Emergency Cardiac Pacing

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Cardiac pacing may be used for emergency control of atrial and ventricular tachyarrhythmias and bradyrhythmias, such as symptomatic sinus bradycardia or atrioventricular block, and it may be used for prophylaxis in patients with myocardial infarction or during pulmonary artery catheter placement.

Important technical factors to consider include energy delivery, waveform, and threshold of initiation. Types of pacing include mechanical, transthoracic, transvenous, transcutaneous, epicardial, and esophageal.

Current guidelines recommend pacing via the transcutaneous or transvenous route in the emergency setting. Correlates of survival include the time until institution of pacing and the underlying systemic or cardiac disorder.

HISTORY

Cardiac resuscitation by electrical means originated as early as the eighteenth century. In a 1788 essay, "On the Recovery of the Apparently Dead," Kite[1] described the use of artificial ventilation and electrical shock in the treatment of drowning. Desantis devised the "Persile Galvanic Pile," an early portable battery used to resuscitate via a gastric-to-cutaneous circuit.[2]

Transvenous pacing was first used by Floresco[3] in a canine model in 1905. Gould administered medication through a left ventricular puncture in a child in asystolic arrest and also described mechanical stimulation in 1929.[4] Hyman[5] manufactured and used an extrinsic artificial pacemaking apparatus in arrest in 1932. The modern resurgence of cardiac pacemaking is attributed to Zoll et al,[6] who used the technique in 1952 for two patients in complete heart block. Zoll later reported successful transcutaneous pacing in 13 of 14 patients.[7]

The transvenous pacing mode requires operator expertise and has a significant complication rate. In many circumstances, it has been supplanted by transcutaneous pacing. The original Zoll device for transcutaneous pacing (ZMI Corporation, Cambridge, MA) had a low impedance, small surface area (3 cm^2), and short impulse signal (3 milliseconds). Modern pacing systems feature high impedance and larger electrodes (10 to 20 cm^2) with longer pulse duration (20 to 40 milliseconds). These modifications selectively stimulate cardiac instead of skeletal muscle, greatly increasing the patient's tolerance.

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Manuscript received April 7, 1992; revision accepted October 2, 1992.

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Key Words: Arrhythmia, cardiac pacemaking, pacing.

0735-6757/93/1102-0016$5.00/0

INTRODUCTION

Artificial cardiac pacemaking is used for treatment of both structural and functional lesions and can be used in pediatric to geriatric patients with little alteration. Common clinical conditions that warrant the use of cardiac pacing include sudden cardiac death, myocardial infarction, and pulmonary artery catheterization in patients with left bundle branch block.[8]

The most common arrhythmia in sudden cardiac death outside the hospital is ventricular fibrillation (62% to 75%), followed by bradyasystole (23% to 31%).[9,10] The prehospital survival rate is generally low (3%) and even poorer in patients with asystole (0%), who frequently receive pacing.[9,10] Arrest survivors include 66% with decreased left ventricular function (cardiac index, $<2.62 \pm 0.72$ L/min/m) due to right coronary thrombosis in 77% of cases.[9,10] Patients who suffer cardiac arrest in the hospital fare slightly better, with 8.2% surviving to discharge and 75% dying in the first 24 hours after arrest.[11]

In patients with myocardial infarction, Col and Weinberg[12] found a 24% incidence of intraventricular conduction delay associated with increased mortality (47% *v* 24% in patients without conduction delay). Conduction abnormalities included left anterior hemiblock (9.4%), incomplete right bundle branch block (7.5%), and left or right bundle branch block (3.7%).[12] In a larger outcome study, Godman et al[13] reported an 8.4% incidence of conduction abnormality with 56% mortality. Conduction defects in these patients were right bundle branch block (3.0%), complete heart block (2.6%), and left bundle branch block (1.6%). Cardiac pacemaking failed to influence survival.[13]

Pulmonary artery catheterization is associated with a 77.5% incidence of arrhythmias, predominantly premature ventricular (45.6%) or atrial (15.5%) contractions and ventricular tachycardia (22.5%). However, only 2.5% of patients progress to right bundle branch block, raising the issue of prophylactic pacing for Swan-Ganz catheter insertion.[14] The cause of right bundle branch block is assumed to be mechanical trauma to the adjacent conduction bundle in right heart catheterization. However, Castellanous et al[15] reported a sympathetic mechanism involving the left fascicle (left anterior or posterior hemiblock) in four patients.

PATHOGENESIS OF CONDUCTION SYSTEM DEFECTS

Conduction system abnormalities generally result when inadequate arterial blood supply causes regional myocardial infarction. The events most commonly associated with conduction delays are right coronary artery occlusion and infarction of the right ventricle.

The sinoatrial node is supplied by the sinoatrial artery, which originates from the right coronary artery in 60% and from the left coronary artery in 40% of patients.[16] The atrioventricular node and artery may be supplied by the right coronary artery (80%), the left coronary artery (10%), or

another artery (10%). Kugel's artery, a branch of the left coronary artery (95%), provides redundant flow to the atrioventricular node.[16] The posterior descending artery feeds the posterior left bundle, whereas the anterior descending artery supplies the inferior left bundle and right bundle branch of the conduction system.

