PAPERS AND ORIGINALS

Selective induction of labour

K O'DRISCOLL, C J CARROLL, M COUGHLAN

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Summary

In a prospective study of 1000 consecutive primigravidae labour was induced on 95 occasions. None of 16 perinatal deaths and none of 4 cases of suspected brain damage occurred after prolonged pregnancy or pre-eclampsia. It is concluded that a low incidence of induction is compatible with good results and that enthusiasm for the statistical concept of high risk in obstetric practice should be reviewed in the interest of mothers and children as individuals.

Introduction

Over the past decade there have been two notable changes in attitudes to labour. The main change concerns management during labour, and it is now widely accepted that once labour has started delivery within a reasonable time should be ensured.[1,2] The other change has been in the attitude to induction, the natural onset of labour being anticipated in more than 50% of cases in some hospitals. This practice has given rise to expressions of concern, which have not been confined to the medical press.[3] It also conflicts with modern principles of management in labour, which aim to reduce stress by restricting the duration of exposure. This paper examines the medical need for induction.

Methods

A prospective study of 1000 consecutive primigravidae was conducted in this hospital between 1 February and 12 June 1975. The study was confined to primigravidae to eliminate the variable feature of parity, which has an important bearing on induction as on almost every other aspect of labour. The mean age at delivery was 24·3 years, the age distribution being: less than 20 years, 192 patients; 20-24 years, 432 patients; 25-29 years, 260 patients; 30-34 years, 80 patients; 35-39 years, 28 patients; and 40 years and over, 8 patients.

In a pilot survey the incidence of induction declined sharply without any change in policy. The indications for induction came under three headings: prolonged pregnancy, pre-eclampsia, and miscellaneous. The two main conclusions were that induction was seldom undertaken in the interest of the mother, and that the indications entered in the records could often not be substantiated in the interest of the child. As a result of the pilot survey a more critical attitude to induction was adopted, and it was decided to avoid vague conventional terms, such as prolonged pregnancy and pre-eclampsia, and to record instead simple facts—blood pressure, proteinuria, and duration of pregnancy.

Results

Altogether 95 of the 1000 patients had labour induced. There were 16 perinatal deaths and four cases of cerebral dysfunction, none of which occurred after induction; 51 patients underwent caesarean sections.

INDICATIONS

The indications for induction were: hypertension with proteinuria (28 cases), pregnancy of 42 weeks or more (24), placental insufficiency (14), antepartum haemorrhage (13), hypertension (8), diabetes mellitus (4), twins (2), and unclassified (2). Labour was induced before 37 weeks on only one occasion; this was at 36 weeks because of hypertension with proteinuria. Placental insufficiency was a primary diagnosis; none of the patients showed signs of toxaemia, and the duration of pregnancy was less than 42 weeks.

PERINATAL DEATHS

Of the 16 perinatal deaths five occurred during pregnancy, one during labour, and 10 after birth. Necropsy was performed in all cases. Four of the deaths during pregnancy were attributed to placental insufficiency and one was attributed to abruption of the placenta. No mother had hypertension or proteinuria and no death occurred after 40 weeks. The deaths attributed to placental insufficiency occurred at 32, 34, 35, and 39 weeks; the weights of these macerated infants were 1040 g, 1400 g, 1420 g, and 2180 g, all of which are below the fifth percentile on the Aberdeen scale. The death attributed to abruption

University Department of Obstetrics and Gynaecology, National Maternity Hospital, Dublin 2

K O'DRISCOLL, MD, FRCOG, clinical professor
C J CARROLL, MRCPI, MRCOG, assistant master
M COUGHLAN, FRCSED, MRCOG, assistant master

of the placenta occurred at 38 weeks; the birth weight was 2200 g, which is also below the fifth percentile.

The one death during labour occurred nine hours after admission at 41 weeks. Birth weight was 3960 g, and the liquor amnii was copious and clear. Necropsy showed evidence of hypoxia.

