Pacemakers and Implantable Cardioverter Defibrillator

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Abstract: The history of cardiac pacing therapy must be viewed within the broader framework of electro-diagnosis and electro-therapy. Electro-therapy has a simple core concept: the use of an outside source of electricity to stimulate human tissue in various ways to produce a beneficial therapeutic effect. This has shown a prolonged, halting development through the ages, sometimes being looked upon as mysterious magic produced by complex machines. The field of paediatric open heart surgery gave a major impetus to the development of pacemakers since heart block often accompanied impeccably performed intra-cardiac repairs of congenital defects. This review deals various of aspects of pacemaker functioning, indications, contraindications and complications.

Keywords: Pacemakers, Codes, Implantable cardioverter defibrillator, Heart block, Conduction system.

INTRODUCTION

Cardiac implantable electronic device (CIED) has been improving over the past few years and the number of patients using this device has continued to grow. The first pacemaker implant was introduced in 1958, which is asynchronous type and functioned for 3years. First on demand pacemaker (DVI) was introduced in 1964. In 1977 first atrial and ventricular demand pacing (DDD) came into use. Later on rate responsive pacing and resynchronization pacing were introduced but the automatic capture detection was introduced only in 1998. First human implants were done in 1980 by cardiac surgeons under general anesthesia. The devices were large and were placed in abdomen and required thorocotomy with perioperative mortality rate upto 9%. They lasted for 1.5years. Today they evolved as small devices placed in pectoral pocket under local anesthesia. Their battery lasted for 9years with perioperative mortality<1%.

PARTS OF PACEMAKERS

A pacemaker is a device that regulates the heart beat. They provide electrical stimuli to cause cardiac contraction when intrinsic cardiac activity is inappropriately slow or absent. It consists of

1. Generator

A Generator is a device containing a battery that provides the energy for sending electrical impulses to the heart and an electric circuitry that controls pacemaker operations, placed subcutaneously or submuscularly, connected to leads. The battery has life span of 5-8yrs and is made of lithium-iodide type [1, 2].

2. Leads

Leads are electronic wires that Deliver electrical impulses from the pulse generator to the heart and sense cardiac depolarization.

3. Electrode

Consists of Cathode (negative electrode), an electrode that is in contact with the heart tissue and Anode (positive electrode), an electrode that receives the electrical impulse after depolarization of cardiac tissue

DEFINITIONS

Pacing Rate

Frequency at which heart is stimulated in the absence of sensed cardiac activity. Mostly pacing pulse width is 0.5-1.5msec width, voltage around 2.5-5volts.

Pacing Threshold

Minimum energy required to capture the atria or ventricle. It is determined by intrinsic excitability of myocardium, duration of electrical pulse. Factors influencing are - Increase: propranolol, verapamil, antiarrhythmics, potassium, insulin, hypothyroidism &MI. Decrease: exercise, hyperthyroidism, hypoxia, glucocorticoids, sympathetic drugs.

Sensing

Sensing is the ability of the pacemaker to “see” when a natural (intrinsic) depolarization is occurring.

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Pacemakers sense cardiac depolarization by measuring changes in electrical potential of myocardial cells between the anode and cathode.

Accurate Sensing

Accurate sensing enables the pacemaker to determine whether or not the heart has created a beat on its own. Ensures that undersensing and oversensing will not occur. Provides proper timing of the pacing pulse.

Undersensing

Pacemaker fails to sense P or R waves that should have been sensed

Oversensing

Pacemaker mistakes extra-cardiac activity for intrinsic cardiac events and generate an impulse.

A-V Interval

Maximum time interval between atrial and ventricular events. Starts with paced or spontaneous atrial beat and termination with initiation of paced or spontaneous ventricular event, takes 150-200msec usually.

Automatic interval

Time in milliseconds between successive pacemaker impulses

Escape Interval

Time the generator takes to emit an impulse in the absence of R wave.

