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Hypothermia for the Treatment of Postsurgical Greatly Accelerated Junctional Ectopic Tachycardia

STEPHEN E. BASH, MD, FACC,* JITENDRA J. SHAH, MD, FACC,*
WILLIAM H. ALBERS, MD, FACC,* DALE M. GEISS, MD†

Peoria, Illinois

Three infants developed greatly accelerated junctional ectopic tachycardia with a heart rate >200 beats/min after open heart surgery. When the heart rate exceeded 200 beats/min for 5 hours, all the infants had congestive heart failure and clinical signs of low cardiac output. Conventional therapy (cardioversion, lidocaine, verapamil, digoxin and ice to face) has been shown in the past to be unsuccessful in controlling the heart rate.

Because hypothermia is known to decrease automaticity of the heart, these patients were treated with induced hypothermia. The goal was to arbitrarily decrease the junctional ectopic rate to <180 beats/min to increase cardiac filling time. The duration of the junctional ectopic tachycardia >180 beats/min ranged from 0.5 to 17 hours after cooling began. The duration of the hypothermia ranged from 4 to 24 hours. Spontaneous reversion to sinus rhythm occurred either during the hypothermia or shortly thereafter in all three patients. The blood pressure and urinary output remained stable during hypothermia.

Hypothermia is an effective means of controlling the rate of greatly accelerated junctional ectopic tachycardia after open heart surgery in infants. Although hypothermia does not convert junctional ectopic tachycardia to sinus rhythm, it slows the rate to a more acceptable level, allowing the infants' survival and eventual recovery of sinus rhythm.

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Various arrhythmias may occur in the immediate postoperative period after open heart surgery in infants and children. Most of the arrhythmias are treatable. However, when junctional ectopic tachycardia occurs at a rate >200 beats/min, hypotension, shock and death may ensue (1,2). Treatment with medication usually has been unsuccessful (2–5) and cardioversion has no effect. However, the tachycardia will most often spontaneously resolve if the infant can survive the postsurgical period.

Methods

Definition and diagnosis. Greatly accelerated junctional ectopic tachycardia is defined in this presentation as a narrow QRS tachycardia with a ventricular rate >200 beats/min. When atrioventricular (AV) dissociation is present, the sinus rate is slower than the junctional rate. This rhythm is assumed to be an automatic focus type of supraventricular tachycardia. Figure 1 demonstrates some of the features of this arrhythmia; the upper panel reveals a slightly irregular narrow QRS tachycardia with a rate of approximately 260 beats/min; P waves are difficult to identify. In the middle panel, it is again difficult to detect P waves. This patient, however, had temporary atrial pacing wires, and an atrial electrocardiogram (ECG) (lower panel), which demonstrated AV dissociation, was obtained. The atrial rate is 136 beats/min, which is slower than the ventricular rate.

However, junctional ectopic tachycardia may occur with AV association if 1:1 retrograde conduction is present. This may make the diagnosis more difficult, because the differential diagnosis then includes reentrant forms of supraventricular tachycardia that would respond to overdrive pacing, AV node blocking maneuvers (both mechanical and pharmacologic) and cardioversion. Sinus tachycardia with first degree AV block must also be excluded, because it may respond to fever control, volume loading or reduction of adrenergic drug infusion.

Clinical features. We report our experience with three infants who developed greatly accelerated junctional ectopic

From the *Department of Pediatrics, Division of Cardiology, and †Department of Surgery, University of Illinois College of Medicine at Peoria, Peoria, Illinois. This work was presented in part at the 1986 Annual Meeting of the American Academy of Pediatrics, Section of Cardiology, Washington, D.C., 1986.

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Address for reprints: Stephen E. Bash, MD, University of Illinois College of Medicine at Peoria, Department of Pediatrics, 530 N.E. Glen Oak Avenue, Peoria, Illinois 61637.