Keller et al[16] reported a 15% incidence of conduction delay associated with 33% mortality in patients with myocardial infarction. First-degree atrioventricular blocks (13%) progressed to second-degree blocks in 5% and to complete heart block in 3% of patients.[16]

The location of an infarction can be correlated with the onset of conduction abnormality. An inferior wall infarction occurs in most cases (80%), but is associated with low mortality (23%).[17] A proximal conduction defect occurs with first- and second-degree atrioventricular block (Mobitz type I); 60% to 75% of such patients progress to complete heart block.[18] However, the escape mechanism is a stable, narrow complex (69%), late onset (64%) rhythm that often responds to atropine.[17,18] This transient escape rhythm improves hemodynamic status and carries a good prognosis, although syncope (42%) or shock (26%) may occur.[17,18]

By contrast, an anterior wall infarction occurs in a minority (20%) of patients, but results in a high mortality rate (76%) when accompanied by a conduction delay.[18] Distal conduction defects may be second-degree atrioventricular block (Mobitz type II) or left or right bundle branch block, with progression to complete heart block in 25% to 40%. The escape mechanism is an unstable, wide complex (31%), early onset (26%) rhythm that is resistant to atropine.[17,18] This escape rhythm deranges hemodynamics, often resulting in syncope (58%) and shock (74%). Its longer duration requires permanent pacing, a poor prognostic indicator.[17,18]

CARDIAC ELECTROPHYSIOLOGY

Cardiac tissue may have contractile or conduction properties. The effects of cardiac pacing on hemodynamics are assessed by analyzing cardiac output, which is determined by heart rate and stroke volume.

In normal patients pacing increases cardiac output 0.2 L/min, or 7%, for each 15-beat increase in heart rate from 60 to 90 beats/min.[19] A plateau effect occurs at higher rates (110 beats/min).[19] Similar results have been demonstrated in patients with impaired cardiac function (New York Heart Association class III or IV) manifesting a 6% increase in cardiac output for a change in heart rate of 15 to 20 beats/min.[20]

Stroke volume is also maximized by allowing adequate time for diastolic filling and avoiding myocardial oxygen consumption-delivery imbalance, which increases the cost of stroke work at higher heart rates. Benchimol et al[21] suggested that stroke work increased with atrial but not with ventricular pacing; in abnormal hearts the difference was greater when cardiac index was measured. A separate study suggested that the increase in cardiac index did not depend on the chamber paced (right atrium, right ventricle, or left ventricle).[22]

The benefits of atrioventricular sequential versus single-chamber pacing have been well substantiated by measurements of increased cardiac output and systemic pressure, as well as decreased central venous and pulmonary artery pressures.[23-25] Sequential chamber pacing is best accomplished by a physiologic PR interval (atrium to ventricle) delay of 150 to 200 milliseconds, which avoids retrograde flow.[26]

MECHANISMS OF PACING

Cardiac pacemakers operate by extrinsically depolarizing myocardial cells. The cathode-to-anode potential change is 100 to 1,000 mV compared with the normal intrinsic change of 30 to 50 mV.[16] This energy may be quantified as equivalent to voltage, current, and duration (E = IVT). The energy level required to initiate depolarization is determined by the electrical resistance of the system, the electrode-myocardial junction, and the myocardial milieu.[16] This threshold varies from 1.0 to 5.0 mA for permanent pacing and emergency pacing, respectively. The threshold level is affected by electrode position, maturity, pulse duration, polarity, surface area, composition, and local myocardial condition. Local myocardial condition may be influenced by drug effects, electrolyte imbalance, or hypoxia and hypercapnia.

The pacing stimulus differs between the two types of pacing used today. Transcutaneous pacing uses a square-wave pattern, which is superior to a sinusoid, spike, or rectilinear configuration, and delivers a signal of 50 to 200 mA for 20 to 40 milliseconds at 15 to 100 V. Transvenous pacing uses a triphasic wave consisting of an intrinsic deflection, far-field potential, and current of injury, delivering a signal of 0.1 to 20 mA for 2 milliseconds at 15 V.[28]

Successful delivery of the signal results in a pacing spike or QRS longer than 0.14 seconds followed by a T-wave and accomplished by the loss of the underlying rhythm.[29,30] Three conditions are important for optimal pacing. Heart rate should be controlled to allow adequate time for diastolic filling. The appropriate power should be used to meet, yet not exceed, the threshold necessary for depolarization. Finally, the duration should be adequate to ensure that the stimulus arrives during the "vulnerable period," when the intrinsic rhythm is amenable to capture.