R-Wave Sensitivity

No: of milivolts required to activate the sensing circuit and inhibit the pacing circuit.

Inhibited Pacemaker

Has a sensing circuit that detects R wave, compares it with standard and finally inactivates the pacing circuit if R-waves meet standard (ie., magnitude >1.5mv, slew rate>0.5v/sec, frequency >70bpm)

Rate Adaptive Pacemaker

Varies its automatic interval with the metabolic state or movement of the patient, it paces at baseline and increases the rate if patient exercises.

Blanking Period

Period after a paced or sensed event during which pacemaker does not respond to any ECG event.

Pacemakers can employ unipolar or bipolar leads. Over the past 15yrs bipolar leads has been in use.

Unipolar Pacemaker

It has only one electrode that contacts the heart at its tip, called the cathode. The anode is the pulse generator. Electromagnetic interference occurs more often in unipolar leads.

Bipolar Pacemaker

In this both anode and cathode are present on the heart. Provides more contact with the endocardium, needs lower current to pace. Less chance for cautery interference.

Table 1: Pacemaker Codes and Nomenculature

I	II	III	IV	V
Chamber Paced	**Chamber Sensed**	**Response to Pacing**	**Rate Modulation, Programmability**	**Anti-Tachycardia Features**
A – Atrium	A – Atrium	T – Triggered	P – Simple	P – Pacing
V – Ventricle	V – Ventricle	I - Inhibited	M–Multi-programmable	S – Simple
D – Dual	D – Dual	D – Dual	R – Rate adaptive	D – Dual
O - None	O - None	O - None	C-Communicating	
			O - None	

PACEMAKER CODES

Most Pacemakers Perform Four Functions:

- Stimulate cardiac depolarization
- Sense intrinsic cardiac function
- Respond to increased metabolic demand by providing rate responsive pacing
- Provide diagnostic information stored by the pacemaker

The North American Society for Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology group (BSEP) has adapted a generic code which has been universally adapted to avoid confusion and improve communication [3].

TYPES OF PACEMAKERS

- Asynchronous / Fixed rate: does not synchronise with intrinsic heart rate. Can be used safely in patients with no inrtrinsic ventricular activity. If patient has ventricular activity it competes with patients own conduction system causing ventricular tachycardia (R –on- T phenomenon) eg: VOO, AOO, DOO.
- Synchronous / Demand: It contains two circuits, one forms impulse, one acts as a sensor. When activated by an R wave, sensing circuit either triggers or inhibits the pacing circuit, called “Triggered” or “Inhibited” pacers. This is most frequently used pacer as it eliminates competition.eg: DDI, VVI/VVT, AAI/AAT.
- Single chamber: The pacing lead is implanted in the atrium *or* ventricle, depending on the chamber to be paced and sensed. It is asynchronous pacing. Single ventricular lead does not provide AV synchrony and single atrial lead does not provide ventricular backup if A-to-V conduction is lost.
- Dual chamber: This has two leads palced one in ventricle and one in atria. It is physiological pacing mode as ventricular systole is preceeded by atrial contraction and the atrial rate is the same as the ventricular rate. It provides atrial kick, optimizes left ventricular filling, AV synchrony and eventually improves cardiac outut. It minimizes AV valve regurgitation that occurs with other modes. Lower incidence of atrial fibrillation, new congestive heart failure, lowers risk of systemic embolism, stroke, mortality and higher survival rates.
- Programmable / nonprogrammable: Recently pacemakers have inbuilt rate modulation or programmability which allows them to increase the heart rate in response to increase physical activity (body movement, excersise) or physiological needs.

INDICATIONS OF PACEMAKERS

AOO: sinus node dysfunction like bradycardia with normal AV conduction.

VOO: significant bradycardia with normal sinus rhythm, chronic AF. It is a temporary mode.

AAI: sick sinus syndrome in the absence of AV node disease or atrial fibrillation.

