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A. Abbreviation for *ampere*.

A (atrial). A is used in most pacemaker marker channels to represent a paced atrial event. See the figure *NBG code*.

A₁. In electrophysiology, the atrial response to S₁, the drive stimulus. See also *drive cycle*, S₁.

A₂. In electrophysiology, the atrial response to S₂, the first extrastimulus. See also *extrastimulus*, S₂.

A2-A3. In an assessment of sinoatrial conduction, the measured distance from an induced atrial premature depolarization (A2) to the next sinus beat (A3); represents the first return cycle.

A3-A4. In an assessment of sinoatrial conduction, the measured distance from the end of the first return cycle after an induced atrial premature depolarization (A3) to the next sinus beat (A4); represents the second return cycle.

AAD. Abbreviation for *antiarrhythmic drug*.

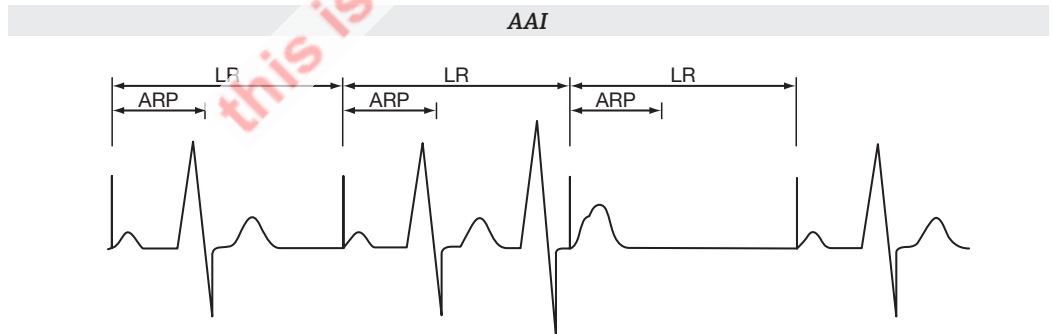
AAI. The NBG code for atrial-inhibited pacing. Pacing and sensing occur in the atrium, and the mode of response to sensed events is inhibited. Portions of the AAI timing cycle that must be considered include the lower rate (LR) and the

“paced” event but may at times be used for an intrinsic atrial event.

AAIR. The NBG code for atrial-inhibited rate-adaptive pacing. Pacing and sensing occur in the atrium, and the mode of response to sensed events is inhibited. The pacemaker also is capable of rate-adaptive pacing, R, via a sensor that monitors some physiologic or nonphysiologic parameter and adjusts the heart rate accordingly. See also *atrial-inhibited rate-adaptive pacing mode*.

AAT. The NBG code for atrial-triggered pacing. Pacing and sensing occur in the atrium, and the mode of response to sensed events is triggered. The atrial output pulse is synchronized with sensed atrial activity and, in the presence of intrinsic activity, does not contribute to atrial depolarization. See also *atrial output pulse*, *atrial-triggered pacing mode*.

A-A timing. A timing scheme used in pacemakers which bases all timing on atrial events, in contrast to ventricular-based timing or timing systems that incorporate a hybrid of atrial and ventricular timing.



ARP, atrial refractory period; LR, lower rate.

atrial refractory period (ARP). AAI pacing also is called atrial demand pacing. See also *atrial-inhibited pacing mode*.

AA interval. The measured distance from atrial event to atrial event. A generally denotes a

AATR. The NBG code for atrial-triggered rate-adaptive pacing. Pacing and sensing occur in the atrium, and the mode of response to sensed events is triggered. The pacemaker also is capable of rate-adaptive pacing via a sensor that monitors

some physiologic or nonphysiologic variable and adjusts the heart rate accordingly. See also *atrial-triggered rate-adaptive pacing mode*.

ABCD. See *Alternans Before Cardioverter Defibrillator*.

aberrancy. Abnormal intraventricular conduction that arises from any of several different mechanisms, such as the Ashman phenomenon, drug toxicity, electrolyte abnormalities, and acceleration-dependent or deceleration-dependent bundle branch block. Fixed bundle branch block is not considered aberrancy. See also *acceleration-dependent aberration*, *Ashman phenomenon*, *bundle branch block*, *deceleration-dependent aberration*.

aberration. See *aberrancy*.

ablate and pace. Terminology for atrioventricular nodal ablation and permanent pacing. Practices vary between placing the pacemaker before the ablation and placing a temporary pacemaker before the ablation and then placing the permanent pacemaker after the ablation.

