SPECIAL ARTICLE

Diagnostic and Therapeutic Uses of Atrial Pacing

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SUMMARY
There are occasions when atrial pacing can be usefully employed both diagnostically and therapeutically. Diagnostically, atrial pacing provides mechanisms for easily, safely, and reproducibly stressing the heart that can help in the evaluation of patients with a variety of cardiac disorders. It is particularly useful in the assessment of patients with coronary disease.

Therapeutically, atrial pacing has been applied in four general areas: (1) to terminate or slow supraventricular tachyarrhythmias; (2) to accelerate the heart rate in patients with sinus bradycardia and intact atrioventricular conduction; (3) to suppress ventricular irritability; and (4) to augment cardiac output. Techniques of atrial pacing are discussed. Currently, a major problem with both temporary and long-term pervenous atrial pacing is erratic pacing. The development of electrodes and pacemakers designed specifically for atrial pacing, as well as further experience with stimulation from the coronary sinus may lead to more reliable pacing.

Additional Indexing Words:
Pacemakers Sinus node disease Atrial tachyarrhythmias
Coronary sinus pacing Overdrive suppression

One of the great advances in cardiology over the past decade has been the development of a variety of means of pacing the heart electrically. For the most part electrical pacing has been applied to the ventricles for the treatment of complete heart block. There are occasions, however, when pacing the atria may be successfully utilized in patients with intact atrioventricular (A-V) conduction. Thus, atrial pacing avoids some of the problems of ventricular pacing and also takes advantage of the well-documented augmentation of cardiac output that occurs when a coordinated atrial systole just precedes ventricular contraction. This report is intended to provide a brief, practical summary of the current status of atrial pacing used both diagnostically and therapeutically.

Techniques and Problems of Atrial Pacing
Atrial pacing can be employed either on a temporary or long-term basis. For diagnostic and other short-term stimulation we generally utilize conventional bipolar pacing electrodes placed under fluoroscopic control and positioned at the junction of the superior vena cava and right atrium. In overdriving ventricular tachyarrhythmias, myocardial infarction, and other such emergencies, floating or semi-
LEAD 2:
Sinus Bradycardia
Rate 46 beats/min.
Acute inferior myocardial infarct

Bipolar electrode being advanced into right atrium
Right atrium entered

Atrial pacing rate 73 beats/min.

Figure 1
(Upper panel) Lead II (2), showing marked sinus bradycardia in a 52-year-old man with an acute inferior myocardial infarct. The rhythm had been unresponsive to 2 mg atropine intravenously. (Middle panel) Electrocardiogram recorded from tip of a semi-floating electrode passed percutaneously at the bedside. The right atrium is entered at the point of abrupt change in size and configuration of the P wave indicated by the arrow. (Lower panel) Atrial pacing at 73 impulses/min.

Floating electrodes can often be passed transvenously at the bedside with electrocardiographic monitoring, as illustrated in figure 1. With currently available electrodes, such problems as erratic atrial pacing or diaphragmatic stimulation may arise. Repositioning the electrodes or adjusting current levels will usually overcome these difficulties.

For long-term atrial stimulation the most reliable method is the surgical implantation of pacing electrodes directly into the wall of either the right or left atrium. Obviously, this requires a thoracotomy, and, therefore, attempts have been made to achieve satisfactory long-term stimulation by percutaneous methods. Our original experience with conventional permanent pacing electrodes used mainly for ventricular pacing was not very satisfactory, as atrial pacing proved to be somewhat unreliable with them. Nevertheless a number of patients were satisfactorily treated with this system. More recently, we have employed an electrode with a J-shaped tip. This is inserted with a stylus in place that straightens out the tip of the electrode. When the stylus is removed, the tip reassumes a curved shape. When inserted, particularly from the left cephalic vein, the electrode tip can usually be directed anteriorly so that it lies in the right atrial appendage, thus effecting good contact and reliable pacing about 85% of the time. Figure 2 illustrates such an electrode in place.