VVI: symptomatic bradycardia caused by sinus node dysfunction, AV block, chronic atrial arrhythmias {AF}.

DVI: useful in dual chamber pacing when atrial sensing is not required.

DOO: highly potential for serious ventricular arrhythmias.

DDD: AV conduction disorders with normal or abnormal sinus node function. Contraindicated in persistant atrial tachyarrhythmias and silent atria. Patients with LVH and LV dysfunction who need coordination of atrial and ventricular contraction to maintain cardiac output.

VDD: AV block with intact sinus node function (particularly in congenital AV block).

The final recommendations for indications for device therapy are expressed in the standard ACC/AHA format:

Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class II a: Weight of evidence/opinion is in favor of usefulness/efficacy.

Class II b: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

Indications for Permanent Pacing in Acquired Atrioventricular Block in Adults

Class I

- Third-degree AV block at any anatomic level associated with any one of the following conditions:
- Bradycardia with symptoms presumed to be due to AV block.*(Level of evidence: C).*
- Arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia. *(Level of evidence: C).*
- Documented periods of asystole ≥3.0 seconds or any escape rate <40 beats per minute (bpm) in awake, symptom-free patients. *(Level of evidence: B, C).*
- After catheter ablation of the AV junction. *(Level of evidence: B, C)* There are no trials to assess outcome without pacing, and pacing is virtually always planned in this situation unless the operative procedure is AV junction modification.
- Postoperative AV block that is not expected to resolve. *(Level of evidence: C).*
- Neuromuscular diseases with AV block such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy. *(Level of evidence: B).*
- Second-degree AV block regardless of type or site of block, with associated symptomatic bradycardia. *(Level of evidence: B).*

Class II a

- Asymptomatic third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster. *(Level of evidence: B, C)*
- Asymptomatic type II second-degree AV block.*(Level of evidence: B)*
- Asymptomatic type I second-degree AV block at intra- or infra-His levels found incidentally at electrophysiological study for other indications. *(Level of evidence: B)*
- First-degree AV block with symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary AV pacing. *(Level of evidence: B)*

Class II b

- Marked first-degree AV block (>0.30 second) in patients with LV dysfunction and symptoms of congestive heart failure in whom a shorter AV interval results in hemodynamic improvement, presumably by decreasing left atrial filling pressure. *(Level of evidence: C)*

Class III

- Asymptomatic first-degree AV block. *(Level of evidence: B*
- Asymptomatic type I second-degree AV block at the supra-His (AV node) level or not known to be intra- or infra-Hisian.*(Level of evidence: B, C)*
- AV block expected to resolve and unlikely to recur (eg, drug toxicity, Lyme disease). *(Level of evidence: B)*

INDICATIONS FOR PERMANENT PACING AFTER THE ACUTE PHASE OF MYOCARDIAL INFARCTION

Class I

- Persistent second-degree AV block in the His-Purkinje system with bilateral bundle branch block or third-degree AV block within or below the His-Purkinje system after AMI. *(Level of evidence: B)*
- 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.*(Level of evidence: B)*
- Persistent and symptomatic second- or third-degree AV block. *(Level of evidence: C)*