ablation. The removal or destruction of tissue. Ablation can be used to eliminate the site of origin of tachycardia or to interrupt the circuit through which the arrhythmia travels. Ablation commonly is used to interrupt accessory pathways such as those found in the Wolff-Parkinson-White syndrome. Ablation techniques include cauterization, cryosurgery, diathermy, direct-current ablation, fulguration, laser ablation, microwave ablation, radiofrequency ablation, and surgical ablation.

abnormal automaticity. The formation of a tachycardia by the emergence of an ectopic rhythm at a rate faster than the normal sinus rate. The underlying mechanism is spontaneous phase 4 depolarization rather than reentry or triggered activity. Tachycardias generated by abnormal automaticity are difficult to identify by electrophysiologic testing because programmed electrical stimuli do not consistently initiate or terminate this form of tachycardia. Abnormal automaticity also is known as enhanced automaticity. See also *reentry*.

aborted shock. An instance in an implantable cardioverter-defibrillator during which the device initiates an energy charge but does not deliver a shock, often due to spontaneous termination of the tachyarrhythmia.

absolute refractory period (ARP). Physiologically, the period of time after cellular activation

during which a response cannot be initiated regardless of the strength of the stimulus. The absolute refractory period typically lasts from the onset of an action potential until repolarization is approximately one-third complete. See also *relative refractory period*.

AC. Abbreviation for *alternating current*.

ACC/AHA/NASPE Guideline for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices. A document endorsed by the major cardiology and heart rhythm societies which provides guidelines for when a pacemaker, implantable cardioverter-defibrillator, and cardiac resynchronization therapy are indicated. The document is updated periodically. The most recently published document is the following: Gregoratos G, Abrams J, Epstein AE, Freedman RA, Hayes DL, Hlatky MA, et al. ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *Circulation*. 2002;106:2145-61 and Gregoratos G, Abrams J, Epstein AE, Freedman RA, Hayes DL, Hlatky MA, et al. ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *J Am Coll Cardiol*. 2002;40:1703-19.

accelerated idioventricular rhythm. An abnormal rhythm that arises from a ventricular focus with a rate similar to and competing with the sinoatrial node. Accelerated idioventricular rhythms are thought to be largely due to abnormal automaticity, with some instances of triggered activity. The rhythm has gradual onset and termination and typically has faster than normal ventricular escape rates (30-40 beats per minute) but is slower than ventricular tachycardia. It is generally thought to be benign, because runs are usually hemodynamically well tolerated and brief.

accelerated junctional rhythm. See *nonparoxysmal junctional tachycardia*.

acceleration. A sudden and sustained increase in the rate of a cardiac rhythm and, thus, a de-

crease in the cardiac interval. Antitachycardia pacing used to terminate an arrhythmia may instead cause acceleration. For example, degeneration of ventricular tachycardia into ventricular fibrillation is acceleration.

acceleration-dependent aberration. Abnormal intraventricular conduction that occurs with gradual increases in heart rate, not necessarily associated with the occurrence of a premature impulse. This type of aberration is pathophysiologic and usually has a left bundle branch block configuration.

acceleration-dependent bundle branch block. Intraheisian conduction block in either the right or the left bundle branch that occurs with an increased supraventricular rate (sinus tachycardia, supraventricular tachycardia).

acceleration time. A programmable variable in rate-adaptive pacing which determines how quickly the pacing rate of an activity-sensing device will increase once an increase in the activity level is detected. See also *reaction time*.

accelerometer. A device used to measure acceleration. In some pacing systems, accelerometers have been incorporated as sensors that detect anterior-posterior motion of the patient for rate-adaptive pacing. See also *activity sensor*.

access code. In pacing, an encoded signal that allows access to the pulse generator for communication. Access is permitted only if the key entered by the person programming the device corresponds to a given identification code.

accessory pathway. An aberrant conduction pathway in which an electrical signal bypasses part or all of the normal cardiac conduction system. Conduction through accessory pathways can cause preexcitation of the ventricles. An accessory pathway, such as a Kent fiber, may provide antegrade or retrograde conduction of impulses. These accessory pathways frequently provide the anatomical basis for reentrant arrhythmias. See also *concealed bypass tract*, *Kent fiber*, *Mahaim fiber*, *Wolff-Parkinson-White syndrome*.

accessory pathway antegrade effective refractory period. During atrial pacing, the longest A_1 to A_2 interval at which A_2 does not conduct to the ventricle over the accessory pathway. The A_1 to A_2 interval is measured from a site as close to the accessory pathway as possible. See also *effective refractory period*.

accordion pacing. See *concertina pacing*.

Accufix/Encor leads. See *Accufix J lead*.

