Cardiol Young 2005; 15: 427–430

ISSN 1047-9511

Brief Report

Successful use of an intravenous infusion of flecainide and amiodarone for a refractory combination of postoperative junctional and ectopic tachycardias

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Abstract After repair of an atrioventricular septal defect with common atrioventricular junction in a 2-month-old girl, rapid atrial tachycardia, in combination with junctional ectopic tachycardia, led to severe postoperative cardiovascular compromise. Intercurrent runs of ectopic atrial tachycardia made atrial pacing impossible, despite high doses of intravenous amiodarone. Following the addition of flecainide to the infusion, we were able to control the rhythm, and when combined with atrial pacing, this led to an immediate haemodynamic improvement. Treatment of refractory supraventricular tachycardias with amiodarone combined with flecainide can be very effective in the setting of postoperative cardiac intensive care.

Keywords: Amiodarone; flecainide; postoperative junctional ectopic tachycardia; ectopic atrial tachycardia; intra-atrial re-entry tachycardia

JUNCTIONAL ECTOPIC TACHYCARDIA IS A potentially life-threatening arrhythmia when producing severe haemodynamic compromise during the postoperative course of congenital cardiac surgery.[1] Intra-atrial re-entry tachycardias are common late after complex atrial surgery, but can also occur early in the postoperative period.[2] Ectopic atrial tachycardias, however, are relatively rare postoperatively. When encountered, they are often associated with some form of surgical disruption of the atrial septum.[3]

We describe a 2-month-old girl in whom a combination of atrial and junctional tachycardias complicated the immediate postoperative course. Only the combination of a continuous infusion of amiodarone together with flecainide permitted us adequately to control the abnormal rhythm.

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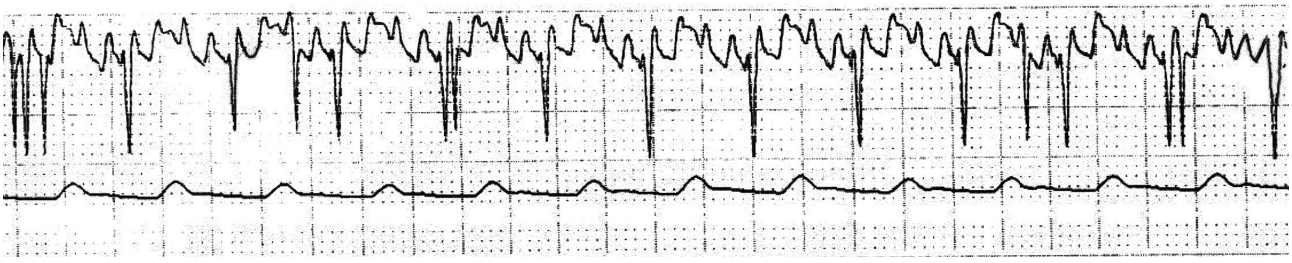
Accepted for publication 15 February 2005

Case report

A 2-month-old girl presented immediately after surgical correction of an atrioventricular septal defect with common atrioventricular valve with a combination of atrial tachycardias and junctional ectopic tachycardia. The ventricular rates varied between 200 and 250 beats per minute. The initial electrocardiogram showed p-waves at a rate of about 400 beats per minute, with variable atrioventricular conduction (Fig. 1a,b). Repetitive trials of atrial overdrive pacing and two synchronised cardioversions were ineffective in terminating the tachycardia. In an attempt to control the atrial rate, we gave a bolus of amiodarone, and within 1 hour of treatment, the atrial tachycardia disappeared, only to be replaced by a rapid junctional ectopic tachycardia at a rate of 200 beats per minute. This impaired the cardiovascular function even more (Fig. 1c). By this time, we were also using dopamine at 5 micrograms per kilogram per minute, and dobutamine at

https://doi.org/10.1017/S1047951105000892 Published online by Cambridge University Press



