implantation through her right axillary vein (panel B) for rhythm stabilization until her neurologic status would allow heparinization for surgical intervention

Cardiac Surgery

Patients who undergo cardiac surgery are at an especially high risk for bradyarrhythmias in the immediate postoperative period, especially when surgery to repair or replace the tricuspid, mitral, or aortic valves is performed. Given the risk for injury or at least transient inflammation at the atrioventricular node or His-bundle tissue, these patients can develop transient or permanent atrioventricular block. Not surprisingly, concomitant placement of temporary epicardial pacing wires is the norm until there is enough evidence of preserved conduction or need for permanent pacing after an appropriate observation period (COR I for tricuspid and aortic surgeries and COR IIa for mitral surgery). Similarly, temporary epicardial pacing is also recommended in patients who undergo surgery for atrial fibrillation (COR I), given their increased risk for sinus node dysfunction and atrioventricular block.

Although isolated coronary artery bypass surgery poses much lower risk for bradyarrhythmias, temporary epicardial pacing is also recommended in this setting according to current guidelines (COR IIa).

Special Circumstances

With increasing utilization and broader indications for transcatheter aortic valve replacement (TAVR), this group of patients has become an important population receiving temporary transvenous pacemakers in the past decade. In addition to allowing rapid ventricular pacing for catheter stabilization during valve deployment, temporary pacing is critical in the post-procedural period given the risk for transient or complete heart block. Even though there is no explicit mention of indication for temporary pacing in TAVR patients, transvenous temporary pacing is routinely employed in these cases similarly to valve surgery.

Another indication for temporary pacing not contemplated on the bradycardia guideline is to prevent or treat tachycardias. In the postoperative period, for example, rapid atrial pacing can terminate atrial tachycardia or atrial flutters, saving the patient the need for external cardioversion. More importantly, in patients who develop recurrent torsades de pointes in the setting of acquired long QT, temporary pacemakers are indicated to pace at faster rates for QT shortening and prevention of pauses that facilitate early afterdepolarizations that may trigger ventricular arrhythmias. This is exemplified in Fig. 22.7, which shows a case of acquired long QT complicated with torsades de pointes that resolved several days later.

Permanent Pacemakers

Unlike temporary pacemakers, permanent pacemaker implantation is usually performed electively and takes more time for completion. Therefore, even when the indication for pacing is urgent, patients are typically stabilized by alternative means before presenting to the electrophysiology laboratory for permanent pacemaker implantation. Although their basic design is essentially unchanged for decades, significant evolution has happened in recent years that have expanded indications for pacing beyond treating bradyarrhythmias. This happened in parallel with novel pacing modalities, which will be reviewed in this section.

Pacing Modalities

The standard pacemaker unit consists of two components: a pulse generator and its respective pacing lead(s). The pulse generator is designed to accommodate the number of leads that will be employed, which depends on the number of pacing sites that are planned. The pacing leads can have different fixation mechanisms, which are critical to prevent lead dislodgement. These can be active fixation mechanisms, with a helix that is screwed into the myocardium, or passive fixation mechanisms, with tines that attach to trabeculated myocardium or pre-shaped leads that stabilize in