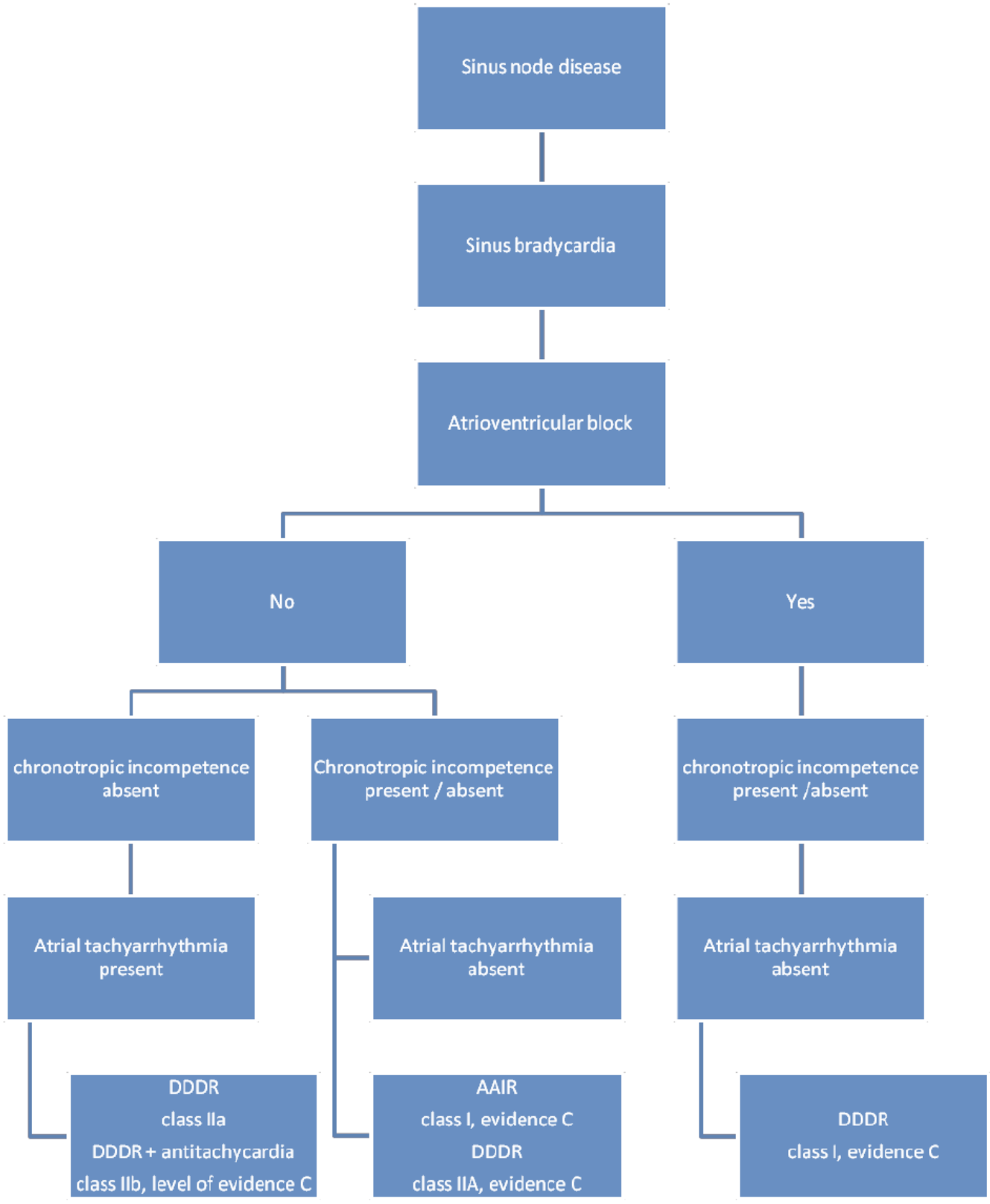

Figure 1: Treatment of Sinus node with Pacemakers.