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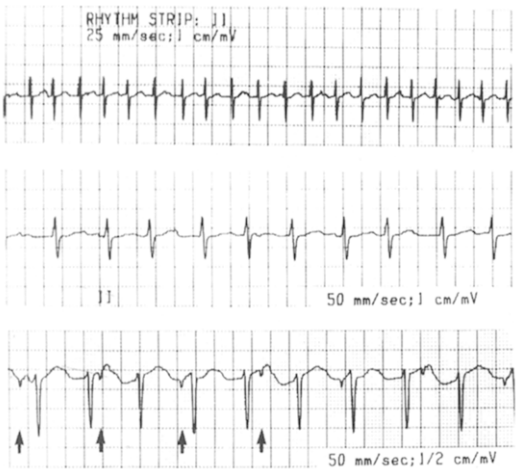

Figure 1. Patient 3. Greatly accelerated junctional ectopic tachycardia. **Upper panel,** A narrow QRS tachycardia with a rate of approximately 260 beats/min is demonstrated; P waves are not readily identified. **Middle panel,** Paper speed increased to 50 mm/s; P waves are still difficult to identify. **Lower panel,** Atrial electrogram using temporary epicardial pacing wires reveals atrioventricular dissociation with an atrial rate of 136 beats/min, which is slower than the ventricular rate of 230 beats/min.

tachycardia after open heart surgery between December 1984 and January 1986 (Table 1). Their ages ranged from 2 days to 4 months. Deep hypothermia and circulatory arrest were used during surgery. The circulatory arrest time ranged from 38 to 78 minutes (mean 60). Hypothermia during surgery, at the lowest rectal temperature, ranged from 12 to 19°C (mean 14.6); esophageal temperature ranged from 9 to 13°C (mean 11).

Preoperatively, all infants had sinus rhythm. Immediately postoperatively, all had sinus rhythm with episodes of accelerated junctional ectopic tachycardia and AV dissociation. Over a period of time, the junctional ectopic rate increased (Fig. 1 and 2). The period of greatly accelerated junctional ectopic tachycardia with a rate period >200 beats/min began 2 to 72 hours after surgery (Table 1). The maximal junctional ectopic rate ranged from 212 to 253 beats/min. Patient 1 had a fever (temperature 39.1°C) at the onset of the accelerated tachycardia; the other two patients were afebrile.

In an attempt to convert or slow the tachycardia before hypothermia, lidocaine (Xylocaine) and digoxin were given to Patient 1 with no effect. Patient 2 received ice to the face and Patient 3 underwent unsuccessful cardioversion three times and received ice to the face and digoxin with no change. Atrial and ventricular pacing were performed and would capture the atrium or ventricle but only at rates greater than the tachycardia. The underlying rhythm was not changed.

Induced hypothermia. These infants all showed clinical signs of poor cardiac output and peripheral vasoconstriction after 5 hours of greatly accelerated junctional ectopic tachycardia. Because hypothermia is known to decrease automaticity of the heart, and because of our poor clinical experience with previous postoperative patients with this arrhythmia, these patients were treated with induced hypothermia. The goal was to arbitrarily decrease the junctional ectopic rate to <180 beats/min to improve cardiac output by increasing cardiac filling time. Cooling was achieved by using two cooling blankets, one on top and one underneath the infant. Esophageal and rectal temperatures were recorded. Paralysis was not induced. Morphine sulfate (morphine), diazepam (Valium), chlorpromazine hydrochloride (Thorazine) or chloral hydrate, or combinations, were used when needed for sedation.

Results

Systemic effects of hypothermia. A graphic representation of the heart rate, temperature, arterial pH and cooling is presented in Figure 3. The lowest rectal temperature achieved ranged from 31.1 to 34.4°C (Table 1). The duration of cooling was 4 to 24 hours (mean 11.7). The time required to lower the heart rate to <180 beats/min was 0.5 to 17 hours (mean 6.3). All patients developed a metabolic acidosis while being cooled. Patient 3 developed a combined metabolic and respiratory acidosis when cooled to 32.7°C. Blood pressure and urinary output remained stable during hypothermia. Cardiac output was not measured. No short-term adverse effects were noted.

