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Bradycardia and Pacemakers/CRT

CHAPTER OUTLINE

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ABBREVIATIONS

ACC	American College of Cardiology
AF	Atrial fibrillation
AHA	American Heart Association
AV	Atrioventricular
BPEG	British Pacing Electrophysiology Group
bpm	Beats per minute
CHB	Complete heart block
CRT	Cardiac resynchronization therapy
CL	Cycle length
cSNRT	Corrected sinoatrial node recovery time
ECG	Electrocardiogram
EP	Electrophysiologic
FDA	Food and Drug Administration
HCM	Hypertrophic cardiomyopathy
HF	Heart failure
HR	Heart rate
HRS	Heart Rhythm Society
ICD	Implantable cardioverter defibrillator
LAFB	Left anterior fascicular block
LBBB	Left bundle branch block
LPFB	Left posterior fascicular block
LQT1	Long QT syndrome type 1
LV	Left ventricular
LVEF	Left ventricular Ejection fraction
MI	Myocardial infarction
NASPE	North American Society for Pacing and Electrophysiology
NYHA	New York Heart Association
PPM	Permanent pacemakers
RA	Right atrium
RV	Right ventricle
RBBB	Right bundle branch block
SA	Sinoatrial

SACT	Sinoatrial conduction time
SND	Sinus node dysfunction
SNRT	Sinoatrial node recovery time
VT	Ventricular tachycardia

DISORDERS OF IMPULSE GENERATION

Sinus Arrhythmia

- **Definition:** Phasic change in heart rate (HR) due to normal respiration
- **Pathophysiology:** Thought to be due to reflex inhibition of vagal nerve tone during inspiration—leading to *increase in HR during inspiration* and slowing during respiration—thought to help improve and synchronize alveolar gas exchange [1]
 - Normal sinus arrhythmia
 - Most pronounced in the young
 - May be associated with sinus pauses for ≥ 2 s
 - Abolishment of sinus arrhythmia
 - Can be achieved through parasympathetic blockade by atropine
 - Autonomic denervation after cardiac transplant
 - Depression of respiratory sinus arrhythmia after MI is associated with an increased risk of sudden cardiac death [2]
 - Non-respiratory sinus arrhythmia [3]
 - In contrast to sinus arrhythmia, non-respiratory sinus arrhythmia is the change of p-p intervals varying at random
 - May be seen in elderly individuals, or reflect digitalis toxicity, intracranial hemorrhage, or ischemic heart disease
- **Treatment:** respiratory sinus arrhythmia is usually not pathologic, even when associated with sinus pauses. When sinus arrhythmia coexists with symptomatic atrial tachyarrhythmias—as sometimes occurs in the case of young athletes—detaining with resultant de-conditioning may resolve the issue.

Sinus Bradycardia

- **Definition:** Defined as sinus node impulse rate ≤ 60 beats per min (bpm)
- **Pathophysiology:** Sinus bradycardia or sinus pauses rarely cause hemodynamic instability, except when associated with extracardiac disturbance. For example:
 - Increased vagal tone in the context of:
 - Nausea and vomiting
 - Bowel obstruction
 - Urinary retention
 - Intracranial mass
 - **Special case:** *Carotid sinus hypersensitivity*
 - Sometimes considered a variant of vasovagal syncope, occurs more frequently in elderly patients and manifests as profound sinus bradycardia with sinus pauses from pressure on the carotid sinus
 - Dual-chamber pacing indicated for patients in whom *recurrent syncope* is caused by spontaneously occurring carotid sinus stimulation and carotid sinus pressure induces *ventricular asystole* lasting ≥ 3 s present (Class I recommendation; see further details) [4]

- Sinus bradycardia can be exaggerated through parasympathomimetic or sympatholytic effect of drugs, notably:
 - β -blockers and calcium-channel blockers
 - Digoxin
- **Treatment:** Identification of underlying etiology and avoidance of culprit agents is first-line treatment.
 - Minimally symptomatic sinus bradycardia with HR less than 40 bpm while awake is Class IIb indication for PPM [4].

Sinus Node Dysfunction (SND)

- **Definition:** First described as *sick sinus syndrome* by Ferrer in 1968 [5], SND subsumes a constellation of abnormalities of the sinus node and surrounding atrial tissue characterized by sinus arrest, inappropriate sinus bradycardia (in the absence of drugs), and chronotropic incompetence. It is often coupled with the concurrent rise of subsidiary pacemakers leading to coexisting atrial tachyarrhythmias (hence, the term *tachycardia-bradycardia syndrome*).
 - Typically diagnosed in seventh and eight decades of life
 - Median annual incidence of complete AV block is 0.6% with a prevalence of 2.1%, suggestive of concurrent specialized conduction system degeneration [6]
- **Pathophysiology:** Most commonly driven by senescence, SND may occur at any age due to destruction of sinus node cells through infiltration, collagen vascular disease, trauma, ischemia, infection or idiopathic degeneration [7]. Drugs can often exacerbate underlying SND (see Table 16-1).
- Predominant clinical manifestations of SND include:
 - Frequent sinus pauses, sinus arrest, or sinus exit block
 - Inappropriate and severe sinus bradycardia with chronotropic incompetence
 - Episodes of bradycardia alternating with atrial tachyarrhythmias (usually atrial fibrillation (AF), although may be other supraventricular arrhythmias)
 - AF with a slow ventricular response or with very slow recovery after spontaneous conversion or cardioversion to sinus rhythm