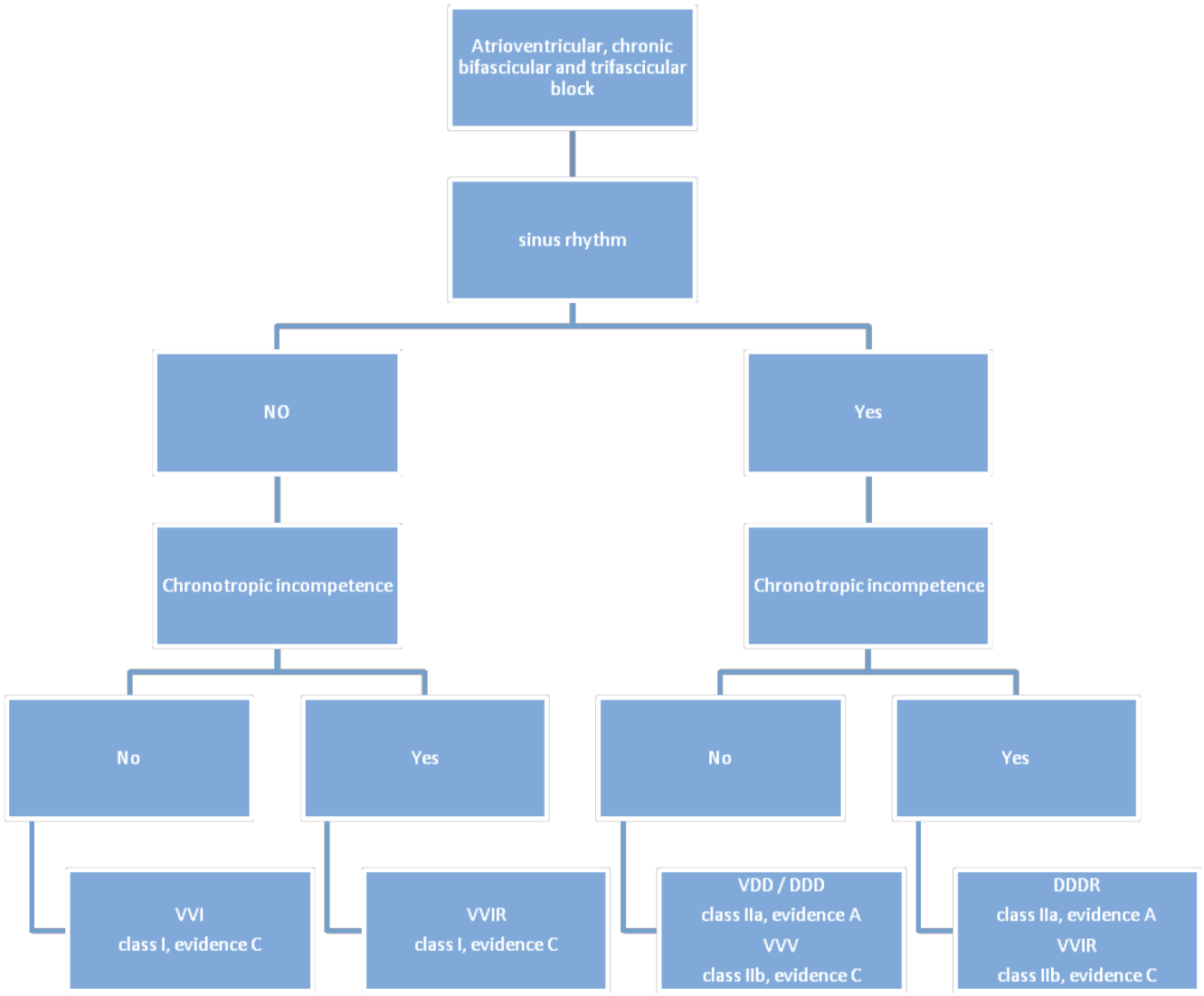

Figure 2: Treatment of heartblock with pacemakers.

Class II a

None.