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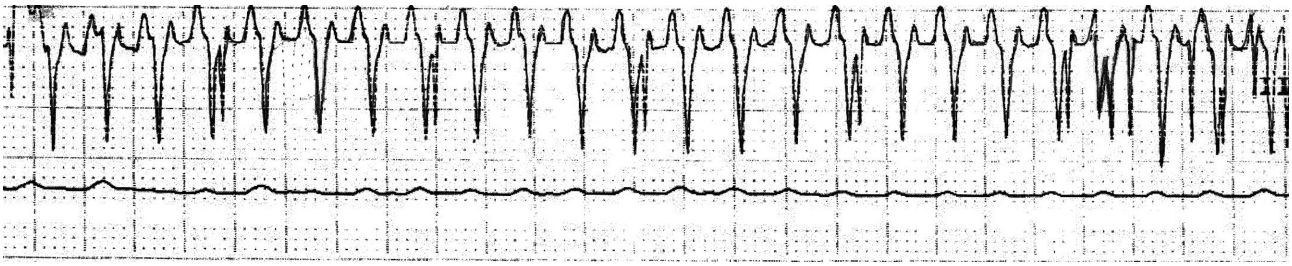

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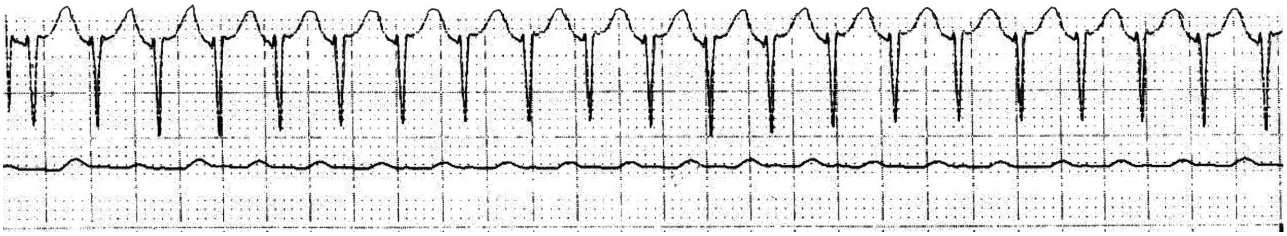

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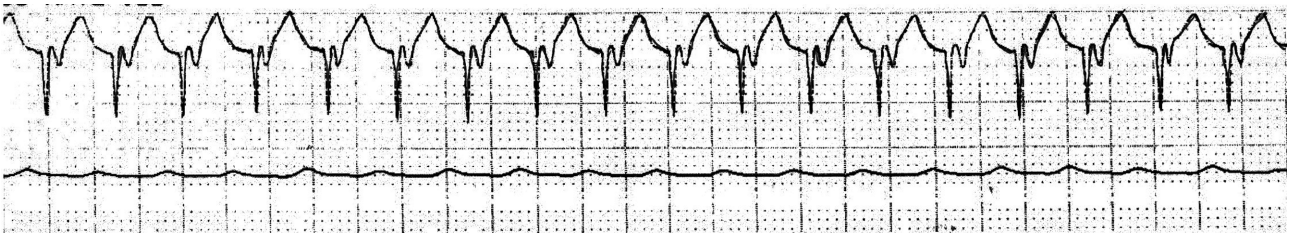

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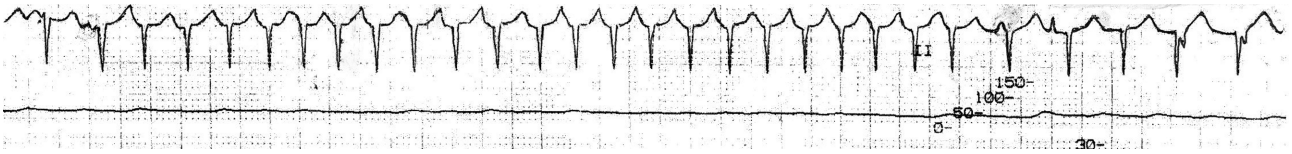

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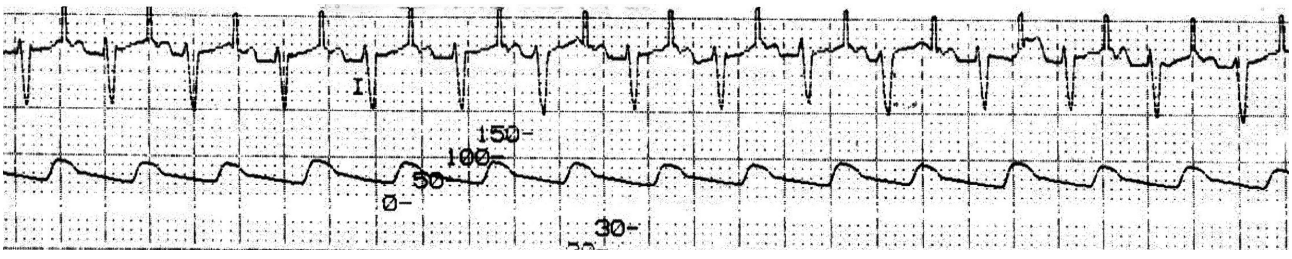