Effect on ECG. An example of the effect of hypothermia on the ECG in Patient 2 is shown in Figure 4. In panel A, at a rectal temperature of 33.3°C, the junctional ectopic tachycardia has slowed to an irregular rate of 144 beats/min. In panel B, the irregular rate has slowed to 108 beats/min. On rewarming (panel C), P waves can be seen after the QRS complex compatible with 1:1 retrograde conduction with a regular junctional rate of 120 beats/min. When the infant became normothermic (panel D), sinus rhythm was present at a rate of 120 beats/min. Figure 5 is an atrial electrogram taken at approximately the same time as panel B in Figure 4. An atrial rate of 113 beats/min with a variable junctional rate of 133 to 144 beats/min and occasional conducting P waves are demonstrated.

Spontaneous reversion to sinus rhythm eventually occurred in all three infants. In Patients 2 and 3 it occurred 4 hours and 9 hours, respectively, after the end of cooling. Patient 1, however, continued to have junctional ectopic tachycardia and it was decided 7 hours after the onset of cooling to pace sequentially the atrium and ventricle at 160 beats/min to obtain AV synchrony. The junctional ectopic rate at that time was <155 beats/min. The patient continued to have junctional ectopic tachycardia with a rate <160 beats/min for 4 days until sinus rhythm eventually returned and the pacing was discontinued.

Follow-up. All three patients continue to have sinus rhythm. They have been followed up from 15 months to 29

Table 1. Summary of Three Cases of Greatly Accelerated Junctional Ectopic Tachycardia: Treatment With Hypothermia

Patient	Age	Sex	Weight (kg)	Cardiac Diagnosis	Onset JET >200 beats/min After Surgery (h)	Maximal Rate JET (beats/min)	Maximal Temperature During JET (°C)	Treatment With Hypothermia: Duration JET >200 Before Hypothermia (h)	Lowest Rectal Temperature (°C)	Duration of Cooling (h)	Time Required to Lower Beats/min to <180 (hr)
1	4 Mo	M	5	VSD, ASD, Sub AoS	2	212	39.1	5	34.5	7	0.5
2	2 Mo	F	4	VSD, ASD	50	220	37.8	5	31.8	24	17
3	2 Days	M	4	TAPVR, PDA	72	253	37.0	18	31.1	4	1.5
Mean			4.3		41	228	37.7	9	32.5	11.7	6.3

ASD = atrial septal defect; F = female; JET = junctional ectopic tachycardia; M = male; PDA = patent ductus arteriosus; Sub AoS = subaortic stenosis; TAPVR = total anomalous pulmonary venous return; VSD = ventricular septal defect.

Figure 2. Greatly accelerated junctional ectopic tachycardia. **Upper panel** (Patient 2), Demonstrating a variable ventricular rate of 214 to 300 beats/min with an atrial rate of 200 beats/min and atrioventricular (AV) dissociation. **Lower panel** (Patient 1), Demonstrating a ventricular rate of 200 beats/min with an atrial rate of 158 beats/min and AV dissociation.

months after treatment and no adverse effects have been detected. Ambulatory 24 hour continuous ECG recordings were normal.

Comparison with three conventionally treated patients. Before the introduction of hypothermic therapy, three patients with postsurgical accelerated junctional ectopic tachycardia were treated with conventional measures. Their ages ranged from 4 to 15 months. Cardioversion, when attempted, was unsuccessful as was treatment with ice to face, lidocaine, verapamil, propranolol and digoxin. Atrial or ventricular pacing would capture the respective chamber but only at rates faster than the intrinsic junctional rate. Burst atrial or ventricular pacing did not convert the tachycardia to sinus rhythm.

These three infants showed signs of poor cardiac output and peripheral vasoconstriction. One patient died 10 hours after the onset of junctional tachycardia and two patients survived but maintained a rate >200 beats/min for 40 and 72 hours, respectively. During this period, both were in a low cardiac output state.

