Cardiac Pacing Update-1.

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The field of cardiac pacing has grown extensively over the last 25 years. Permanent cardiac pacemaker implantation has become the established treatment for management of heart block and bradyarrhythmias. The oldest pacemakers were large, bulky units. Since then both the indications and the technology have changed considerably. This unbridled growth of a highly technological field has led to the use of smaller, microprocessor based, sophisticated, longer lasting and "Smart" pacemakers that can pace either or both chambers of the heart, are multiprogrammable and make available to the clinician data on both the patient and the performance of the device. Patient survival has improved both quantitatively and qualitatively due to establishment of AV synchrony and the greater hemo-dynamic benefit from the newer types of dual chamber pacemakers.

This monograph will focus on prodiding the clinician with clear and concise analysis of the indications, types and practical management of modern cardiac pacemakers. The question the clinician faces today are not only who requires pacing, but what type of pacemakers will be most beneficial to the patient. For the most part, the discussion will be limited to pacemaker use in patients with bradyarrhythmias. Devices for treatment of tachyarrhythmias are becoming more and more available, but they are still investigational and going through an evolution and are beyond the scope of this review.

Indications For Pacing In Bradyarrhythmias

Although clinically there are clear-cut indications for cardic pacing, there are certain areas in which the use of pacemakers remains controversial. A physician, in deciding to implant a permanent pacemaker, has to consider factors other than just statistical numbers. These may include for example, the general condition of the patient and the presence of associated illness and the specific constraints imposed by patient and his or her family.

Indications will be discussed under the following five headings:

1) Indications For Pacing In Sinus Bradycardia

2) Indications For Pacing In Patients With AV Block

3) Indications For Pacing In Chronic Intraventricular Conduction Abnormalities

4) Indications For Pacemaker In Acute Myocardial Infarction

5) Indications For Pacing In Sick Sinus Syndrome

1) INDICATIONS FOR PACING IN SINUS BRADYCARDIA

Sinus bradycarcia may be a natural phenomenon in athletes or may result from various other factors. The treatment may be simple in certain clinical circumstances, for example, requiring stopping the drug causing it or correction of underlying electrolyte abnormalities. Indications for permanent pacemaker for sinus bradycardia are really limited. This includes patients with symptomatic bradycardia due for patients who have symptomatic sinus bradycardia on therapeutic doses of drugs which

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they must take. However, the majority of these patients have sick sinus syndrome that is just unmasked by drug treatment.

2) PERMANENT PACEMAKING IN INTRAVENTRICULAR BLOCK

Av conduction disorder encompasses a spectrum of abnormalities. Therefore, patients may exhibit an array of symptoms directly related to bradycardia or none at all. However, the decision to implant a permanent pacemaker is based on either a) appearance of AV block known to have grave prognosis or b) appearance of symptoms related to bradycardia.

Etiology of intraventricular block: There are many conditions associated with intraventricular block: i) physiologic, which may occur due to increased vagal tone, 2) pathologic, which may either be congenital or acquired. The treatment of intraventricular block depends on the etiology, degree and the presence of associated symptoms.

The commonest abnormality causing AV block is fibrosis of conduction system. This commonly results in high grade permanent heart block.

Congenital AV block: This is usually associated with frequent block in or around AV node. This results in escape rhythm with narrow QRS complex. The escape rhythm in congenital heart block is often stable compared to that seen in adult acquired heart disease. They rarely need a permanent pacemaker except for those who are symptomatic because of braducardia, especially when they occur on exercise or if the QRS complex is equal to or greater than 0.12 seconds.

Acquired intraventricular block Causes: A) Iatrogenic following open heart surgery. This is commonly seen in children following repair of septal defects or tetralogy of Fallot; in postop aortic valve replacement and IHSS following Swan-Ganz catheterization. This results in temporary iinterruption of right bundle branch B) Infiltrative diseases of the heart.

Therefore the indications for permanent pacemaker in intraventricular block can be summarized as: i) intermittent or permanent complete heart block; 2) second degree AV block with symptomatic bradycardia; 3) atrial fibrillatin with complete heart block or slow ventricular response; 4) asymptomatic, Type II second degree AV block?; 5) symptomatic congenital AV block or asymptomatic congenital AV block with wide QRS escape rhythm.

