Rapid overdrive pacing for refractory tachyarrhythmias in patients after open-heart surgery

The efficacy of rapid ventricular pacemaker overdrive in the treatment of supraventricular and ventricular tachyarrhythmias is presented as a new approach to the management of these rhythm disorders in patients after cardiac surgery. This mode of therapy is exemplified in the control of heart rate and return of normal sinus rhythm in patients with both types of tachyarrhythmias refractory to conventional antiarrhythmic agents. In addition, the pathogenesis and mechanisms of pacemaker overdrive in terminating these rhythm disturbances are delineated.

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Although ventricular and supraventricular tachyarrhythmias, usually manageable by standard pharmacologic agents, frequently occur after cardiac surgery, rapidly deteriorating hemodynamic function owing to rhythm disorders refractory to conventional antiarrhythmic agents are occasionally encountered. Recently, in two individuals who had had open-heart surgery, rapid overdrive pacing at rates in excess of the intrinsic rhythms was the only successful method of terminating these life-threatening ectopic electrophysiological disturbances. Further continued pacing at moderately elevated rates was necessary to avoid the re-emergence of tachyarrhythmias. The effectiveness of pacemaker overdrive suppression of electrical irritability in these patients provides new perspective for the management of refractory tachycardia when conventional pharmacologic and electric countershock techniques do not rapidly terminate potentially lethal arrhythmias after cardiac surgery.

Case reports

CASE 1. A 47-year-old man with rheumatic aortic valve disease had developed severe aortic regurgitation following bacterial endocarditis 20 years previously. Two years prior to the present admission, he began having episodes of dizziness, syncope, and increasing dyspnea. The resting electrocardiogram (Fig. 1, A) showed normal sinus rhythm, left axis deviation, and left ventricular hypertrophy with strain. Cardiac catheterization revealed an elevated pulmonary artery pressure of 35/16 mm. Hg, a mean pulmonary capillary wedge pressure of 19 mm. Hg, a left ventricular end-diastolic pressure of 20 mm. Hg, and a cardiac index 2.88 L. per minute per square meter. An aortic root angiogram demonstrated marked aortic regurgitation, left ventricular hypertrophy with dilatation, and reduced ejection fraction of 40 per cent without mitral regurgitation. Findings from selective coronary arteriography were normal. The patient then underwent aortic valve replacement with a No. 27 Björk-Shiley aortic prosthesis with the aid of regional cardiac (4° C. Ringer's lactate solution) and systemic (30° C.) hypothermia. On the first postoperative day, a sustained rapid arrhythmia at 200 beats per minute with wide QRS complexes, and hypotension developed (Fig. 1, B). Carotid sinus massage and maximum therapeutic doses of levarterenol, lidocaine, diphenylhydantoin, propranolol, and propranolol were ineffective in controlling the rhythm disorder. At this point the patient's hemodynamic status was deteri-
Rapid pacing for refractory tachyarrhythmias

**CASE I**

Fig. 1. Representative electrocardiograms from Case 1. Panel A (preoperative) is the standard twelve-lead scalar electrocardiogram showing normal sinus rhythm, left axis deviation, and left atrial and left ventricular hypertrophy with strain. Panel B obtained in the immediate postoperative period demonstrates a regular tachycardia (rate 220 beats per minute) with wide QRS complexes which could represent either supraventricular tachycardia with bundle branch block or ventricular tachycardia.

orating and overdrive ventricular pacing by the temporary epicardial electrode implanted during the operation was instituted. Initially this therapeutic approach was not able to abolish the tachyarrhythmia because the first external pulse generator (Medtronics Model 5880A) used was capable of delivering impulses up to only 150 per minute. However, a special R wave-synchronized pulse generator (Medtronics Model 5837) with a maximum rate of 600 impulses per minute was successful in capturing ventricular rate by electrically pacing at 215 impulses per minute. In this manner the rapid rhythm was converted to a controlled, paced rate which was gradually reduced to 110 per minute with immediate improvement in hemodynamics. Because of a high pacing threshold of the epicardial electrode, a transvenous bipolar pacing electrode was positioned in the right atrium. When endocardial pacing from different atrial sites was unsuccessful, the electrode was advanced to the apex of the right ventricle and capture was easily achieved at an acceptable lower current threshold. Following further reduction of the ventricular paced rate, termination of external pacing allowed re-emergence of normal sinus rhythm at a rate of 80 beats per minute (Fig. 2, A). A twelve-lead electrocardiogram showed right bundle branch block and left anterosuperior hemiblock pattern (Fig. 2, B), similar to the QRS configuration observed during the tachyarrhythmias, (Fig. 1, B), which suggested that the original tachyarrhythmia was supraventricular in origin. The patient was then treated with digoxin, quinidine, and diphenylhydantoin for arrhythmia prophylaxis, and his subsequent postoperative convalescence was without untoward events.

