One of the major problems in the practice of obstetric anaesthesia has been the conflicting requirements of the two patients—up to the time of delivery—when general anaesthesia is provided for operative delivery (currently almost invariably by Caesarean section). The mother wishes to be rendered and to remain unconscious; the infant to be spared the effects of depressant drugs derived transplacentally. However, an additional requirement is for both patients to be sustained in biochemical stability, which in this context may be related simplistically to acid-base and respiratory gas status.

During the 1940s and 1960s, most attention and effort were directed to minimizing “drug induced depression” of the neonate. One consequence was a high incidence of maternal awareness. It seems probable that the pendulum has now swung in the opposite direction. This movement was initiated probably by Moir’s demonstration [1] that maintenance of anaesthesia with an inspired mixture containing 0.5% halothane in 50% oxygen could ensure maternal unconsciousness (or at least, lack of recall after operation) without harm to the neonate. Indeed, there is what some might term a rather cavalier approach which suggests that the small mass of a volatile inhalation agent reaching the fetus during the interval between induction of anaesthesia and delivery is of little or no consequence to the ultimate well-being of the infant [2] and that the maternal stress response likely to be evoked by awareness is potentially much more harmful to the infant [3].

As a consequence, maternal awareness during Caesarean section should have been virtually eliminated from current practice. Discounting the very unusual case in which the mother requires vigorous intraoperative resuscitation (massive haemorrhage or severe bronchospasm, for example), or instances of technical mismanagement, there are classically three periods when awareness has occurred: during or shortly after tracheal intubation (which usually implies that the anaesthetist has administered an inadequate dose of induction agent [4]); during insertion of the final skin sutures (the anaesthetist has been in a hurry to get away from the theatre or to press on with the list); and at the time of delivery (insufficient volatile agent has been provided). The former two have always been unforgivable. In the light of current practice referred to above, the third situation should no longer occur.

So much for drug related effects, but what of biochemical problems? Much of the danger of asphyxia to which the abdominally delivered infant was exposed 2–3 decades ago (especially if the induction-delivery (I-D) interval was prolonged) has been averted by the avoidance of maternal aorto-caval compression, accomplished usually by the provision of a lateral tilt, although it is recognized that prolongation of the uterine incision to delivery (U-D) interval can, under the conditions of general anaesthesia, promote fetal asphyxia [5]. What is not yet clearly defined is the influence upon the neonate of the choice of oxygen concentration in the maternally-inspired mixture.

For many years standard teaching was based upon the advice of Rorke, Davey and Du Toit [6] and Marx and Matteo [7], although it is likely that, in practice, it has been honoured more in the breach than in the observance. Briefly, it was advocated that nitrous oxide should be administered in a concentration of 33% in oxygen, the objective being to maintain a maternal arterial $P_{O_2}$ of approximately 40 kPa. Values less than this were associated with diminished fetal oxygenation. A further increase in maternal hyperoxia produced feto-placental vasoconstriction and thus, again, reduction in fetal oxygenation. Baraka [8] agreed with the first part of this teaching, but reported that increasing the inspired concentration of oxygen from 50 to 100% was associated with no change in fetal oxygenation.

As the authors of the two articles in the present...
issue point out [9,10], current understanding suggests that these studies were seriously flawed. Relief from aorto-caval compression was not ensured by Rorke and colleagues [6] and Baraka [8]; little significance was attached to the duration of the I-D and U-D intervals in the control groups of subjects used for comparison, and the technique of anaesthesia was not standardized. Furthermore, no attention was paid to the effect of carbon dioxide. It has been suggested [11,12] that a result of maternal hypocapnia is a reduction of fetal \( P_{O_2} \) and an increase in fetal base deficit and lactic acid concentration. Duncan and colleagues [13] claim that the provision of a fresh gas flow equivalent to 100 ml kg\(^{-1}\) min\(^{-1}\) guards against maternal hypocapnia and hypercapnia.

The questions therefore remain, what is the optimum concentration of oxygen to be delivered to the mother, and is there a maximum beyond which no benefit derives to the fetus? Oxygen is transferred from maternal to fetal blood almost entirely in response to a pressure gradient. However, the system is rendered more complex by the presence of counter-currents which characterize the anatomy of the intervillous space, and also by the possibility that "hyperoxia" can indeed provoke vasoconstriction in the feto-placental circulation.

Possibly the most informative pointer to answer these questions is contained in a report by Ramanathan [14] of the consequences of providing different concentrations of oxygen to mothers undergoing elective Caesarean section under extradural analgesia. Fetal oxygenation was found to correlate significantly with maternal \( P_{A_{O_2}} \), possibly confirming reports, as discussed by Norris and Dewan [15], that materno-fetal oxygen equilibration is reached within less than 6 min. Ramanathan and colleagues also reported that "hyperoxic" fetuses were less acidic than were "normoxic" ones.

As with the majority of studies of placental transfer, the baseline investigation must be confined to cases of elective section which satisfy the criteria of the "clinically acceptable ideal case" [16]. Studies of groups of patients with and without pathology possibly indicative of placental dysfunction, and of elective and emergency procedures, render untenable any conclusions of fetal well-being drawn from the results. The mother should not have been exposed to aorto-caval compression, either during her journey to the anaesthetic room or on the operating table. The anaesthetic technique should be standardized, the only variable being the ratio of flow rates of oxygen and nitrous oxide (it would be as well to study the three advocated concentrations of oxygen— 50, 67 and 100\%). Maternal normocapnia should be maintained. In each case, the U-D interval should be within an acceptable range (up to 90 s) for this type of investigation. Traditionally—and I believe correctly—analysis of umbilical artery rather than umbilical vein blood has been quoted as indicative of the acid-base and respiratory gas status of the neonate. The Apgar-minus-colour scores at 1 and 5 min and the time to sustained respiration of the infant should be assessed (and recorded in detail) by a paediatrician known to be competent and reliable in the exercise.

A definitive investigation such as this has yet to be reported. Once the results of such a study are known and validated, consideration may then be given to the situation of the potentially less than "ideal" infant, in regard to whom it must be remembered that, owing to the characteristics of the fetal oxyhaemoglobin dissociation curve, a small increase in fetal \( P_{A_{O_2}} \) causes a significant increase in oxygen saturation, thus improving fetal oxygen stores preparatory to the interval between the process of delivery and the initiation of active resuscitation.

However, extrapolation of the results of a study as outlined, to the conduct of cases characterized by fetal distress, will pose many more problems. The abolition merely of uterine contractions by the general anaesthetic probably contributes to the unexpectedly healthy state of the infant frequently seen in such circumstances (an aspect of "fetal resuscitation"). If the intra-uterine distress results from massive abruption or from a severely constricted umbilical cord, with consequent gross disturbance of fetal haemodynamics, varying the concentration of maternally-inspired oxygen concentration is unlikely to make a discernible difference to the umbilical artery acid-base and respiratory gas values. Thus a very large series of subjects, grouped with meticulous care, would be required to investigate this question.

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REFERENCES