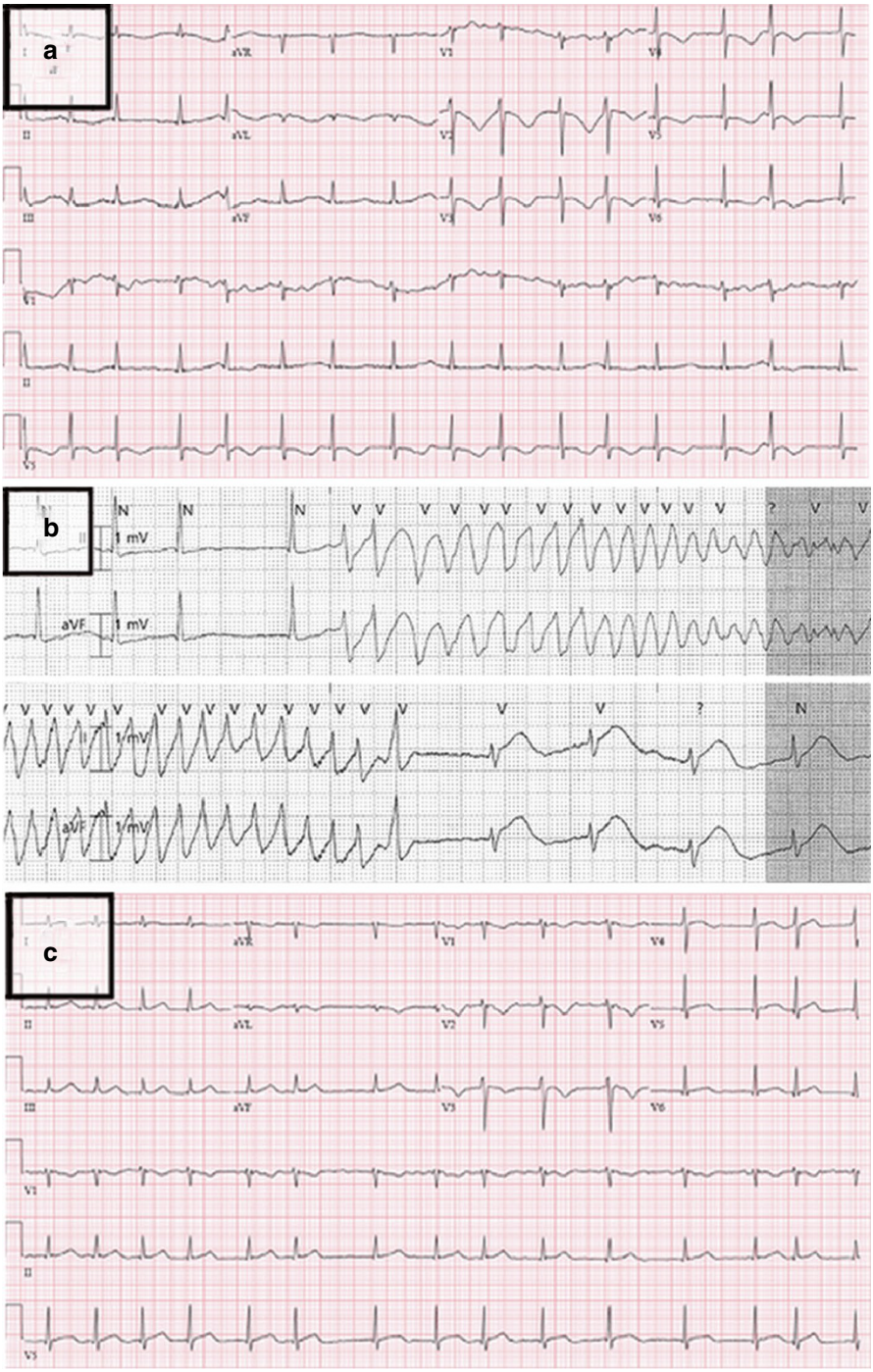

Fig. 22.7 Acquired long QT in the setting of stress-induced cardiomyopathy with concomitant QT prolonging drugs (**a**) leading to recurrent episodes of torsades de pointes (**b**). This patient's QT interval completely normalized after some recovery from cardiomyopathy and removal of offending drugs (**c**), precluding need for permanent device implantation

coronary venous branches for left ventricular pacing. Permanent pacemakers can be used for single-chamber pacing (atrial or ventricular), dual-chamber pacing, and biventricular pacing for cardiac resynchronization. In addition, directly pacing the conduction system (His bundle, left bundle) can also achieve resynchronization without need for pacing in the coronary venous system. A detailed description of pacemaker implantation technique is beyond the scope of this chapter, and cardiac resynchronization therapy will be reviewed in further detail in Chap. 25.

