Editorial II

Oxygen and elective Caesarean section

It was during the 1960s that it became accepted that additional oxygen was required to maintain adequate saturation during general anaesthesia. If there was a seminal contribution to this process it was the publication of Nunn's serial researches into respiratory function.[1] There has never been a similar defining moment for regional anaesthesia, which can also impair respiratory performance. High blocks that involve paralysis of the intercostal muscles will reduce the contribution of the rib cage to breathing. Satisfactory regional block for Caesarean section requires that Aβ fibres are blocked to the level of the T5 dermatome,[2] and as a consequence there are reductions in maternal peak expiratory flow rate, forced vital capacity, and forced expiratory volume. Despite these changes, maternal oxygen saturation is maintained.[3] Although breathing oxygen enriched air increases maternal oxygen saturation, a maternal rationale for supplementary oxygen has failed to become established. In the 1984 edition of his textbook, Crawford wrote 'it is advisable to provide the mother with supplemental oxygen, via a loosely fitting face-piece, until the time of delivery'. His opinion, widely disseminated through his writings, helped establish the practice of oxygen therapy at Caesarean section, but solely for fetal reasons.[4]

Increasing the inspired maternal oxygen fraction increases oxygen delivery to the fetus. The relationship between maternal and umbilical vein Pa_{O_2} during general anaesthesia for Caesarean section was shown by Marx and colleagues in 1971.[5] Their study demonstrated improvements in time to sustained respiration, and Apgar scores, that would not have been so impressive if they had not included hypoxic and normoxic women with those receiving increasingly hyperoxic mixtures.[5] Improvements in neonatal outcome were not maintained with increasing hyperoxia, but despite this a firm link was established between oxygen therapy and neonatal welfare before the obstetric regional anaesthesia revolution occurred. Ramanathan and colleagues[6] broadly repeated, in essence, the study of Marx and colleagues, but on women receiving epidural anaesthesia. They included a normoxic group, but no women were hypoxic. There was a correlation between maternal and umbilical arterial oxygen tensions, but Apgar scores and umbilical artery pH did not improve with increasing maternal hyperoxia. There was a small difference in base excess between the normoxic and hyperoxic groups that barely justified the authors' claims that hyperoxia improved neonatal acid–base status.[6] Uncritical readers of this study will find support for the fetal rationale of higher inspired maternal oxygen fractions, but the difference reported failed to achieve clinical significance. Following uncomplicated vaginal delivery, umbilical artery pH is below 7.3, and base deficit will be between –6 and –10 mmol litre^{-1}.[7] Any deterioration in acid–base status that is too small to impinge on this range cannot be considered important. Babies born to mothers who breathe air at elective Caesarean section, do not display an important increase in metabolic acidosis.[8]

When the neonatal impact of oxygenation is formally assessed, there is an assumption that potentially confounding factors do not have a significant role. Prolonged uterine incision to delivery time produces worrying metabolic acidosis,[9] but problem cases can be excluded. Aortocaval compression causes hypotension, which, if severe enough, will also produce worrying metabolic acidosis,[7 10] but careful vasopressor use will not produce acidosis of concern.[11] Umbilical artery pH is a reflection of both respiratory and metabolic acidosis, does not correlate with Apgar scores and is a poor indicator of outcome.[12] Apgar scores are not a specific marker for asphyxia, being reduced by drug effects which tend to be benign and reversible, and are of poor predictive value as far as outcome is concerned. In contrast, standard base excess reflects only the metabolic component of acidosis and correlates with neonatal outcome, values greater than –12 mmol litre^{-1} having an association with moderate to severe newborn encephalopathies.[13] As far as elective surgery is concerned, if cord biochemistry is to act as a surrogate marker for neonatal morbidity, then standard base excess is the variable to follow. However, it should be recognized that maternal hyperoxia is unlikely to achieve changes of the magnitude required to alter outcome during elective surgery.

One aspect of elective spinal anaesthesia that is often overlooked by anaesthetists is that there is a small but persistent incidence of neonatal acidosis associated with this technique. One hundred consecutive elective, normoxic, spinal anaesthetics for Caesarean section, in this maternity unit, yielded a mean (SD) base excess of –2.26 (3.3) mmol litre^{-1}. The mean value represents a highly satisfactory state of affairs, but the size of the standard deviation indicates some variability. In fact, 11 women had values consistent with the range for normal vaginal delivery, but one value was in excess of –12 mmol litre^{-1}. Whereas mean values are often reassuring, any potential benefit of maternal hyperoxia on the incidence of neonatal base excess beyond –6 or –10 mmol litre^{-1} does not appear to have been assessed, perhaps because a large study would be required. When the arrival of pulse oximetry allowed women with unsatisfactory respiratory performance to be identified, the routine provision of supplementary oxygen at uncomplicated deliveries ceased in this hospital. Others

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have taken the view that maternal oxygen does no harm, but might possibly do a little good, and have continued its use on that basis.

One early concern about raising oxygen tension in the umbilical vein relates to the ductus arteriosus, which constricts in response to high local oxygen tensions as part of the transition from the fetal to neonatal circulation. With the passage of time this is clearly a theoretical risk that is now largely ignored. In this issue of the Journal, a further concern is raised. Khaw and colleagues[14] have randomized women undergoing elective Caesarean section under spinal anaesthesia to breathe air or oxygen enriched air. The effects of breathing oxygen were confirmed by maternal and cord blood gas analysis, and markers of free radical activity were measured in cord blood at birth. The results show a clear difference between the groups, with greater free radical activity in the neonates born to mothers breathing oxygen enriched air.[14] An immediate caveat is that increased free radical activity may represent yet another surrogate marker for an unfavourable neonatal outcome. At present, we have no means of linking free radical formation with neonatal outcome following elective Caesarean section. The authors point out that in a low risk situation such as elective Caesarean section, a favourable outcome is unlikely to be influenced by maternal hyperoxia. The significance of this finding relates to the use of higher inspired maternal oxygen fractions for the delivery of compromised and premature infants. Indeed, it is possible that as far as hyperoxia is concerned, it might be inappropriate to group compromised term and premature babies together, as prematurity and potential asphyxia may be different risks. The authors note the involvement of hyperoxia in a host of neonatal disorders, from retinopathy to intracranial haemorrhage. One of the studies they list reports an improved outcome in neonates resuscitated with air, as opposed to oxygen, that is attributed to less free radical activity. This finding has changed practice, and this year a neonatal resuscitation manual has ceased to recommend the use of oxygen.[15]

Will this study change anaesthetic practice? One thing that will change is the ability to justify maternal oxygen therapy at elective Caesarean section on the basis that it might do some good, but will not do any harm. The latter position is much weakened by this study, and some will revert to air breathing for elective cases. Giving higher inspired oxygen fractions to mothers whose babies are distressed is a deep-seated reflex. Where gas exchange across the placental unit is threatened, the provision of a steeper gradient may improve exchange without necessarily achieving hyperoxia in the umbilical vein. As the anaesthetist can only speculate on what might be occurring, this is a theoretical point. Maternal oxygen might do some good, and might do a little harm, but which, we can only guess. Whereas some may hesitate, a widespread change in practice in the management of the fetus at risk, resulting from this study alone, is not expected.

S. K. Backe
Department of Anaesthesia
The Royal Hospitals
Belfast
UK
G. Lyons
St James' University Hospital
Leeds LS9 7TF
UK

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