INDICATIONS FOR PACING

Table 1 summarizes the indications for emergency cardiac pacing in 1699 patients from 1971 to 1985.[31-45]

The use of atrial pacing requires an intact conduction pathway. In one series atrial pacing succeeded in 61% of 36 patients, including all those with paroxysmal atrial tachycardia and 81% with atrial flutter, but in none of the patients with atrial fibrillation.[46] The technique includes a cycle length 10 milliseconds shorter than the intrinsic cycle and a progressive rate increase to 400 to 450/min. Two-to-one block of atrial fibrillation results and may be controlled with digitalis.[31,47] In 22 patients with atrial flutter, this approach had a success rate of 16.6% with pacing alone and 100% with procainamide added.[31]

Ventricular pacing is more commonly used for emergency intervention. Of 57 patients this technique was effective in 67% overall, and was more effective with slower cycle lengths (>350 milliseconds) (81% *v* 51%), multiple stimuli (61% *v* 18%), and antiarrhythmic agents to prolong the cycle length to approximately 400 milliseconds.[48] Thus, pacing for ventricular tachycardia is more efficacious for heart rates less than 170 beats/min (80%) than for higher rates.[48] Fisher et al[49] reported a 93% success rate (n = 573) overall in this setting; specifically, 89% for rates less than 200 beats/min,

TABLE 1. Indications for Emergency Cardiac Pacing in Series Collected From 1971 to 1985 (n = 1699)

Indication	Incidence (%)
Sinoatrial node	3.9
Sick sinus syndrome	7.1
Sinus bradycardia	1.9
Sinus pause	1.5
Drug effect	
Digoxin	3.2
Calcium channel blocker	0.3
Beta-blocker	—
Class IA antiarrhythmics*	—
Supraventricular tachycardia	1.7
Atrial flutter	1.5
Atrial fibrillation	3.1
Paroxysmal atrial tachycardia	—
Atrioventricular node	
1st degree AVB	2.2
2nd degree AVB, Mobitz type I	8.7
2nd degree AVB, Mobitz type II†	—
3rd degree AVB†	27.1
Junctional	
Bradycardia‡	0.5
Tachycardia	—
His-Purkinje	
LBBB plus 1st degree AVB†	7.1
RBBB plus left anterior or posterior hemiblock†	0.6
Ventricular	
Asystole	1.7
Ventricular tachycardia/fibrillation	5.0
Idioventricular rhythm	0.6
Torsades de pointes	0.2
Miscellaneous	
Myocardial infarction prophylaxis	16.4
Diagnostic electrophysiologic study	6.0
Post cardioversion	0.8
Permanent pacemaker failure	0.6
Metabolic (increased K+, decreased PO_2)	—
Vagal hyperresponsiveness	—
Trauma	—
Pulmonary artery catheterization with LBBB	—

ABBREVIATIONS: AVB, atrioventricular block; LBBB, left bundle branch block; RBBB, right bundle branch block.
* Vaughan-Singh-Williams classification system.
† Myocardial infarction.
‡ Any symptomatic bradycardia may require pacing.
Data from selected investigations.[31-45]

3% for rates less than 300 beats/min, and 1% for higher rates. He derived a "best fit equation" (rate = VT + 47/0.969 [R = .89]) suggesting the intrinsic ventricular tachycardia rate plus 56 as the pacing frequency. Torsades des pointes due to an "abnormal dispersion of refractoriness" is amenable to therapeutic pacing, which restores homogeneous depolarization.

Myocardial infarction is perhaps the most common indication for prophylactic or therapeutic cardiac pacing. The use of pacing is based on the presence of symptoms or the likelihood of progression based on prior clinical experience. Syncope was suggested to be a decisive symptom in 60% of patients; other symptoms are hypotension, angina, ischemia, pulmonary dysfunction, altered mentation, or the onset of significant ectopy.[5] Syncope is present more frequently in distal (75%) than in proximal (25%) myocardial infarctions, requiring pacing in 100% versus 75%, respectively.[50]

The incidence of conduction deficit in myocardial infarction is substantial (n = 57), with right bundle branch block predominant in inferior lesions (84.6%) and left bundle branch block in anterior lesions (27.7%).[13] A history of prior myocardial infarction, a current anterior wall myocardial infarction, or Killip class III or IV failure predisposes patients to progression of conduction delay.[51] Progression to complete heart block is implicit with trifasicular block and likely with bifasicular block if it is associated with left bundle branch block and right bundle branch block (100%), right bundle branch block or left bundle branch block with first-degree atrioventricular block (38% to 100%), right bundle branch block or left bundle branch block (66.6%), and right bundle branch block along with left anterior hemiblock or left posterior hemiblock (31% to 54.5%).[13,15] Progression is unlikely with unifasicular block (12.7%).[13]

When prophylactic pacing was used in patients with antecedent left bundle branch block undergoing pulmonary artery catheterization, the incidence of new right bundle branch block (n = 150) was 5% with a mean duration of 9.5 ± 3.1 hours and a range of 11 seconds to 17.5 hours.[37] Predisposing events included a prolonged catheter insertion time, hypokalemia, and hemodynamic instability.[37] Thus, it would seem prudent to have transcutaneous pacing available during pulmonary artery catheterization and to be familiar with the Paceport (Baxter-Edwards, Irvine, CA) pulmonary artery catheter.

