Current Obstetrics and Gynecology Reports (2023) 12:173–177
https://doi.org/10.1007/s13669-023-00363-w

REVIEW

Intrapartum Oxygen for Fetal Resuscitation: State of the Science

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Accepted: 27 April 2023 / Published online: 18 May 2023

Abstract

Purpose of Review This review aims to summarize the current evidence regarding maternal oxygen supplementation for Category II fetal heart tracings (FHT) in labor. We aim to evaluate the theoretical rationale for oxygen administration, the clinical efficacy of supplemental oxygen, and the potential risks.

Recent Findings Maternal oxygen supplementation is an intrauterine resuscitation technique rooted in the theoretic rationale that hyperoxygenating the mother results in increased oxygen transfer to the fetus. However, recent data suggest otherwise. Randomized controlled trials on the efficacy of oxygen supplementation in labor suggest no improvement in umbilical cord gases or other adverse maternal and neonatal outcomes compared to room air. Two meta-analyses demonstrated that oxygen supplementation is not associated with an improvement in umbilical artery pH or reduction in cesarean delivery. Although we lack data on definitive clinical neonatal outcomes with this practice, there is some suggestion of adverse neonatal outcomes with excess in utero oxygen exposure, including lower umbilical artery pH.

Summary Despite historic data suggesting the benefit of maternal oxygen supplementation in increasing fetal oxygenation, recent randomized trials and meta-analyses have demonstrated a lack of efficacy of this practice and some suggestion of harm. This has led to conflicting national guidelines. Further research is needed on short- and long-term neonatal clinical outcomes following prolonged intrauterine oxygen exposure.

Keywords Oxygen · Intrapartum resuscitation · Non-reassuring fetal status

Introduction

Prior to 2022, the American College of Obstetricians and Gynecologists (ACOG) recommended maternal supplemental oxygen administration as part of the management of abnormal fetal heart tracings (FHT) in labor with the belief that this would resolve fetal hypoxia, thereby preventing fetal acidemia. Most protocols for this practice involve administering a laboring patient 5–10 l/min of supplemental oxygen via a non-rebreather mask in response to Category II FHT. This is often combined with other resuscitative maneuvers, including maternal repositioning, intravenous fluids, amnioinfusion, and management of contractions, either by stopping Pitocin or giving Terbutaline. Oxygen as an intrauterine resuscitation technique became common practice following animal and human observational studies performed over 50 years ago that showed an increase in fetal partial pressure of oxygen (PO_2) via scalp sampling and resolution of late decelerations with oxygen supplementation in laboring patients [1•, 2•]. In 2014, it was estimated that 2 out of 3 laboring patients received supplemental oxygen in response to FHT [3•].

Contemporary evidence within the last decade has challenged the dogma of oxygen supplementation for non-reassuring FHT in labor. In order to comprehensively evaluate the use of supplemental oxygen as an intrauterine resuscitation technique, we must subject it to the same scrutiny as any other drug in pregnancy with careful evaluation of the three tenants of a therapeutic intervention — absorption, efficacy, and safety.

Absorption

Maternal oxygen supplementation is performed with the intent of increasing oxygen transfer to a hypoxic fetus. Fetal hypoxia leads to anaerobic metabolism and

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subsequent metabolic acidosis. Fetal heart rate monitoring aims to predict acidemia with the ultimate goal of preventing adverse neurologic outcomes and fetal death, so it is reasonable to believe that supplemental oxygen results in more oxygen transfer to the fetus, thereby resolving hypoxia and preventing acidemia.

In 1971, a study of fetal scalp blood sampling after maternal hyperoxygenation demonstrated an increase in fetal PO_2 [1•]. A 2006 study of fetal pulse oximetry in labor demonstrated that for fetuses with non-reassuring FHT, maternal oxygen administration with 40 or 100% inspired FiO_2 increased fetal oxygen saturation by 4.9% (40% FiO_2 group) to 6.5% (100% FiO_2 group) when monitored with an N400 fetal pulse oximeter after rupture of membranes [4•].

