Management of premature rupture of membranes

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Today I will attempt to put into perspective the opposing philosophies of whether or not to intervene in pregnancies complicated by premature rupture of the membranes which I define as positive evidence of leaking amniotic fluid before the 38th week of pregnancy calculated from the first day of the last menstrual period.

I would like to start by presenting some relevant facts and figures on preterm delivery (whether complicated or not by PROM). This occurs with a frequency that varies from 5-10 per cent (Rush et al. 1976; Daikoku et al. 1981), tending to be higher in urban populations with social and medical problems. Fetal growth retardation complicates preterm delivery in about 25 per cent of cases and fetal malformations are three times higher than in the population as a whole (Howie and Liggins, 1977). In Howie and Liggins series of preterm labours, 23 per cent had complications of pregnancy such as fulminating toxaemia (8.3%) and placenta praevia of a major degree (2.2%) necessitating immediate delivery, regardless of whether the membranes were ruptured or not. Intrapartum asphyxia occurs more commonly in preterm labour. Hobel (Hobel et al. 1972) has shown that the asphyxiated preterm fetus is more likely to develop RDS and to die than the well-oxygenated fetus of an equivalent weight. Figure 1 shows how neonatal care has significantly improved the prognosis of the preterm fetus to the extent that after 32 weeks of age, survival is almost assured (Mutch et al. 1981). These are all important considerations when determining the management of premature rupture of the membranes (PROM).

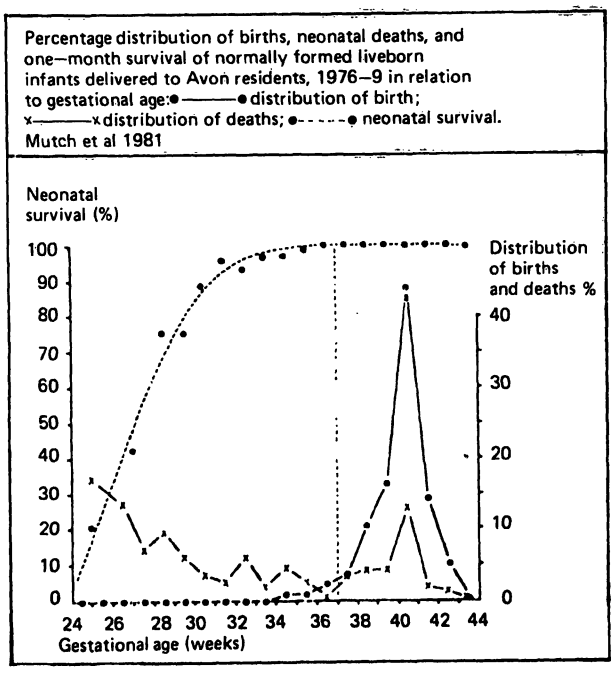

Fig. 1

Preterm labour is preceded by PROM in 40% of cases (Gillibrand, 1967 - 38%, Daikoku et al. 1981 - 42%) the remaining 60% going into preterm labour with intact membranes. In the group with intact membranes

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labour usually progresses, but in the PROM group it is delayed more than 24 hours in 40-50% of cases. In this group, the earlier PROM occurs in pregnancy the more prolonged is this latent period (Miller et al. 1978).

Traditionally, management of PROM is confined to expectant observation on the rationale that as the fetus becomes more mature so the mortality and complications of prematurity diminish. There is good evidence to support this view for, not only do fetal lungs become functionally more mature with advancing gestation, but it is now proven beyond doubt that between 30 and 34 weeks gestation there is a significant reduction in the incidence of respiratory distress (RDS) with prolongation of the latent period between membrane rupture and delivery. This effect is well shown in Figure 2 from a recent study in which there was a 50% reduction in RDS that can be ascribed to PROM (Varner and Galask, 1981; Howie and Liggins, 1977). However, with the advent of neonatal intensive care, a more active form of management with early delivery after PROM has a more logical basis. The rationale underlying this approach is that a proportion of babies of mothers with PROM will be born in poor condition due to intrauterine infection or asphyxia. The preterm baby is known to have a reduced resistance to infection (Levy and Arquembourg, 1981) so that, ergo - the longer the latent period between membrane rupture, the greater the risk of neonatal infection, respiratory distress and perinatal mortality. Logically, early delivery is likely to avoid these complications, but this benefit has to be balanced against the risk of pulmonary immaturity. Let us examine the evidence for and against active management of PROM.