3) PERMANENT PACEMAKER INSERTION IN CHRONIC INTRAVENTRICULAR CONDUCTION ABNORMALITIES

Although the chronic diseases of conduction system have been well studied, it remains a controversial subject. The question of permanent pacing in chronic bi and trifascicular block has been extensively debated and is the source of controversy. The dilemma compounding the problem in delineating the prognosis in patients with chronic bifascicular block is the fact that EKG may not show the status and therefore does not give a clue as to progression to high degree AV block. It is in this situation that EPS (electrophysiologic study) may be useful to delineate the nature of the conduction abnormalities.

Generally speaking, sudden death due to bradyarrhythmias is uncommon in patients with chronic bifascicular block and intact AV conduction. Permanent pacemakers in these patients is reserved for those with established symptomatic bradyarrhythmias. In contrast, patients with bifascicular block who have transient high degree AV block or trifascicular block are at high risk of sudden death. Permanent pacemaker may prolong survival in this group of patients. In a prospective study when patients with chronic bifascicular block undergoing EPS were found to have HV interval of greater than 70 msec., there was a high incidence of development to high degree AV block. In addition, the study showed that a markedly increased HV interval (equal to or greater than 100 msec) was associated with high incidence or progression to complete heart block.

If chronic intraventricular conduction disease is associated with second or third degree AV block, then permanent pacemaker is usually indicated. Thus, the indication for permanent pacemaker insertion in chronic bifascicular block can be summarized as follows.

1) Bifascicular block with intermittent complete heart block and bradycardia:

2) Bifascicular block with intermittent Type II second degree AV block and symptoms.

3) Bi/trifascicular block with syncope.

4) PERMANENT PACEMAKER INSERTION IN THE SETTING OF ACUTE MYOCARDIAL INFARCTION

Generally speaking, various types of AV block in the setting of myocardial infarction occur because of the nature of the damage to conductin system from ischemic event; and it is not unfair to say that the prognosis for survival in patients with myocardial infarction is dependent more on the myocardial factor than the conduction disturbances.

Permanent pacemaker in acute myocardial infarction should be reserved for patients with advanced second or third degree AV block and those who have symptomatic bradycardia associated with new intraventricular conduction disturbance.

However, a patient who may require remporary pacing during the acute phase of myocardial infarction may not necessarily need permanent pacemaker insertion, especially in the setting of an inferior wall myocardial infarction.

Overall the incidence of bundle branch block during myocardial infarction is around 20% and about 20% of these patients progress to high degree AV block. In the presence of new bifascicular block, 30% of the patients generally progress to advanced AV block. These are the patients who have extensive myocardial damage.

Overall mortality is high in patients who present with myocardial infarction complicated by bundle branch block. This is usually due to extensive myocardial damage and pump failure. In this subset of patients, fatal ventricular tachyarrhythmias is an additional factor which contributes to the increased mortality.

5) PERMANENT PACEMAKER IN SICK SINUS SYNDROME AND ITS VARIANT

In the U.S. sick sinus syndrome is one of the major indications of cardiac pacemaker insertion. This syndrome includes a spectrum of abnormal ities resulting from sinus nodal dysfunction. 1) Persistent severe sinus bradycardia; 2) Sinoatrial block. 3) Sinus arrest; 4) Chronic atrial fibrillation with a slow ventricular rate in the absence of drugs; 5) Sudden sinus arrest by carotid sinus massage greater than 3 seconds; 6) Tachy/brady syndrome.

The causes of sick sinus syndrome include: 1) coronary artery disease, especially acute myocardial infarction is the commonest cause of sick sinus syndrome; 2) degenerative disease of the conduction system; 3) iatrogenic secondary to surgical trauma; 4) congenital heart disease; 5) rheumatic heart disease; 6) myocarditis; 7) cardiomyopathies; 8) pericarditis.

In patients with sick sinus syndrome the value of ambulatory monitoring cannot be overstated as the relationship between symptoms and bradycardia must be documented before the insertion of a permanent pacemaker is decided upon.

Generally speaking, in patients with symptoms due to sick sinus syndrome, long term drug treatment has been unsatisfactory. The treatment of choice, therefore, in these patients is permanent pacemaker. An artificial pacemaker should be considered in symptomatic patients with sick sinus syndrome with repeated episodes of sinus arrest and a systolic interval of equal to or greater than 3 seconds.

Indications of permanent pacemaker in sick sinus syndrome therefore should be summarized as: 1) symptomatic bradycardia; 2) asymptomatic bradycardia with pauses greater than 3 seconds; 3) carotid sinus hypersensitivity syndrome; 4) tachy/brady syndrome when drug treatment results in an unavoidable severe bradycardia.