In recent years, leadless pacemakers have been developed so the whole unit comprises the equivalent of a generator and pacing electrode. This miniaturized generator is implanted directly into the right ventricular myocardium, with capabilities comparable to standard single-chamber pacemakers (pacing, sensing, and rate responsiveness). Since they preclude the need for any transvenous component and creation of a pocket to accommodate the generator unit, leadless pacemakers are not associated with long-term vascular complications (such as stenosis or thrombosis) and are less prone to infection compared to standard pacemakers. In general, leadless pacemakers are indicated for any patient for whom single-chamber ventricular pacing (VVI mode) is appropriate, such as patients with permanent atrial fibrillation, patients who require infrequent pacing, or patients with comorbidities that are significant enough to determine overall survival.

For instance, patients who develop bradycardia due to high-grade AV block while in atrial fibrillation have no indication for atrial lead and should be good candidates for either standard single-chamber pacemaker or leadless pacemaker implantation. In patients who could benefit from an atrial lead for atrial pacing or for sequential pacing, leadless pacemakers are still an alternative if the risk of placing transvenous leads outweighs the benefits from dual-chamber pacing. This is the case in hemodialysis patients who are expected to have low pacing burden, for whom vascular access issues can have a significant impact on survival; in frail patients who may not heal from pacemaker pocket surgery; in patients at high risk for blood stream infections, such as immunocompromised patients; and even in patients with pacemakers and active endocarditis who require system extraction but are pacemaker-dependent [15]. In the future, the issue of asynchronous pacing at risk for pacemaker syndrome (when there is atrial contraction against a closed atrioventricular valve) may be circumvented by an accelerometer-based AV synchronous pacing algorithm, which may expand current indications for leadless pacemakers [16].

Indications for Permanent Pacemakers

Similar to temporary pacemakers, permanent pacemaker implantation is generally indicated in symptomatic bradycardia and when there is risk for unreliable ventricular escape rhythms, i.e., in infranodal block. Unlike temporary pacing, however, permanent pacemakers are only indicated when reversible causes have been discarded and are usually not performed emergently since rhythm stabilization is achieved much faster with temporary pacing. In addition, pacemaker implantation is indicated for resynchronization therapy in patients at risk for pacing-induced

Table 22.2 Summary of indications for permanent pacemakers according to current bradycardia and conduction delay guidelines

Permanent pacing recommendation	COR	LOE
Symptomatic bradycardia due to SND or as a consequence of GDMT when there is no alternative treatment and continued treatment is clinically necessary	I	C
Tachy-brady syndrome with symptoms attributable to bradycardia	IIa	C
Acquired 2° AV block Mobitz II, high-grade AV block, or 3° AV block not attributed to reversible causes, regardless of symptoms	I	B
Permanent AF with symptomatic bradycardia	I	C
Symptomatic AV block as a consequence of GDMT when there is no alternative treatment and continued treatment is clinically necessary	I	C
Asymptomatic adults with congenital complete AV block	IIa	B
Neuromuscular diseases associated with conduction disorder who have 2° AV block, 3° AV block, or prolonged HV interval (70 ms or greater) regardless of symptoms	I	B
Neuromuscular diseases with PR longer than 240 ms, QRS longer than 120 ms, or fascicular block	IIa	C
Lamin A/C mutation with PR longer than 240 ms and LBBB	IIa	B
Anderson-Fabry disease with QRS longer than 110 ms	IIb	C
Significant 1° AV block or 2° AV block Mobitz I with symptoms clearly attributable to the AV block	IIa	C
Syncope with bundle branch block and HV 7 0 ms or greater or infranodal block	I	C
Alternating bundle branch block	I	C
Cardiac resynchronization therapy in heart failure with cardiomyopathy with EF 36–50% and LBBB with QRS 150 ms or greater	IIb	C
New LBBB after transcatheter aortic valve replacement	IIb	B

Based on data in Kusumoto et al. [14]

ACC American College of Cardiology, *AHA* American Heart Association, *AF* atrial fibrillation, *AV* atrioventricular, *CABG* coronary artery bypass grafting, *COR* class of recommendation, *EF* ejection fraction, *GDMT* guideline-directed medical therapy, *HRS* Heart Rhythm Society, *LOE* level of evidence, *LBBB* left bundle branch block, *MI* myocardial infarction, *SND* sinus node dysfunction

cardiomyopathy or in patients with cardiomyopathy and conduction disease. Finally, there are special conditions with high risk for bradyarrhythmias for whom pacemaker implantation is also indicated but are not contemplated in the more traditional recommendations. Current indications for permanent pacemaker implantation, as described in the 2018 guideline on evaluation and management of patients with bradycardia and cardiac conduction delay, are summarized in Table 22.2 and will be discussed below [14].

