ventricle and pulmonary artery. Half of all emboli to the right ventricle cause a major complication or death.[4]

Before 1970 embolic fragments were either left or removed at thorocotomy. Various less invasive techniques have been employed using snares, hooks, forceps, and ureteric stone baskets.[2-5] The percutaneous snare technique is widely practised and has minimal morbidity and a high success rate in the hands of a skilled cardiac catheteriser. The femoral vein approach is preferred, as a formal cutdown is not necessary and the risk of dislodgment of the proximal end of the catheter is less than when an antecubital or neck vein approach is used. Catheter fragments in the pulmonary artery may necessitate a longer catheter or an approach from an antecubital or neck vein.[3]

Catheter emboli should be removed as soon as possible by skilled operators and, when transvenous methods fail, direct operative intervention is advised, provided the patient's condition will tolerate it. If the infraclavicular subclavian vein is used the catheter should be inserted through a polyethylene introducer and not a metal needle.

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Propranolol in hypertension during pregnancy

The use of beta-blocking drugs during pregnancy has been questioned for two main reasons: their possible stimulant action on uterine muscle,[1 2] and their pharmacological effects on the fetus.[3 4] In an earlier retrospective study of 10 hypertensive pregnant patients propranolol was used with success.[5] We report a prospective study of nine similar high-risk pregnancies followed to term in which propranolol was used to treat hypertension.

Method and results

Three primiparous and six multiparous patients received propranolol for hypertension. In one patient treatment had been started before pregnancy; in five, it was started within three months of the onset of pregnancy; and in three it was started between the fourth and fifth month. The patients' ages, doses used, and any additional antihypertensive treatment given are shown in the table. Patients were examined monthly until seven months, then every two weeks until delivery. Oedema, weight changes, and proteinuria were recorded. Supine blood pressure after five minutes' rest was recorded and urinary oestradiol titrated. Uterine contractions and fetal heart rates were recorded at each visit from seven months. At birth the newborn was weighed, the Apgar index calculated, and plasma glucose concentrations recorded.

The table shows the blood pressure readings. One patient only had a final blood pressure of >145/95 mm Hg. No proteinuria or clinical oedema was recorded and in no case was the weight gain over 12 kg. In seven cases urinary oestradiol concentrations were within normal limits to the end of pregnancy. In the other two patients, the levels stagnated at the lower limit of normal (these two patients had caesarean sections at 35 weeks). Only one patient showed any abnormality of uterine contractions, weak contractions occurring at 7½ months which disappeared at the next recording in spite of continuing propranolol treatment. Labour was normal at 40 weeks.

Except in case 3, the fetal heart rates were between 120 and 150 beats/minute. Apgar index at 1 minute was 10 in five cases, but lower in the others (8, 7, 1); Apgars at 5 minutes were 10 in these eight cases. Blood glucose concentrations recorded in seven of these eight infants were normal (⩾2·2 mmol/l (⩾40 mg/100 ml)) in five and at the lower limit of normal in the remaining two. In case number 3 the infant was born at 35 weeks by caesarean section for fetal distress (heart rate 110 beats/minute). Infant weight was 1320 g and the Apgar index was 0 at one minute and 8 at five minutes but the plasma glucose concentration was above 2·2 mmol/l. Despite immediate referral to intensive care this child died at three months of hyaline membrane disease with acute on chronic distress. Of four previous pregnancies in this patient, there had been two abortions, one premature delivery of a stillborn child, and one premature delivery of a living child.

Comment

The blood pressure was controlled in eight of the nine patients during pregnancy by propranolol with no effect on uterine contractions or increased frequency of abortion and premature labour. No congenital malformations were seen in the six patients started before three months and propranolol did not seem directly responsible for fetal or maternal distress. Of the four patients who had had previous complicated pregnancies including hypertension, three had a much more satisfactory course with propranolol and in the fourth patient no improvement over previous pregnancies was seen. These results need confirmation but indicate that propranolol is effective in reducing high blood pressure in pregnancy without increasing mortality in babies already at risk.

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Blood pressure readings in nine mothers treated with propranolol

Case No	Age	Dose of propranolol (mg/24 h)	Additional treatment	BP before treatment (mm Hg)	Last BP before delivery (mm Hg)	Infant birth weight (mg)	Onset of labour (weeks)	Caesarean sections and reason
1	23	40–120	Triamterene Polythiazide Dihydrallazine	155/90	120/88	1900	35	Caesarean section for stagnation of urinary oestradiol concentrations (two previous abortions)
2	19	40–60	Spironolactone Dihydrallazine	175/105	130/85	3100	40	Caesarean section for obstetrical reasons
3	25	60	Spironolactone Furosemide Dihydrallazine	170/100	130/90	1320	35	Caesarean section for weakening of fetal heartbeat
4	28	60		155/95	140/80	3200	40	
5	35	40	Dihydrallazine	170/130	110/80	2950	37	Caesarean section for obstetrical reasons
6	22	60–40		160/100	135/80	4730	40	
7	28	60	Alphamethyldopa Dihydrallazine	160/110	130/80	3180	41	
8	32	40–100		190/90	150/80	2800	42 (?)	
9	32	40–240	Spironolactone Dihydrallazine	150/105	160/110	2830	35	Caesarean section for intractable hypertension in spite of treatment