B Blockers

Calcium channel blockers (e.g., verapamil, diltiazem)

Sympatholytic antihypertensives (e.g., α -methyldopa, clonidine, guanabenz, reserpine)

Cimetidine

Lithium

Phenothiazines (rarely)

Antihistamines

Antidepressants

Antiarrhythmic agents

May cause sinus node dysfunction (SND) in normal subjects: amiodarone

Frequently worsens *mild* SND: flecainide, propafenone, sotalol

Infrequently worsens *mild* SND: digitalis, quinidine, procainamide, disopyramide, moricizine

Rarely worsens *mild* SND: lidocaine, phenytoin, mexiletine, tocainide

Opioid blockers

TABLE 16-1

CARDIOACTIVE DRUGS THAT MAY INDUCE OR WORSEN SINUS NODE DYSFUNCTION

Adapted from Cardiac Arrhythmia by Podrid and Kowey (Lippincott Williams and Wilkins, Philadelphia 2001) [8]

- **Diagnosis:** Usually made by clinical history of presyncopal symptoms or palpitations and confirmation on electrocardiogram (ECG). Other options include
 - Ambulatory ECG monitoring
 - Exercise testing to evaluate chronotropic competence
 - Electrophysiologic (EP) study may be diagnostic, and there is a class I indication to pursue EP study in patients with symptomatic bradycardia in whom a causal relationship between SND and symptoms has not been established [9]. Criteria evaluated include:
 - *Sinoatrial node recovery time (SNRT):* SA node is overdrive suppressed with atrial pacing, and the time from last paced atrial beat to the first spontaneous sinus beat is measured. Centers differ on normal SNRT, although <1500 ms is conventional. A corrected SNRT (cSNRT) is the SNRT minus the sinus cycle length (CL), and is typically <550 ms
 - *Sinoatrial conduction time (SACT):* is the time required for the sinus impulse to capture the atrium. Typically it is between 50 and 115 ms, and is often prolonged during SA block
- **Treatment:** Largely depends on the diagnosis of symptomatic bradycardia, for which the only effective treatment is permanent cardiac pacing. Guideline recommendations are presented below [4]
 - **Class I indications** for permanent pacing in SND:
 - SND with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - Symptomatic chronotropic incompetence
 - Symptomatic sinus bradycardia from required drug therapy for medical conditions
 - **Class II indications** for permanent pacing in SND:
 - Symptomatic bradycardia with HR <40 bpm, although without documentation of level bradycardia (class IIa)
 - Syncope of unexplained origin with abnormal EP study (class IIa)
 - HR <40 bpm while awake with minimal symptoms (class IIb)

DISORDERS OF IMPULSE PROPAGATION

Disorders of impulse propagation: may occur at any point in the conduction system. Importantly, conduction block is distinct from the normal physiologic phenomenon of *interference*, in which a preceding impulse causes a period of refractoriness due to inactivation of ion channels.

Sinoatrial Exit Block

- **Definition:** also called SA exit block, it manifests as sinus arrest of variable length on surface ECG. Prevalence is 1% in otherwise normal subjects [10].
- **Pathophysiology:** defect of impulse propagation within the SA node
 - First-degree SA exit block cannot be detected on surface ECG because sinus node depolarization is not inscribed separately from atrial depolarization (i.e., the *p* wave)
 - Second-degree SA exit block
 - Type 1: progressive prolongation of conduction block within the sinus node until complete exit block occurs (surface ECG demonstrates progressive shortening of *p–p* intervals before block)
 - Type 2: spontaneous block of sinus impulse leading to sinus pause which is an *exact multiple* of the preceding *p–p* interval

- Third degree SA exit block: simply manifests as sinus arrest, usually with eventual appearance of subsidiary pacemaker (i.e., junctional escape rhythm)

■ **Treatment:** Sinoatrial exit block is usually treated in the context of SND, as indicated above

Atrioventricular (AV) Block

■ **Definition:** By convention, first-degree ‘block’ refers to impulses that are delayed, second-degree block refers to intermittent block of impulse conduction, and third-degree to complete block. Further specific terminology is described below.

- First-degree AV block is defined as PR interval >0.20 s; generally due to block at the level of the AV node, although when associated with bundle branch block, may occur more distal in the His-Purkinje system. Prevalence is 0.65% in healthy adults [11]. Largely benign by itself, recent data from the Framingham cohort suggest that PR prolongation may be associated with increased risks for AF, pacemaker implantation, and all-cause mortality over time [12]
- Second-degree AV block was first classified into two types by Mobitz in 1924

■ **Mobitz Type 1 (Wenkebach) AV block:** characterized by progressive prolongation of the PR interval before non-conduction. Also generally associated with block at the level of the AV node.