Perhaps the most dependable method of long-term percutaneous atrial pacing reported to date is that of pacing the left atrium by an...
Figure 2
Posteroanterior and lateral X-rays of the chest showing the tip of a J-shaped electrode directed to the left and anteriorly into the right atrial appendage.

Figure 3
Posteroanterior and lateral X-rays demonstrating long-term pervenous pacing with an electrode in the coronary sinus. In the lateral view, the extreme posterior position of the electrode in the heart is characteristic.

electrode placed in the coronary sinus (fig. 3). The most experience with this method has been that of Kramer and Moss, who now have initiated long-term pacing from the coronary sinus in 14 patients. In 10 of these patients, they were able to achieve effective long-term pacing for periods as long as three years. In 4 patients it was not possible to achieve
adequate stimulation by this route for a variety of reasons. To date they have experienced no serious complications from coronary sinus pacing. Presently, if long-term percutaneous atrial pacing is contemplated, coronary sinus pacing or use of the J-tipped electrode would seem the most reliable method, although further follow-up is necessary to establish the ultimate safety and reliability of these techniques.

The threshold for atrial pacing is generally two to five times higher than for ventricular pacing. In addition, pacing thresholds may double or triple their initial values with the passage of time. Therefore it would not seem advisable to try long-term atrial pacing if the threshold as determined at the time of pacemaker insertion is above 4 ma.

Ideally, for long-term atrial stimulation, demand pacing is preferable to asynchronous pacing, but there may be problems in sensing from the atrium. It is generally, but not always, possible to find a position either in the coronary sinus or right atrium where a large enough P wave or even an R wave can be sensed. Examples of this are shown in figure 4. Additional problems may arise if the pacemaker senses the R wave and not the P wave, as illustrated in figure 5.

A very imaginative ventricular-inhibited pacemaker has recently been introduced by Berkovits, Castellanos, and Lemberg. This so-called “bifocal” pacemaker is designed specifically for patients with sinus bradycardia with or without A-V block. The pacemaker involves the use of two electrodes, one positioned in the right atrium and one in the right ventricle, with both atrial and ventricular pacing circuits. The ventricular electrode senses the ventricular electrocardiogram and sequentially paces the atrium and ventricle on demand. The atrium is paced in the presence of sinus bradycardia and intact A-V conduction, but the ventricular pacer is inhibited by the
Demand Pacemaker: Electrode in Coronary Sinus

Not Sensing "R" Waves
Paced Rate 95 impulses/min.

Sensing "R" Waves
Paced Rate 65 impulses/min.

640 ms

280 640 920 ms

Figure 5

Slowing of the discharge rate of a demand atrial pacemaker caused by the pacemaker sensing the R wave. (Left) The R wave is not sensed, and the pacemaker is discharging at a rate of 95 impulses/min. The interval between pacemaker impulses is 640 msec. (Right) The pacemaker is now sensing the R wave. However, the R wave does not occur until 280 msec after the pacemaker discharge—that is, the P-R interval. The pacemaker is now reset from the peak of the R wave, and does not fire again for another 640 msec. The interval between successive pacemaker impulses is now 920 msec, and the discharge rate falls to 65 impulses/min.

Conducted impulse. This pacemaker is now in clinical use, and the early results with it appear promising.

Currently one of the major problems lies in the reliability of both short- and long-term pervenous atrial pacing. The development of more satisfactory pacemakers and electrodes designed specifically for atrial pacing as well as further experience with pacing from the coronary sinus may resolve some of these problems.

Diagnostic Atrial Pacing

Diagnostic atrial pacing has been employed primarily as a means of stressing the heart by selectively increasing heart rate. Obviously the heart can be stressed and the heart rate increased by the use of exercise or by employing drugs like beta-adrenergic agents or atropine. However, atrial pacing has several advantages over these other methods. First, with atrial pacing, the only variable is the heart rate. There are no hemodynamic or metabolic effects except those caused by changes in heart rate alone. Second, with atrial pacing, studies are easily performed and require no effort on the part of the patient. Third, the desired heart rate is totally controllable and can be accurately reproduced. Fourth, adverse effects caused by the tachycardia, should they occur, can be immediately reversed by discontinuing pacing.