The contraindications to cardiac pacing are the subject of debate and are best viewed as relative; the technique may be used with caution if warranted by the patient's condition. The use of any form of pacing in hypothermia may precipitate ventricular fibrillation due to myocardial irritability.[52] Bradycardia secondary to hypothyroidism is best treated with hormone replacement, with an auxiliary role for pacing. The use of pacing with prolonged arrest times (30 to 45 minutes) is often unsuccessful. The use of the transvenous pacing route may be unwise in patients who have digoxin toxicity, a prosthetic tricuspid valve, sepsis, or coagulopathy.[53]

METHODS OF CARDIAC PACING

Cardiac pacing may involve mechanical, transthoracic, transvenous, transcutaneous, epicardial, or esophageal routes.

Mechanical Pacing

In 1928 Condorelli[54] described intermittent "fist pacing" over a 2-hour period resulting in the patient's survival.[54] This method is suggested to generate 4 to 5 joules of energy, effecting depolarization. Iseri et al[55] reported a series of canine and human observations comparing fist pacing to cardiopulmonary resuscitation noting improvement in blood pressure (60% *v* 24%) and cardiac output (77% *v* 38%) and a twofold increase in coronary blood flow.

Transthoracic Pacing

The transthoracic route was revitalized by Theverent et al[56] in 1958, but it had major complications and was superseded by transcutaneous pacing. Transthoracic pacing still

may be used in prolonged asystole or other nonviable rhythms. Contraindications include moderate impairment and bleeding diathesis.

A stylet and needle are placed via the parasternal route (beginning at the left fifth intercostal space, entering cephalad to the right costochondral junction at 30°, and advancing 5 to 10 cm) or the left subxiphoid route (directed to the right second costochondral junction at 30°). The stylet is removed, the blood return is assessed, and medications are administered. The entire J wire is then inserted and the needle is removed.

Accurate placement of the pacing wire is more likely via the parasternal route (r = .85) than with the subxiphoid route (r = .46).[57] Brown et al[58] further suggest that the parasternal route is the safest. This route avoids such catastrophic complications as tamponade, found in 4.7% to 16.6% of cases, pneumothorax, and laceration of the internal mammary artery or the liver.[59,60] Transthoracic pacing is predominantly used late in the course of bradysystolic arrest. Tintinalli and White[60] (n = 21) used the technique after failed pressor therapy with electrical capture in 38% and mechanical capture in 9%, but none of their patients survived. White and Brown[61] (n = 48) found that electrical and mechanical capture rates of 23% and 17% could be improved twofold by administration of epinephrine and bicarbonate through the pacing port. Ornato et al[42] (n = 54) found similar results with late application (26.9 ± 17.7 minutes) during arrest; 1.2% of their patients survived, whether transvenous or transthoracic pacing was used. Transthoracic pacing appears to yield results similar to other methods with increased risk, but provides rapid access for intravascular medication.

Transcutaneous Pacing

The modern resurgence of transcutaneous pacing began with Zoll,[6] using the NTP pacemaker. This device is indicated for use in most bradysystolic arrests, because of ease of application, but may be contraindicated in thoracotomy. The apparatus has been improved by increasing electrode surface area (10 to 100 cm^2) and pulse duration (2 to 40 milliseconds). These changes meet the increased threshold requirements in patients with myocardial ischemia.[62]

In a recent comparison of available transcutaneous pacers, Heller et al[63] suggest a superior capture rate (80% v 50%) and threshold (66.5 mA v 104 mA) and less discomfort associated with the Zoll NTP compared with the Life Pack 8 (Physiocontrol, Redmond, WA). Proper electrode position is essential to minimize threshold requirements. Capture is superior with the cathode (negative terminal) in the anterior parasternal position and the anode (positive terminal) in the posterior right or left scapular position.[64]

A study of patients undergoing catheterization paced to 85% of maximal heart rate demonstrated increases in heart rate, arterial venous oxygen difference, oxygen consumption, and central venous, pulmonary artery, and mean arterial pressures; no change in cardiac index and systemic vascular resistance; and a decrease in systolic blood pressure.[65] Angina was induced in 50% of patients, indicating the cost of chronotropic work in the dysfunctional heart.[65]

Clinical trials performed in prehospital and hospital settings suggest that the application of transcutaneous pacers is delayed (24.2 minutes), but a reasonable threshold (57 mA; range, 25 to 107 mA) and duration of therapy (24 minutes; range, 4 minutes to 8.5 hours) may be maintained.[8,43,45,66-73] In 740 patients, electrical capture rate was 76.7%; mechanical capture rate was 20.6%, and cumulative survival was 24.8% (Table 2).

Case control studies have demonstrated no difference in mortality.[67,68,73] The correlation between the initial rhythm and subsequent outcome was variable.[66,67,73] Interestingly, studies using both transvenous and transthoracic pacing found equivalent response rates.[70,71]

Conditions related to decreased efficacy include an increased anteroposterior thoracic diameter requiring a higher threshold (MA = 142).[74] Postoperatively in cardiac patients, increased threshold requirement is correlated with chest closure and increases in cardiac output and core temperature.[8]

Transcutaneous pacing is generally a safe technique, using 1 in 1,000 of the energy of defibrillation.[75] Occasionally it may cause reproducible ventricular tachycardia.[76] Syverud et al[77] in a canine model undergoing transcutaneous pacing

TABLE 2. Results of Transcutaneous Pacing Series Collected From 1983 to 1989 (n = 740)