Sinus Node Dysfunction

In patients who exhibit symptoms that correlate with bradycardia due to sinus node dysfunction, pacemaker implantation is indicated to improve symptoms (COR I). This includes patients with symptomatic sinus bradycardia, chronotropic incompetence, symptomatic sinus pauses greater than 3 s while awake, and those with bradycardia due to necessary medical therapy, such as beta-blockers for cardiomyopathy treatment. In addition, pacemaker implantation is also recommended in patients with tachycardia-bradycardia syndrome with symptoms attributable to bradycardia

(COR IIa). Although these patients only need atrial pacing to treat their symptoms, the guidelines acknowledge the possibility of both single-chamber or dual-chamber pacemaker implantation even in the presence of intact atrioventricular conduction and no conduction abnormalities (COR I).

Invasive assessment of sinus node function is not performed routinely because pacing indications for sinus node dysfunction are typically evident clinically. In very specific circumstances, invasive evaluation of sinus node function can be performed to help guide treatment decision (Fig. 22.8); however, an isolated finding of long sinus node recovery time in an asymptomatic patient has no clinical importance and is not by itself an indication for pacing. Significant sinus bradycardia and asystole can also be observed as part of neurocardiogenic syncope, a clinical entity in which patients can have a mixed vasodepressive and cardioinhibitory exacerbated response that leads to transient loss of consciousness. Many of these patients may continue to have syncope despite pacemaker implantation due to the vasodepressive response leading to transient hypotension. This scenario is not explicitly addressed in the most current guideline document for pacing, but the 2008 version would consider permanent pacemaker implantation in highly symptomatic patients with neurocardiogenic syncope associated with bradycardia documented spontaneously or with tilt-table testing (COR IIb) [17].

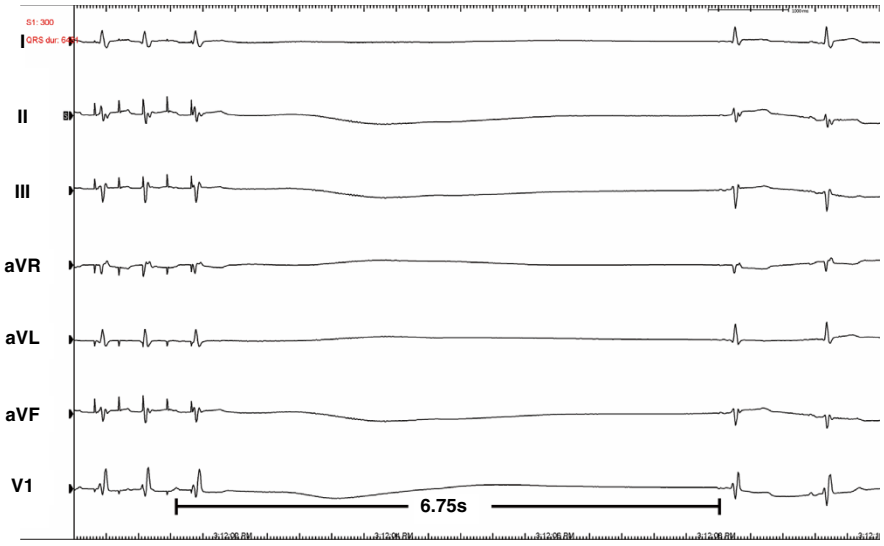

Fig. 22.8 Sinus node recovery time of 6.75 s, defined as the time elapsed until the first sinus depolarization following burst atrial pacing. The longer the recovery time, the more evidence of sinus node dysfunction. This patient developed sinus arrest following cardioversion while receiving intravenous amiodarone, beta-blockers and propofol, but when he spontaneously converted from atrial fibrillation to sinus rhythm his post-conversion pause less than 3 s. This finding helped indicate pacemaker instead of attributing sinus arrest to the medications being administered at the time of cardioversion

