Flecainide induced ventricular fibrillation in a neonate
F Ackland, R Singh, S Thayyil

CASE DETAILS
The baby was born by emergency lower segment caesarean section for fetal tachycardia at 38 weeks of gestation. Birth weight was 2.9 kg and Apgar scores were normal. Heart rate was noticed to be persistently high at 240–260 beats/min and ECG was suggestive of supraventricular tachycardia. The baby was initially symptomatic and was treated with ice pack adenosine but with no response. Flecainide 2 mg/kg orally was begun when the baby was 24 hours of age and the heart rate settled from 240 beats/min to 170 beats/min. At 48 hours old, the baby’s ECG showed broad complex tachycardia at 170 beats/min and therefore flecainide was stopped (after four doses). Six hours later the baby became poorly perfused and went into shock requiring ventilation. ECG showed fine ventricular fibrillation and poor pulses. Blood gas showed severe metabolic acidosis. Flecainide concentrations were normal (630 µg/l, 24 hours after the last dose). The baby required prolonged resuscitation with cardioversion, adrenaline, calcium gluconate, magnesium, and bicarbonate infusion. Treatment with amiodarone 5 mg/kg intravenously was begun. Perfusion started improving after two hours of resuscitation and ECG showed broad complex tachycardiac arrests and re-entrant supraventricular tachycardia with a heart rate of 180 beats/min unresponsive to adenosine. Arrhythmia was finally controlled with digoxin and amiodarone, but treatment was subsequently withdrawn following evidence of irreversible cerebral damage on serial electroencephalograms and computed tomography.

DISCUSSION
The treatment of children, especially neonates, with tachyarrhythmias remains a complex task. Flecainide induced QRS widening is a serious proarrhythmic effect that can occur at therapeutic blood concentrations. Extreme caution needs to be exercised when using flecainide for supraventricular tachycardias in neonates.

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