Reference	Population	n	Capture Rate (%)		Survival (%)
			Electrical	Mechanical	
Dunn and Gregory[45]	Hospital	37	91	—	81.8
Paris et al[66]	Prehospital	105	52	8	0
Hedges et al[67]	Prehospital	202	—	0.9	5.9
Eitel et al[68]	Prehospital	91	93	11	0
Kelly et al[8]	Postoperative	23	100	100	95.6
Olson et al[69]	Prehospital	12	—	8.3	8.3
Madsen et al[70]	Hospital	35	94	94	94
Vukov et al[7]	Aeromedical	23	—	—	52
Falk et al[72]	Prehospital	19	36.8	10.5	0
Zoll et al[43]	Hospital	134	78.3	—	50
Clinton et al[73]	ED	33	—	24.2	15.1
Falk et al[72]	Hospital	26	—	—	7.6
Cumulative			76.7	20.6	24.6

ABBREVIATION: ED, emergency department.

for 4 hours found no electrocardiographic changes, a total creatine phosphokinase increase of 78 units, but an unchanged fraction of MB isoenzyme. However, pathologic analysis of a similar model revealed gross pallor and microscopic myofascial coagulation necrosis in the right (5%) and left (1%) ventricles in all animals.[78] These lesions were not hemodynamically significant.

Transvenous Pacing

Furman and Schwedel,[79] in 1959, initiated the use of transvenous pacing for therapy of Stokes-Adams seizures. This modality may be used for therapy and prophylaxis for all forms of hemodynamically unstable arrhythmias. However, it may be contraindicated in patients with extreme hypothermia (<28°C), which causes ventricular irritability.

The transvenous pacing electrode may be rigid (5 to 7 french), which is placed under fluoroscopic guidance, semirigid (4 french), or balloon-tipped (6 french), which requires adequate blood flow for placement.[18] The lead configuration may be unipolar or bipolar with distal pacing and proximal sensing leads. More complex quadripolar or hexapolar varieties allow simultaneous sensing of intracardial atrial and ventricular electrocardiograms. A J-shaped distal tip ensures wall contact or specific atrial placement; a stylet-tipped version is used in tricuspid disease.[18]

Transvenous pacemakers may be placed by several routes. Successful placement has been demonstrated in 67% of 142 cases, with a mean insertion time of 2 minutes (range, 40 seconds to 30 minutes).[40,80] The right internal jugular route is recommended for low-flow states and has the highest rate of placement (40%).[81] The right subclavian route is also successful (30% to 98.9%), followed by the left subclavian (20%) and the internal jugular (10%) approaches.[81,82] The Paceport (Baxter-Edwards) pulmonary artery catheter has been used for pacing with an 80% success rate (n = 10) and insertion times of 30 seconds to 10 minutes.[83] However, a high threshold (MA = 3.6) requirement and difficulties with hemodynamic stability were encountered.[83]

If time allows, intracardiac monitoring should be used during transcutaneous pacing. In the method described by Bertrand et al[84] the V lead of the electrocardiograph is attached to the distal pacing probe to act as a sensing electrode. The five-stage pattern includes a preatrial stage (p inverted), an atrial stage (p deep), a postatrial stage (p upright), a ventricular stage (QRS), and a current of injury (ST elevation) indicating wall contact.[85] More simply, these changes may be designated as the atrial (PQ elevation) and the ventricular (ST elevation) phases. An amplitude decrease indicates passage of the pacing wire into a neck vein, negative polarity designates placement in the inferior vena cava, and positive polarity suggests positioning in the coronary sinus.

Under emergency conditions, intracardiac monitoring may be performed by a flow-directed or fluoroscopic technique. The lead is advanced until it makes contact. The asynchronous pacing mode is used in ventricular stand-still, whereas the demand mode may be used if an intrinsic escape rhythm is present to avoid competing with the rhythm and generating arrhythmias. Energy is then increased from 1 to 20 mA or until capture. The threshold is identified by reversing this energy progression until capture is lost. The energy level is then returned to the point of capture, and 1.0 mA is added as a safety margin. Placement is confirmed radiographically. A right posterior position should be noted on pulmonary artery and lateral films, respectively. Catheters (n = 36) placed 35 cm from the right internal jugular position were found most commonly in the right atrium (50%), followed by the right ventricle (28%). Right ventricular placement provided the highest rate of capture.[86]

Cardiac pacing modes used for atrial ventricular tachycardia allow placement of an extrinsic depolarization signal into a vulnerable period of the intrinsic rhythm, the "tachycardia termination zone."[87] The underdrive mode, which is at 75% of the intrinsic rate, may be used for ventricular tachycardia.[87] The overdrive mode, at 125% to 135% of the baseline, is used for atrial ventricular tachycardia. Burst stimuli or a rapid, grouped sequence of six to eight beats is often successful.[87] The extra stimuli mode is used with a known coupling interval.[87] Automatic decremental or incremental programs allow progressive decrease or increase in cycle length of the pacing stimuli.[87] Specifically programmed PASAR sequences may be used as an adjunct to electrophysiologic testing.[87]