Class II b

- Persistent second- or third-degree AV block at the AV node level. *(Level of evidence: B)*

Class III

- Transient AV block in the absence of intraventricular conduction defects. *(Level of evidence: B)*
- Transient AV block in the presence of isolated left anterior fascicular block. *(Level of evidence: B)*
- Acquired left anterior fascicular block in the absence of AV block. *(Level of evidence: B)*
- Persistent first-degree AV block in the presence of bundle branch block that is old or age indeterminate. *(Level of evidence: B)*

IMPLANTABLE CARDIOVERTERDEFIBRILATOR (ICD'S)

These are designed to treat cardiac tachydysrythmias. They are capable of detecting a ventricular arrhythmia and delivering a defibrillator shock/cardioversion. They have antitachycardia pacing activity and have pacemaker function also(combo devices). Present day ICD's can terminate 98% of

Table 2: Defibrillation Codes for AICD

I	II	III	IV
Shock Chamber	**Antitachycardia Chamber**	**Tachycardia Detection**	**Antibradycardia Pacing**
A – Atrium	A – Atrium	E – EKG	A – Atrium
V – Ventricle	V – Ventricle	H - Hemodynamics	V – Ventricle
D – Dual	D – Dual		D – Dual
O - None	O - None		O – None

ventricular fibrillation episodes. These devices consists of electronic circuitry, power source, microprocessor, memory, leads and discharges from 1v-750v.

ICD's measure each cardiac R-R interval. IT reconfirms VT/VF and can differentiate VT from SVT. It gives a shock usually at 25 J. it takes 5-20 seconds to sense VT/VF and 5-15 sec to charge and 2.5-10 sec delay before next shock is administered. Delivers totally 5 shocks then pauses. During shock delivery if the patient has to be touched (while doing CPR) rubber gloves have to be worn for insulation.

INDICATIONS

Survivors of cardiac arrest due to VF or hemodynamically unstable VT

Structural heart disease and spontaneous sustained VT

LVEF <35% due to prior MI who are atleast 40dys post MI and DCM with EF<35% who are in NYHA class II or III

Non sustained VT due to prior MI, LVEF<40% and inducible VF or sustained VT at electrophysiological study.

Unexplained syncope, nonischemic DCM

Patients with long QT syndrome

COMPLICATIONS

Pacemaker

Early complications include pneumothorax, subcutaneous emphysema, myocardial perforation, arterial lead placement, brachial plexus injury. Late complications include pulse generator erosion, thromboembolism, lead defects, battery depletion, allergy, twiddler syndrome, failure to capture and sensing abnormalities.

Pacemaker syndrome: symptoms like weakness, lethargy, lassitude, syncope, light headedness, cough, chest pain, dyspnea and signs like orthostatic hypotension, cannon A waves, decreased cardiac output, CHF are seen in patients with pacemaker. These are due to retrograde atrial activation by non-synchronous ventricular pacing modes.

The complication of *ICD's*are same as pacemakers except that there can be inappropriate or ineffective cardioversion/ defibrillation.

Electromagnetic interference (EMI): Any device that emits radiofrequency waves between 0 and 10 Hz cause EMI and interfere with pacemaker/ICD function. Eg: metal detectors, cell phones, MRI, electrocautery, ESWL, electroconvulsive therapy, radiofrequency ablation, shivering, fasciculations and large tidal volumes. High frequency waves (X rays, alpha waves, infrared and ultraviolet waves) are unlikely to interfere with CIED. EMI interference can result in-

Inappropriate inhibition or triggering of a paced output

Asynchronous pacing

Reprogramming into a backup mode (VOO/VVI)

With ICD's inappropriate delivery of defibrillator shock occurs.

These problems have been overcome by modern pacemakers because of the availability of bipolar leads, filters and circuit shields.