Discussion

The cause of junctional ectopic tachycardia after open heart surgery is unknown, but it is thought to be secondary to trauma or inadequate protection in an area around the AV node during surgery (2,6,7). Because junctional ectopic tachycardia is assumed to be an automatic type of supraventricular tachycardia, controlling the rate or converting the abnormal rhythm to sinus rhythm has been difficult. Cardioversion is unsuccessful and use of conventional medications has been associated with poor results. The use of propafenone in controlling the greatly accelerated junctional ectopic tachycardia rate in four infants after open heart surgery has been reported (8). However, propafenone has a

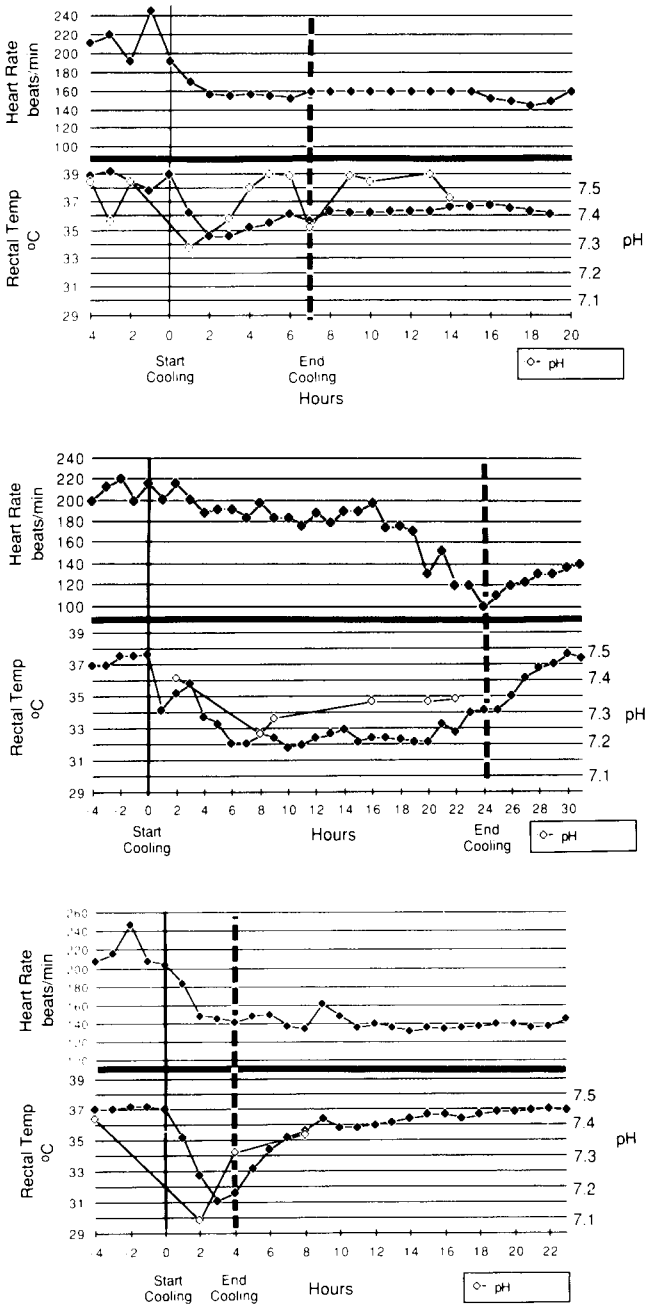

Figure 3. Greatly accelerated junctional ectopic tachycardia in three infants. Effects of hypothermia on heart rate, rectal temperature and pH. **Top panel,** Patient 1; **middle panel,** Patient 2; **lower panel,** Patient 3.