Pacemaker malfunction may be noted as a loss of capture, sensing, or pacing artifact.[88] Loss of capture is seen as a pacing spike without a QRS complex. It is due most commonly to lead displacement, or to myocardial metabolic or perfusion abnormalities, postcardioversion block, or the use of intraarterial or intracardiac antiarrhythmics. Loss of sensing results in uncoordinated extrinsic and intrinsic activity. Undersensing occurs when failure to detect a low-amplitude R wave causes excessive extrinsic firing. Oversensing occurs when the pacemaker is suppressed by nonsignificant electromagnetic discharge resulting in absence of pacing. Finally, loss of pacing artifact signifies a mechanical problem with the pacemaker-myocardial circuit. For emergency pacing maximal energy (MA = 20) should be delivered in the asynchronous (VOO) mode. Proper lead contact should be assured by radiography, and the patient should be placed in the right lateral decubitus position. In bipolar systems, polarity can be reversed to allow pacing of the operational probe. If the system fails, the generator should be replaced, and transcutaneous pacing should be used.

In clinical trials transvenous pacers have been placed for myocardial ischemia in 48.6% of patients, for atrioventricular block in 29.9%, for asystole in 6.7%, and for complete heart block in 6.4%.[30,80,82,89-92] In several series, an overall capture rate of 78.8% was followed by survival in 55.9% (Table 3). Compared with transcutaneous pacing, transvenous pacing yields an equivalent rate of electrode capture (77% *v* 79%) and greater survival (56% *v* 25%), which likely reflects patient selection.

In a controlled study, outcome did not improve when patients (n = 52) were transferred to transvenous pacing after failed transcutaneous pacing. Survival rates for the two methods were 2% and 8%, respectively.[30] Studies examining concurrent use of these two methods suggest that transvenous pacing produces better hemodynamics and rates of arrhythmia termination.[81,93] Transvenous pacing also proved to be equivalent to transvenous cardioversion.[94] Transfer requires an operating energy of 2 to 2.5 times the threshold level, or less than 10 mA for transvenous pacing and less than 100 mA for transcutaneous pacing.[64]

TABLE 3. Results of Transvenous Pacing in Series Collected From 1970 to 1990 (n = 675)

Reference	Population	Diagnosis	n	Capture Rate (%)	Survival (%)
Bartecchi et al[89]	Elderly	MI (48.6)	36	100	55.5
Macaulay and Wright[82]	Hospital	CHB (30.7)	91	100	74.7
Davis[80]	Hospital	CHB (65)	20	95	60
Abinader et al[90]	Hospital	AVB (46.5)	339	81	—
Rosenberg et al[91]	Hospital	AVB (33.3)	98	—	82.6
Hazard et al[92]	Hospital	Asyst (33.3)	39	62	20.5
Dalsey et al[30]	ED	Asyst (57.6)	52	15	8
Cumulative				78.8	55.9

ABBREVIATIONS: MI, myocardial infarction; CHB, complete heart block; AVB, atrioventricular block; Asyst, asystole; ED, emergency department.

The overall incidence of complications in several series (n = 1533) is 18.5%.[40,41,95,96] The most common occurrence is lead dysfunction in 30% (Table 4), which is strongly correlated with the access route used. The antecubital route is most often (30% to 37%) and the internal jugular route is least often (15.3%) associated with malfunction.[41,96] Lead malfunction tends to occur early in therapy, with 50% of instances within 24 hours and 86% within 48 hours.[96]

Arrhythmia is the second most common complication (6.6%). Predisposing factors include acute myocardial infarction, right ventricular infarction, and central versus peripheral placement.[90,97,98] Ventricular ectopy includes premature ventricular contractions in 28.8%, ventricular tachycardia in 12.8%, and ventricular fibrillation in 5.6% with myocardial infarction.[90] New-onset right bundle branch block occurs in 3%, but not in patients with pre-existing left bundle branch block (0%) (n = 279).[37] Deep venous thrombosis occurs in 34% of stable and 85% of unstable patients, and subsequent pulmonary embolism is diagnosed in 60%.[99]

Problems with the generator include the "runaway pacemaker" or pacemaker-induced ventricular tachycardia. Two cases, caused by body fluid leakage and a short circuit, were reported in patients with VVI pacemakers.[100] Therapy includes decreasing the delivered energy and placing a magnet over the pacemaker to convert it to fixed-rate mode. Alternatively, the pacemaker may be disconnected and replaced by transcutaneous pacing. Antiarrhythmics and defibrillation may be ineffective.[101] Pacemaker syndrome due to atrial-ventricular asynchrony may occur. Therapeutic radiation doses administered in malignancy have rendered certain pacemakers inoperative.[102] Hysteresis, or a programmed firing delay based on the preceding intrinsic impulse, may cause sinus pauses.[103] Electrode malfunctions include perforation of the low-pressure right ventricle, causing the paced left bundle branch block to become a right bundle branch block pattern. The symptoms may include focal motor twitching, imitating seizure activity, or diaphragmatic stimulation.[104] A chest radiograph is diagnostic in 3% to 14% of cases.[105] Posttraumatic electrode fracture and subsequent failure have been reported.[106] Pacemaker-induced pericardial friction rub disappears when the electrode is removed.[107]