Atrioventricular Block

In patients with 2° AV block Mobitz II, high-grade AV block, or 3° AV block without reversible causes, pacemaker implantation is recommended regardless of symptoms (COR I). This is justified, given the risk for pauses without reliable escape in infranodal conduction block, which poses challenges when patients have 2:1 AV block. Clues for infranodal block are concomitant infranodal conduction disease and shorter PR intervals, but infranodal block can also be determined invasively during electrophysiologic study (Fig. 22.9). The same recommendation is true for patients with symptomatic AV block due to necessary medical therapy (COR I) and for patients with permanent atrial fibrillation who have symptomatic bradycardia (COR I). It is also reasonable to implant pacemakers in patients with asymptomatic congenital complete heart block (COR IIa).

Conduction Disorders with 1:1 Conduction

In special groups of patients who are at higher risk of progressive cardiac conduction disease, pacemaker implantation can be indicated even with 1:1 atrioventricular conduction when other signs of conduction disorders are present. This is true for patients with neuromuscular diseases (such as muscular dystrophy and Kearns-Sayre

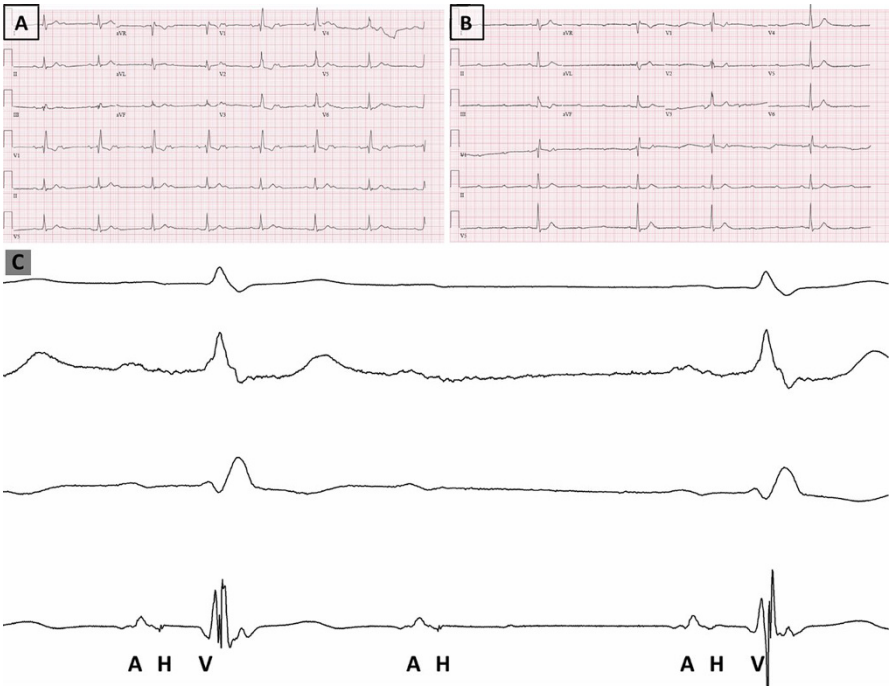

Fig. 22.9 Electrocardiogram showing 2:1 AV block in an asymptomatic patient (**a**), with clues for infranodal block given concomitant infranodal conduction disease (right bundle branch block) and periods of high-grade AV block (**b**). During His-bundle pacemaker implantation, infranodal block was confirmed with His-bundle recording during atrioventricular block (**c**). A atrial electrogram, H His electrogram, V ventricular electrogram

syndrome, a mitochondrial disease associated with heart block) with 2° AV block Mobitz I or HV interval 70 ms or greater without symptoms (COR I) or who have 1° AV block with PR longer than 240 ms, QRS longer than 120 ms, or fascicular block (COR IIa). Lamin A/C mutation, which also can cause progressive cardiac conduction disease, can receive pacemakers if they exhibit PR longer than 240 ms and left bundle branch block (COR IIa). Finally, in patients with Anderson-Fabry disease (an inherited lysosomal disorder that causes myocardial accumulation of glycosphingolipids), pacemaker can be considered when QRS is longer than 110 ms.