The question of absorption, however, becomes more complex with consideration of hemoglobin and oxygen dissociation curves. While oxygen saturation refers to hemoglobin-bound oxygen, PO_2 refers to oxygen molecules dissolved in plasma. Fetal hemoglobin has a higher affinity for oxygen than does adult hemoglobin, and this difference in dissociation curves contributes to the saturation of fetal hemoglobin even in the setting of moderate maternal hypoxia. Moreover, in the setting of normal oxygen saturation, the maternal PO_2 is at a plateau where any additional oxygen supplementation is unlikely to affect how much oxygen is either dissolved or bound to hemoglobin and how much is being unloaded at the level of the uterus and placenta. Data on whether oxygen supplementation increases fetal PO_2 is mixed. A recent meta-analysis demonstrated no increase in umbilical artery PO_2 in three randomized trials of oxygen delivery versus room air in labor [5•]. A second meta-analysis of 16 randomized trials in laboring and non-laboring patients demonstrated that oxygen supplementation was associated with an increase in umbilical artery PO2; however, after stratifying by the presence or absence of labor, this increase in PO2 was only seen with oxygen given at the time of scheduled cesarean delivery and not during labor [6••] A recently published secondary analysis of a randomized trial of oxygen among laboring patients also demonstrated no difference in umbilical artery PO2 between groups [7•]. Overall, these data indicate that maternal oxygen supplementation in labor does not increase UA PO_2. Perhaps most importantly, longer duration of oxygen exposure was recently linked with a *lower* PO2 compared to those exposed to oxygen for shorter periods of time. In a secondary analysis of the Raghuraman RCT, patients were compared between those who had a length of oxygen exposure < 75th percentile and ≥ 75th percentile. In those with prolonged oxygen exposure, umbilical vein partial pressure of oxygen was significantly lower. These data suggest that prolonged hyperoxia may actually prevent transplacental oxygen transfer [8•].

Efficacy

While the existing data suggest that maternal oxygen supplementation is unlikely to increases fetal PO2 in laboring patients, it is important to recognize that umbilical artery PO2 is a poor marker of neonatal morbidity [9•]. Most importantly, the burden for the value of oxygen supplementation as a treatment lies in the question of whether it prevents neonatal acidemia or operative deliveries for non-reassuring fetal status.

Effect on Electronic Fetal Monitoring

In a study of 21 pregnant women performed in 1967 by Althabe et al., oxygen administration was associated with resolution of late decelerations [2•]. A 2021 secondary analysis of data from a randomized trial investigating intrapartum fetal electrocardiographic ST-segment analysis demonstrated that two-thirds of Category II FHT improved with oxygen supplementation within 60 min, with the strongest improvement in tracings with absent accelerations or absent variability. However, this analysis did not have a room air comparator, and thus, we do not know if the FHT improvement was spontaneous or due to oxygen given that this was an observational study [10•].

In the 71 patients analyzed in a randomized trial by Moors et al., there was an improvement in FHTs in the oxygen group with lower rates of "deterioration" as compared to the placebo group [11••]. In a separate secondary analysis of a randomized trial, oxygen treatment did not increase the rate of resolution of recurrent late and/or variable decelerations or high-risk Category II features [12•].

Effect on Mode of Delivery

To date, five RCTs have been completed that examine the administration of oxygen during labor [11••, 13•, 14•, 15•, 16••]. In the first three studies, oxygen was administered in a "prophylactic" fashion to non-selected patients (i.e., no requirement for non-reassuring FHT) in the second stage [13•, 14•, 15•]. They were compared to a control group in which patients did not receive oxygen. There have been two additional randomized controlled trials that examined the effect of oxygen for non-reassuring fetal heart tracings in labor [11••, 16••]. When the data from these trials were pooled in a meta-analysis, on the whole, there was no difference in the rate of cesarean delivery, operative vaginal delivery, or spontaneous vaginal delivery when oxygen was applied. This lack of difference held when oxygen was given prophylactically or for non-reassuring fetal heart tracings [5•].

Effect on Cord Gases and Acidemia

In a meta-analysis of RCTs on peripartum oxygen supplementation, [11••, 13•, 14•, 16••, 17•], there was no

difference in umbilical artery (UA) pH, UA pH < 7.2, or UA base excess [6••]. These data were not analyzed by the presence or absence of non-reassuring fetal heart tracing.

Effect on Surrogate Neonatal Outcomes

Secondary and surrogate neonatal outcomes have been evaluated in five randomized trials of supplemental oxygen [11••, 13•, 14•, 15•, 16••]. Oxygen was associated with similar 1-min Apgar scores < 7, 5-min Apgar scores < 7, and NICU admissions as room air comparators. These outcomes remained similar regardless of whether oxygen was given prophylactically or in response to non-reassuring FHT [5•, 6••].

Real-World Implementation Studies

Subsequent to the above RCTs and meta-analyses comparing oxygen to room air, there have been two "real-world" implementation studies looking at the clinical effects of de-implementing oxygen supplementation for NRFHT. In one study, 22.6% of patients in the pre-deimplementation phase were treated with oxygen as opposed to 0.6% in the post phase, indicating excellent acceptance of the initiative. In a cohort of 1334 patients, no differences were noted in the rates of cesarean or operative vaginal deliveries, Apgar scores < 7, NICU admissions, or neonatal deaths after de-implementing oxygen administration [17•]. In the second study, 4932 patients treated after de-implementation of oxygen for Cat II or III FHTs were compared with 4906 patients from the 6 months prior to the policy change. Oxygen suspension was associated with an increase in composite adverse neonatal outcomes (3.8% vs. 2.4%), and independently higher rates of arterial pH < 7.1. There was also a higher rate of cesarean for non-reassuring FHT after de-implementation [18•]. Both of these implementation studies gathered data in 2020 at the beginning of the COVID-19 pandemic, potentially biasing results given the major shifts in healthcare during that time. Further implementation work is needed to observe the acceptability of oxygen de-implementation and to assess real-world outcomes.

