

Treatment of fetal supraventricular tachycardia with flecainide acetate after digoxin failure

To the Editors: After reviewing the interesting case report by Kofinas et al. (Kofinas AD, Simon NV, Sagel H, Lyttle E, Smith N, King K. Treatment of fetal supraventricular tachycardia with flecainide acetate after digoxin failure. *AM J OBSTET GYNECOL* 1991;165:630-1), readers may be interested in a further case of fetal supraventricular tachycardia treated by maternal administration of flecainide after unsuccessful treatment with digoxin that was reported in the British literature.¹ A 26-year-old gravida 2, para 1 woman at 28 weeks' gestation had fetal tachycardia >200 beats/min on routine auscultation with a Pinard stethoscope. Fetal M-mode echocardiography showed a ventricular rate of 240 beats/min with a 1:1 atrioventricular relation. The heart was structurally normal, but moderate pericardial effusion and ascites were noted. The mother was given three oral doses of digoxin 500 µg every 8 hours and then 500 µg daily. Echocardiography showed a persistent supraventricular tachycardia in spite of trough maternal serum digoxin levels of 1.5 µg/L and amniotic fluid levels of 0.2 µg/L. Flecainide 110 mg (1.4 mg/kg) was then given intravenously under maternal echocardiography and fetal echocardiographic control. Fetal sinus rhythm at 120 beats/min supervened. Oral flecainide 100 mg every 8 hours was substituted for digoxin treatment. Trough maternal serum flecainide concentration was 656 µg/L (adult therapeutic range 400 to 1000 µg/L). The fetus stayed in sinus rhythm, and all traces of pericardial and ascitic effusion resolved within 10 days. Labor was induced at 38 weeks' gestation. A 12-lead electrocardiogram on the 3450 gm female infant showed sinus rhythm with no evidence of preexcitation. Concentrations of flecainide in maternal and cord plasma obtained simultaneously 5 hours after the last oral dose were 833 and 533 µg/L, respectively, confirming good transplacental passage of flecainide. The baby remained in sinus rhythm without further medication.

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REFERENCE

1. Macphail S, Walkinshaw SA. Fetal supraventricular tachycardia: detection by routine auscultation and successful in-utero treatment. Case report. *Br J Obstet Gynaecol* 1988;95:1073-6.

Reply

To the Editors: I thank Mills for his interest in our article. We are aware of his case and another case published by Wren and Hunter.¹ Unfortunately, the JOURNAL allows only two references in brief communication articles, and we chose the two that were most appropriate for our paper.

We believe that flecainide acetate is a safe alternative for fetuses with hydriopic changes unresponsive to di-

goxin. Flecainide acetate has been associated with lethal ventricular tachyarrhythmias in patients who received it to suppress arrhythmias caused by myocardial infarction. Certainly we must keep this in mind for maternal safety, but it is very unlikely that the heart of a healthy pregnant woman would be subject to the same effect. It is prudent, however, to administer this medication in a hospital setting under electrocardiographic observation of the maternal heart, especially when the medication is administered intravenously. Baseline electrocardiography before treatment with follow-up studies until therapeutic levels are achieved in the mother may be satisfactory in patients who receive the medication orally.

For these reasons it becomes clear that flecainide acetate should be used in the treatment of fetal supraventricular tachycardia only when other safer measures have failed to help the fetus. We hope that future cases will also be published so that we can gain more experience with the use of this medication and increase its safety margin.

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REFERENCE

1. Wren C, Hunter G. Maternal administration of flecainide to terminate and suppress fetal tachycardia. *BMJ* 1988; 296:249.