

BRIEF REPORTS

Management by Hypothermia of Junctional Ectopic Tachycardia Appearing after Pediatric Heart Surgery

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Junctional ectopic tachycardia (JET) is a complication of the repair of congenital cardiac malformations that responds poorly to conventional treatment. We report our experience with the use of moderate hypothermia in its management. Twelve infants with postoperative JET treated with hypothermia were reviewed. The mean interval between the diagnosis of JET and initiation of hypothermia was 1.5 ± 0.5 hours. In the first 24 hours of hypothermia, central temperature and heart rate decreased significantly. Arterial pressure and diuresis tended to increase and central venous pressure tended to decrease. No direct adverse events occurred. All the patients but one survived and are alive and free of neurological deficits after 15 ± 12 months.

Key words: *Tachycardia. Arrhythmia. Surgery. Congenital cardiac malformations. Pediatrics.*

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Tratamiento mediante hipotermia de la taquicardia ectópica de la unión tras cirugía cardíaca infantil

La taquicardia ectópica de la unión (TEU) es una complicación que aparece tras las reparaciones de cardiopatías congénitas, con una escasa respuesta al tratamiento convencional. Describimos nuestra experiencia en el uso de hipotermia moderada como parte de su tratamiento. Se revisaron las historias de 12 pacientes con TEU postoperatoria tratados con hipotermia. El intervalo entre el diagnóstico de TEU y el inicio de la hipotermia fue de $1,5 \pm 0,5$ h. Durante las primeras 24 h hubo un descenso significativo de la temperatura y la frecuencia cardíaca, con tendencia al aumento de la presión arterial y de la diuresis y al descenso de la presión venosa central. No hubo complicaciones relacionadas con la hipotermia. Todos excepto un paciente sobrevivieron y tras un seguimiento de 15 ± 12 meses permanecen vivos y sin secuelas neurológicas.

Palabras clave: *Taquicardia. Arritmia. Cirugía. Cardiopatías congénitas. Pediatría.*

INTRODUCTION

Acute junctional ectopic tachycardia (JET) can appear after surgery for congenital heart defects. Although it is self-limiting within 48-72 h, mortality in the absence of prompt treatment is 40%¹ because of elevated heart rate (HR) and atrioventricular dissociation (AVD). The poor response to pharmacological treatment has led to a search for other treatment options, including moderate hypothermia.² This treatment, first described in 1987,³ has been reported in the literature infrequently, and all reports involve low numbers of patients.

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Received 14 March 2002.

Accepted for publication 12 November 2002.

PATIENTS AND METHOD

Patients

We reviewed the clinical records of patients operated on for congenital heart defects between September 1999 and October 2001 at the Área Infantil del Corazón (Pediatric Heart Service) of the Juan Canalejo Hospital Complex in A Coruña, northwestern Spain. Standard electrocardiograms were examined, as were atrial electrograms recorded during episodes of arrhythmia with the atrial electrodes placed during surgery or with esophageal electrodes. Only patients with confirmed or probable JET who were treated with hypothermia were included in this study.

Diagnostic criteria

– Confirmed JET: a) tachycardia with a QRS com-

ABBREVIATIONS

JET: junctional ectopic tachycardia
HR: heart rate
AVD: atrioventricular dissociation

plex similar to the basal tracing or to a tracing obtained by atrial stimulation, together with b) AVD with slower atrial rate than ventricular rate.

– Probable JET: recorded when criterion 1 as noted above was satisfied but it was not possible to demonstrate AVD, or when there was 1:1 retrograde conduction and arrhythmia did not respond to electrical cardioversion or atrial overstimulation.

Treatment

Treatment of JET was based on: a) basic measures (decreased use of inotropic agents, sedation, correction of electrolyte imbalance); b) pacemaker (to restore atrioventricular synchrony by establishing a HR faster than that caused by JET); c) hypothermia, and d) antiarrhythmic drugs. Digoxin was used for inotropic and diuretic support in some patients.

Hypothermia was begun when hemodynamic status deteriorated despite basic measures, or when it was not possible to maintain a pacemaker rate faster than the JET rate. All patients were sedated and placed on mechanical ventilation with muscle relaxants. Fans, cold packs placed on the skin, and lavage with cold physiological saline via a nasogastric tube were used. The temperature was lowered until the JET rate decreased, but never to below 33 °C as measured with a rec-

tal thermometer. The patients were rewarmed slowly (1 °C/8 h) after at least 36 h of hemodynamic stability (adequate central venous pressure, arterial pressure and diuresis) by increasing external heat sources under constant electrocardiographic monitoring. In patients treated with a pacemaker, this treatment was interrupted when normal sinus rhythm appeared or when the JET rate decreased to below the 75th percentile of normality according to age.