Accufix J lead. A specific preformed atrial J lead (Telectronics, Inc., Englewood, Colorado). The lead incorporated a “retention wire” to maintain the J shape. In some patients, the retention wire fractured, an event leading to cardiac perforation or tamponade. Another atrial lead manufactured by Telectronics, Inc., the Encor, had similar problems.

Accufix retention wires. See *Accufix J lead*.

acebutolol. A β_1 -selective, β -adrenergic blocking agent with intrinsic sympathomimetic activity. Acebutolol may increase pacing and sensing thresholds. Because of its β_1 -selectivity, acebutolol may tend to cause less bronchospasm than some other β -adrenergic blocking agents. Because of its intrinsic sympathomimetic activity, acebutolol may tend to cause less fatigue and bradycardia than some other β -adrenergic blocking agents. Acebutolol is metabolized by the liver but has an active metabolite that is excreted by the kidneys. For side effects, see *β -adrenergic blocking drugs*.

ACE inhibitor. Abbreviation for *angiotensin-converting enzyme inhibitor*.

acetylcholine. A neurotransmitter that stimulates muscarinic receptors and causes postganglionic efferent parasympathetic nerve transmission. Acetylcholine also is a preganglionic neurotransmitter that stimulates nicotinic receptors in both the sympathetic and the parasympathetic nervous systems.

acetylcysteine. A mucolytic agent that can be given prophylactically, in addition to hydration, to prevent the reduction in renal function sometimes induced by contrast agents in patients with heart failure. Generic name for Mucomyst.

N-acetylprocainamide (NAPA). The major active metabolite of procainamide. N-Acetylprocainamide has class III activity and is being investigated as an antiarrhythmic drug. It prolongs refractoriness in atrial, ventricular, and accessory pathway tissue. It has efficacy in the treatment of supraventricular and ventricular arrhythmias. Unlike procainamide, NAPA does not seem to cause a lupus syndrome. The plasma half-life of NAPA is 8 hours. It is excreted by the kidneys. Side effects include gastrointestinal and central nervous system effects and proarrhythmia.

acetylstrophanthidin. A cardiac glycoside used experimentally to block the sodium-potassium pump.

acidosis. A pathologic condition caused by the accumulation of acid or loss of base in the body. Acidosis is characterized by increased hydrogen ion concentration in body tissues (decreased pH). Acidosis may alter pacing thresholds. See also *pH sensing*.

aconitine. A toxin that blocks the inward fast sodium current across cell membranes. Aconitine is used experimentally in vitro and in vivo to induce sustained arrhythmias.

acquired AV block. Atrioventricular block that is not congenital. Causes of acquired atrioventricular block are shown in the table below.

action potential is characterized by rapid depolarization followed by repolarization. During depolarization, a reversal of polarity causes the inside of the cell to become positive with respect to the extracellular fluid. During repolarization, the cell returns to its resting potential. Action potentials occur in response to intrinsic or extrinsic stimulation.

action potential duration (APD). The amount of time required for one complete action potential to occur. Action potential duration is measured from the onset of phase 0 to the return to resting membrane potential. Action potential

Acquired Atrioventricular Block: Causes

Idiopathic (senescent) block

Coronary artery disease

Calcific valvular disease

Postoperative or traumatic

Atrioventricular node ablation

Therapeutic radiation to chest

Infectious

- Chagas' disease
- Diphtheria
- Infective endocarditis
- Lyme disease*
- Syphilis
- Toxoplasmosis
- Tuberculosis
- Viral myocarditis (e.g., Epstein-Barr, varicella)

Collagen-vascular

- Ankylosing spondylitis
- Dermatomyositis
- Marfan's syndrome
- Polyarteritis nodosa
- Rheumatoid arthritis
- Scleroderma
- Systemic lupus erythematosus

Infiltrative

- Amyloidosis
- Hemochromatosis
- Malignant disease (lymphomatous or solid tumor)
- Sarcoidosis

Neuromuscular

- Limb-girdle dystrophy
- Myotonic muscular dystrophy
- Peroneal muscular atrophy, Charcot-Marie-Tooth disease
- Progressive external ophthalmoplegia, Kearns-Sayre syndrome
- Scapulothoracic syndrome

Drug effect

- Amiodarone
- β -Blockers
- Calcium blocking agents
- Class IC agents: propafenone, encainide, flecainide
- Digoxin
- Procainamide

* Should not require permanent pacing; temporary pacing only until infection is treated.

