

## **SUPPLEMENTARY OXYGEN ADMINISTRATION DURING REGIONAL ANAESTHESIA FOR LSCS – IS IT JUSTIFIED?**

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Maternal oxygen supplementation during regional anaesthesia for caesarean section has been routinely practiced for many years. It was Crawford who advocated the “use of maternal oxygen therapy until the time of delivery” in the 1984 edition of his book on obstetric anaesthesia. This opinion of Crawford established the practice of supplementing oxygen in all elective caesarean sections (1), the basic aim being to prevent maternal haemoglobin desaturation and improve maternal and neonatal oxygenation and overall neonatal outcome.

Satisfactory regional block for caesarean section requires a block of A $\beta$  fibres up to the level of T5 dermatome leading to reductions in maternal peak expiratory flow rates, forced vital capacity and forced expiratory volume. In the normal healthy parturient these changes in ventilatory parameters accompanying spinal anaesthesia are well tolerated (2).

However Mueller et al 1997 (3), found the incidence of foetal acidaemia to be higher during regional anaesthesia as compared to general anaesthesia. The cause of this foetal acidaemia was attributed to the hypotension which occurred during spinal anaesthesia, leading to decreased uteroplacental perfusion and decreased intervillous blood flow.

The routine provision of supplementary oxygen to uncomplicated pregnancies subjected to LSCS under regional anaesthesia has recently been dogged by controversy. Previous studies have shown that maternal administration of oxygen does improve foetal oxygenation and acid base balance along with biophysical profile during foetal hypoxia (4,5). However, other studies have demonstrated that administration of oxygen does not have any beneficial effects on the foetal oxygenation or acid-base status. Ramanathan and colleagues in 1982 (6) have reported that administration of 35% FIO<sub>2</sub> did not cause any change in the fetal umbilical vein pO<sub>2</sub> (UVpO<sub>2</sub>). This UVpO<sub>2</sub> was improved when maternal FIO<sub>2</sub> was increased to 47%-100%, but not to the same proportion as increase in maternal pO<sub>2</sub>. There was a correlation between maternal umbilical arterial O<sub>2</sub> tensions but APGAR scores and umbilical artery pH did not improve with increasing maternal hyperoxia. Lawes et al, 1988 (7) also found no difference in foetal outcome or acid-base status when maternal FIO<sub>2</sub> was decreased from 0.5 to 0.33, in 35 patients undergoing LSCS under general anaesthesia. Perreault et al, 1992 (8) designed a study to determine if whether foetal arterial and venous PO<sub>2</sub> could be increased by increasing maternal FIO<sub>2</sub> in the period between hysterotomy and birth. They concluded that maternal hyperoxia does not result in any increase in foetal PO<sub>2</sub>. Similarly Cogliano et al, 2002 (9) studied the effects of supplementary oxygen to mothers undergoing elective LSCS under spinal anaesthesia. They found no significant differences in

the umbilical arterial or venous pH, partial pressures of O<sub>2</sub> and CO<sub>2</sub> in the event of a prolonged uterine incision to delivery (U-D) interval during LSCS. Recently, Khaw et al, 2004 (10) studied the effects of maternal hyperoxia in parturients with a uterine incision – delivery interval of more than 180 secs. They found no differences in UV or UA blood gases, oxygen content or APGAR scores between cases with and without prolonged U-D intervals. They concluded that their data did not support the routine administration of supplementary oxygen during elective LSCS under spinal anaesthesia.

Thus there appears to be no significant increase in the maternal – foetal O<sub>2</sub> transfer rate when O<sub>2</sub> tension is raised on the maternal side, since with the increase in O<sub>2</sub> tension of the perfusing blood, there is probably a concomitant vasoconstriction which negates any positive effects that might be expected as a result of increasing the maternal-foetal O<sub>2</sub> gradient. Also the combined O<sub>2</sub> consumption of the whole placenta and foetal membranes is a considerable fraction of the O<sub>2</sub> consumption of the total uterine contents. It was shown that there is a difference in dynamics of O<sub>2</sub> transfer by the lung as compared to that of placenta where a linear relationship between maternal and foetal blood exists only when maternal arterial pO<sub>2</sub> is <100mmHg. The placenta thus limits O<sub>2</sub> transfer regulating foetal O<sub>2</sub> tension to ensure patency of the ductus arteriosus.