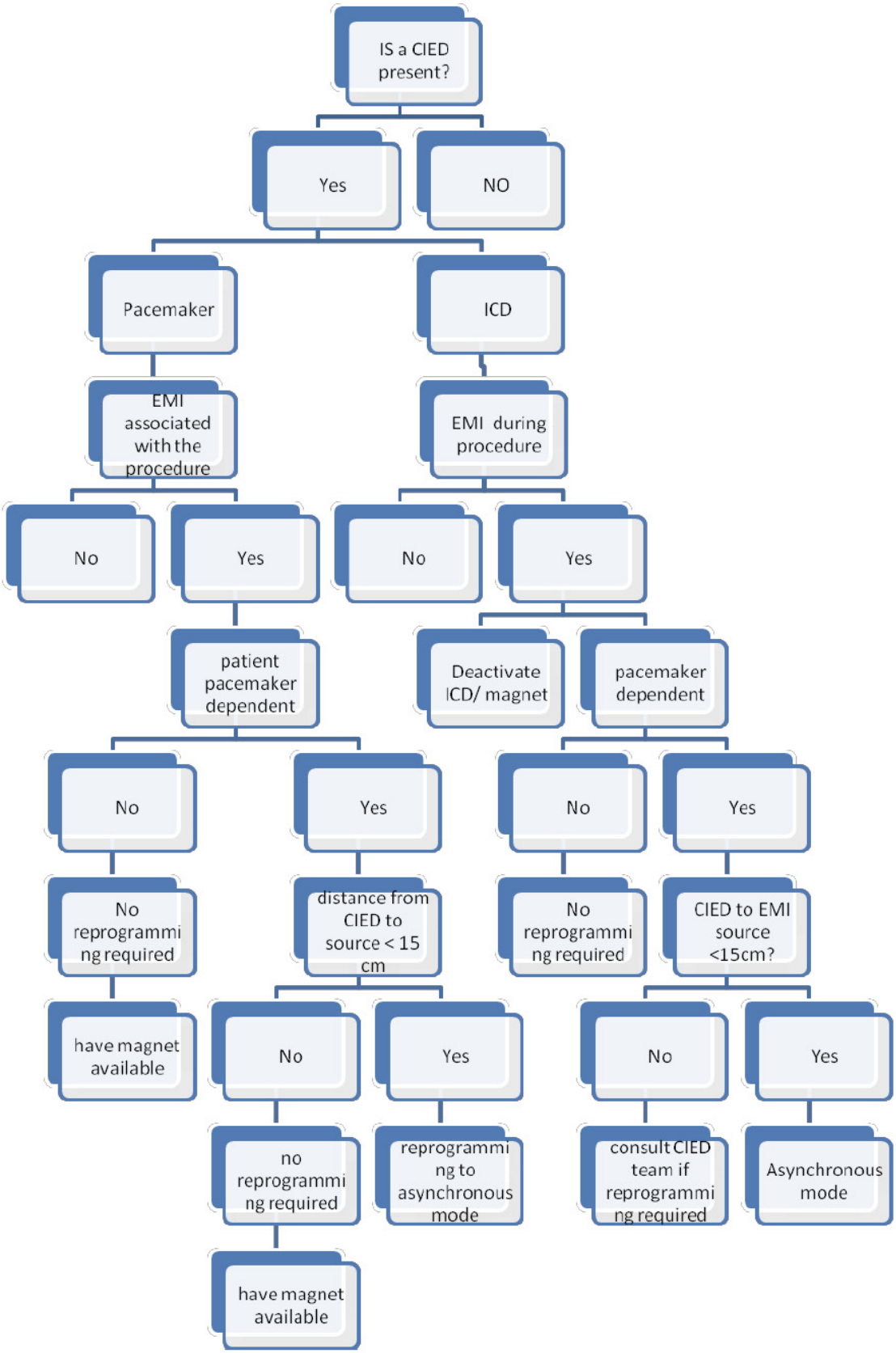

Figure 3: Perioperative management of Pacemakers and AICD.