The neonatal deaths were attributed to congenital malformation in four cases, premature delivery in four cases, traumatic intracranial haemorrhage in a breech presentation complicated by cord prolapse in one case, and rupture of a vasa praevia late in labour in one case. The congenital malformations were Edwards's syndrome, Potter's syndrome, diaphragmatic hernia, and anencephalus. The deaths attributed to premature delivery were caused by respiratory distress after spontaneous labour at 29, 29, and 31 weeks, and after caesarean section at 33 weeks; necropsy showed hyaline membrane disease, with intraventricular haemorrhage and pulmonary haemorrhage in two cases. A rupture of the tentorium cerebelli was found in the case of the breech presentation and exsanguination in the case of the vasa praevia.

BRAIN DAMAGE

In the four cases of cerebral dysfunction no mother had hypertension or proteinuria and no case of cerebral dysfunction occurred after 40 weeks. The durations of pregnancy were 37, 37, 38, and 38 weeks, and the birth weights were 3150 g, 2920 g, 3620 g, and 2830 g, all of which are above the tenth percentile. Paediatric assessments of the gestational ages of the newborn infants were 37, 38, 40, and 38 weeks. The infants were resuscitated by intubation for 3, 0, 3, and 10 minutes, and the Apgar scores were 7, 8, 7, and 7 at five minutes. Cerebral dysfunction was attributed to trauma in the first case because blood was found in the cerebrospinal fluid. Evidence of cerebral dysfunction persisted at discharge from hospital only in the last case; the mother, aged 18, had been in labour for two hours, when forceps were applied for fetal distress.

CAESAREAN SECTION

Of the 51 caesarean sections three were performed after induction of labour, once for fetal distress and twice for failed induction.

Discussion

The results of this study provide a basis for comparison with centres at which the incidence of induction may be greater by a factor of five or more, and are therefore worthy of close scrutiny. An impressive finding was that in none of the 16 deaths and four cases of cerebral dysfunction was prolonged pregnancy or pre-eclampsia a feature.

Although all five infants who died during pregnancy were of low birth weight, in no case was there a conventional indication for induction. A decision to deliver would need to have been based on a diagnosis of retarded fetal growth on an individual basis, and if a liberal policy of induction had been pursued none of these cases would have been included. The birth weight was normal in the only case in which death occurred during labour, and the circumstances suggest that the problem originated in labour; this was the only death after 40 weeks. The 10 neonatal deaths were certainly not amenable to delivery at an earlier date. All four infants with evidence of cerebral dysfunction were of normal birth weight; the evidence suggests that the damage was inflicted during labour and would not have been averted by induction; all were born before 40 weeks.

The concept of high risk has greatly influenced contemporary obstetrics. Butler and Bonham[4] firmly established that prolonged pregnancy and pre-eclampsia are associated with an increased perinatal mortality. Thus in many hospitals labour is induced at 42 weeks precisely, and in every case of pre-eclampsia, no matter how trivial. Our results raise doubts about the validity of this statistical approach to clinical practice.

The vague terminology in current use tends to obscure the fact that in most cases induction of labour is performed for the benefit of the fetus, and this should be acknowledged by expressing the indication for induction in fetal instead of conventional maternal terms. Placental insufficiency is almost the sole fetal indication for induction and may occur as a primary condition without any maternal abnormality; conversely, prolonged pregnancy and pre-eclampsia are usually not associated with placental insufficiency. The release of a copious flow of clear liquor at routine induction for prolonged pregnancy presents a paradox, because the action that commits the patient to delivery shows that the fetus is not in danger, while a similar conflict of purpose is introduced in pre-eclampsia every time a fetus who is considered to be at risk is subjected to an extended period of stress with oxytocin.