PRINCIPLES OF PACEMAKING

"Voltage," "current," 'resistance," "pulse width," and "energy" are commonly used terms in pacemaker therapy. Since physicians need to understand these terms, electrical aspects of pacing are reviewed. The basic law of the electrical circuit, including pacemaker circuity, is ohm's law, which states the relationship between voltage, current and resistance: Voltage equals current times resistance. Voltage, measured in volts, is the electrical charge of potential that causes electrons to flow in an electrical circuit. Electrical flow rate, current, is measured in milliamperes (mA), and the resistance to flow is measured in ohms.

The electrical aspects of cardiac pacing are based on the same laws as any electrical circuitry. Sensing circuits can be designed to react to P

and R waves and ignore extracardiac signals or electrical activity from muscle movement or environment.

A second equation measures the energy delivered by an electrical impulse: Energy equals current times voltage times pulse width. In other words, the energy (measured in microjoules) delivered by an electrical circuit is the product of current,voltage and the pulse width of the electrical impulse, measured in milliseconds (msec). A record is made of an electrical impulse recorded from a pacemaker's pulse generator using an oscilloscope. The exact shape of the electrical impulse depends on the type of power source, which in this case is a voltage gnerator. Amplitude is measured either in volts, as illustrated, or in milliamperes for a constant-current generator. Pulse width is measured as illustrated.

The terms and concepts reviewed above apply primarily to the pacing circuit of a pacemaker. The sensing circuit is also based on some elementary concepts. First, an electrical signal is characterized according to its frequency or wave length, amplitude and slew rate. Frequency is the number of signal cycles per unit of time, and wave length is the length of one cycle. Frequency and wave length have an inverse relationship; high frequency signals have shorter wave lengths and low frequency signals have longer wave lengths. The third term, "slew rate," refers to the slope or rate of change of signal amplitude per unit of time.

On the basis of these three properties, sensing circuits have been designed to sense P wave or R waves and ignore other intracardiac signals such as T waves. Electrical activity from muscle movement (myopotentials) and from electrical mechanical interference from the environment usually do not affect moderate sensing circuits. The great majority of pulse gnerators function in the inhibited mode. That is, whenever the sensing circuit of a pulse generator senses the proper signal, the pacing circuit's output is inhibited. A second method of sensing is called the triggered mode. In this mode, the output pulse occurs shortly after the detection of the R wave or P wave, and the stimulus fallsin the absolute refractory period. Both modalities are designed to avoid competing rhythms and stimulation of the myocardium during the vulnerable periods of the atrium or ventricle. In the second part of this series, electrocardiogram strips will illustrate both types of sensing modes.

Most pulse generators manufactured in the United States are constant-voltage generators, which are designed to provide a fixed level of voltage output, e.g. 3.5 V, at all levels of lead resistance. However, constant current pulse generator are also available. Constant-current pacemakers are designed to provide a fixed level of current until the upper limit of the voltage source is reached. In other words, a constant-current pulse generator with a maximal voltage supply of 3.5 V will provide a current of 7 mA as long as the resistance does not exceed 500 Ohms (lead resistance measurements are usually lower), according to a calculation using Ohm's law.

Both types of pulse generators are powered by cells or batteries which can vary according to the manufacturer. A cell is an energy source consisting of a negatively charged anode and a positively charged cathode with an electrolyte interface. A battery is a series of two or more cells. The earlier generators used zinc at the anode and mercury at the cathode. However, almost all modern pulse generators are lithium-anode generators, since the maximal life of a lithium generator is between ten and fifteen years (depending on the amount of lithium used). This compared favorably with the two to three year maximal life of the mercury-zinc system.

Two other notable types of power sources are the rechargeable nickel-cadmium and the nuclear-powered pulse generator. Nickel-cadmium pulse generators are not popular because the total charge loss is 15% per week compared with 1% per year for the lithium cell. The nuclear generators are powered by plutonium 238, which produces heat that is converted that is converted to energy by thermocouples. Nuclear-powered units can potentially function for the entire life of the recipient. However, use has been limited due to the licensure by the Nuclear Regulatory Commission, the paperwork and follow-up involved for nuclear safety, fear of environmental contamination and the cost, which is three times that of a lithium system.