TABLE 4. Complications of Transvenous Pacing in Series Collected From 1969 to 1984 (n = 1533)

Complication	Incidence (%)
Complications	18.5
Lead dysfunction	30.0
Generator failure	9.3
Arrhythmia	6.6
Pericardial rub	5.3
Pulmonary embolism	4.7
Perforation	3.7
Hematoma	3.5
Poor connection	2.0
Infection	1.4
Phlebitis	1.1
Arterial puncture	1.0
Diaphragmatic stimulation	0.9
Pneumothorax	0.1

Data from selected investigations.[40,41,95,96]

Epicardial Pacing

Epicardial pacing, first described by Hyman,[5] is indicated when the heart is exposed intraoperatively or in trauma. A bleeding diathesis theoretically contraindicates its use. The technique involves surgical fixation of electrodes approximately 2 cm apart on the right atrium and ventricle.

In 94% of postoperative cardiac patients (n = 44) with hemodynamic instability, epicardial pacing increased cardiac output from 3.17 ± 0.10 to 4.17 ± 0.20 L/min.[108] The technique is also used for supraventricular tachycardia (9%), heart block (6%), and arrhythmia diagnosis.[108] The capture rates for transvenous and epicardial pacing are 71% and 97%, respectively.[109]

Esophageal Pacing

The esophageal route provides a less invasive means of atrial pacing for diagnosis and therapy. It is contraindicated when nasogastric or orogastric access is not possible. The apparatus consists of a pill or semi-rigid bipolar electrode. The proximal atrial (low-output) lead is separated by 15 to 30 mL from the distal ventricular (higher-output) electrode. The device is inserted to the point of maximal atrial sensing. Esophageal pacing has been used successfully (80%, n = 5, and 100%, n = 7), but in small numbers of patients.[61,110]

Permanent Pacing

The VVI pacemaker, described by Leatham et al,[111] is indicated when symptoms continue during temporary pac-

ing. Permanent implantation is contraindicated by active infection.

The electrode may be implanted via the coronary transvenous or routine intracardiac route. The generator is based on a primary cell composed of mercury or zinc, a secondary cell composed of rechargeable nickel and cadmium, a solid-state unit constructed of lithium, or a nuclear-powered source.[112] Early types of generator failed precipitously, whereas later models exhibit a gradual decline in rate.[113]

In an emergency the type of pacemaker should be ascertained, if possible, to allow appropriate intervention or reprogramming. Parsonnet et al[114] suggested the ICHD three-code classification system, designating position I as the chamber paced, position II the chamber sensed, and position III the mode of response. They later expanded this code to include positions IV, programmable functions, and V, antiarrhythmic functions.[115]

This classification system has been used to label the common pacemaker models.[44,116] The synchronous (VVO) pacemaker was the first available and is appropriate for complete heart block without premature ventricular contractions. The ventricular (VVI) pacer is most widely used, and offers both positive and negative hysteresis. The ventricular trigger (VVT) version converts abnormal intrinsic depolarization of an adequate rate into a normal waveform, but is subject to electromagnetic interference. The atrial synchronous (VAT) pacer generates an atrial contribution to cardiac output in young patients with an adequate atrial rhythm. The atrial-ventricular synchronous (VDD) pacer senses both chambers but paces the ventricle when atrial conduction is abnormal. The atrial-ventricular sequential (DVI) is the most efficient model but requires an adequate atrial rate. The select atrial pacers (AAI, AAT) require coronary sinus implantation in sick sinus syndrome and allow rapid atrial rhythm to suppress ectopic beats. Last, the most versatile variant (DDD) senses and paces all chambers sequentially.

The 1-year failure rate of permanent pacemakers is 7.4% to 15%. Most failures occurred within 30 days of implantation.[117,118] Failure was associated with dual-chamber pacing (62% *v* 35% for single-chamber pacing), use of a bipolar electrode, a large right ventricle, an energy threshold greater than 0.5 mA, and a paced QRS-ST deviation less than 2.0 mm.[117,118] Difficulties encountered include inadequate sensing in 32%, usually due to electrode dislocation (6.8%), battery failure and muscle stimulation in 17%, and inadequate capture (12%).[118,119]

Complications are discovered by patient reports of recurrent symptoms and physical examination findings of infection or disconnection. Pacing dysfunction may occur due to loss of capture with absence of a pacing spike, sensing with rate competition, and absence of a pacing artifact, suggesting mechanical difficulties. Chest radiography allows confirmation of electrode position. Placement of the magnet perpendicular to the generator converts the system to a more reliable, fixed ventricular pacing mode.

CURRENT ISSUES

Several aspects of pacing in cardiac emergencies bear discussion and further investigation.

The literature regarding the use of transcutaneous pacing in pediatric patients is sparse. The most common pediatric arrhythmias are paroxysmal ventricular tachycardia and complete atrioventricular block, both of which are amenable to pacing in adults.[120] The most common pacing routes in children are the endocardial and transesophageal routes.[121] The use of transcutaneous pacing in children, although unproved, is reportedly efficacious for overdrive pacing of tachyarrhythmias.[121,122] Its safety and ease of use suggest that transcutaneous pacing should be attempted for brady-tachyarrhythmias in children.

