

Umbilical cord clamping in preterm infants

EDITOR,—The possibility that late clamping of the umbilical cord may lead to a reduction in respiratory distress syndrome is not a particularly new finding, but S Kinmond and colleagues offer a more scientific approach in evaluating this phenomenon.¹ However, they give little information about the obstetric features of their cases, which might be quite important since matching is essential with such small numbers—for example, were the babies predominantly born during preterm labour or were they delivered electively for some obstetric complication? Also not clear is whether any of the mothers received steroids to accelerate fetal maturity before delivery.

Although these matters may not be particularly important as far as the haematological features are concerned, they certainly are with respect to ventilation. I believe that more information on them would be helpful in the interpretation of the paper.

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1 Kinmond S, Aitchison TC, Holland BM, Jones JG, Turner TL, Wardrop CAJ. Umbilical cord clamping in preterm infants: a randomised trial. *BMJ* 1993;306:172-5. (16 January.)

EDITOR,—S Kinmond and colleagues' results require further consideration.¹ Immediate cord clamping may deprive the infant of placental blood, and previous reports suggest that a long delay may lead to adverse effects due to hypervolaemia.² Holding the infant 20 cm below the introitus for 30 seconds before clamping the cord probably enables an intermediate and beneficial volume of placental blood to enter the fetal circulation.

The question arises as to why a longer (60 second) delay should allow a larger and thus excessive transfusion. Almost all women in Britain receive oxytocin-ergometrine (Syntometrine) at delivery, which causes an increase in plasma oxytocin concentration within 45 seconds.³ The resulting contraction could force an excessive volume of blood into the neonate. If so, the cord should be clamped before the increase in plasma oxytocin concentration. The time between administration of an oxytocic agent and cord clamping rather than fetal delivery and cord clamping may thus be critical. The authors do not state whether their patients received an oxytocic agent or document its time of administration.

Further consideration of the physiological process of labour at term raises an additional point. Even when an oxytocic agent is not given a small proportion of patients show an increase in plasma

oxytocin concentration during the late second⁴ or early third⁵ stage of labour. This increase in endogenous oxytocin could theoretically cause uterine contraction and excessive transfusion within 30 seconds of delivery. Did the authors record the time of uterine contraction after delivery and, if so, was there any relation to neonatal outcome?

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- 1 Kinmond S, Aitchison TC, Holland BM, Jones JG, Turner TL, Wardrop CAJ. Umbilical cord clamping and preterm infants: a randomised trial. *BMJ* 1993;306:172-5. (16 January.)
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EDITOR,—S Kinmond and others' study of early versus late umbilical cord clamping shows an apparent improvement in outcome with a simple intervention but raises some questions.¹ Firstly, is the position of the baby or the timing of the clamping of the cord the primary factor? If the position of the baby is important maybe the two babies in the intervention group who were not held below the introitus should not have been included in the final analysis. Equally, one infant in the random group was held below the introitus, and the position of the others is not stated. Over the whole study was position a significant variable independent of the study group?

We note that the unit's policy was consistent during the study. One of the significant outcomes was the volume of blood transfused, and therefore it is important for the transfusion policy to be stated, especially as the text does not suggest that the staff were blind to the study groups. On the basis of the figures given for the number of infants requiring blood transfusion there is no significant difference between the two groups for either ventilated infants or all infants (χ^2 test, two by two analysis).

There were three babies with chronic lung disease in the random group and none in the regulated group, but the total in the study is too small to say if either figure is different from the expected incidence of about 10% in this population of babies. The blood pressure stability is said to have been no different between the two groups. Was this an overall effect on blood pressure or specifically the beat to beat variability? Beat to beat variability has been used as an indicator of hypovolaemia in preterm infants at risk of intraventricular haemorrhage.²

The proposed mechanism of improvement of placental transfusion of volume and red cells is attractive. As the authors have pointed out previously,³ packed cell volume is a poor indicator of red cell mass and a better measure would have been useful. The improvement may well be largely related to an increase in volume leading to an

increase in blood volume in the lungs—an effect shown by transthoracic impedance techniques.⁴

As the authors state, it is important for neonatal research to look at simple interventions such as this. We believe, however, that further study is needed before firm recommendations about umbilical cord clamping can be made.

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EDITOR,—The paper by S Kinmond and colleagues suggests that modification of a common practice—clamping of the umbilical cord—may have important implications for the wellbeing of immature babies.¹ Most of the previous research into the timing of cord clamping has concentrated on babies born near term.² Rigorous steps to avoid selection biases seem to have been taken in only two of the four studies of immature babies—namely, those of Hofmeyr *et al.*^{3,4}

The first of these two studies is referred to in Kinmond and colleagues' paper as showing a reduction in the incidence of periventricular and intraventricular haemorrhage with delayed cord clamping.³ In addition, however, this policy was associated with more neonatal deaths. As the authors explain, the small sample size (38 babies) led to imbalances in the baseline variables, which could explain the differences in outcome. Because of this the investigators subsequently carried out a larger trial (in 86 babies), which did not confirm either the reduction in haemorrhage or the increase in mortality.⁴ There also seem to have been considerable baseline imbalances in the trial reported by Kinmond and colleagues, judged on the few characteristics described in table I, and so the same concerns must apply.

The inclusion of birth weight as one of the enrolment characteristics is probably inappropriate. Increased placental transfusion in the regulated group should surely have resulted in an increased birth weight, as found in other studies in which cord clamping was delayed.³ Birth weight is therefore more reasonably considered to be an outcome variable.

The principal outcome, and the measure on which the trial sample size was based, is the number of transfusions. In appraising the trial's results it would therefore be helpful to know the indications for transfusion that applied at the Queen Mother's Hospital during the trial and whether the decision to transfuse was made without knowledge of the policy on cord clamping.

Immature babies are even more likely than others to have their umbilical cords clamped and cut immediately after birth to allow resuscitation and transfer to neonatal intensive care. Kinmond and colleagues' study suggests that a short delay

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