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10 micrograms per kilogram per minute, to maintain the blood pressure at 60 over 40 millimetres of mercury.

After two additional boluses of amiodarone at 5 milligrams per kilogram, and an infusion at 20 milligrams per kilogram per day, equivalent to 14 micrograms per kilogram per minute,[1] the rate of the junctional ectopic tachycardia decreased to 170 beats per minute (Fig. 1d). Despite additional treatment with administration of a bolus of magnesium at 0.5 millimols per kilogram, and verification that levels of potassium in the blood were in the high normal range, we were unable adequately to control the rhythm with atrial pacing because of intercurrent runs of atrial tachycardia (Figs 1e and 2). The patient deteriorated over the following 3 hours, requiring an infusion of noradrenaline at 0.1 micrograms per kilogram per minute to maintain the blood pressure. Subsequently, output of urine stopped, necessitating peritoneal dialysis. Echocardiography at this time revealed poor cardiac function, with left ventricular fractional shortening of 20 per cent, and a calculated cardiac index of 1.5 litres per minute per metre squared.

Over the following 6 hours, we were unable to achieve any significant improvement. It proved necessary to increase the dose of noradrenaline to 0.2 micrograms per kilogram per minute so as to maintain the blood pressure, and the patient became acidotic. Flecainide was started at a rate of 0.25 milligram per kilogram per hour, equivalent to 6 milligrams per kilogram per day, and increased to 0.5 milligrams per kilogram per hour, or 12 milligrams per kilogram per day, after 2 hours of treatment, at the same time maintaining the infusion of amiodarone at 20 milligrams per kilogram per day. Within 4 hours, the runs of atrial tachycardia had disappeared, the rate of the junctional ectopic tachycardia had decreased to 150 beats per minute, and atrial pacing at 155 beats per minute could be maintained without further problems (Fig. 1f). It then proved possible to discontinue the noradrenaline and dopamine, and decrease the dobutamine to 5 micrograms per kilogram per minute while maintaining a blood pressure of 85 over 45 millimetres of mercury. Urinary output also improved. Echocardiography after 24 hours showed a left ventricular fractional shortening of 30 per cent, and a calculated cardiac index of 2.5 litres per minute per metre squared.

The QT-interval corrected for heart rate raised from a baseline of 0.41–0.43 seconds during the infusion of amiodarone, peaking at 0.48 seconds subsequent to the addition of flecainide, when the level of flecainide in the blood was 0.7 milligrams per litre, the recommended range being from 0.2 to 1.0. Sinus rhythm was re-established 1 day later, permitting the amiodarone to be stopped. The child was extubated within the following 48 hours, and transferred to the ward 1 day later. We continued with the flecainide for another month, stopping after that period without recurrence of the supraventricular tachycardias.

Discussion

Adequate control of heart rate and rhythm is crucial when caring postoperatively for patients with congenital heart defects. Junctional ectopic tachycardia is associated with significant haemodynamic impairment, and even death.[4] Modern protocols for junctional ectopic tachycardia usually include the administration of amiodarone.[1,4,5] Intravenous flecainide has also been shown recently to be effective in managing postoperative junctional ectopic tachycardia.[6]

Our patient presented initially with a very rapid atrial tachycardia, which failed to respond to repetitive trials of atrial overdrive pacing and synchronised cardioversion. Within 1 hour of administering amiodarone, we saw junctional ectopic tachycardia at a rate of 220 beats per minute. The combination of the two arrhythmias caused major haemodynamic compromise, posing a major therapeutic dilemma. It was flecainide used in combination with amiodarone that permitted us to control the arrhythmias and stabilise the haemodynamic situation.

Postoperative atrial tachycardias may be due to an ectopic focus, or due to macro-re-entry in the atrium.