As noted above, the longevity of the popular lithium generator may be as long as 15 years but occasionally is as short as four or five years. Larger pulse generators, which contain a greater quantity of anode and cathode material, have more battery capacity and a longer predicted life. Longevity is also dependent on and inversely

proportional to battery or current drain. Current drain is highest in those patients who are paced 100% of the time or who require output in order to pace the ventricle (high threshold). The complexity of pulse generator design also increases current drain. Fortunately, most lithium, generators are designed to develop a gradual decrease in voltage or current. A precipitous drop in output leads to abrupt pacemaker failure, which is dangerous for a pacemaker patient. A decrease in pulse generator output is usally indicated by a drop in paced or magnet rate, which serves as a signal for elective replacement of a generator.

In early pacemakers, the leads were inserted transthoracically. Most leads are now inserted transvenously, with less risk of complication.

The type of pacemaker electrodes that are used depends on whether they are inserted via the transthoracic or transvenous route. When first used, transthoracic electrodes were sutured to the ventricular epicardium, necessitating a thoracotomy. This method resulted in a mortality rate of between 1.0% and 7.5%, with complications such as postpericardiotomy syndrome, arrhythmias and complications of thoracotomy and general anesthesia. However, over the last several years, newer transthoracic electrodes have been developed for ventricular pacing that do not require an extensive tharacotomy. The corkscrew electrode requires a minimal subxiphoid incision and, therefore, has a decreased complication rate. Insertion of epicardial atrial leads still requires an open thoracotomy, so these leads usually are inserted when open-heart surgery is being performed for another reason.

Most commonly, pacemaker leads are inserted transvenously, an approach that has a small complication rate. Many veins can be used, such as the internal jugular, external jugular and femoral veins. However, the cephalic vein and the subclavian vein are preferred since the risk of lead fracture or skin erosion is less in these areas. A percutaneous technique of inserting pacemaker leads using the subclavin vein has recently been introduced and this will soon be the most popular method. With the transvenous approach, the ventricular electrodes are positioned in the right ventricular apex, and the atrial electrodes are positioned in the right atrial appendage. The lead contains a portal for a thin guidewire stylet, which can be shaped prior to insertion to give the desired configuration to the flexible intracardiac lead. This facilitates intracardiac maneuvering and positioning. After adequate placement, the stylet is permanently removed.

Ventricular screw-in or pronged lead tips are the most stable but elicit greater fibrosis. Helical-coil and tined lead tips are less invasive. Porous tip leads are the most recent advance in ventricular lead design. The tined J-lead up tip is preferred among atrial leads.

Recent innovations in lead designs, especially in lead tip configurations, have resulted in improved electrode stability. Ventricular leads usually have less than a 5% displacement rate when they are implanted by experienced physicians. Lead tip configurations include invasive screw-in or pronged tips that actually penetrate the endocardium Although theoretically the most stable type of leads, invasive tips are associated with a higher chronic threshold the higher invasive lead elicits. The less invasive lead tips include helical-coil or tined tips that fix into the trabeculae of the ventricular apex. Porous tip electrodes containing highly porous platinum-iridium mesh fibers are the most recent advance in tip design. Fixation is facilitated by tissue ingrowth rather than by active fixation. Preliminary results indicate that porous tip electrodes may offer the advantage of less fibrotic reaction and lower threshold while still providing stability.

The atrial leads are historically more difficult to keep in position. The atrial leads are of several types but are usually a J-lead with or without tines, an endocardial screw-in or a coronary sinus lead. The tineless J-leads were prone to dislodgement, but tined atrial leads usually have a less than 7% dislodgement rate. As a results, the tined J-lead is probably the lead of choice for atrial pacing. The atrial lead can also be placed in the coronary sinus with a bipolar electrode, using the proximal electrodes for pacing or a special coronary sinus lead with a "rattail" configuration that contains a soft tip to minimize trauma. Although the dislodgment rate over two years has been only 5%, the coronary sinus lead is a distant second choice to the tined lead because it is more difficult to position. The screw-in atrial lead is also not as popular because of the higher incidence of atrial perforation and phrenic nerve irritation.

The pacemaker current that depolarizes the heart completes the circuit between the anode and the cathode. The electrode system can be bipolar or unipolar. In the bipolar system, the cathode is the terminal at the tip of the pacemaker lead; the anode is more proximal but within the cardiac chamber. In the unipolar system the

cathode is at the lead tip and the anode is extracardiac, usually encased in the generator pack.