Our series shows what can be achieved by a policy of selection. Induction was performed in only 24 out of 126 cases in which pregnancy was prolonged to 42 weeks or more, and in only eight cases of pre-eclampsia without proteinuria. Selection was based on careful clinical assessment of fetal growth and liquor volume by the same obstetrician at every antenatal visit, and by admission of suspect cases to hospital for further evaluation of placental function. Special care was taken not to intervene when the circumstances were unfavourable for induction without clear evidence that the fetus was in danger. There were no routine inductions.

Thus the time seems opportune to consider again the evidence on which the concept of high risk is based before the welfare of patients as individuals is submerged in statistics. Butler and Bonham[4] showed that when pregnancy was prolonged to 42 weeks or more perinatal mortality was increased by 72% over the mortality at term. In our series there were four perinatal deaths among 800 deliveries that occurred between 38 and 42 weeks. On the basis of Butler and Bonham's statistics seven perinatal deaths would have been expected if all these 800 pregnancies had continued to 42 weeks or more. It may be deduced from these figures that routine induction at 42 weeks would have exposed the 800 mothers, to whom prolonged pregnancy presented no danger, and 800 babies to the disadvantages of induction to prevent three perinatal deaths. This is a crude approach to a common problem, yet Cole *et al*[5] suggested that routine induction might be practised at 41 weeks or even at 39 weeks to reduce perinatal mortality.

The other main indication for induction, pre-eclampsia, is open to even more stringent criticism. Butler and Bonham showed that there is no increase in perinatal mortality in pre-eclampsia without proteinuria when the diastolic blood pressure is below 100 mm Hg; as the diastolic pressure in pre-eclampsia does not often exceed 100 mm Hg without proteinuria this means that pre-eclampsia without proteinuria is seldom a valid indication for induction.

There is a subtle influence in obstetrics that operates to absolve a doctor who intervenes in the course of normal pregnancy and which, by implication, exposes his conservative colleague to censure for inactivity when an infant is born dead. This places a premium on intervention as a form of personal insurance for the doctor, although the consequences are detrimental to some patients. Vague descriptive terms, such as prolonged pregnancy and pre-eclampsia, encourage intervention because they are sufficiently elastic to include almost every patient. Sturrock[6] drew attention to an attitude of mind that gains momentum when a policy of intervention is adopted; this has special significance in teaching hospitals.

The conclusion is that good results may be achieved with a low incidence of induction when patients are treated as individuals; an uncritical acceptance of the statistical concept of high risk has led to a steady increase in interference with the course of normal pregnancy.

ADDENDUM—The four infants suspected of brain damage have been reviewed since this paper was submitted for publication. At 6 months of age two infants showed evidence of cerebral palsy and mental handicap and two appeared normal. The

infants who showed evidence of permanent brain damage are the first and last cases described in the text.

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SHORT REPORTS

Retrobulbar neuritis and infectious mononucleosis

In a recent leader in the *British Medical Journal*[1] on the aetiology of optic neuritis it was stated that, "By no means all cases of optic neuritis are due to multiple sclerosis and other causes, particularly in the older patients, should be remembered." We would like to support this statement by reporting a case of retrobulbar neuritis complicating infectious mononucleosis in an 18-year-old male student.

Case Report

An 18-year-old student was admitted to hospital with a 24-hour history of loss of vision. Two weeks previously he had had an upper respiratory tract infection and for one week bitemporal headache, pain when moving both eyes, and tiredness. On examination he was found to have bilateral retrobulbar neuritis, in addition to palatal petechiae and generalised lymphadenopathy and splenomegaly. Apart from the ocular signs no other neurological abnormality was shown then or subsequently.

Investigations showed haemoglobin 14·4 g/dl, erythrocyte sedimentation rate 28 mm in 1 h, white blood count $6{\cdot}2 \times 10^9$/l (6200/mm^3) (neutrophils 19%, lymphocytes 76%, monocytes 5%). Blood film showed many atypical lymphocytes. A Monospot test gave a strongly positive result; the titre of the Paul-Bunnell test was 1/1286; cerebrospinal fluid and Lange's test result were normal; and the Epstein-Barr virus titre (fluorescent antibody test) was 80. Vision in the right and left eyes was 6/36-1 and 6/60 respectively.