Figure 1.

*Initially, there is a high atrial rate of more than 400 beats per minute, with a variable atrioventricular conduction (**a**). The mean ventricular rate, however, is about 150 beats per minute. The blood pressure (see curve below) is adequate. A subsequent trace (**b**) shows that the atrial rate is more than 400 beats per minute. There is now, however, a 2:1 conduction, leading to a ventricular rate of more than 230 beats per minute. The blood pressure (see curve below) has now dropped significantly. In a further tracing (**c**), the electrocardiogram shows a junctional ectopic tachycardia at a rate of about 200 beats per minute. The blood pressure (see curve below) is still significantly impaired. The next electrocardiogram (**d**) shows a junctional ectopic tachycardia at a rate of about 170 beats per minute, after two boluses of amiodarone given at 5 milligrams per kilogram, and under a continuous infusion of amiodarone at 20 milligrams per kilogram per day. There is a visible retrograde p-wave after each QRS-complex. The blood pressure (see curve below) is still significantly impaired. The next electrocardiogram (**e**) shows one of the intercurrent rapid runs of ectopic atrial tachycardia, at a rate of 220 beats per minute. During this, the blood pressure (see curve below) is dramatically reduced. Finally, after the combination of amiodarone and flecainide (**f**), adequate atrial pacing was possible, at a rate of 155 beats per minute, with excellent haemodynamic response and increase of the blood pressure.*

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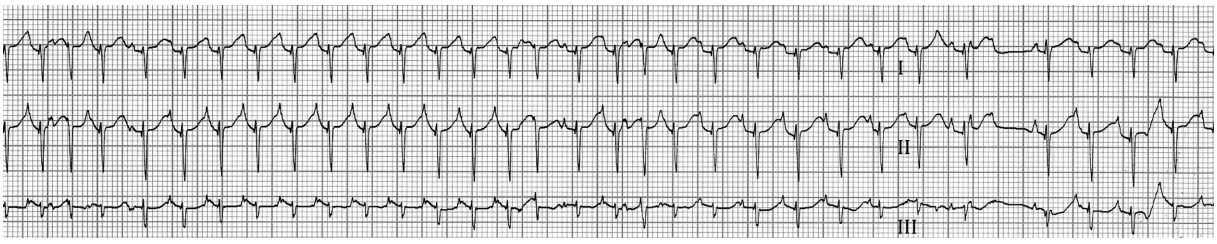

Figure 2.
This 3-lead electrocardiogram shows one of the intercurrent rapid runs of ectopic atrial tachycardia, at a rate of 220 beats per minute. There is different p-wave morphology and various P-P-intervals.

They are often found in postoperative patients after the Fontan procedure.[3,7] An ectopic focus is typically characterised by a "warm up" phase, indicated by a progressive shortening of the PP-interval, and in many cases by a "cool down" phase, with lengthening of the PP-interval before termination of the tachycardia. The variable atrial rate, and failure to response to electrical cardioversion, perhaps makes an ectopic focus more likely than a re-entrant tachycardia in our patient, but either or both forms may have been present at different times. The repetitive bursts of tachyarrhythmia rendered impossible the use of atrial or dual chamber pacing (Fig. 2).

Amiodarone and flecainide used independently have proven effective in the treatment of a variety of supraventricular tachyarrhythmias in children.[8] Single drug therapy, as well as standard combination medical therapy for tachyarrhythmias in children, nonetheless, sometimes fails. Fenrich et al.[9] have used amiodarone in combination with flecainide administered orally in 9 patients, with 6 of them having various supraventricular tachycardias. The drugs used in combination for postoperative junctional ectopic tachycardia were first described in a child following the Fontan operation, flecainide being given orally 3 days after a postoperative junctional ectopic tachycardia was controlled with amiodarone.[10]

The combination of the two potent antiarrhythmic agents raises concerns about the potential for proarrhythmogenic effects. Due to this, it is important to monitor carefully the levels of flecainide in the blood, along with the serum electrolytes and QT-intervals. An awareness of potential interactions with other medications used in intensive care is also important. Our experience, nonetheless, shows that amiodarone and flecainide can be used effectively in combination in children in the management of life-threatening postoperative tachyarrhythmias.

Acknowledgment

Financial support: normal hospital support.

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