Atrial pacing has been employed in the investigation of a wide variety of cardiac disorders but it has been particularly useful in three areas: (1) in the study of patients with coronary disease; (2) in the assessment of patients with valvular heart disease; and (3) in studies of A-V conduction.

Perhaps the great value of diagnostic atrial pacing lies in the assessment of patients with coronary disease in whom rapid pacing has been used to induce coronary ischemia. Using as an indicator of myocardial oxygen consumption a tension-time index calculated as a product of heart rate, mean systolic ejection...
pressure, and ejection time, Sowton and co-workers found a threshold for angina in patients with coronary disease that could be consistently reproduced by episodic stimulation of the right atrium. This group also found that the threshold for angina in any given patient was similar whether produced by atrial pacing or by exercise.

Parker and his colleagues have performed some outstanding studies of hemodynamic, metabolic, and electrocardiographic changes induced by right atrial pacing in patients with coronary disease. Using atrial pacing at relatively rapid rates, they have been able to induce typical coronary pain in these patients. In addition, they have shown that the iatrogenic tachycardia is accompanied by the production of excessive lactate and the appearance of abnormal concentrations of potassium in coronary sinus blood, ischemic ST-segment depression on the electrocardiogram, and elevation of the left ventricular end-diastolic pressure. This response of left ventricular end-diastolic pressure is abnormal. In normal individuals cardiac output is altered little or is slightly increased with atrial pacing. Accordingly, as the heart rate rises, stroke index and stroke work index fall, and there is a corresponding decrease in left ventricular end-diastolic pressure in accordance with the Starling mechanism. The rise of left ventricular end-diastolic pressure as stroke index falls implies an actual decrease in left ventricular contractility during coronary insufficiency. All of the abnormal changes reverse themselves shortly after pacing is discontinued. Similar findings have been reported by other groups.

Thus atrial pacing may help in the evaluation of patients suspected of having coronary disease. The development of characteristic pain and ischemic, metabolic, and electrocardiographic changes in response to an increase in heart rate induced by pacing may be of value in the differential diagnosis of chest pain. Perhaps further correlations between pacemaker-induced changes and coronary artery anatomy may help in defining more accurately the significance of sclerotic lesions demonstrated angiographically in the coronary arteries. Responses to therapy such as nitrates, beta-adrenergic blocking agents, and, especially, surgical revascularization procedures can be more precisely assessed by this highly reproducible means of cardiac stress.

Atrial pacing has been used to study patients with valvular heart disease, especially those with mitral stenosis, a lesion to which tachycardia is generally deleterious. Not surprisingly, it has been shown that tachycardia produced by pacing increases the gradient across the mitral valve when it is stenosed.

Interesting investigations have also been carried out in several centers using atrial pacing to help define mechanisms of A-V conduction. In particular, Damato and his group and Narula et al. have done extensive work using recordings of A-V nodal, His, bundle, and bundle-branch potentials obtained by positioning special sensing electrodes in proximity to these structures. With atrial pacing they have shown that the P-R interval gradually lengthens as the heart rate increases. It has been found that the delay is located largely between the atrium and His bundle. Such studies of A-V conduction in patients with sinus node disease have actually helped clinically in defining the best mode of pacing in such individuals. If, for example, atrial pacing at relatively slow rates results in second or third degree A-V block, long-term ventricular rather than atrial pacing is preferable.

In summary, diagnostic atrial pacing is, as Balcon has said, "a way of exercising the heart without exercising the patient." It has been particularly useful in the evaluation of patients with coronary disease, but its application might be considered in any situation where the assessment of the effects of an increase in rate upon the heart might be desirable. In addition to these clinical diagnostic uses, atrial pacing has been used extensively for research purposes, and has greatly enhanced understanding of the effects of an increase in heart rate upon normal and abnormal hearts.