In patients without inherited disorders but with ominous signs suggestive of infranodal block, even when not clinically apparent, pacemaker is also considered. This is the case in patients with syncope who have bundle branch block and HV prolongation (70 ms or greater) or infranodal block (COR I), as well as patients who have alternating bundle branch block regardless of symptoms (COR I). In addition, when patients exhibit 1° AV block (or 2° AV block Mobitz I) with symptoms attributable to AV block, pacemaker can be implanted (COR IIa).

Another group contemplated with recommendation for pacemaker implantation despite 1:1 conduction is that of patients who undergo transcatheter aortic valve replacement. New left bundle branch block following these procedures is associated with higher incidence of syncope and complete heart block requiring pacemakers, although their overall mortality was not impacted by this finding [18, 19]. Therefore, pacemaker implantation may be considered if in patients who undergo transcatheter aortic valve replacement who develop new left bundle branch block (COR IIb), even without development of further infranodal conduction disease (Fig. 22.10).

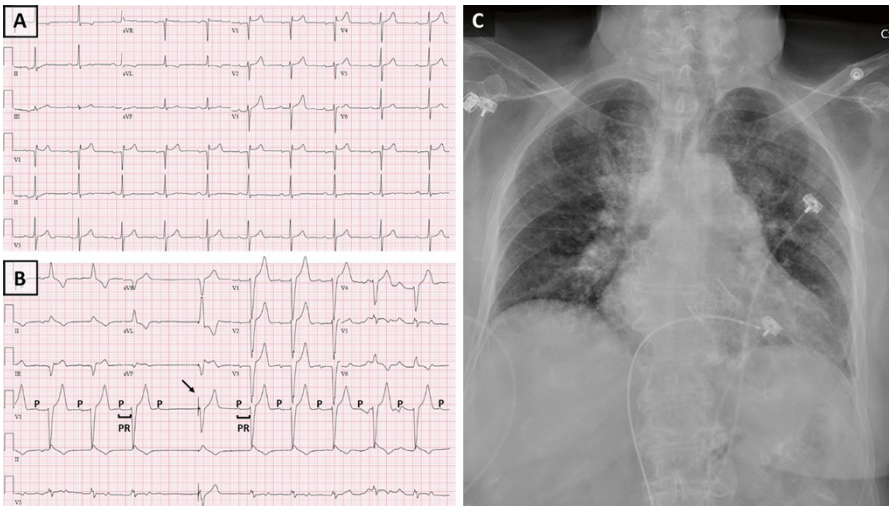

Fig. 22.10 Baseline electrocardiogram from a patient who underwent transcatheter aortic valve replacement showing baseline incomplete RBBB, which increases risk of post-procedural heart block (**a**). Post-procedural electrocardiogram showed new LBBB, so transvenous pacemaker was left in place from the femoral vein for several days. As seen in panel **b**, this patient developed 2° AV block Mobitz II 3 days after TAVR, with clearly no PR prolongation before the blocked P wave and backup pacing (arrow) set at VVI 30. Panel **c** shows the transvenous pacemaker in place from a femoral vein access, as well as a valve-expanding TAVR in situ

Complications and Monitoring

Generally speaking, there are only two types of issues related to pacing itself: underpacing and overpacing. Underpacing can happen due to subthreshold output, such that the pacing impulse fails to capture enough myocardium to cause depolarization, or because of oversensing, when electrical noise leads to pacing inhibition. The former is observed as a pacing spike that fails to generate myocardial depolarization, while the latter is observed as lack of pacing when pacing would be appropriate. Conversely, overpacing would happen when the device is unable to sense an underlying rhythm (observed when pacing happens at a rate above the set rate) or when the device is inadvertently set to pace asynchronously when there is an underlying rhythm.

