Beta blockers in pregnancy and their effect on regional Doppler ultrasound and fetal weight

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Summary: Beta-blockers taken throughout pregnancy lead to abnormalities in the fetoplacental circulation manifested by abnormal Doppler ultrasound and impaired growth.

Key words: Beta-blockers; Doppler; Growth restriction.

SUBJECTS AND METHODS

I studied five women who took atenolol throughout pregnancy with doppler ultrasound and one who was treated with acebutalol.

Flow velocity waveforms were obtained using a Hewlett Packard scanner from the umbilical artery, middle cerebral artery and descending abdominal aorta, and the A/B ratio calculated as an average of three measurements. The last result obtained prior to delivery was documented along with the umbilical artery and vein acid base status.

RESULTS

Weight

All the babies were severely growth retarded with birth weights < 3rd centile when corrected for sex and gestational age (corrected ultrasound) based on charts by Gairdner & Pearson and confirmed by the standard birthweight deviation scores for all babies with the exception of Case 1 (too premature for the nomograms (Altman & Coles, 1980 ([1])).

Doppler

Two of the six women showed normal umbilical artery flows while in four it was severely abnormal - absent end diastolic flow being demonstrable in all four. The fetuses in these cases also showed absent end diastolic flow in the descending aorta and significant brain sparing in the middle cerebral arteries in three of the four as shown by a low A/B ratio. The two women with normal U/A flows also demonstrated abnormally low middle cerebral ratios.

DISCUSSION

Medical treatment of essential hypertension during pregnancy has been the subjects of much debate, particularly in relation to the fetal outcome. Beta blo-

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Table - *Patients details.*

Case	Drugs	Indication	DOPPLER		GASES			Birth-weight	Gestation (weeks)	Centile	Standard Birth-weight Deviation Score	Delivery	Reason
			Umbilical Artery	Aorta	Middle Cerebral	Umbilical pH	Artery BE						
1	Atenolol 100 mg	Essential Hypertension	AEDF	AEDF	2.9	7.227	–1.4	1080 g	32	<3rd	N/A	LUSCS	Abnormal Antenatal CTG
2	Atenolol 50 mg	Wolff Parkinson White	AEDF	AEDF	2.7	7.308	–5.9	1850 g	35	<3rd	–1.9	LUSCS	Fetal distress post ARM
3	Atenolol 100 mg	Essential Hypertension	2.4	–	2.2	–	–	2280 g	39	<3rd	–2.5	SVD	
4	Atenolol 50 mg	Supraventricular Trachycardia	AEDF	AEDF	2.0	7.32	3.2	1650 g	35	<3rd	–2.1	LUSCS	Abnormal Antenatal CTG
5	Atenolol 100 mg Nifedipine 20 mg bd Alphamethyldopa 250 mg bd	Essential Hypertension +superimposed P.I.H.	AEDF	AEDF	4.1	7.309	–0.9	1740 g	35	<3rd	–2.8	LUSCS	Abnormal Antenatal CTG
6	Acebutalol	Essential Hypertension	3.2	13.2	2.0	7.28	3.0	2480 g	39	<3rd	–2.8	LUSCS	Abnormal Antenatal CTG

ckers have been shon to decrease the mean fetal birth weight at delivery when compared to a control group (Butters, *et al.*, 1990) ([2]). This does not seem to cause any long term harmful effects on these infants (Reynolds *et al.*, 1984) ([3]).

Atenolol, a B1 selective blocker has become accepted in pregnancy (Rubin *et al.*, 1984) ([4]). The use of long term atenolol treatment in women wishing to conceive has not been studied although there has been one case report in the literature to date with a successful outcome in a woman with essential hypertension (Fowler *et al.*, 1984) ([5]). It is known that growth retarded fetuses are more likely to exhibit abnormal doppler velocity flow waveforms in the fetal and uteroplacental circulations but the significance of this finding is as yet nuclear. Reversed or absent end diastolic flow in the umbilical artery is however thought to be associated with an increased incidence of "fetal distress" (Reuwer *et al.*, 1987) ([6]) and resultant perinatal morbidity and mortality (Brar & Platt 1988 ([7]), Johnstone *et al.*, 1988 ([8])).

From the results it can be seen that two of the six women described showed normal umbilical artery flows while in four it as severely abnormal - absent end diastolic flow being demonstrable in all four. The fetuses in these cases also showed absent end diastolic flow in the descending aorta and significant brain sparing in the middle cerebral arteries in three of the four as shown by a low A/B ratio.

The two women with normal U/A flows also demonstrated abnormally low middle cerebral ratios. It is interesting hoever that none of the five fetuses from whom cord gases were obtained showed any evidence of significant intrauterine hypoxia on testing despite the fact that all five were delivered for "fetal distress".

CONCLUSION

Beta blockers used throughout pregnancy including the time of conception interfere with the fetoplacental circulation causing abnormalities which can be demonstrated by doppler ultrasound leading to compromised fetal growth, although this has not had any obvious harmful effects on the pregnancies reported here.

From this work it cannot be confirmed that AEDF is a reliable predictor of fetal distress.

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