A further concern regarding maternal O<sub>2</sub> supplementation is related to the increased oxygen free radical (OFR) activity in both mother and baby. Khaw and colleagues, 2002 (11) investigated the effect of high FiO<sub>2</sub> on maternal and foetal oxygenation and oxygen free radical activity in parturients having LSCS under spinal anaesthesia. They randomized 44 healthy parturients to breathe either 21% (air group) or 60% oxygen (oxygen group) intraoperatively via a ventimask. They found the oxygen group had greater maternal arterial pO<sub>2</sub> (P<0.001) and greater umbilical venous PO<sub>2</sub> (P=0.04) compared with the air group. Maternal and umbilical plasma concentrations of lipid peroxides (8-isoprostane, MDA, OHP) were greater in the oxygen group than the air group (P<0.05). Thus, they concluded that breathing high FiO<sub>2</sub> modestly increased foetal oxygenation but caused a concomitant increase in OFR activity in both mother and foetus. Stipek et al, 1995 (12) tested the lipid peroxidation caused by reactive oxygen species which are produced as a consequence of tissue reoxygenation and the inactivation of these species by the maternal and newborn superoxide dismutase. They concluded that maternal and foetal antioxidant defence systems can be overloaded during deliveries with abnormal oxygenation, where increased lipid peroxidation occurred. Recently, the effects of maternal hyperoxia on foetal oxygenation and lipid peroxidation following foetal asphyxia in late gestation goats has been investigated by Yamada et al, 2003 (13). They found that maternal hyperoxia increased foetal oxygenation but caused a concomitant increase in lipid peroxidation in the foetus. Foetal plasma MDA levels were found to significantly increase after initiation of maternal oxygen supplementation and further increased following foetal asphyxia and after cord occlusion (p<0.005). These values were significantly higher than those in fetuses without oxygenation.

Thus changes in lipid peroxidation and antioxidant status during uncomplicated vaginal delivery have been observed which may affect the foetus by creating oxidative stress (14,15). Even various obstetric complications per se can result in oxidative stress to the mother and foetus like prolonged labour, foetal distress, oligohydramnios and tight nuchal cord entanglement (14,16,17,18). In these conditions, free radicals can be generated by two different pathways. It can be generated via the pathways involving hypoxic stress and ischaemia – reperfusion injury or by direct electron transfer. During ischaemia, vascular endothelial xanthine dehydrogenase (XD) is converted to xanthine oxidase (XO) which in the presence of molecular oxygen catalyses the formation of hydroxyl radicals from the breakdown of purine metabolites, xanthine and hypoxanthine (19). Thus enhanced tissue damage by oxidants occurs when reperfusion takes place after temporary vascular occlusion. Hyperoxia per se also generates OFR via an alternative pathway involving direct electron transfer, with no concurrent formation of purine metabolites. Hyperoxia as a mediator of tissue injury has also been implicated in bronchopulmonary dysplasia, retinopathy of prematurity, persistent ductus arteriosus, necrotizing enterocolitis and intracranial haemorrhage (20-22). It has been further observed that neonates resuscitated with oxygen resulted in poorer neonatal outcome because of generation of OFR as compared to resuscitation with air (11). This has led to a change in practice and one of the neonatal resuscitation manuals has ceased to recommend the use of oxygen (23).

## CONCLUSION

The clinical practice of administering a high  $\text{FiO}_2$  to all the parturients undergoing elective LSCS under regional anaesthesia is questionable provided that continuous monitoring with pulse oximetry is available. It has been established, beyond doubt that maternal oxygen supplementation in 'without risk' mothers undergoing elective LSCS under regional anaesthesia does not improve neonatal outcome. In, my opinion, it is more important for the anaesthetist to avoid haemodynamic alterations associated with regional anaesthesia, rather than resort to increasing the mother's discomfort and anxiety with an oxygen face mask.

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