Safety

Medical interventions in pregnancy are always weighed as potential for benefit over potential for risk. There are some who argue that there may be value to oxygen administration as it is "low risk" even if it is ineffective. However, excess oxygenation is associated with harm as it can produce toxic free radicals. In contemporary medicine, oxygen free radicals are thought to be a major contributor of hypoxia-reperfusion injuries that occur when adequate blood flow and oxygenation are restored [19•].

In adult populations, multiple randomized trials have demonstrated harm secondary to the administration of oxygen. Hyperoxia in sepsis has been linked with increase rates of death [20•] and conservative as opposed to conventional oxygen therapy is associated with lower mortality rates in the ICU [21•]. A randomized trial of oxygen therapy in myocardial infarctions similarly demonstrated that oxygen supplementation without hypoxia led to higher rates of recurrent myocardial infarctions, cardiac arrythmias, and increased infarct size, reinforcing the concept of the hypoxia-reperfusion injury with excess oxygenation. [22•].

Free radical-induced oxidative cell damage is also an underlying contributor to neonatal adverse outcomes including necrotizing enterocolitis, intraventricular hemorrhage, and bronchopulmonary dysplasia [23•]. To this end, multiple systematic reviews have demonstrated that neonatal resuscitation with room air, as opposed to 100% oxygen, is associated with a decrease in neonatal harms, including hypoxic ischemic encephalopathy and death [24•, 25•, 26•].

In pregnancy specifically, the creation of oxygen free radicals with hyperoxygenation during umbilical artery compression has been demonstrated in ewe models [27•]. However, to date, there have been no demonstrated increases in free radicals in randomized trials of oxygen administration in labor. Specifically, markers of oxidative stress (methemoglobin, malondialdehyde (MDA), or superoxide dismutase and glutathione) were studied in three studies with no difference between markers [5•]. Notably, however, supplemental oxygen for women with normal oxygen saturations at the time of cesarean delivery is associated with an increase in free-radical production without any demonstrated benefit to the neonate [28•].

There is some concern for harm with hyperoxygenation in labor. In a secondary analysis of 7789 patients with cord gas values, those with intrauterine hyperoxemia (defined as an UA $PO_2 \geq 90^{th}$%ile) were compared to those without hyperoxemia. While there were similar rates of acidemia between groups, for those with acidemia and hyperoxemia, there was an increase in neonatal morbidity [9•]. A recent study on the effect of maternal hyperoxygenation on maternal circulation additionally raises concerns about this intervention. In non-laboring patients in the third trimester, hyperoxygenation was associated with a decrease in stroke volume and a rise in systemic vascular resistance (SVR). The increase in SVR, notably, did not recover within 10 min of stopping oxygen therapy. These findings raise concern that hyperoxygenation may be associated with decreased placental perfusion, rather than increased passage of oxygen [29•]. This could be adding insult to injury by attempting to hyperoxygenate a fetus that is hypoxic.

Conclusion

The above data reviewed the absorption, efficacy, and safety of maternal oxygen supplementation and call into question the validity of this practice. We reviewed that supplemental oxygen does not appear to be absorbed by the fetus, as umbilical artery PO2 does not increase with oxygen supplementation in laboring patients. Oxygen does not appear to have efficacy in preventing operative deliveries, improving umbilical cord gas values, or ameliorating surrogate neonatal outcomes in RCTs performed thus far. There are conflicting data pertaining to oxygen's effect on FHTs. Finally, while there is theoretical concern that oxygen may produce excess free radicals, this has not been demonstrated in RCTs in labor. There are data that with oxygen supplementation there are changes in maternal circulation that could negatively impact placental perfusion.

Based in part on these data, in January 2022, ACOG joined the UK (NICE guidelines) in recommending against oxygen supplementation in labor among normally oxygenated women [30•, 31••]. However, a subsequent statement released by the Association of Women's Health, Obstetric, and Neonatal Nurses (AWOHNN) in March 2022 advocates against abandoning oxygen use, stating that the studies were heterogeneous and there is some evidence of improvement in FHT [32••]. They call for a large, randomized trial to assess meaningful clinical maternal and neonatal outcomes, rather than surrogate outcomes such as pH. Thus, although the available data suggest we move toward de-implementation of this practice, conflicting national guidelines demonstrate continued equipoise with a need for more definitive data best obtained in large RCTs.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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