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Editorial Comment

Fetal tachycardia

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THE REPORT OF TRIEDMAN AND COLLEAGUES IN THIS issue of the Journal[1] details the management of two cases where there was successful conversion of fetal tachycardia after maternal loading with procainamide. This needs to be interpreted with caution. The use of this drug in the fetus was first suggested by Dumesic and co-workers in 1982.[2] Since then, it has been tried frequently in cases of refractory arrhythmias, but without consistent success.[3,4] Many different drugs have been used in the attempt to achieve control of fetal tachycardia. Eight are listed by the authors, and I can add adenosine to the list. The reason so many drugs have been tried is that there is no "magic bullet" or drug that will work in all circumstances without fetal risk. This may be because of differences between patients in the underlying conduction defect triggering the tachycardia.

It could be argued that, in the first case the authors describe, a more appropriate drug would have been flecainide,[5] as placental transfer is known to be good. Although there is a risk of a proarrhythmic effect, this was the drug which was eventually used postnatally, three other drugs having failed. In addition, the authors note that procainamide can have a proarrhythmic effect but are reassured by the absence of this effect in these two cases. But in our series using flecainide, we had no fetal losses in the first 15 cases. We then had three in the next 15, presumably from a proarrhythmic effect. Thus, it is clear that any drug needs extensive evaluation in this setting.

I agree with the contention of the authors that, too frequently, drugs are tried and abandoned before they have had a real chance of working. This criticism could also be made, nonetheless, of the use by the authors of digoxin. In neither case was an adequate dose of digoxin used, nor were adequate maternal serum levels obtained. Recommended levels of maternal digoxin are 2 micrograms/liter, as placental transfer in the hydropic fetus is of the order of 40-50%.[6] At the levels achieved by the authors in the maternal serum, the fetus was not in the therapeutic range. Because this is the safest drug, I still feel it should probably be the drug of choice as a starting point, despite the fact that it will work in only about half the cases, and needs to be used in near toxic doses.

The main message from this report is that fetal tachycardias, particularly when the fetus is hydropic, are difficult to treat. Whatever drug is used, adequate dosage is required, allowing for increased maternal blood volume and drug clearance. The authors state correctly that control and complete clearance of hydrops can take one to two weeks, depending on the severity of the cardiac failure. Persistence is needed with the form of therapy chosen before it is replaced by another potentially dangerous antiarrhythmic drug. No single drug will be efficacious in every circumstance. Those of us who see cases of fetal tachycardia in large numbers need to become familiar with the use of a small selection of agents with which our arrhythmology colleagues are experienced.

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