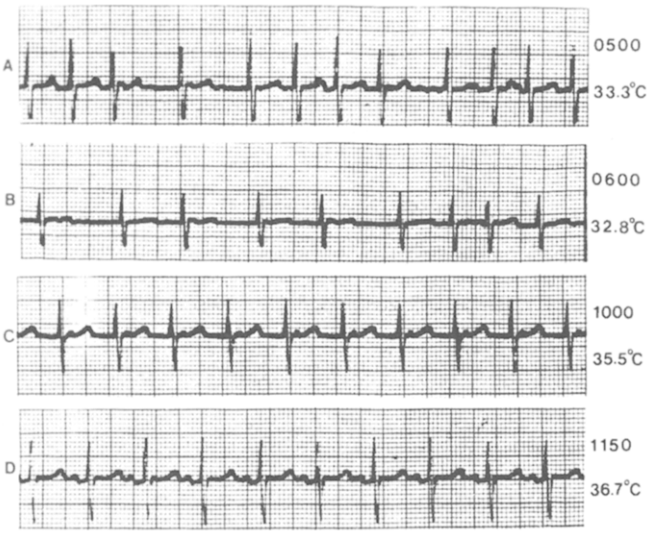

Figure 4. Patient 2. Effects of hypothermia on junctional ectopic tachycardia. **A,** At 5 AM, temperature was 33.3°C rectally. A junctional rhythm is shown with an irregular ventricular rate of 144 beats/min. **B,** An irregular ventricular rhythm with a rate of 108 beats/min. **C,** Regular junctional rhythm with a rate of 120 beats/min; P waves follow each QRS. **D,** Sinus rhythm with a rate of 120 beats/min is present after rewarming.

definite negative inotropic effect and further clinical trials are needed to establish its role in the management of junctional ectopic tachycardia.

Paired ventricular pacing as suggested by Waldo et al. in 1976 (1) might be helpful, but their results have not been duplicated on a regular basis. We did not attempt paired ventricular pacing in these infants. Cryoablation or surgical ablation is also possible (9) but should be avoided in postoperative greatly accelerated junctional ectopic tachycardia because of the transient nature of the arrhythmia.

Role of hypothermia. Hypothermia producing sinus bradycardia has been reported as early as 1912 (10). This slowing of heart rate is secondary to depression of automaticity (11). Because greatly accelerated junctional ectopic tachycardia is thought to be an automatic type of supraventricular tachycardia, it may also respond to hypothermia by slowing. A surgeon in Tucson originated this concept and A. Garson, Houston, Texas, encouraged it (personal communication). In our experience, congestive heart failure began occurring when the junctional ectopic rate exceeded 200 beats/min. All of our infants had overt failure by 5 hours after the onset of greatly accelerated junctional ectopic tachycardia. Controlled hypothermia was used to decrease the tachycardia rate to <180 beats/min, which would allow for increased diastolic filling time resulting in improvement

Figure 5. Patient 2. Atrial electrogram taken at approximately the same time as Panel B in Figure 4 at twice the paper speed. The atrial rate is 113 beats/min with a variable junctional rate of 133 to 143 beats/min and occasionally conducted P waves.

of cardiac output. Once a stable heart rate <180 beats/min was achieved, the patient was allowed to rewarm.

ECG diagnosis. It is imperative that the diagnosis of junctional ectopic tachycardia be made correctly. With a narrow QRS tachycardia >200 beats/min with AV dissociation, the diagnosis of greatly accelerated junctional ectopic tachycardia is fairly simple. However, if AV association is present and junctional ectopic tachycardia is not definitely established, cardioversion and possible medical management should first be attempted. If the P waves are not readily identifiable, we found the atrial electrogram to be extremely helpful. An esophageal atrial electrogram would also be helpful to demonstrate P waves in patients who did not have temporary atrial pacing wires.

Possible side effects of hypothermia. Hypothermia in itself can induce cardiac arrhythmias, including asystole, during both the cooling and rewarming periods (12). None of our patients had problems with cardiac rhythm during induction of hypothermia or rewarming. However, we did induce a metabolic acidosis during the hypothermia. Moreover, it also became difficult to follow the status of these infants clinically because they all exhibited peripheral vasoconstriction during the period of induced hypothermia. Shivering was not a problem, but agitation required intermittent sedation. Nevertheless, the condition of all three patients improved and sinus rhythm eventually returned.

Conclusions. Hypothermia is an effective means of controlling the rate of greatly accelerated junctional ectopic tachycardia after open heart surgery in infants. Though hypothermia does not convert junctional ectopic tachycardia to sinus rhythm, it slows the rate to a more acceptable level, allowing the survival of the infant and eventual recovery to sinus rhythm.

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