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Conversion of atrial flutter to normal sinus rhythm using rapid atrial pacing. This arrhythmia occurred in a 63-year-old man following surgery for coronary disease. The upper panel shows atrial flutter with 2:1 A-V block. Atrial pacing is begun in the middle panel, first at 180 impulses/min, and is increased abruptly to 360 impulses/min. At the point indicated by the arrow, the flutter is terminated and the atrium is captured by the pacemaker. Lower panel, atrial pacing is discontinued, and sinus rhythm supervenes.

**Therapeutic Atrial Pacing**

Atrial pacing has been applied therapeutically in the management of four general categories of cardiac problems: (1) the termination or slowing of certain supraventricular tachyarrhythmias; (2) the therapy of bradycardia in the presence of intact A-V conduction; (3) the overdrive suppression of refractory ventricular irritability; and (4) the enhancement of cardiac output.

**Termination of Supraventricular Arrhythmias**

Rapid atrial stimulation may effectively terminate many different supraventricular arrhythmias such as atrial flutter, paroxysmal atrial tachycardia, and junctional and reciprocating tachycardias. There are probably several mechanisms by which atrial pacing accomplishes this. First, it is likely that some ectopic foci may actually be overdriven or suppressed by rapid atrial stimulation. Second, a self-sustaining supraventricular tachyarrhythmia may be converted to one which is not, for example, the conversion of atrial flutter to atrial fibrillation, which then reverts to sinus rhythm. Third, it may be possible to interrupt fixed circus or reciprocating pathways through the atria and area of the A-V junction by introducing properly-timed electrical stimuli. This latter mechanism has been documented in certain patients with the Wolff-Parkinson-White syndrome. Figure 6 illustrates an example of the termination of atrial flutter by rapid atrial stimulation, and figure 7 shows the termination of supraventricular tachycardia, the precise nature of which is unclear, by atrial pacing.
Atrial Pacing

Conversion of a supraventricular tachycardia by rapid atrial pacing. Left upper panel shows the arrhythmia with a rate of 160 beats/min recorded in Lead II. (Right upper panel) An electrocardiogram taken from an atrial electrode clearly shows a P wave between successive QRS complexes. This is either an atrial tachycardia with first degree A-V block, or a junctional or possibly even a reciprocal tachycardia. (Second panel) Atrial pacing is commenced at a rate of 200 impulses/min. Midway through the tracing the rhythm becomes irregular, indicating interruption of the arrhythmia. (Third panel) The pacemaker is turned off, and a very slow escape rhythm is present. (Lower panel) Because of the bradycardia, slower atrial pacing is initiated.

In our experience, when rapid atrial stimulation has been used in this way it has been possible to convert atrial flutter, paroxysmal atrial tachycardia, and junctional tachycardias about 70% of the time. Atrial fibrillation can generally not be terminated by this method.

When should rapid atrial stimulation be considered in preference to external electrical conversion? There are certain instances when it can be applied easily and usefully. For example, in arrhythmias that arise during the course of cardiac catheterization it is simple to guide a pacing electrode into the right atrium and attempt to terminate the arrhythmia by this means. Similarly in postoperative cardiac patients, most of whom have atrial electrodes in place in our institution, this technique has been extremely valuable. Also, in individuals who are seriously ill or in whom there is actual or potential digitalis intoxication, the risks of external cardioversion may be sizable and cardioversion may even be contraindicated. In such instances, rapid atrial stimulation may be easier and safer. However, the overall rate of successful conversion with rapid pacing is not as great as with cardioversion. When converting arrhythmias in this way, it is essential to establish at the outset that the electrode is not in the right ventricle. When this has been ascertained, stimulation can be started at a rate of 100 to 150 impulses/min, and gradually increased to as rapid a rate as 500 or 600 impulses/min until either termination has been effected or the method has been unsuccessful.
In addition to rapid asynchronous stimulation, coupled or paired atrial pacing has been used to slow the heart rate in cases of sinus tachycardia as well as other supraventricular tachyarrhythmias. With this technique, a pacemaker impulse is delivered to the atrium shortly after the spontaneously occurring P wave (coupled pacing), or after an initial electrical stimulation (paired pacing). In both instances it is applied to the atrium shortly after the end of its refractory period. This results in electrical depolarization of the atrium but little or no mechanical response. When properly timed, the second impulse depolarizes the atrium prematurely, blocking the next spontaneous beat, but occurring too early to be conducted across the still-refractory A-V junction. This results in slowing of the ventricular rate. We have found the application of coupled or paired pacing to be limited by the propensity of the stimuli to produce atrial fibrillation.