- Progressive shortening of $R-R$ intervals prior to a dropped beat; shorter PR interval immediately after dropped beat
- Irrespective of QRS width, usually represents an appropriate physiologic response to increasing HR through decremental conduction in the AV node

■ **Mobitz Type 2:** characterized by sudden non-conduction of atrial impulse without change in preceding PR interval. Usually represents infranodal disease and as such is accompanied by wider QRS compared to Mobitz I.

- Care should be taken to differentiate Mobitz II from a premature atrial complex (examine preceding $p-p$ intervals) which causes physiologic interference and not conduction block
- Some authors refer to multiple consecutive non-conducted impulses as ‘high-degree’ or ‘advanced’ heart block prior to true third-degree AV block
- In the setting of AF, a prolonged pause ≥ 5 s is suggestive of underlying advanced second-degree AV block

■ **2:1 AV block:** characterized by sudden non-conduction of atrial impulse without change in preceding PR interval after a single QRS complex. Based on surface ECG, it is not possible to discern whether the location of 2:1 block is within the AV node or below the level of the node (i.e., infranodal). In patients with 2:1 AV block, evaluation of contemporaneous conduction disturbances (e.g., Wenkebach-type Mobitz I) is used to help infer level of block (see Fig. 16-1)

- Third-degree AV block, or complete heart block, occurs with absence of atrial impulse propagation to the ventricles and may manifest as ventricular standstill in the absence of an escape rhythm. When reversible etiologies are present (e.g., electrolyte disturbance, non-anterior ischemia, Lyme disease), temporary pacing is usually indicated

■ **Pathophysiology:** There are numerous potential etiologies for AV block.

- Physiologic AV block (first-degree or second-degree Type 1) is commonly due to enhanced vagal tone.
- Idiopathic fibrosclerosis of the conduction system (i.e., Lev’s disease affecting the old and Lenegre’s affecting the young),



FIGURE 16-1

Ladder diagram of 2:1 AV block and Wenckebach type block. The rhythm strip shows a 2:1 block followed by short-stretch of Wenckebach and followed again with 2:1 block. The location of the block is inferred to be in the AV node due to the presence of Wenckebach, although cannot be determined conclusively without further information

- Infiltrative cardiomyopathy such as amyloidosis or sarcoidosis.
- Peri-AV nodal inflammation
 - Lyme disease
 - Myocarditis
 - Systemic lupus erythematosus
 - Dermatomyositis
- Endocrinologic states
 - Thyroid storm or myxedema
- Severe electrolyte disturbance
 - Hyperkalemia
- Drug toxicity or overdose, particularly when agents are added in combination or if either renal or liver insufficiency occurs, which leads to accumulation of the drugs.
 - β blockers
 - Calcium channel blockers
 - Amiodarone
 - Digoxin
- Iatrogenic etiologies of AV block are becoming increasingly common
 - Surgical or transcatheter aortic valve replacement
 - Alcohol septal ablation for hypertrophic cardiomyopathy
 - Transcatheter closure of ventricular septal defects
 - Complication of ablation during EP procedures.
- Congenital etiologies are other rare but predictable causes of AV block:
 - Familial AV conduction block
 - Sequela of neonatal lupus syndrome (particularly in babies born of mothers that are positive for antinuclear antibodies SSA/Ro and SSB/La)
 - Hereditary neuromuscular diseases such as myotonic dystrophy

Recommendations in acquired atrioventricular block in adults

Third-degree and advanced second-degree AV block at any anatomic level associated with bradycardia and symptoms (including heart failure) or ventricular arrhythmias presumed to be due to AV block
Third-degree and advanced second-degree AV block at any anatomic level associated with arrhythmias and other medical conditions that require drug therapy that results in symptomatic bradycardia
Third-degree and advanced second-degree AV block at any anatomic level in awake, symptom-free patients in sinus rhythm, with documented periods of asystole ≥ 3.0 s or any escape rate less than 40 bpm, or with an escape rhythm that is below the AV node
Third-degree and advanced second-degree AV block at any anatomic level in awake, symptom-free patients with AF and bradycardia with 1 or more pauses of at least 5 s or longer
Third-degree and advanced second-degree AV block at any anatomic level after catheter ablation of the AV junction
Third-degree and advanced second-degree AV block at any anatomic level with postoperative AV block that is not expected to resolve after cardiac surgery
Third-degree and advanced second-degree AV block at any anatomic level associated with neuromuscular diseases with AV block, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy, with or without symptoms
Second-degree AV block with associated symptomatic bradycardia regardless of type or site of block
Asymptomatic persistent third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster if cardiomegaly or LV dysfunction is present or if the site of block is below the AV node
Second- or third-degree AV block during exercise in the absence of myocardial ischemia

Recommendations in chronic bifascicular block

Advanced second-degree AV block or intermittent third-degree AV block
Type II second-degree AV block
Alternating bundle-branch block

SND sinus node dysfunction, *AV* atrioventricular, *SVT* supraventricular tachycardia, *VT* ventricular tachycardia, *MI* myocardial infarction

