

# Clamping the umbilical cord

**Professor Peter Dunn** of Bristol University Medical School argues that, when collecting umbilical blood for banking, it is important to consider the interests of the infant. After all, harvesting 100 ml of blood from a newborn is equivalent to taking 2.5 pints from an adult

**T**he discovery in the 1980s that umbilical cord-blood provided a rich source of haematopoietic [blood-forming] stem cells, which could help children with diseases such as leukaemia, led to the organised collection of blood in the 1990s. The blood was traditionally held in cord blood banks until required and, in some countries, the purchase and sale of cord-blood was commercialised.

In seeking consent to the collection of cord-blood, mothers were usually informed that, once the cord had been clamped, the blood remaining in the placenta was no longer of use to her baby, but might help to save another child's life. However, this advice neglected discussion of the timing of cord occlusion [clamping], and the significance that this might have on the transitional umbilical circulation at birth, on the blood volume of the newly born infant, on adaptation of the fetus to life in the outside world and on the delivery of the placenta.<sup>1-3</sup>

Collectors of cord-blood for banking prefer to harvest a volume of 100–200 ml. To achieve this, early cord occlusion is advised, as illustrated by the following statement from *The Sunday Times* (March, 1996): “It helps if the midwife cuts the cord as soon as possible to enable us to collect as much blood as we can (otherwise it drains into the baby)”.

The collector's aim is all too often fortuitously aided by the fact that many obstetricians and midwives already practice early cord-clamping either to get the baby ‘out of the way’ to expedite delivery of the placenta, to obtain samples of arterial and venous cord-blood for acid-base and blood-gas evaluation, or to transfer the child to the resuscitation area.

Thus, a typical obstetric instruction on this matter reads: “A segment of the cord must be isolated before the first breath, as less than 10 seconds of neonatal breathing can radically alter the cord blood gases.”

Now let us consider how this potentially iatrogenic intervention may affect the baby at a most critical time in its life.

Cardiorespiratory adaptation following birth<sup>1</sup> depends primarily on the achievement of adequate ventilation of the alveoli [tiny air sacs] which leads to and is accompanied by a fall in pulmonary vascular resistance [the resistance on the pulmonary arteries to the blood flow through the lungs]. This results in a greatly increased flow of blood

to the lungs, and of oxygenated blood to the left side of the heart and systemic circulation.

In turn, this leads to secondary changes in the circulation, including the closure of two shunts—the foramen ovale and ductus arteriosus, which allow blood to bypass the lungs during fetal life—as well as the cessation of the umbilical circulation.

The key to successful adaptation appears to be the replacement of the lung fluid by air that fills the alveoli prior to labour. Evacuation of lung fluid is achieved in part by the surge of catecholamines (adrenaline-like substances) that accompanies labour. This cuts down lung fluid secretion and also promotes its absorption. Thoracic

compression, which occurs as the infant's chest passes through the birth canal during the second stage of labour (80 mmHg or more), also helps to squeeze fluid out of the lungs through the mouth or nose, or causes it to be swallowed.

After delivery, the remaining lung fluid is mainly drained through the pulmonary lymphatics into the central venous pool at the level of the clavicles [collar bones], propelled by the ‘milking’ action of respiration. Failure to

adequately clear lung fluid at and soon after birth contributes to a variety of maladaptation syndromes, of which the best known are respiratory distress syndrome, mainly confined to the preterm infant, and transient tachypnoea of the newborn, which is more commonly seen in term infant.<sup>5-7</sup>

A major factor contributing to fetal maladaptation at birth is premature occlusion of a vigorously pulsating umbilical cord prior to the establishment of respiration. This intervention cuts off the low-resistance placental circulation and leads to a sharp rise in the infant's systemic blood pressure. In the presence of a continuing high pulmonary vascular resistance, the heart may, in these circumstances, exhibit transitory failure, with raised pulmonary and central venous pressures.

Raised pulmonary venous pressure may then lead to pulmonary oedema, with the extravasation [escape] of plasma proteins. This, in turn, causes inactivation and displacement of surfactant [a substance lining the alveoli and essential for the prevention of alveolar collapse], and increased surface tension which, especially with the preterm infant, may lead to such medical complications

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***‘Some mothers indicate they do not wish the umbilical cord to be ligated until after all pulsation has ceased—causing a delay of 10 minutes or more.’***

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