**Treatment of Bradycardia in the Presence of Intact A-V Conduction**

This method has been particularly applicable to patients with an inadequate sinus mechanism—the so-called “sick sinus syndrome.” These patients have marked sinus bradycardia, often with sinus arrest, with or without paroxysmal supraventricular tachycardias. When episodic tachycardia complicates the bradycardia, the clinical picture has been described by Short as “the syndrome of alternating bradycardia and tachycardia.” Adams-Stokes symptoms may arise in association with bradycardia, occurring either spontaneously as a result of sinus arrest or as a consequence of long periods of asystole following conversion of the supraventricular arrhythmias. Since these patients usually have

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**Figure 8**

Use of atrial pacing in a patient with the “brady-tachy” syndrome. (A) Atrial fibrillation with a rapid ventricular response of 150 beats/min. (B) Later the same day, sinus bradycardia at a rate of 38 beats/min. The patient experienced episodes of dizziness and syncope at times of conversion of the tachycardia. She was successfully treated with a percutaneous atrial pacemaker set at a rate of 100 impulses/min (C), which is shown still capturing the atrium over a year later (D).
intact A-V conduction, atrial pacing is possible. Figure 8 illustrates the electrocardiograms of a patient with the syndrome of alternating bradycardia and tachycardia ("brady-tachy" syndrome) treated with an atrial pacemaker.

The manifestations of bradycardia can be eliminated by atrial pacing, but the frequency of the episodes of tachycardia may or may not be influenced by the pacemaker. In our own experience pacing per se has been rather disappointing in preventing the troublesome episodes of recurrent tachycardias. However, this has not been true in some other series. Nonetheless, with bradycardia prevented by a pacemaker antiarrhythmic agents can be given with considerably more safety, and the combination of pacing plus the administration of these agents usually does decrease the frequency of the tachycardias.

In the treatment of this syndrome, ventricular pacing—especially of the demand mode—can also be employed, but atrial pacing, because of the better cardiac output that results, is preferable if it can be effectively established. An additional disadvantage of ventricular pacing in the presence of intact A-V conduction is that retrograde atrial activation may occur. This can initiate reciprocal rhythms and even reciprocating tachycardias.22

Nonetheless, in our own experience, and as has been pointed out by several workers, individuals with disordered sinus mechanism may also have impaired A-V transmission.14, 23 The A-V conduction of any patient with the sick sinus syndrome should be tested with atrial pacing in advance of final electrode placement. If by this maneuver second or third degree block is elicited with relatively slow pacing rates—for example, rates less than
Effect of atrial pacing in a 31-year-old man after repair of tetralogy of Fallot. Upper tracing shows the electrocardiogram, Lead III (3); middle tracing, the radial arterial pressure in mm Hg; and lower tracing, the central venous pressure. Note the fall in radial arterial pressure and rise in central venous pressure, with cannon waves appearing with cessation of atrial pacing and appearance of a junctional rhythm in the center of the figure.

Atrial pacing may also be effectively employed to accelerate slow sinus rates induced by drugs or following cardiac surgery.

**Overdriving Ventricular Irritability**

There are occasional patients whose life-threatening ventricular arrhythmias cannot be managed by any or all conventional antiarrhythmic medications. In such instances, particularly when there is a slow basic heart rate and intact A-V conduction, rapid atrial pacing may safely and successfully abolish the irritability.25, 26

Overdrive suppression can be accomplished by pacing either from the right atrium or the right ventricle, but atrial pacing has distinct advantages. Specifically, there is no danger that the electrode or the electrical stimuli will themselves contribute to ventricular irritability. Furthermore, cardiac output may be considerably better with atrial than with ventricular pacing.