Adapted from the "2008 ACC/AHA/HRS Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities." JACC 2008; 51: e1-e62 (4)

TABLE 16-2

ACC/AHA/HRS CLASS I
RECOMMENDATIONS FOR
PERMANENT PACING

- Myocardial ischemia is an important cause of AV and infranodal block
 - Many forms of AV block commonly seen in acute inferior MI, most often due to increased vagal tone, rarely due to AV nodal infarction
 - AV block and infranodal block due to acute anterior wall MI most often due to infarction of the conduction system
- **Treatment:** the initial course of treatment is to identify and remove any potential reversible offending agents. The decision for permanent pacing is often left to the discretion of the cardiologist, based on an appreciation of the relative stability of the underlying rhythm and the risk associated with developing symptoms. Class I indications are as outlined in Table 16-2

Intraventricular Block

- **Definition:** Failure in normal ventricular activation due to block in the His-Purkinje system.
 - The left and right bundle branches are commonly divided into a trifascicular system, consisting of the right bundle branch and the left anterior and posterior fascicles [13].

Although a septal fascicle has also been identified in anatomic studies, ECG manifestations of septal conduction block are debated and remain to be defined [14].

- Beyond commonly recognized right and left bundle branch blocks (RBBB and LBBB), other commonly used terminology for intraventricular block include:
 - **Bifascicular block:** Block is present when either the left anterior or left posterior fascicular block (LAFB or LPFB, but not both) is associated with RBBB
 - Most often precedes third-degree AV block, although rate of progression is variable and often slow [15]
 - **Trifascicular block:** Evidence of disease of all three fascicles present on success ECG tracings.
 - Typically manifests as *alternating BBB*. For example, RBBB + LAFB may be seen to alternate with RBBB + LPFB
 - Care should be made to contrast true trifascicular block from patients who demonstrate first-degree AV block in association with bifascicular block. This does *not* constitute evidence of trifascicular disease. When symptomatic, however, bifascicular AV block and *advanced AV block* (second-degree AV block with multiple non-conducted beats) is associated with increased mortality
- **Pathophysiology:** Intraventricular block may be due to a broad array of etiologies, similar to what was described above as causes of atrioventricular block. The special case of intraventricular block in the setting of MI deserves special mention
 - **Inferior MI:** usually associated with varying degrees of AV block from AV nodal artery ischemia or enhanced vagal tone from exaggeration of the Bezold-Jarisch reflex, *infranodal block is uncommon*
 - **Anterior MI:** can be associated with ischemia of the fascicles directly leading to true intraventricular block. In the pre-thrombolytic era, new fascicular or bundle branch blocks were common after an MI and were associated with a significantly increased risk of mortality [16]. Development of new bifascicular block after anterior MI is a Class I indication for pacing.
 - A simple scoring model characterizing the risk of progression to complete heart block after MI was developed by Lamas based on ECG criteria [17].
 - The complete heart block (CHB) scoring model assigned one point to the presence of well-recognized conduction disturbances, including: first-degree AV block, second-degree block (both type I and type II), LAFB and LPFB, RBBB, or LBBB. The risk of CHB after MI was linearly correlated with the total score (or simply, the sum of number of conduction disturbances) found on their presenting ECG. For example, a patient who's ECG after MI demonstrated 1st degree AV block and right bundle branch block would have a score of 2.
 - According to Lamas' study, patients with a CHB risk score of 0 had a 1.2% chance of developing complete heart block. CHB score of 1 was associated with 7.8% risk, 2 with 25% risk, and a CHB score of 3 or higher was associated 36% risk.
- **Treatment:** Permanent pacing is considered first-line in the treatment of bifascicular block with evidence for concurrent advanced AV node block or intermittent trifascicular block (see Table 16-2).
 - Treatment of intraventricular block in the setting of symptomatic left ventricular dysfunction is a special case which will be discussed further below in the cardiac resynchronization therapy section
 - Because of the relatively common incidence of bradycardia after MI (as indicated above based on the CHB score model), the American College of Cardiology (ACC)/ American Heart Association (AHA) have clear guidelines on intervention, including the use of temporary pacing, for AV and intraventricular disturbance post MI (see Table 16-3)

Application of transcutaneous patches and standby transcutaneous pacing

TABLE 16-3

Class I

Normal AV conduction *or* first-degree AV block *or* Mobitz type I second-degree AV block with new bundle branch block

Normal AV conduction *or* first-degree AV block *or* Mobitz type I second-degree AV block with fascicular block + RBBB

First-degree AV block with old or new fascicular block (LAFB or LPFB) *in anterior MI only*

First-degree AV block *or* Mobitz type I *or* type II second-degree AV block with old bundle branch block

Mobitz type I *or* type II second-degree AV block with normal intraventricular conduction

Mobitz type I *or* type II second-degree AV block with old or new fascicular block (LAFB or LPFB)

Class IIa

First-degree AV block with old or new fascicular block (LAFB or LPFB) *in non-anterior MI only*

Class IIb

Alternating left and right bundle branch block

Normal AV conduction with old bundle branch block

Normal AV conduction with new fascicular block (LAFB or LPFB)