Effect of duration of rupture of membranes by gestational age on the incidence of RDS.
(Varner and Galask, 1981. Am J Obstet. Gynec. 140, 39–45)

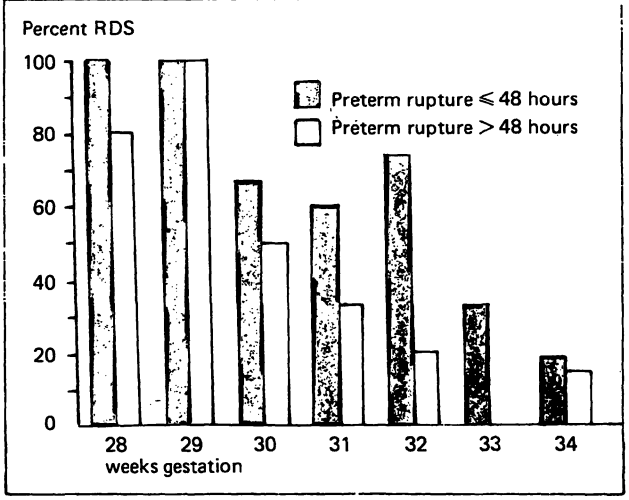

Fig. 2

	PROM+ve	PROM−ve	Incidence of sepsis in whole preterm labour group: Maternal	Neonatal
Daikoku et al (1981)	203	274	–	13.3 (6.2)
Miller et al (1978)	151	184	8 (1)	13 (3.0)
Overall Results	354	458		13.2 (5.0) $p < 0.001$

Incidence of all types of maternal and neonatal infections in those with premature rupture of membranes (PROM+ve) and those without (PROM−ve). Figures in brackets relate to the incidence in full term pregnancies.

Table 1

Table 1 shows the results of two recent studies (Miller et al. 1978; Daikoku et al. 1981) which comprises 354 mothers with PROM who were not in labour and 458 who went into preterm labour without PROM. In

neither of these studies were antibiotics used unless there was clinical evidence of infection, and labour was allowed to start spontaneously. In both studies there was a preterm labour group as compared with full term babies, but no difference between those with intact compared with ruptured membranes. In the study by Miller et al. (1978), which also reported on maternal infections, although the incidence of 8 per cent was small it is significantly higher than in full term deliveries and all the cases occurred in mothers with PROM. Table II shows in the recent study by Daikoku et al.(1981) that

		Preterm (<37/52)		Term (>37/52)
	Total (%)	PROM+ ve (%)	PROM – ve (%)	(%)
PNM	16.3 (78/477)	13.8	18.3	4.4
Infection	1.3	2.0	0.7	0
RDS	5.9	3.9	7.3	0.5
Anoxia	7.3	6.4	8.2	0.3
Anomaly	1.7	1.5	1.8	0.2

Perinatal Mortality (PNM) and major causes of PNM in preterm (477) and term (2300) groups and related to premature rupture of membranes (PROM+ve) and intact membranes (PROM–ve).
Daikoku et al (1981). Obstet. Gynec. 58:417–425.

Table II

(1) although perinatal mortality (PNM) amongst preterm babies was four times that among the mature babies in the control group, there was no significant difference in PNM in the groups with and without PROM,

(2) infection only contributed to 14 per cent of the perinatal deaths although it was higher in the PROM group. Respiratory distress (28%) and anoxia (46%) were much more important causes in the PROM group. Thus it seems that although the infection rate is significantly increased in mothers who go into preterm labour, and in their babies, there is no significant difference between those with and without PROM. Likewise the incidence of intrapartum anoxia as a cause of perinatal death is not significantly higher in the PROM group as compared with those with intact membranes.

Thus, on the face of it, there appears to be a good case for conservative management of PROM rather than proceeding to immediate delivery. Unfortunately the matter is not quite so clear cut as that. The benefit from a policy of awaiting the spontaneous onset of labour in those with PROM comes from a lower incidence of RDS amongst the newborn and possibly a reduced rate of operative delivery. However, one has to take into consideration the evidence of Liggins who has shown convincingly in preterm babies of up to 34 weeks gestation born during the first week after PROM that the administration of steroids to the mother reduces the incidence of RDS and that in this series, 58 per cent of these women had delivered by the end of the first week. He showed that in the remaining 42 per cent who were delivered after the first week, the incidence of RDS fell regardless of whether steroids were administered or not. Thus if steroids are administered to the mother immediately after PROM and delivery then expedited, it is possible that the fetus

will not only have a reduced risk of RDS but may well be delivered in better condition than if a more conservative approach had been adopted. The more immature fetus of 28-32 weeks of gestational age is most likely to benefit from this approach. The mother is also spared a long miserable wait before her baby is delivered safely.

It would be fair to say that there is insufficient evidence to say, with confidence, whether an active or conservative form of management of PROM is better. With the exception of this study by Liggins, there are no large prospective randomised controlled trials in this field. All that it is possible to say is that awaiting spontaneous onset of labour in women with PROM does not carry a major risk of maternal or neonatal infection. Our own experience suggests that the prophylactic use of steroids in pregnancies up to 34 weeks gestation given as soon as the mother comes into hospital after PROM is probably justified. This is particularly so in maternity units in which neonatal intensive care facilities are not readily available. There is no doubt that the solution to the problem of PROM lies in determining the cause, or causes, of the condition. Until the condition can be actually prevented, whatever is done in the interests of mother and fetus will inevitably be a rather unsatisfactory compromise.

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