Statistical analysis

Quantitative variables are expressed as the mean±standard deviation, and the median is also reported for variables whose distribution was markedly asymmetric. Categorical variables are expressed as absolute values (percentages). Changes in hemodynamic values obtained at 1, 2, 4, 8, 12 y 24 h from the start of hypothermia were analyzed with MANOVA for linear trends for repeat measures; normal distribution of the data was verified beforehand with the Shapiro-Wilk's test. Differences were considered significant at $P<.05$. The statistical analysis was done with the SPSS 10.0.5 (Chicago, Illinois) and Stat View programs.

RESULTS

A total of 138 patients underwent pediatric heart surgery with cardiopulmonary bypass; 21 (15.2%) developed confirmed or probable JET, and hypothermia was used in 12 patients analyzed here (9 girls, mean age 2.1 ± 2.0 months) (Table 1). The onset of JET was recorded 6.3 ± 11.9 h after the operation (median, 2 h). At that moment, temperature was 37.0 ± 0.7 °C (Table 2). Hypothermia was started 1.5 ± 0.5 h after the onset of JET (median, 1.0 h), and lasted 74 ± 42 h (range, 36-144 h). During hypothermia, digoxin was used in 6 patients, and in 4 of these amiodarone was also given for

TABLE 1. Characteristics of the patients

Patient	Sex	Age	Diagnosis	Surgery	JET	Onset of JET (postoperative h)	Start of hypothermia (h after onset of JET)
1	M	7 months	Single ventricle SLL	Glenn, widening VSD	P	1	1
2	F	8 days	Pulmonary atresia, VSD	Repair	C	3	1
3	M	14 days	TGV, ASD	Arterial switch, closure ASD	P	0	2
4	F	5 months	Complete AV canal	Repair	C	3	6
5	M	12 days	Truncus	Repair	C	27	0
6	M	4 months	TOF	Repair	C	0	4
7	F	2 months	TAPVC, VSD	Repair	C	2	0
8	F	13 days	TGV	Arterial switch	P	0	0
9	F	1.5 months	ASD, VSD	Repair	C	2	1
10	F	1.5 months	Partial AV canal	Repair	P	0	1
11	F	2.5 months	TOF	Repair	S	36	2
12	F	1 month	TGV, IASD, IVSD	Arterial switch, repair	S	2	0

AV indicates atrioventricular; ASD, atrial septal defect; VSD, ventricular septal defect; TAPVC, total anomalous pulmonary venous connection; P, probable; C, confirmed; TOF, tetralogy of Fallot; TGV, transposition of the great vessels.

TABLE 2. Treatment with hypothermia

Patient	CAD	Initial central temperature (°C)	Initial HR (beats/min)	Minium central temperature	HR Minimum	PM	Duration of hypothermia (h)	Recovery sinus R (h)	Recooling	Course
1	Digox	37.9	157	35.0	122	Esoph	48	60	no	Lived
2	Digox, amio	37.0	214	32.4	122	AAI	-	-	-	Died
3		37.0	192	34.3	118	-	70	41	No	Lived
4		38.3	177	35.8	157	AAI	40	60	No	Lived
5		36.0	188	34.0	120	AAI	120	33	No	Lived
6	Digox	36.7	182	33.5	162	AAI	36	72	No	Lived
7	Digox, amio	36.6	200	33.2	153	DDD	144	168	yes	Lived
8	Digox, amio	36.0	200	33.3	120	AAI	144	72	yes	Lived
9		36.5	180	35.1	134	-	40	28	No	Lived
10		36.5	213	32.5	12	DDD	84	72	Yes	Lived
11	-	37.8	170	34.8	148	AAI	48	40	No	Lived
12	Digox, amio	37.1	216	34.4	147	AAI	44	72	No	Lived

AAI indicates atrial pacing and sensing, inhibition response; amio, amiodarone; DDD, dual-chamber pacing and sensing, dual response (inhibition and triggering); digox, digoxin; esoph, esophageal electrode; CAD, coadjuvant antiarrhythmic drugs; HR: heart rate; PM: pacemaker; R: rhythm.