First-degree AV block with normal intraventricular conduction

Mobitz type II second-degree AV block with new bundle branch block

Mobitz type II second-degree AV block with fascicular block + RBBB

Class III

Normal AV conduction with normal intraventricular conduction

Temporary transvenous pacing

Class I

Alternating left and right bundle branch block

Mobitz type II second-degree AV block with new bundle branch block

Mobitz type II second-degree AV block with fascicular block + RBBB

Class IIa

First-degree AV block *or* Mobitz type I second-degree AV block with new bundle branch block

First-degree AV block *or* Mobitz type I second-degree AV block with fascicular block + RBBB

Mobitz type II second-degree AV block with old bundle branch block

Mobitz type II second-degree AV block with normal intraventricular conduction

Mobitz type II second-degree AV block with old or new fascicular block (LAFB or LPFB) *in anterior MI only*

Class IIb

Normal AV conduction with new bundle branch block

Normal AV conduction with fascicular block + RBBB

Mobitz type I *or* type II second-degree AV block with old bundle branch block

Mobitz type II second-degree AV block with old or new fascicular block (LAFB or LPFB) *in nonanterior MI only*

Class III

Normal AV conduction *or* first-degree AV block *or* Mobitz type I second-degree AV block with normal intraventricular conduction

Normal AV conduction *or* first-degree AV block *or* Mobitz type I second-degree AV block with old or new fascicular block (LAFB or LPFB)

Normal AV conduction with old bundle branch block

ACC/AHA GUIDELINES FOR TREATMENT OF ATRIOVENTRICULAR AND INTRAVENTRICULAR CONDUCTION DISTURBANCES DURING STEMI^A AND ACC/AHA/HRS RECOMMENDATIONS FOR POST-MI PERMANENT PACING

TABLE 16-3*Class I recommendations for permanent pacing after the acute phase of myocardial infarction*

(CONTINUED)

 Persistent second-degree AV block in the His-Purkinje system with alternating bundle-branch block or third-degree AV block within or below the His-Purkinje system after ST-segment elevation MI

 Transient advanced second- or third-degree infranodal AV block and associated bundle-branch block. If the site of block is uncertain, an electrophysiological study may be necessary

 Persistent and symptomatic second- or third-degree AV block

Adapted from the "2004 ACC/AHA Myocardial Infarction." *Circulation* 2004; 110: e82-e293 [18], and the "2008 ACC/AHA/HRS Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities." *JACC* 2008; 51: e1-e62 (4)

AV, atrioventricular; BBB, bundle branch block; BP, blood pressure; LAFB, left anterior fascicular block; LBBB, left bundle branch block; LPFB, left posterior fascicular block; MI, myocardial infarction; RBBB, right bundle branch block

^aExcept where specified, all indications include anterior *and* nonanterior MI

PERMANENT PACING

PPM utilize placement of pacing electrodes within (or to the epicardial surface of) the heart attached to a pulse generator.

- Modern options in PPM selection include single-chamber atrial pacemaker (rarely used), single-chamber ventricular pacemaker, and dual-chamber pacemakers.
- Selection of device is driven by indication for pacing (usually SND or AV block) and whether or not there is a desire (or substrate) for rate responsiveness.
- As devices have become more sophisticated, the nomenclature used to define PPM functionality has been updated. A brief review is provided below

Coding/Nomenclature

- **Background:** The first coding system for PPM was proposed in 1974 [19] and was jointly updated by the North American Society for Pacing and Electrophysiology (NASPE) and British Pacing Electrophysiology Group (BPEG) in 2002 [20].
- The combined NASPE/BPEG generic code, or NBG code, outlines five distinct positions to describe PPM activity (see Fig. 16-2).
- **Commonly used codes:**
 - **VVI or VVIR:** also called “ventricular demand pacing”—this code is used in single-chamber ventricular lead devices in which the ventricle is paced, sensed, and inhibited in response to a sensed beat. It is commonly employed in patients with chronic atrial fibrillation and slow ventricular response. Two important caveats:
 - AV synchrony is not maintained in VVIR mode, and chronic right ventricular pacing is associated with an increased risk of heart failure (HF) hospitalization and AF due to increased ventricular dyssynchrony [21, 22]
 - In addition, some patients with chronic VVI pacing develop the pacemaker syndrome. Similar to what is seen in complete heart block, patients manifest a reduction in stroke volume, and may also demonstrate atrial contraction against a closed tricuspid or mitral valve. Reported symptoms include weakness, lightheadedness, a sensation of throat fullness, palpitations, near syncope and syncope
 - **DDD or DDDR:** represents dual-chamber pacing which is the most “physiologic.” Requiring the use of an atrial and ventricular lead, the PPM is typically programmed

The Revised NASPE/BPEG Generic Code for Antibradycardia Pacing					
Position:	I	II	III	IV	V
Category:	Chamber(s) Paced	Chamber(s) Sensed	Response to Sensing	Rate Modulation	Multisite Pacing
	O = None A = Atrium V = Ventricle D = Dual (A + V)	O = None A = Atrium V = Ventricle D = Dual (A + V)	O = None T = Triggered I = Inhibited D = Dual (T + I)	O = None R = Rate modulation	O = None A = Atrium V = Ventricle D = Dual (A + V)
Manufacturers' designation only:	S = Single (A or V)	S = Single (A or V)			

