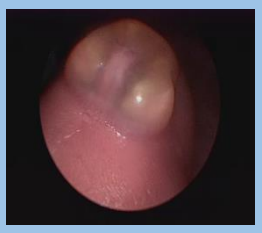

Fetal respiratory acidosis can be compensated by maternal hyperventilation in fetoscopy using carbon dioxide insufflation

Ana Paola ABN Souza, Gregório L Acácio, Renato AM Sá, Denise AL Pedreira
Fetal Therapy Center - Hospital Israelita Albert Einstein and on behalf of Brazilian Fetal Network

Introduction

The increasing complexity of surgical techniques needed to operate the fetus led to the need to use amniodistension with CO2.
In the ovine model, CO2 amniodistension causes fetal acidosis. But, the respiratory nature of this acidosis can apparently be corrected and it appears to be transient. The occurrence of brain damage consequent to this reversible acidosis is largely unknown. If the same acidosis occurs in humans and if it causes any harm, is yet to be proven. To measure acidosis in the human fetus is not harmless and even if we prove it actually occurs, it may not lead to neurodevelopmental abnormalities.

Objectives

To evaluate the occurrence of fetal acidosis during CO2 insufflation into the uterine cavity, in sheep.

Methods

The key words or expression: "carbon dioxide" , "fetal surgery", "acidosis", "experimental", "fetoscopy", "amniodistension" and "sheep" were searched in MEDLINE 1950-2015. We have searched for experimental studies of CO2 amniodistention in sheep, from which baseline and post-insufflation pH were available. All the statistics were done in Review Manager 5.3 software from Cochrane.

Results

We have found a total of 5 studies. The analysis showed that fetal respiratory acidosis occurred in all cases beginning at an average of 20,6 minutes after starting the insufflation and ending after 25,6 minutes of the CO2 suspension. Maternal hyperventilation led to respiratory alkalosis that was able to interfere significantly improving fetal acidosis.

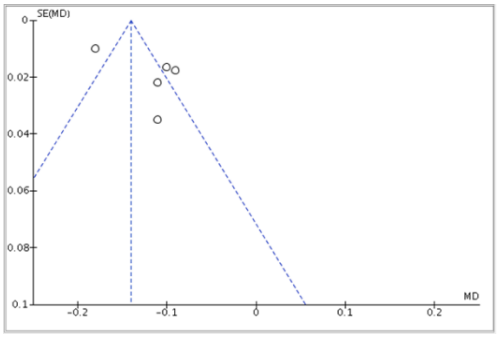

Figure 1. Funnel plot of comparison: CO2 insufflation in sheep under fetoscopy: comparing fetal surgery x no fetal surgery.

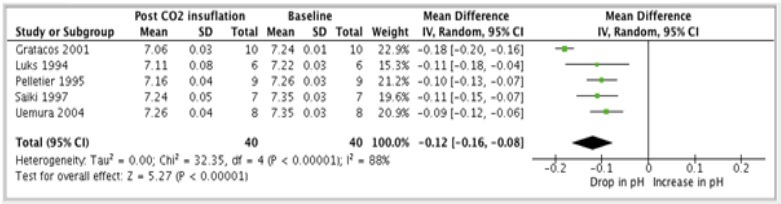

Figure 2. Forest Plot of comparison: CO2 insufflation in sheep under fetoscopy: comparing fetal pH at the beginning x at the end of procedure.

Conclusion

Carbon dioxide insufflation into uterine cavity leads to fetal reversible respiratory acidosis, which can be ameliorated by maternal respiratory alkalosis, in sheep. The probable mechanism for this improvement may be related to the bicarbonate buffering on placental level. If this type of acidosis, that is essentially respiratory, has the same impact on posnatal neuropsicomotor development as the metabolic acidosis consequent to chronic hypoxia (related to placental insufficiency) is yet to be proven. However, fetal respiratory acidosis caused by CO2 insufflation is likely to be controlled and reversible in humans, by adjusting maternal parameters during anesthesia, without leading to long term neurological impairment.

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