Each system has its advantage. For example, a unipolar lead can be introduced more easily through a small vein because it is smaller than a bipolar lead. Another advantage of a unipolar pacing system is that the pacer blip is usually larger, thus facillitating electrocardiogram analysis. Furthermore, in some cases threshold for capture may be lower with unipolar pacer leads because the resistance of the return circuit is lower. However, since both terminals are intracardiac, a bipolar pacing system has the advantage of being less susceptible to electromagnetic interference. Muscle stimulation is also less of a problem with bipolar systems; in unipolar systems the muscle underlying the pulse generator may be stimulated as the pacemaker current travels through muscle in order to complete the electrical circuit.

Pacers must have their pacing and sensing system tested routinely. Portable pacing system analyzers are manufactured by most companies to test their own products.

An important part of the method of implantation is the routine testing of the pacing and sensing circuits of the pacing system by measuring the parameters discussed above. The baseline test parameters are recorded in the chart and are available for comparison. Pacing system analyzers have simplified pacer and lead testing since their introduction in 1974. Most United States firms manufacture portable pacing system analyzers designed to thet their own products. Some hospital operating rooms need several different analyzers and the multiplicity of equipment is one of the major drawbacks of pacing system analyzers. Nevertheless, the analyzers are still the most popular method of testing pacing system in the United States.

The pulse generator is generally tested first by hooking it up to a pacing system analyzer using sterile cables. Output, pulse width and pacing rate are easily read from the digital display and compared with the manufacturer's specificatins. The analyzer also checks the sensing circuit using test signals that have similar amplitude and frequency as intracardiac signals. Significant variation from the manufacturer specifications is commonly due to error in testing procedures, such as using another company's analyzer. If an error in procedure is ruled out, then the pulse generator is probably a defective unit. In this case, a different pulse generator is put through the same testing procedures.

After the pulse generator passes testing, the surgeon positions the pacing lead. Endocardial leads are usually positioned using fluoroscopy, but epicardial leads are inserted under direct vision, commonly via a limited thoracotomy. Next, lead parameters are tested to serve as a check that the lead is in proper position for pacing and sensing. Testing lead position for capture is done by hooking the lead pins to the pacing system anzlyzer and measuring threshold. Threshold for capture is the lowest pulse amplitude, measured in either milliamperes or volts, that maintains consistent pacer capture. Threshold is always measured with the analyzer adjusted to the pulse width at which the pacemaker will be set. This is important because the energy delivered by a pacing pulse varies with both amplitide and pulse width.

Optimal threshold values for modern ventricular leads (8mm^2 to 12 mm^2 tips) are in the range of 0.6 mA and 0.3 V with a pulse width of one msec. Despite improvements in atria leads, which now have excellent stability, acute threshold values for atrial leads are commonly three times higher than ventricular leads. Acute threshold values must be low for both types of pacing because threshold values frequently rise threefold due to reaction inflammatory changes around the lead tip. A rule of thumb that many physicians use to provide the patients with a margin of safety is that acute threshold should not exceed 15% of the pulse generator's output. Chronic threshold values are usually higher but should remain less than 70% of the pulse generator's output in order to maintain a margin of safety.

After the threshold is measured in milliamperes and volts, the lead resistance can be calculated as a ratio of the voltage and current thresholds. Normal values depend on the type of lead but are usually less than 900 ohms for electrodes with a surface area of 8 mm^2 to 12 mm^2 or less. Measurement of resistance serves as one more check on the integrity of the pacing system because a high impedance value may indicate a lead fracture, and a very low impedance value may indicate a rupture in the lead insulation.

The pacing system analyzer also checks the lead position for sensing by measuring the amplitude of the sensed R wave. Most manufacturers recommend a signal amplitude of at least 5.0 mV

to 6.0 V for ventricular leads, which exceeds the sensitivity setting of most pacemakers by an adequate margin. The amplitude of sensed P wave from an atrial lead is smaller and can be in the range of 1 mV to 2 m V. Since most pacing system analyzers are not accurate in this range, some physicians prefer to measure the P-wave amplitude by recording an intravardiac electrogram on an oscilloscope. If high thershold values or poor signal amplitude are obtained, the physician will try to resposition the leads. However, an optimal lead position sometimes cannot be found, especially if there is diffuse myocardial fibrosis. In these cases, the higher threshold or poor signal amplitude may have to be accepted. Difficult cases might require specially made high-output or high-sensitivity pulse generators to prevent pacing failure.