Oxytocin and obesity: 2015-2017 literature review on oxytocin use in obese women

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Abstract
As obesity becomes more prevalent, research regarding oxytocin use is increasing as well. A literature review from 2015-2017 assessing oxytocin use in obesity highlighted this evolving field. Body Mass Index (BMI) alone appears to contribute to blunted myometrial and therefore contractile responses seen in obese women. The interplay of elevated progesterone and leptin contribute to this phenomenon, and maybe explain the elevated oxytocin dosing seen amongst this population. None the less, the effects of obesity on the mode of delivery is still controversial, with some investigators claiming the interval from induction to delivery, delivery within 24 hours, vaginal delivery within 24 hours, and the cesarean delivery rate did not vary between stratified classes of obesity. Conversely, the authors concluded that as BMI increases the women undergoing induction with misoprostol have a longer time to delivery, require greater quantities of misoprostol, longer duration of oxytocin, and increased cesarean section rates. Amongst the literature, there is a consistent message that obese women are somehow different. Investigating the information retrospectively highlights areas, which necessitate prospective trials and assessment.

Abbreviations and symbols: BMI: Body Mass Index, CI: Confidence Interval, PROM: Premature Rupture of Membranes

Introduction
As obesity becomes more prevalent, research regarding oxytocin use in obesity is increasing. In the most recent National Center for Health Statistics (NCHS) the prevalence of obesity amongst women aged 20-39 was 36.5% and from 1999–2000 through 2015–2016, a significantly increasing trend in obesity was observed in both adults and youth [1]. Complications of maternal obesity include maternal complications such as gestational diabetes mellitus, hypertensive disorders in pregnancy, and long-term cardiovascular implications, as well fetal complications including stillbirth, macrosomia, childhood obesity, and others [2-5]. Due to these complications, obese women are at risk of labor induction and subsequent failure [6]. It appears obesity impacts progression of labor with longer median duration when compared to normal weight women [7]. This phenomenon is multifactorial though oxytocin pharmacokinetics and metabolism differ. Reviewing the literature regarding this specific patient population allows reflection on current advances and directs future research in hopes of optimizing oxytocin administration and clinical care.

Materials and methods
A literature review of studies between the years 2015 to 2017 was conducted. Keywords words such as oxytocin, obesity, induction of labor, and augmentation of labor were used in PubMed. The analysis involved eight articles, with a selected subset of six pertaining to oxytocin and obesity. Of these six articles, there was five retrospective cohort studies and a single randomized control trial. The following paper reviews the most recent literature regarding oxytocin use in obese women.

Results
Oxytocin response is unpredictable and further studies are needed to investigate the characteristics that determine the sensitivity or blunting of response, which is thought to occur in obesity. Carlson NS et al. specifically examined 136 spontaneous laboring obese women (BMI ≥ 30 kg/m²) undergoing augmentation with regards to hourly doses of oxytocin. After controlling for maternal, fetal, and labor characteristics, BMI alone explained 16.56% (95% Confidence Interval (CI)13.7-20.04) of the variance in hourly oxytocin doses. The authors concluded, obesity contributes to blunting of myometrial contractility and therefore a response to oxytocin [8]. This effect was seen in Hill M et al. study as well. In a retrospective review, amongst a cohort of 54-term women undergoing induction of labor with subsequent vaginal deliveries, obese women (BMI > 40 kg/ m²) required more oxytocin than lean women during the first stage of labor when matched for parity, diabetes, epidural use, birth weight, pre-induction dilatation, and station [9]. In attempts of explaining this phenomenon, Hajagos-Toth et al. reviewed the influences of increased body weight on multiple endocrine and paracrine systems. For example, increased body weight leads to elevations in progesterone, which contribute to poor contractility. Obesity also increases leptin, which inhibits both spontaneous and augmented or induced uterine contractions in animal models as well as isolated human myometrium [10]. These articles highlight the complexity of obesity on oxytocin administration, efficacy, and management.

Whether obesity impacts mode of delivery for women undergoing induction of labor is still controversial. In a randomized control trial conducted by Mackeen et al., 201 women underwent randomization between induction with concurrent Foley catheter and oxytocin versus oxytocin alone. The women were stratified based on BMI into the

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The articles' strongest attribute is the consistent message that obese women are somehow different. Investigating the information retrospectively highlights areas, which necessitate prospective trials and assessment. Ideally collecting a large patient population across geographical locations with consistent BMI groups enables greater generalizability. Starting with basic science research, the pathogenesis of obesity derived myometrial blunting to clinical outcomes, oxytocin use in obese women is an evolving area of research.

**References**