The use of transcutaneous pacing in patients outside the hospital has been debated. Early use of transcutaneous pacing may provide the best chance of survival for patients with amenable arrhythmias.[123] The modality is safe, easily used, has minimal side effects, and is well tolerated by conscious patients.[124] Thus, transcutaneous pacing should be available for field care or interhospital transport. Isolated evidence suggests that electromagnetic interference during helicopter transport may disrupt pacemaker function.[125] However, a clinical trial in 297 patients found a 48% transvenous and a 43% transcutaneous capture rate during helicopter transport in patients with arrhythmias refractory to drug therapy.[71]

Pacing techniques are commonly used for atrioventricular block and tachyarrhythmias. The use and timing of pacing for bradysystolic cardiac arrest are less certain. A prehospital clinical trial suggests that pacing speeds the return of circulation and increases survival.[126] This study also found that pacing begun in patients without a palpable pulse resulted in no survivors.[126] In trials of pacing in the emergency department and in hospitalized patients, the highest success rate was observed with bradycardia due to atrioventricular block, and essentially 100% mortality was seen after asystole.[127,128] Thus, if used in pulseless, asystolic patients, pacing must be started promptly.

Recently, the use of transcutaneous pacing for tachyarrhythmias has been explored. Endocavitary recording found that simultaneous ventricular and atrial pacing is possible, with atrial capture occurring at higher thresholds. The findings suggest that this mode may be useful in the treatment of tachyarrhythmias.[129] A clinical trial in 31 patients demonstrated successful pacing of supraventricular (73%) and ventricular (40%) tachycardias by overdrive and underdrive mechanisms in 67% and 29%, respectively.[130] However, malignant ventricular arrhythmias were induced in 6% of cases.[130]

Cardiac pacing may present therapeutic dilemmas concerning cardiac pacing in patients with acute myocardial infarction. Myocardial infarction may be complicated by ventricular fibrillation (57%), asystole (20%), and atrioventricular block (10%).[131] Patients with suspected myocardial infarction may be classified into high- and low-risk groups based on certain clinical criteria. More common in the high-risk groups are overall complications (64% *v* 26%), malignant arrhythmias (11% *v* 0.9%), mechanical intervention such as pacing (20% *v* 2%), and mortality (8.2% *v* 0.4%).[132] The need for pacing may also be predicted by the location of the infarction (62% for anterior and 29% for inferior), which reflects the extent of involvement of the conduction system.[133] Also, late onset of arrhythmia (>4 hours after infarction) requires pacing in 76% of cases, indicating more serious structural involvement than found in initial "stunned myocardium."[133]

Therapies used for myocardial infarction often predispose the patient to arrhythmias best treated by cardiac pacing. Bradyrhythmias are common after defibrillation for ventricular arrhythmias.[134] The administration of thrombolytic agents should lower the threshold for prophylactic pacing, because reperfusion is associated with the onset of bradycardia or tachyarrhythmia.[135] These arrhythmias are felt to be due to bradycardia and hypotension (the Bezold-Jarisch cardioinhibitory reflex), which may follow recanalization of the right coronary artery (65%) and resultant reperfusion of the inferoposterior wall.[136] Clinically, the administration of the recombinant tissue-type plasminogen activator to patients with documented coronary thrombosis increased the ischemic pacing threshold.[137] Thus, prophylactic pacing should be instituted for patients with late onset anterior wall myocardial infarction and Mobitz type II conduction delay or progressive atrioventricular block.

In general, transcutaneous pacing should be instituted initially, because of ease of application, safety, and efficacy. Among patients with myocardial infarction, although no serious side effects of transcutaneous pacing occurred, 27% required sedation and 88% had increased chest pain during the procedure.[138] Although the efficacy of converting from transcutaneous to transvenous pacing for refractory arrhythmias has not been well documented, hemodynamic status often improves.[7,30,81,93] Thus, to avoid pain and the resultant sympathetic discharge in patients with myocardial ischemia, conversion to transcutaneous pacing should be attempted when patients in critical care require pacing for more than 12 hours.[138] However, the slightly better capture rate and patient comfort must be balanced against the 5% incidence of arrhythmias generated by transvenous catheter placement.[81,90,93]

CONCLUSION

The utility of cardiac pacing in the emergency setting is well described. Cardiac pacemaking is primarily instituted for symptomatic arrhythmias, such as atrioventricular block, atrial tachyarrhythmias, ventricular tachyarrhythmias, and prophylactically, in the setting of myocardial infarction or pulmonary artery catheterization.

Transcutaneous pacing may be substituted when there are contraindications to the transvenous route due to myocardial irritability, such as digoxin toxicity, drug overdose, and hypothermia. There is subtle but inconclusive evidence to suggest the superiority of transvenous to transcutaneous pacing in the acute setting. However, the early use of pacing leads to a better outcome than does pacing instituted late in the patient's course.

The author expresses sincere appreciation to Devera Davis for manuscript preparation.

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