FIGURE 16-2

The revised NBG coding system. From: "The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group." *Pacing Clin Electrophysiol* 2002; 25: 260-64 (19)

to maximize appropriately timed and intact native A-V conduction (i.e., through self-inhibition), add ventricular paced beats in the presence of significant AV delay or block (after allowing for native atrial depolarization), or synchronously add paced atrial and ventricular beats (in the setting of SND or asystole)

- **VOO or DOO**: are commonly employed "asynchronous" pacing modes in which the device paces without respect to native conduction. These modes are usually only employed for limited periods (e.g., surgeries, emergencies) in which there is high possibility for errors in sensing

■ Special terminology:

- **Rate-modulation**: also referred to as 'rate responsiveness' or 'rate adaptation,' rate modulation is a programmable device feature in which the pacing rate varies dependent upon patient activity, as detected by device sensors
- **Hysteresis**: also called AV-search hysteresis, this is a feature available in dual-chamber devices in the DDD mode in which the pacemaker will periodically lower its pacing rates in order to allow for potential intrinsic activity below the programmed lower rate (or sensor rate). It is often misinterpreted for oversensing with pauses.

Additional Indications

- The most common indications for pacemaker device therapy is SND or AV block.
- There has also been active research regarding specific indications for pacing beyond conventional SND or AV block. Some of these are briefly outlined below (see Table 16-4)

TABLE 16-4

ACC/AHA/HRS RECOMMENDATIONS FOR PERMANENT PACING IN SPECIFIC CONDITIONS

<i>Recommendations for pacing in hypersensitive carotid sinus syndrome and neurocardiogenic syncope</i>
Recurrent syncope caused by spontaneously occurring carotid sinus stimulation and carotid sinus pressure that induces ventricular asystole of ≥ 3 s (Class I)
Reasonable for syncope without clear, provocative events and with a hypersensitive <i>cardioinhibitory</i> response of ≥ 3 s (Class IIa)
May be considered for significantly symptomatic neurocardiogenic syncope associated with bradycardia documented spontaneously or at the time of tilt-table testing (Class IIb)
<i>Recommendations for pacing after cardiac transplantation</i>
Persistent inappropriate or symptomatic bradycardia not expected to resolve and for other Class I indications for permanent pacing (Class I)
May be considered when relative bradycardia is prolonged or recurrent, which limits rehabilitation or discharge after postoperative recovery from cardiac transplantation (Class IIb)
<i>Recommendations for pacing in neuromuscular diseases</i>
Permanent pacemaker implantation may be considered in the setting of neuromuscular diseases such as myotonic muscular dystrophy, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy with bifascicular block or any fascicular block, with and without symptoms (Class IIb)
<i>Recommendations for pacing to prevent tachycardia</i>
Sustained pause-dependent VT, with or without QT prolongation (Class I)
Reasonable in high-risk patients with the congenital long-QT syndrome (Class IIa)
May be considered for prevention of symptomatic, drug-refractory, recurrent AF in patients with coexisting SND (Class IIb)
<i>Recommendations for pacing in patients with hypertrophic cardiomyopathy (HCM)</i>
Indicated for SND or AV block in patients with HCM per usual indications (Class I)
May be considered in medically refractory symptomatic patients with HCM and significant resting or provoked LV outflow tract gradient. When risk factors for SCD are present, consider DDD-ICD placement (Class IIb)
<i>Recommendations for permanent pacing in children, adolescents, and patients with congenital heart disease</i>
Indicated for advanced second- or third-degree AV block associated with symptomatic bradycardia, ventricular dysfunction, or low cardiac output (Class I)
Indicated for SND with correlation of symptoms during age-inappropriate bradycardia (Class I)
Indicated for postoperative advanced second- or third-degree AV block not expected to resolve or that persists at least 7 days after cardiac surgery (Class I)
Indicated for congenital third-degree AV block with a wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction (Class I)
Indicated for congenital third-degree AV block in the infant with a ventricular rate ≤ 55 bpm or with congenital heart disease and a ventricular rate ≤ 70 bpm (Class I)

SND sinus node dysfunction, AV atrioventricular, SVT supraventricular tachycardia, VT ventricular tachycardia, MI myocardial infarction

Adapted from the "2008 ACC/AHA/HRS Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities." JACC 2008; 51: e1-e62 (4)

CARDIAC RESYNCHRONIZATION THERAPY (CRT)

Although pacemakers have been conventionally used in the primary treatment of arrhythmia, pacing for hemodynamic indication has been recognized of increasing importance in patients with heart failure due to systolic left ventricular (LV) dysfunction.

Background

- CRT is the use of a biventricular pacemaker with three electrical leads to coordinate myocardial contraction.