He was treated with 1 mg ACTH (Synacthen Depot) twice weekly. The patient reported some minimal improvement in his vision, but there was no objective evidence of this. After nine weeks the ACTH was increased to three times weekly but the optic atrophy progressed, particularly on the left. Because of the severity of the visual defect a prolonged course of treatment seemed justified. Treatment was finally stopped after a further nine weeks, his visual acuity remaining unchanged.

Discussion

Neurological complications in infectious mononucleosis are well known but seldom lead to significant permanent damage. The ocular manifestations of infectious mononucleosis were reviewed by Tanner[2] in 1954. He divided the clinical features into two groups: those possibly due to direct involvement of the eye, such as conjunctivitis, eyelid and periorbital oedema, episcleritis, uveitis, optic neuritis, papilloedema, retinal oedema, and haemorrhage; and those due to a more remote lesion in the central nervous system such as extraocular muscle palsies, ptosis, and disturbances of conjugate deviation.

Probably the earliest report of optic neuritis in glandular fever was by Clemens[3] in 1907, but corroborative details are lacking. The first authenticated report of retrobulbar neuritis without other central nervous system involvement was by Ashworth and Motto[4] in 1947; subsequent reports, together with a case report, were reviewed by Frey[5] in 1973—a total of 8 cases.

All the cases reported to date did well, with or without steroid therapy, and complete recovery was the rule. Unfortunately our patient did not follow this pattern. Possibly he has some other condition, but as yet none has been shown.

We suggest, therefore, that the possibility of infectious mononucleosis as a cause of retrobulbar neuritis should not be overlooked.

We thank Dr C R S Jackson and his staff, at Princess Alexandra Eye Pavilion, Royal Infirmary, Edinburgh, for their help in management of this case, and Mrs G B McKenzie for her secretarial help.

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Department of Infectious Diseases, City Hospital, Edinburgh EH10 5SB
S PICKENS, MB, MRCP, senior registrar
G SANGSTER, MD, MRCP, consultant physician

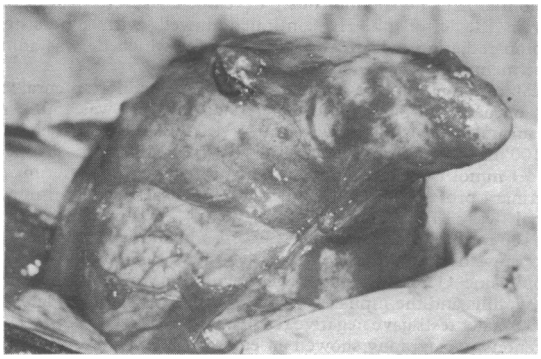
Hydatid cysts in pancreas

In only two out of 780 cases of hydatid disease affecting various tissues and organs operated on in one surgical unit in Baghdad during the period 1963-75 was the cyst located in the pancreas—an incidence of 0·25%.

Case reports

The first case was that of a 27-year-old woman complaining of a gradually growing abdominal lump and heaviness in the left upper abdominal quadrant for the past year. There was a palpable mass ($7{\cdot}6 \times 10{\cdot}2$ cm) notched in its lower pole, in the left hypochondrium. It moved vertically with respiration, was smooth, mobile, and not tender and was thought to be a large spleen. The results of *x*-ray examination of the chest and abdomen, routine haematological investigation, liver function tests, and Casoni's test were all normal.

At laparatomy a large hydatid cyst was found protruding below the stomach and adherent to the transverse colon, mesocolon, and omentum. It arose from the body of the pancreas at its junction with the tail and had a bud on its lower pole, producing the notch mentioned above (see fig). The liver and other abdominal viscera were normal. The cyst cavity was full of milk-coloured hydatid fluid, with many daughter cysts and shredded membranes.



Hydatid cyst of body of pancreas.