acquired long QT syndrome. Prolongation of the QT interval which is not congenital and associated with subsequent susceptibility to ventricular arrhythmias, especially torsades de pointes. Acquired long QT syndrome most frequently is due to antiarrhythmic drugs that prolong repolarization. See also *congenital long QT syndrome, torsades de pointes*.

actin. One of several genes identified as responsible for familial dilated cardiomyopathy.

action potential. A sudden transient change in the electrical potential (voltage) across the cell membrane of muscle or nerve cell tissue. An

duration often is measured as a percentage of full recovery; for example, APD_{90} is equal to the action potential duration measured to 90% recovery of repolarization. In antiarrhythmic therapy, increasing the action potential duration, and thus refractoriness, may control arrhythmias. However, in some disease states, a long action potential may cause prolonged repolarizations and lead to arrhythmias.

activated state. One of the three states of an ion channel. According to the modulated receptor hypothesis, ion channels can exist in three states: activated (A), inactivated (I), resting (R).

In the activated state, a channel in the cell membrane is open and may conduct ions. See also *inactivated state, ion channel, modulated receptor hypothesis, resting state*.

activation front. The leading edge of depolarization propagating through tissue. See also *depolarization*.

activation mapping. A catheter mapping technique used to pinpoint the focus and circuit of a tachycardia and thus guide therapeutic intervention, such as ablation. Activation mapping uses a roving catheter in the atrium or ventricle to identify locations of the earliest signal or a progression of activation for an arrhythmia.

activation recording. Similar to activation mapping, but it also may be done for a nonmapping issue, such as a trigger for a therapy.

activation sequence. The order in which various cardiac structures are depolarized by an impulse. For example, sinus rhythm occurs in a high-to-low atrial activation sequence; that is, impulses normally spread downward from the sinus node, through the atrial myocardium, to the atrioventricular node. Similarly, impulses normally spread downward from the atrioventricular node, through the His bundle, the bundle branches, and the Purkinje fibers to the ventricular myocardium.

activation time. The amount of time required for an impulse to travel from one point to another. Activation time cannot necessarily be translated into conduction velocity because the pathway of activation is unknown and may not be linear.

active electrode. The part of a pacing lead that delivers energy to the heart from the pulse generator; usually the tip electrode of a transvenous pacing lead.

active fixation. Embedment of the distal tip of an endocardial or epicardial pacing lead directly into the myocardium by means of a special fixation device designed to ensure stable electrode placement. See also *active fixation lead*.

active fixation lead. A pacing lead with a screw, barb, prong(s), hook(s), or some other mechanical device affixed to the lead tip. During lead positioning, the device is embedded into the myocardium to ensure stable electrode placement. Active fixation leads may be particularly useful in clinical situations that necessitate atypical lead placement. See also *endocardial screw-in lead, epicardial screw-in lead*.

activities of daily living (ADL) rate. Heart rate target that a patient is expected to reach during moderate exercise, that is, with activities that would occur during the course of a normal day.

activity sensing, accelerometers in. See *accelerometer*.

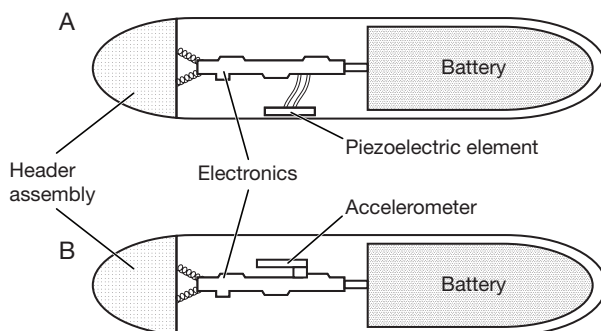
activity sensing, gravitational. A device configuration in which a moving magnetic ball is surrounded by a wire coil in the pacemaker housing. Movement of the ball gives rise to an electrical signal that is interpreted into a pacing rate. Several variations of gravitational sensors have been studied as part of rate-adaptive pacing systems.

activity sensing, magnetic ball. See *activity sensing, gravitational*.

activity sensing, piezoelectric vibration sensors in. See *piezoelectric crystal*.

activity sensing, in rate-adaptive pacing. A nonmetabolic variable for rate control in pacing that has achieved wide clinical acceptance. Activity-sensing pacemakers typically react quickly to the start and end of exercise, although their performance in other areas,

Activity Sensors



Modified from Millerhagen JO, Combs WJ. Activity sensing and accelerometer-based pacemakers. In: Ellenbogen KA, Kay GN, Wilkoff BL, editors. *Clinical Cardiac Pacing and Defibrillation*. 2nd ed. Philadelphia: W.B. Saunders Company; 2000. p. 249-70. Used with permission.