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Managing the umbilical cord at birth

It is common practice at birth to clamp and divide the umbilical cord at once in order to separate mother and baby and to allow the collection of cord blood for acid base/blood gas studies or to harvest stem cells. But this intervention abruptly interrupts the fetoplacental circulation and may have a profound influence on the baby's health and welfare. For instance, the timing of cord ligation may have a major impact on the baby's blood volume. During the second stage of labour the soft walled umbilical vein becomes compressed against the side of the birth canal with the result that bloodflow back to the baby is reduced and the placenta becomes congested. Immediate cord clamping then results in an engorged placenta which may lead to third stage problems, and also to a baby who is hypovolaemic and hypotensive with peripheral vasoconstriction at the very time when the infant is attempting to adapt to extrauterine life. On the other hand, delayed cord ligation beyond one minute after birth with the placenta still *in situ*, is likely to result in an over-large placental transfusion, especially if oxytocic agents have been administered. This in turn may result in transitory circulation overload and to polycythaemia. However, the value of a *normal* placental transfusion should not be overlooked; it not only restores the baby's blood volume but also provides baro- and chemo-receptor stimuli to the onset of respiration¹⁻⁶.

Normal fetal adaption to breathing requires first the evacuation of the lung fluid filling the alveoli before birth and its replacement with air following the onset of respiration. This in turn is accompanied by circulatory changes in the lungs, heart and umbilical arteries. Premature cord occlusion reverses the order in which these events take place, imposing circulatory changes prior to the onset of respiration. This may be harmful to the newborn infant, especially when premature or delivered by caesarean section.

The sudden clamping of a vigorously pulsating cord in an infant that has not yet taken a breath will, by cutting off the low resistance placental circulation, lead to a sharp rise in systemic blood pressure. This in turn may lead to transitory 'heart failure' with raised pulmonary as well as systemic venous blood pressure, especially if the baby is hypervolaemic due to a large placental transfusion.

At the same time the raised systemic blood pressure will be transmitted through the patent

ductus arteriosus to the pulmonary arteries just as lung expansion with air is leading to a dramatic fall in pulmonary vascular resistance. Indeed, occlusion of a vigorously pulsating cord is itself a powerful, though not ideal, stimulus to the onset of respiration, probably due to the excitation of a baro-receptor reflex response to raised pulmonary artery blood pressure. Thus the pulmonary capillary bed may be simultaneously subjected to pressure-stress from both arterial and venous sides at the very time when the pulmonary arterial resistance is falling abruptly following the intake of air into the alveoli.

Alveolar over-expansion following spontaneous or artificial ventilation may cause even further stress damage to the structurally fragile pulmonary capillary bed. Premature infants are especially at risk as they tend to lack alveolar surfactant, a substance that helps to protect the capillaries from pressure-stress during pulmonary inflation.

Stress failure of the capillaries leads to pulmonary oedema with leakage of plasma proteins into the alveolar ducts and alveoli where they form the so-called 'hyaline membranes,' destroy and impair the action of surfactant, and lead to atelectasis and respiratory distress¹⁻⁶.

Ideally the mother, preferably in an upright position, should be encouraged to deliver both the baby and then the placenta with the umbilical circulation still intact. Lying alongside each other, normo-volaemia will then be re-achieved, the baby will establish respiration and the umbilical vessels will cease to pulsate. Only then should the cord be ligated and divided.

This indeed was the common practice among many so-called primitive peoples before the advent of western medicine. However, if for any reason it is necessary to clamp and divide the cord before placental delivery, it is best to delay this intervention for between 30-45 seconds after birth in order to allow a partial placental transfusion and also to provide the opportunity for the baby to breathe spontaneously.

If in spite of these measures the newborn infant remains blue and apnoeic, the baby should be resuscitated in a head-up (rather than head-down) position as this aids pulmonary lymphatic drainage and diaphragmatic respiration. Should positive ventilation be required, it should be delivered gently, preferably using a face mask and bag and room air rather than an endotracheal tube and 100% oxygen¹⁻⁶.

References

1. **Dunn P.M.** Caesarean section and the prevention of RDS of the newborn. In: Bossart H., Cruz J.M. et al, eds. 3rd European Congress of Perinatal Medicine, Lausanne, April 1972. Publ. Hans Huber. Bern: 1973: 138-45.
2. **Dunn P.M.** Management of child birth in normal women: The third stage and fetal adaptation. In: Clinch J., Matthews T., eds. Perinatal Medicine. Proc. IXth European Congress Perinatal Medicine, Dublin, September 1984. Lancaster: MTP Press: 1985: 47-54.
3. **Dunn P.M.** In the Delivery Room. In: Fleming PJ., Speidel B.D., Dunn P.M. eds. A Neonatal Vade-Mecum. London: Lloyd-Luke Ltd. 1986: 15-32.
4. **Dunn P.M.** Perinatal factors influencing adaptation to extrauterine life. In: Belfont P., Pinotti J.A., Eskes T.K.A.B., eds. Advances in Gynecology and Obstetrics, Vol. 5, Pregnancy and Labor. Proc. 12th World Congress Obstetrics Gynecology, Rio de Janeiro, October 1988. Carnforth, Lancs: Parthenon Publ. 1989: 15: 119-23.
5. **Dunn P.M.** Stress failure of pulmonary capillaries at birth. *Lancet* 1993; **341**: 120.
6. **Dunn P.M.** Clamping the umbilical cord. *Aims* 2004-5; **16**: 8-9.