**CASE 2.** A 49-year-old man was hospitalized with an acute extensive anterior myocardial infarction complicated by cardiac arrest. Following successful resuscitation, the patient was in hemodynamically stable condition until the eighth
hospital day, when suddenly he developed repeated episodes of ventricular tachycardia and fibrillation requiring repeated electrical cardioversion. Because of recurrent refractory tachycardias accompanied by marked pump dysfunction, cardiac catheterization was performed. Pulmonary arterial pressure was $42/20$ mm. Hg, mean pulmonary wedge pressure $19$ mm. Hg, left ventricular end-diastolic pressure $22$ mm. Hg, and the cardiac index was $2.38$ L. per minute per square meter. Coronary cineangiograms revealed complete obstruction of the proximal left anterior descending coronary artery proximal to the first septal perforator without collateral filling of the vessel distally. The circumflex and right coronary arteries were not diseased. Left ventriculograms demonstrated a large anterolateral dyskinetic area and markedly reduced ejection fraction of $37$ per cent without evidence of mitral regurgitation.

Because several episodes of ventricular tachycardia and fibrillation followed catheterization despite intensive antiarhythmic therapy, emergency left ventricular infarctectomy was performed. An estimated $40$ per cent of the left ventricular muscle mass, involving mainly the anterolateral segment, appeared to be infarcted. An extensive area of the anterior wall of the left ventricle from the septum to the lateral border of the anterior wall was resected. Opening the left ventricle disclosed that the transmural infarction extended to but did not include the papillary muscles. Further, a fresh thrombus was removed from the left ventricular apex. It was not possible to remove the entire infarcted myocardium, because an adequate left ventricular chamber had to be preserved. On completion of the infarctectomy and after cessation of cardiopulmonary bypass, epicardial ventricular pacing and vasopressor agents were necessary to maintain a systolic blood pressure of $90$ mm. Hg.

During the postoperative period, episodes of ventricular tachycardia returned and overdrive epicardial ventricular pacing was initially ineffective because of high current threshold. Therefore, a transvenous right ventricular electrode was inserted which did successfully capture the ventricle and was able temporarily to suppress the irritability. Diagnostic electrograms of the His bundle were performed to identify the ectopic site; this study revealed that the arrhythmia was indeed ventricular in origin (Fig. 3). Pacemaker capture was then achieved at a rate of $150$ beats per minute (Fig. 4, Panel A). Gradual reduction of the paced rate to $110$
Rapid pacing for refractory tachyarrhythmias

Fig. 3. His electrogram with simultaneously recorded standard scalar Leads I, II, and V₆ are shown from Case 2 during the postoperative arrhythmia. The His bundle spikes (H) occurring before QRS complexes in beats 1, 2, and 5 indicate that these beats are normally conducted from the sinus node through the atrioventricular node and His-Purkinje system, and identify a QRS complex as being sinus or supraventricular in origin. In contrast, the ectopic irregular tachyarrhythmia is defined as ventricular in origin since the QRS beats 3, 4, 6, and 7 occur without a prior His spike.

Discussion

Rhythm disorders after cardiac surgery occur frequently because of varying degrees of hypoxia, increased intramyocardial tension, altered membrane permeability, and disturbed cellular electrolyte balance. These pathological disturbances may result in abnormalities of either impulse formation or propagation and thereby provoke tachyarrhythmias.

The surface electrocardiogram alone provides limited information concerning the pathogenesis and perpetuation of rhythm disturbances. In Case 1, although the tachyarrhythmia was refractory to pharmacologic therapy, rapid overdrive ventricular pacing for even relatively brief periods was able to restore the sinus rhythm (Fig. 2, A). This sequence of events strongly supports the hypothesis that the supraventricular tachyarrhythmia in this patient was re-entry in origin. The short train of stimuli through retrograde invasion of the atrioventricular node appeared to interrupt the delicate balance of abnormal impulse conduction necessary for perpetuation of the circus mechanism. It should be noted that a brief period of ventricular pacing would not be expected to eliminate an irritable focus of impulse formation which would recur spontaneously after cessation of such a short duration of rapid electrical stimulation.