- Two leads are endocardial, placed in the right atrium (RA) and right ventricle (RV), while a third lead is placed in a tributary of the coronary sinus overlying the epicardial surface of the LV.
- CRT exerts its physiological impact via synchronizing ventricular contraction, leading to improved left ventricular filling, reverse remodeling with reduced left ventricular volumes and increased ejection fraction, and reducing functional mitral regurgitation.

Impact

Multiple prospective randomized studies have shown that CRT yields long-term clinical benefits, including improved quality of life, increased exercise capacity, reduced heart failure hospitalization and decreased all-cause mortality [23–28] in patients meeting traditional CRT criteria (New York Heart Association (NYHA) Class II–IV, LV ejection fraction (LVEF) $\leq 35\%$, QRS width ≥ 120 ms) [29, 30]. Note: *HF symptom status should be assessed after medical therapy has been optimized for at least 3 months.*

Patient-Selection

Beyond traditional criteria for CRT, there are subsets of patients who derive particularly substantial benefit

- Female patients
- LBBB morphology
- QRS width ≥ 150 ms
- Patients with history of non-ischemic cardiomyopathy

Recommendations

ACC/AHA/Heart Rhythm Society (HRS) Recommendations

- Class I
 - In patients with NYHA Class II, III or ambulatory Class IV heart failure symptoms, LVEF $\leq 35\%$, LBBB, QRS ≥ 150 ms, and sinus rhythm, CRT with or without implantable cardioverter defibrillator (ICD) is indicated
 - Class II
 - CRT reasonable in similar patients to above with LBBB and QRS 120–149 ms (IIa) or non-LBBB and QRS ≥ 150 ms (IIa)
 - CRT reasonable in similar patients as above in AF if (a) patient requires ventricular pacing and (b) AV node ablation or pharmacologic rate control will allow near 100% biventricular pacing (Class IIa)
 - CRT can be useful in patients with LVEF $\leq 35\%$ undergoing new or replacement device placement with anticipated requirement for significant ($>40\%$) ventricular pacing.
 - CRT may be considered for patients with NYHA Class I symptoms, ischemic etiology of heart failure, LVEF $\leq 30\%$, sinus rhythm, LBBB and QRS ≥ 150 ms (class IIb)
 - CRT may be considered for patients with LVEF $\leq 35\%$, sinus rhythm, non-LBBB, NYHA class III/ambulatory class IV, and QRS 120–149 ms (IIb) or similar non-LBBB with NYHA II symptoms and QRS ≥ 150 ms (IIb)
 - Class III
 - CRT not indicated for asymptomatic patients with reduced LVEF in absence of other indications for pacing
 - CRT not indicated for patients whose functional status and life expectancy are limited by predominantly noncardiac conditions

Biventricular Pacing in Patients with atrioventricular block and Systolic Dysfunction

Based on randomized controlled data (BLOCK-HF), biventricular pacing was superior to conventional right ventricular pacing in patients with atrioventricular block and LV systolic dysfunction (LVEF < 50%) with NYHA I–III HF symptoms [31]. The primary benefit was a reduction in HF related hospitalizations.

QUICK REVIEW

TOPIC	KEY POINTS
Etiology of bradyarrhythmia	Can be broadly classified into (a) failure of impulse generation or (b) impulse propagation
Primary cause of bradyarrhythmias due to failure of impulse generation	Sinus node dysfunction
SND is characterized by:	<ol style="list-style-type: none"> 1. Frequent sinus pauses, sinus arrest, or sinus exit block 2. Inappropriate sinus bradycardia with chronotropic incompetence 3. Episodes of bradycardia alternating with atrial tachyarrhythmias 4. AF with slow ventricular response or very slow recovery after conversion
Treatment of choice for SND:	PPM
Selected indications:	<i>Symptomatic</i> bradycardia <i>Symptomatic</i> chronotropic incompetence
Primary cause of bradyarrhythmias due to failure of impulse propagation	Atrioventricular block
Types of AV block:	<ol style="list-style-type: none"> 1. First-degree AV block (usually supranodal) 2. Second-degree AV block <ol style="list-style-type: none"> a. Mobitz I/Wenkebach (usually at the level of the node) b. Mobitz II (often infranodal) 3. Third-degree AV block (usually infranodal)
Treatment of choice for AV block:	<i>'Advanced-AV block' or 'advanced second-degree AV block' refers to second-degree AV block with multiple nonconducted beats</i>
Selected indications:	Eliminate offending agent; consider PPM Advanced second- or third-degree AV block with <i>symptomatic</i> bradycardia, ≥ 3 s pauses in NSR, ≥ 5 s pauses in AF, or escape rate <40 bpm
Selected specific situations beyond SND or AV block that require pacing	<ul style="list-style-type: none"> ■ <i>Carotid sinus hypersensitivity</i>: recurrent syncope, documented ≥ 3 s aystolic pauses with regular activity, and predominant cardioinhibitory response ■ <i>After anterior MI</i>: alternating bundle-branch block ■ <i>After cardiac transplantation</i>: persistent inappropriate sinus bradycardia ■ <i>To prevent tachycardia</i>: due to pause-dependent with or without QT prolongation and in high-risk patients with congenital long QT syndrome ■ <i>In hypertrophic cardiomyopathy (HCM)</i>: may be considered in medically refractory symptomatic patients with HCM and significant resting or provoked LV outflow gradient
Indications for CRT	<ul style="list-style-type: none"> ■ LVEF $\leq 35\%$ ■ NYHA Class II, III, ambulatory class IV on optimal medical therapy ■ QRS ≥ 150 ms, LBBB (Class I) ■ QRS 120–149 ms, LBBB or QRS ≥ 150 ms, non-LBBB (Class IIa) ■ LVEF $\leq 35\%$ with anticipated RV pacing >40% or LVEF <50%, NYHA I–III and complete AV block (BLOCK-HF)