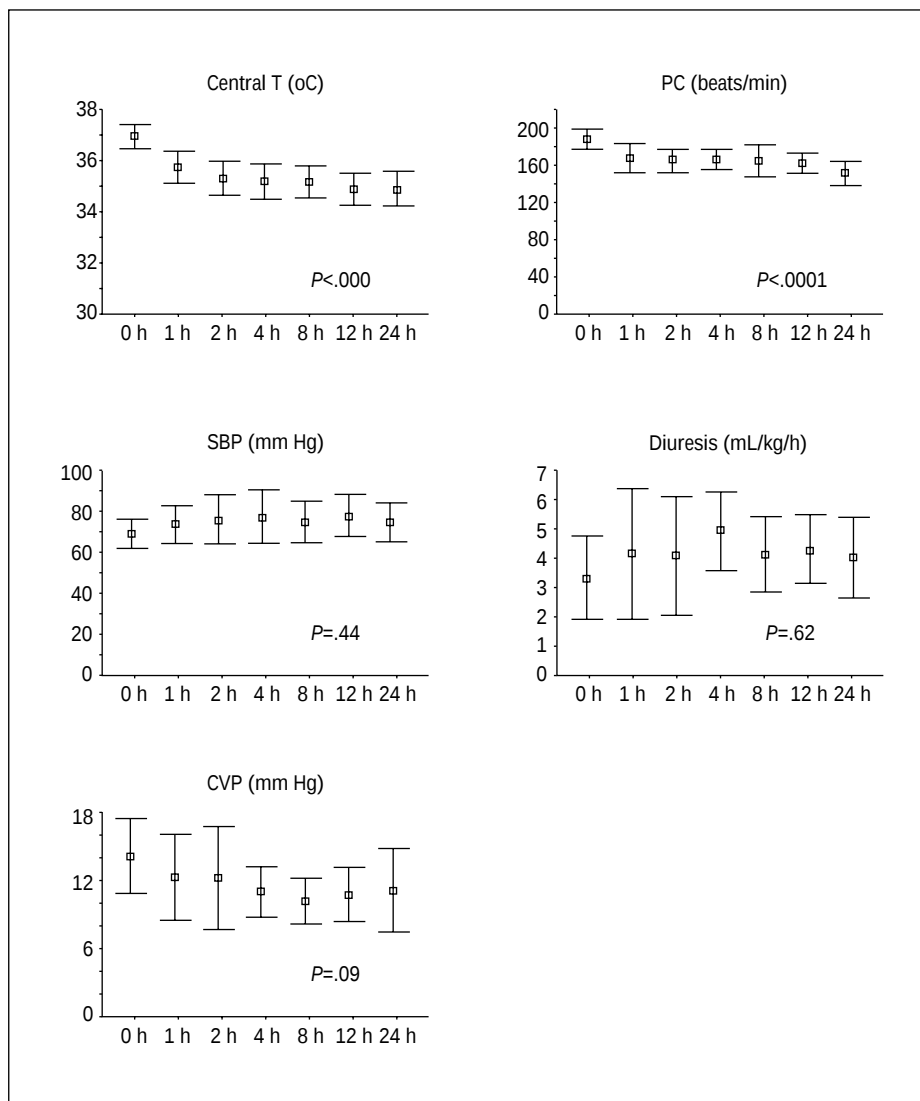
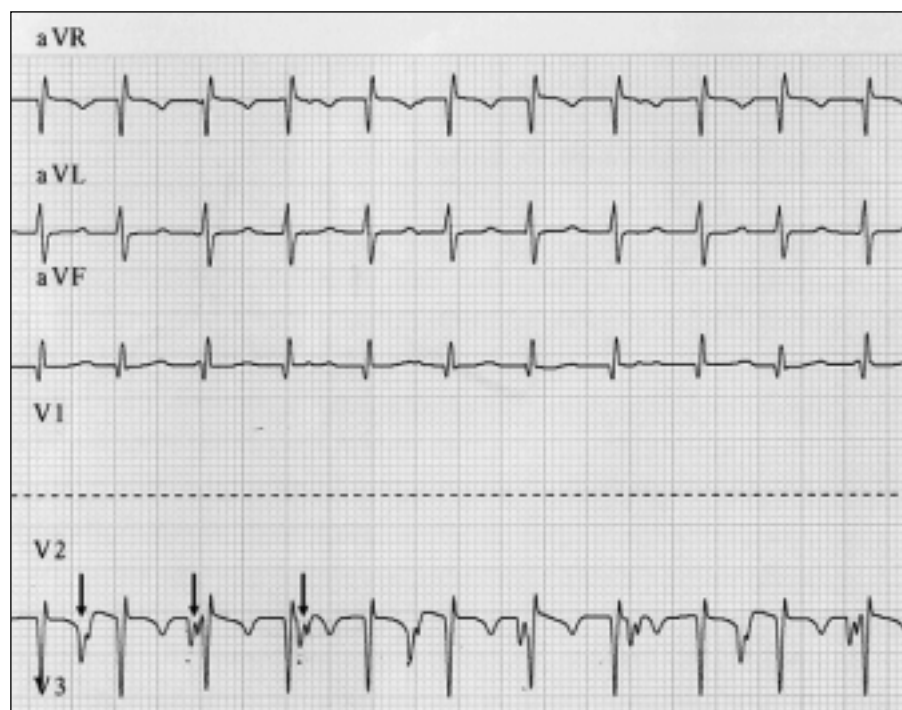


Fig. 1. Course during the first 24 h of hypothermia. Lines illustrate means and 95% confidence intervals. P values refer to MANOVA linear trends tests for repeat measures. HR indicates heart rate; beats/min, beats per minute; T, temperature; SBP, systolic blood pressure; CVP, central venous pressure.