The phenomenon of sudden spontaneous re-emergence of ventricular extrasystoles after discontinuation of pacemaker overdrive was seen in Case 2 (Fig. 4). In contrast to Case 1, grouping of ectopic beats observed in this patient suggested an irritable focus of abnormal impulse formation with periods of exit block. Further, the difference in QRS morphology during the tachyarrhythmia favored multifocal areas of irritability (Fig. 4). The electrophysiological disturbance associated with increased automaticity exemplified in this patient therefore required continuous overdrive suppression of the ectopic foci.

Microelectrode analysis of abnormalities of impulse formation has shown that the fundamental disturbance in this type of tachyarrhythmia resides in the cellular membrane. Thus the slope of phase 4 depolarization of the action potential is increased, and this results in enhanced ectopic automaticity (Fig. 5, A). Less frequently increased automaticity may result from factors which increase the resting membrane potential or decrease the threshold potential. The ventricular tachyarrhythmia manifested in Case 2 is representative of abnormal impulse generation (Fig. 5, A).

In contrast, abnormalities in electrical wave propagation may result in the re-entrant type of tachyarrhythmias owing to slowed conduction and unidirectional block involving the atrioventricular node (Fig. 5,
Fig. 4. Postoperative continuous electrocardiogram. Lead V1 from Case 2, demonstrating control of the ventricular tachyarrhythmia by rapid pacemaker overdrive suppression (strips A, B, and C). In strip D, cessation of pacing resulted in resumption of the multifocal ectopic ventricular arrhythmia identified by the phenomenon of group beating (strips D, E, and F).

B) or Purkinje network (Fig. 5, C) which allow retrograde activation of the blocked pathway. The supraventricular tachyarrhythmia observed in Case 1 is representative of the re-entry mechanism shown in Fig. 5, B. This re-entry phenomenon is thought to be the basis for onset and persistence of most ventricular and supraventricular tachycardias. In ventricular rhythm disturbances the re-entry circuit consists of Purkinje fibers and ventricular muscle cells (Fig. 5, C). This latter mechanism of tachyarrhythmia is referred to as micro-re-entry because it usually involves minute areas of ventricular myocardium and therefore is difficult to identify anatomically. On the other hand, the supraventricular re-entry phenomenon is termed macro-re-entry because it usually involves a considerably larger pathway consisting of the atrioventricular node and an adjacent area of atrial myocardium (Fig. 5, B).

In the management of tachyarrhythmias based on enhanced automaticity (repetitively firing focus), it is highly unlikely that an isolated electrical pacemaker stimulus of short duration would be effective in suppressing the extrasystoles. Instead, continuous pacemaker overdrive stimulation of the ectopic focus is necessary to suppress the irritable myocardium (Fig. 4). Ectopic irritable foci appear to be less sensitive to overdrive suppression by pacing than re-entry pathways within the conduction system. Consequently, following abrupt cessation of rapid electrical pacing, re-entrant arrhythmias remain suppressed longer than automaticity-induced tachyarrhythmias, so that the dominant normal pacemaker activity of the heart can resume. In addition, gradual slowing of prolonged rapid overdrive pacing in tachyarrhythmias owing to abnormal impulse formation allows normal sinus rhythm to return.

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Fig. 5. Diagrammatic representation of the mechanisms involved in ventricular and supraventricular tachyarrhythmias. Panel A demonstrates the mechanism of enhanced automaticity with increased diastolic depolarization (phase 4 portion of the intracellular action potential). The superimposed dashed action potentials illustrate the increased rhythm frequency produced by this phenomenon, which occurred in Case 2. Panel B shows the re-entry mechanism of supraventricular tachycardia which generated the rhythm disorder in Case 1. The area of unidirectional block within the (atrioventricular) node is shown by the double-dashed horizontal lines terminating antegrade conduction (solid lines) at the bifurcation of the conduction pathways. The retrograde re-entry conduction is indicated by the broken arrows. Panel C illustrates the re-entry mechanism characteristic of ventricular tachyarrhythmias. The area of unidirectional block within limb B of the Purkinje network is indicated by the double-dashed lines. The retrograde re-entry circuit is completed by the wavy lines in limbs A, M, and B.

REFERENCES