QUESTIONS AND ANSWERS

Question 1

A 21-year-old male was found down outside of a college dormitory. An automated external defibrillator placed in the field recommended defibrillation. He converted to sinus bradycardia at 35 beats per minute after one shock. He was subsequently transported to the hospital where therapeutic hypothermia was initiated. On physical exam he was intubated and nonresponsive to painful stimuli. His pupils were dilated, but reactive to light symmetrically. His past medical history is notable for two prior fainting episode in adolescence after diving into a cold pool. His parents deny knowledge of a history of substance abuse. His family history is notable for a brother who died suddenly at the age of 32. On echocardiography, he was found to demonstrate a reduced left ventricular ejection fraction of 25% with normal wall thickness and without evidence of asymmetric left ventricular hypertrophy. No significant valvular abnormalities were noted. ECG on presentation was notable for a QT of 600 ms (QTc 458 ms) and intraventricular conduction delay with QRS width of 130 ms. The patient's resting HR remained in normal sinus, but remained below 45 beats per minute even after return of normothermia and complete neurological recovery.

All of the following statements are true except?

- The patient meets secondary-prevention criteria for an implantable cardioverter-defibrillator due to aborted sudden cardiac death.
- The patient meets criteria for cardiac resynchronization therapy given his low ejection fraction and QRS width >120 ms.
- The patient's family history of sudden cardiac death is a pertinent risk factor.
- The patient will require a dual-chamber chamber pacemaker.
- Medical therapy with beta-blockade should be initiated.

Answer

1. The correct answer is A

This patient demonstrates classic findings of the congenital long QT syndrome. The description of a prior syncopal episode while swimming is characteristic of an Long QT syndrome type 1 (LQT1)-type trigger. LQT1 is the most common form of congenital long QT syndrome, and is due to a loss-of-function mutation in KCNQ1, which encodes IKs. The patient's current presentation is consistent with an aborted sudden-cardiac death event. Polymorphic Ventricular tachycardia (VT) (i.e., torsades de pointes) was the likely inciting ventricular arrhythmia. The mainstays of therapy include beta-blockade, which suppresses the adrenergic surge which leads to polymorphic VT. In addition, there is a role for DDD pacing in some patients who exhibit pause-dependent VT. Although this cannot yet be substantiated in this

patient, he demonstrates an inappropriate degree of bradycardia, which would likely worsen after beta-blockade. If his LVEF does not recover after this event over time, he may eventually become a candidate for CRT, although his heart failure symptom status cannot be established from this event (at least 3 months must progress on optimal medical therapy, including titrating of diuretic therapy to maintain normal volume status).

Question 2

An 84-year-old woman presents to her primary care physician with complaints of near-fainting spells and "lack of pep." Her past medical history is only notable for hypertension, for which she is maintained on propranolol extended release once daily. She is found to demonstrate normal sinus rhythm on her ECG with a heart rate of 65 beats per minute. She reports feeling reasonably well at home while in bed watching TV, but is often light-headed with quick standing or while reaching/bending over to clean. A tilt-table test is performed which reveals cardioinhibitory response and bradycardia. The patient is awake throughout testing, and reports feeling fatigued, but is conversant throughout. When asked to walk on a treadmill, the patient's HR increases to 70 beats per minute and she again asks to stop due to fatigue.

Which of the following statements are true?

- The patient meets criteria for a permanent pacemaker to treat the hypersensitive carotid sinus syndrome.
- The patient meets criteria for a permanent pacemaker due to symptomatic bradycardia.
- The patient meets criteria for a permanent pacemaker due to symptomatic chronotropic incompetence.
- The patient will require a dual-chamber chamber pacemaker.
- Definitive recommendation regarding permanent pacing cannot be made at this point.

Answer

1. The correct answer is E

The patient demonstrates features suggestive of the carotid sinus syndrome (symptoms while stretching/bending) and with a concerning tilt-table test result. With that said, she reports no history of frank syncope and demonstrated only lightheadedness when bradycardic during testing. A history of syncope is mandated in order to meet Class I or II indication for permanent pacing. The patient does demonstrate evidence of chronotropic incompetence on treadmill testing and this is likely the primary cause of her symptoms. With that said, however, her beta-blocker has not yet been discontinued and recommendation regarding pacing cannot be made until all potentially offending agents have been discontinued. In this woman's case, her past medical history does not mandate therapy with nodal blocking agents, and an alternative antihypertensive could be employed.

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