Table 3: Step Wise Approach to the Perioperative Treatment of the Patient with CIED [4]

Preoperative evaluation	Patients with CIED	Focused history and physical examination
	Determine the type of CIED - pacemaker/ICD	Identify the CIED, manufacture details Chest x-ray, past medical records, cardiology consultation
	Determine whether patient is dependent on CIED	History Bradyarrythmiasymptoms Atrioventricular node ablation No spontaneous ventricular activity
	Determine CIED function	Comprehensive CIED evaluation Determine whether pacing pulses are present and create paced beats
Preoperative preparation	EMI unlikely during the procedure	No special precautions are required
	EMI likely, patient has pacemaker	Reprogramme to asynchronous mode when indicated Suspend rate-adaptive functions
	EMI likely, patient has ICD	Suspend antitachyarrythmia functions If patient is dependent on pacing alter pacing function as above
	EMI likely, all CIED	Use bipolar cautery, ultrasonic scalpel Temporary pacing and external cardioversion-defibrillation should be available
	Intraoperative physiologic changes likely (eg: bradycardia, ischemia)	Plan for possible adverse CIED -patient interaction
Intraoperative management	Monitoring	ECG, SpO2, etc as per ASA standards
	Electrocautery interference	Avoid proximity of cautery tool (CT) to pulse generator(PG)/leads. Short bursts at lowest possible energy should be used. Use bipolar cautery, ultrasonic scalpel.
	Radiofrequency catheter ablation.	Avoid contact of radiofrequency catheter with PG/leads Radiofrequency current path far away from PG/leads Discuss these concerns with the operator
	Lithotripsy	Do not focus lithotripsy beam near PG If R wave triggers lithotripsy, disable atrial pacing
	MRI	Generally contraindicated If required, consult operating physician, cardiologist, radiologist and manufacturer
	Radiation therapy	PG/leads must be outside of RT field Possible surgical relocation of PG Verify PG function during/after RT course
	Electroconvulsive therapy	Consult with ordering physician, patient's cardiologists, CIED service / manufacturer
Emergency defibrillation/cardioversion	ICD: magnet disabled	Terminate all EMI sources Remove magnet to re-enable therapies Observe for appropriate therapies
	ICD: programming disabled	Programming to re-enable therapies or proceed directly with external cardioversion-defibrillation
	ICD: either of the above	Minimize current flow through PG/leads External pads and paddles for cardioversion/defibrillation (PP) to be placed as far as possible from PG PP to be placed perpendicular to major axis PG/leads PP placed in anterior-posterior location
Postoperative management	Immediate postoperative period - regardless of CIED type	Use clinically appropriate cardioversion/defibrillation energy Monitor cardiac rhythm & rate continuously Backup pacing and cardioversion/defibrillation capability
	Postoperative interrogation & restoration of CIED function	Interrogation to assess function Appropriate settings to be done, ICD : early restoration of anti-tachyarrythmia function Use cardiologist/pacemaker-ICD service if needed

CONCLUSION

The prevention and treatment of life-threatening brad arrhythmias have been revolutionized in the last half century by electronic pacemakers. The primary purpose of a pacemaker is to maintain an adequate heart rate, either because the heart's natural pacemaker is not fast enough, or there is a block in the heart's electrical conduction system. Most of the scientists and physicians involved in electro-therapy faced significant criticism and sometimes even derision by the contemporary scientific community. Yet the specialty moved steadily on gaining medical respectability and now helps countless patients all over the world.

REFERENCES

[1] Practice Advisory for the Perioperative Management of Patients with cardiac rhythm management devices: pacemakers and ICD's: a report by the American Task Force on perioperative management of patients. Anesthesiology 2005; 103: 186-198.

[2] Practice Advisory for the Perioperative Management of Patients with Cardiac Electronic Devices: Pacemakers and Implantable Cardioverter Defibrillators. An updated report by the American Society of Anesthesiologist Task Force on Perioperative Management of patients with cardiac Implantable Electronic Devices. Anesthesiology 2011; 114: 47-261

[3] Bernstein AD, Daubert JC, Fletcher RD etal. The revised NASPE / BPEG generic code for antibradycardia, adaptive rate and multi-site pacing. Pacing clinical Electrophysiology 2001; 25: 260-264.

[4] Paul G. Barash, Bruce F. Cullen, Robert K. Stoelting Clinical anesthesia sixth edition, Appendix: Electrophysiology 1586-1587.

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