Fig. 2. Electrocardiographic tracing of JET, including aVR, aVL and aVF leads. The lower tracing shows the atrial electrogram, and was obtained by connecting the lead usually used for the V2 electrode to the atrial epicardial electrode. This tracing shows increased atrial activity (arrows); atrioventricular dissociation is evident from the slower atrial rate compared to the ventricular rate.



support during hypothermia. A pacemaker was used in 10 patients: in 9, epicardial electrodes were placed in the operating room, and in 1 an esophageal electrode was used.

Changes in central temperature and hemodynamic status during the first 24 h are shown in Figure 1. The greatest improvements were obtained during the first 4 h of hypothermia, after which patients remained stable.

All patients except one survived. The infant who died had low output status secondary to JET, which was refractory to treatment. There were no complications directly attributable to hypothermia. At the time of rewarming, 5 patients continued to have JET; one required recooling because of a significant increase in HR. Predominantly sinus rhythm reappeared 65 ± 38 h after the start of hypothermia. Two of the 6 patients who recovered a normal sinus rhythm during hypothermia required recooling because JET and rapid HR reappeared.

The 11 patients who were discharged remained alive and had no neurological sequela after a mean follow-up period of 15 ± 12 months (range, 2-36 months). Sinus rhythm was maintained without arrhythmia during follow-up.

DISCUSSION

Postoperative JET arises from an ectopic focus located in the nodal tissue or bundle of His, and is induced by mechanical irritation during surgery.² Its ectopic nature makes it sensitive to sympathetic-vagal balance and resistant to atrial overstimulation or electrical car-

dioversion. The high HR and AVD decrease cardiac output, and this in turn accelerates arrhythmia because of increased adrenergic tone, thus creating a vicious circle.^{1,2} Because the process is self-limiting within 48-72 h,² the main goal of treatment is maintenance of appropriate heart output until the arrhythmia remits spontaneously.

Treatment should include^{1,2,4,5} correction of electrolyte imbalance and acid-base imbalance, removal of the adrenergic stimuli (pain, irritation, anxiety), appropriate sedation, and decreased levels of inotropic and vasodilating agents, which accelerate HR and JET.² The response to antiarrhythmic drugs is poor,² and digoxin is not effective.⁵ Propafenone appears to have some benefit,⁶ despite the fact that it is a negative inotropic agent. Amiodarone is useful^{7,8} although in our experience it may require volume expansion because of the appearance of hypotension. Procainamide is effective in combination with hypothermia only when hypothermia alone is insufficient.⁵ The role of beta blockers is controversial,² and calcium antagonists are not recommended. Other treatment options are various modes of stimulation with pacemakers, and prompt ablation of the bundle of His in refractory cases.^{1,2}

Early diagnosis is fundamental. When JET is suspected, a 12-lead electrocardiogram and atrial electrogram should be done.⁹ In our series of patients, atrial electrography made it possible to unmask AVD in 2 patients and confirm the suspicion in 6 patients in whom the surface electrocardiogram was suggestive of AVD (Figure 2).

Treatment with hypothermia is based on the observa-

tion that decreased central temperature reduces the automaticity of pacemaker cells. Since the first report of this treatment was published by Bash et al, in 1987,³ scattered reports have appeared of the successful use of hypothermia in small numbers of patients.^{9,10} The aim of these interventions was to attain a central temperature of 32-35 °C. Our goal was to reduce HR until appropriate pacemaker treatment became possible, and to thus avoid deep hypothermia and its possible side effects. In 2 patients pacemakers were not used because atrial epicardial electrodes were not available. In view of their good clinical course we decided not to use an esophageal electrode. Because of our study design, it was not possible to judge the relative usefulness of digoxin and amiodarone given during hypothermia in controlling HR.

In this series of patients hypothermia associated with the use of a pacemaker was an effective, safe option. Hemodynamic changes occurred within a few hours, and no side effects directly attributable to hypothermia were seen. Although the small number of patients we studied does not allow us to generalize, the mortality rate in our series of 8.3% was clearly lower than the 40% figure reported for conservative treatment.¹ The infant who died was one of the first patients in the series and had very rapid tachycardia, which might account for the failure of hypothermia.

Rewarming was begun after at least 36 h of hemodynamic stability without awaiting recovery of a stable sinus rhythm. The likelihood that recooling would be needed (27%) was not related with the sinus rhythm or JET, and we therefore feel that rewarming can be timed on the basis of hemodynamic criteria alone.

The limitations of our study are those inherent in any retrospective, observational analysis based on a

small number of patients with no control group. Nevertheless, we believe our findings, together with other published reports, support the use of hypothermia as an effective and safe treatment for the management of postoperative JET.

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