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120 beats/min—permanent ventricular rather than atrial pacing is preferable.

There are many other instances of transient sinus slowing that are amenable to atrial pacing. Benchimol and McNally have advised right atrial pacing for the prevention of the almost invariable slowing of the heart rate and occasional serious arrhythmias accompanying coronary angiography.24 In the sinus bradycardia associated with acute myocardial infarction—especially inferior or posterior infarction—atrial pacing may be very effectively applied if drugs are unsuccessful and A-V block is absent. In such patients we often pass floating or semi-floating electrodes at the bedside from a peripheral vein into the right atrium using electrocardiographic monitoring. Adequate atrial pacing can be achieved by this method about 80% of the time. Figure 1 illustrates an example of a patient with a diaphragmatic infarct and marked sinus bradycardia treated by transvenous atrial pacing.
ATRIAL PACING

Overdrive pacing can usually be considered a temporary measure to tide patients over a critical period, until the irritability is diminished by the beneficial effects of pacing, or until transient factors causing the irritability are eliminated—among these, digitalis intoxication, electrolyte abnormalities, adverse effects of various drugs, or the arrhythmogenic aspects of cardiac surgery or acute myocardial infarction. In rare instances, long-term atrial pacing has been necessary. We have treated four patients with permanent atrial pacing for refractory ventricular irritability, and in all instances the arrhythmias have been considerably diminished or eliminated by combining long-term atrial pacing with antiarrhythmic drugs. Figure 9 illustrates the use of long-term pacing from the coronary sinus to overcome refractory ventricular irritability in a 60-year-old man.

Cardiac Output Augmentation

Simple sinus bradycardia alone and, particularly, slow junctional rhythms may reduce cardiac output, which may then be increased with atrial pacing. This application of atrial pacing has been particularly useful immediately following cardiac surgery, when slow junctional rhythms are fairly common. Figure 10 illustrates the increase in systemic blood pressure and reduction in venous pressure afforded by atrial pacing in a patient with a junctional rhythm following repair of a tetralogy of Fallot. However, even in patients with A-V block, cardiac output may be augmented by producing a properly-timed atrial systole in advance of ventricular contraction. Indeed, this is the rationale behind synchronous or P-wave triggered pacemakers. We have employed sequential A-V pacing in patients with heart block accompanying acute myocardial infarction. This technique involves placing pacing electrodes both in the right atrium and the right ventricle. By means of a coupled-pulse generator, the atrium and ventricle are paced in sequence at an interval that can be preset. In nine patients with acute myocardial infarction, there was an increase in cardiac output of 24% when sequential A-V pacing was employed in contrast to ventricular pacing at the same rate. Improvement in a few patients has been dramatic.

When using sequential pacing, it is usually necessary to pace the heart 10 beats/min faster than the basic sinus rate in order to capture the heart. In instances of marked sinus tachycardia, it may be impossible to establish adequate sequential pacing.

The effect of enhanced atrial contractility with paired atrial pacing has also recently been explored by Chamberlain et al. They have shown that the same extrasystolic augmentation of contractility that occurs in the ventricles with paired pacing also occurs in the atria. As a consequence of paired atrial pacing, there is a tremendous increase in the force of left atrial contraction, resulting in an increase in left ventricular end-diastolic pressure. This causes an increase in the force of left ventricular contraction as a result of the Starling mechanism. Though of considerable interest, this method has not yet been applied clinically.

Pacing After Heart Surgery

As has been previously indicated, atrial pacing has been extremely valuable in the management of patients following open heart surgery. At our institution, atrial and ventricular pacing wires are routinely inserted into all patients at the time of open heart surgery except for those individuals with very long-standing atrial fibrillation or greatly enlarged atria, in whom only ventricular electrodes are used. Atrial pacing subsequently is utilized in about 60% of patients postoperatively, for the treatment of all of the problems previously discussed.

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