When transvenous pacing is being employed, loss of capture is usually an indicative of lead dislodgement. This can be temporized by increasing the pacing output until the pacing lead can be readjusted for better pacing threshold. In epicardial temporary pacing, however, loss of capture may represent lead failure, such that a more definitive pacing modality may have to be employed if a patient requires ongoing pacing. Sensing issues, on the other hand, can easily be addressed by manually adjusting the sensitivity or by pacing asynchronously in patients who are dependent. Failure to capture may happen because of inappropriate transcutaneous pad placement leading to poor myocardial pacing vector, as well as electrical current diversion if a patient's skin is wet from sweat, for instance. As discussed previously, documentation of myocardial capture with transcutaneous pacing should be performed by observing a measurable pulse on invasive arterial blood pressure monitor or by arterial pulse palpation. One should never rely on telemetry tracings for myocardial capture since chest wall myopotentials can mistakenly be interpreted as myocardial capture. In permanent pacemakers, loss of capture from lead dislodgment would indicate need for lead revision and is the main reason why some centers observe patients overnight after a new implant.

As an invasive procedure, transvenous pacing carries risks related to both vascular access and intracardiac lead positioning. The preferred vascular access site for temporary pacing is the right internal jugular vein or the left axillary or subclavian vein, which allows the lead to follow the natural curve of the venous system to enter the heart. This is less important for permanent pacemakers, since fluoroscopy will guide lead positioning. Preferential access site relates more to lower complication risk in these patients, so cephalic and axillary veins are usually preferred over subclavian to decrease the risk of pneumothorax. Besides, access site complications include hemothorax and inadvertent arterial puncture, so careful technique for venous access must be employed—ideally under ultrasound guidance in temporary pacing and certainly under ultrasound or fluoroscopy guidance in permanent pacing to decrease the risk of complications.

Complications related to lead positioning include cardiac perforation and arrhythmias. Cardiac perforation should be suspected when a patient develops chest pain with pericarditis symptoms, a sudden change in paced axis from left bundle branch block morphology to right bundle branch morphology (indicating

the pacing site is no longer the right ventricle), development of pericardial effusion with possible tamponade, poor pacing capture, or even diaphragmatic capture. Although highly unlikely, given their profile and flexibility, balloon-tipped catheters may cause perforation if significant torque is applied to the lead, i.e., if the catheter is advanced much more than necessary into the right ventricular cavity. Semi-rigid pacing leads, however, without a balloon at their tip, can increase the risk of perforation if placed against the true right ventricular apex or the right ventricular free wall. Active fixation leads can also cause perforation if secured to myocardium other than the interventricular septum, so usual fluoroscopic clues for septal orientation in left anterior oblique view should also be employed. Cardiac arrhythmias usually happen from catheter-induced ectopy in the right ventricle, typically in the right ventricular outflow tract, so if these are observed, the pacing lead usually needs repositioning.

In patients who are receiving temporary pacing, recognizing when temporary pacing is no longer indicated is as important as knowing when it was indicated. The longer a patient has a temporary pacing lead, the higher the risk of complications described above, as well as blood stream infections and deep venous thrombosis associated with the pacing lead. Therefore, besides daily checks for pacing threshold, pacing morphology, underlying rhythm, and lead position, reassessment for ongoing temporary pacing needs or for definitive pacemaker implantation should also be done.

Finally, in patients who undergo permanent pacemaker implantation, pocket site complications can also happen and should be monitored. The most concerning is pocket infection, which complicates 1–2% of implants and requires device extraction to prevent infective endocarditis [20]. Pocket hematoma, which contributes to the risk of infection, can also happen if the patient is at high risk of bleeding (such as patients with coagulopathy or on uninterrupted anticoagulation), so careful operative technique should be employed to ensure good hemostasis before pocket closure.

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