

Amniotic Liquid Lactate (AFL): An Indicator of Work Result in Dystocic Labor

Abstract

The goal of this review is: Even today, childbirth is responsible for the deaths or severe complications of hundreds of thousands of women. Stopped labor progress, also known as dystocia of labor, is one of the most common causes. Since Friedman developed the Partogram in the 1950s, there have been no advances in the diagnosis or treatment of dystocic deliveries. Dystocic labor can only be treated with oxytocin. Oxytocin can sometimes save a woman's life, especially in severe postpartum hemorrhages. In addition, it is one of the drugs used the most frequently in obstetric care. The purpose of this review article is to provide a brief summary of the current understanding of labor metabolism, uterine lactate production, and their connection to labor dystocia. The article also aims to provide a glimpse into the future of dystocic labor management and to reflect innovative approaches to practical recommendations for treating labor dystocia.

Keywords: Childbirth • Dystonic delivery • Oxytocin • Hemorrhage • Labor • Instrumental delivery

Introduction

Hundreds of thousands of women still die during childbirth or suffer severe complications. Over 295,000 women died during childbirth in 2017. Even though there have been recent advancements, labor dystocia continues to be one of the greatest threats to women and their unborn children during labor. The uterus experiences regular contractions, the cervix becomes dilated, and the fetus passes through the birth canal during normal labor. The clinical term for labor dystocia is "slow or arrested progress of labor." Since Friedman introduced the partogram in the 1950s there have been no significant advancements in the diagnosis of labor dystocia. The reason why labor progress stops is still unknown. The partogram is a time-series graph of cervical dilatation. A permanent partogram delivery is recommended by the WHO [1]. The partogram's primary objective is to identify individuals at risk for prolonged dystocic labor. The current understanding of the uterine metabolism, uterine lactate production, and their connection to labor dystocia will be discussed in this review.

Amniotic fluid lactate during labor

The utero placental blood flow decreases

during normal labor contractions but quickly returns after the contraction is over. At the point when work withdrawals are irregular and insufficient, the utero placental blood flow is answered to be decreased for a more expanded period continually. As a result, in dystocic labor, the fetal-maternal unit's gas exchange may be disrupted, leading to lactic acidosis and hypoxia-ischemia in the fetus. The objective of all obstetric consideration is to convey a healthy woman and a sound infant. Even though the fetus is monitored in a manner that we believe to be optimal, newborns still lack oxygen. This could be because the staff is overwhelmed with information or because the monitoring methods aren't specific enough. For example, the Ctg brings about countless false positives. Out of those with an impacted Ctg follow, only a little extent are hypoxic upon entering the world. The recommendation is to use an additional method of fetal monitoring if there is any doubt regarding a Ctg interpretation. Fetal blood inspecting is an intrusive and non-continuous method [2, 3].

It has been reported that up to 20% of sampling attempts fail. Since the 1970s, it has been known that amniotic fluid contains a high concentration of lactate, but this has never been used for analyzing fetal wellbeing during labor.

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Additionally, the measurement of pH and lactate in fetal scalp blood has an unsatisfactory and low predictive value for adverse fetal outcomes at birth. 850 primiparous women in labor were included in a 2011 study at Soder Hospital in Stockholm. Within 30 minutes of the delivery, the amniotic fluid was sampled, and the AFL value was analyzed blindly. All conveyances had a Ctg recording that was reviewed after conveyance, indiscriminately and concurring to FIGO rules. Neonatal adverse outcomes (pH 7.05 and BD>12 in the umbilical artery, Apgar score 7 at 5 min, meconium aspiration, hypoxic-ischemic encephalopathy grade 1–3) were significantly higher in the group with an AFL>10.1 mmol/L in the final sample taken before delivery. More newborns were admitted to the NICU for longer than 24 hours and more were given resuscitations [4].

Two newborns were born with grade 2 HIE; both were in the group with a high AFL level (>10.1 mmol/L). In the last sample taken before delivery, almost 80 percent of deliveries with a negative neonatal outcome and a pathologic Ctg in the last 30 minutes also had a high AFL level. When combined with a Ctg recording, high AFL levels were found to have a greater predictive value for adverse neonatal outcomes. These results suggest that high AFL levels are a useful indicator of uterine hypoxia. The AFL method is simple, noninvasive, and safe for the mother and her unborn child. Taken together with a high AFL level before delivery, functional bradycardia indicated an almost fourfold risk of adverse neonatal outcomes. Children are stillborn with unexpected adverse neonatal outcomes despite what is considered careful fetal surveillance, so these findings have important clinical implications [5].

Effect on uterus

All human cells undergo glycolysis to produce lactic acid when exerting them. Glycolysis is most common in hypoxia, but the uterus is unique in that it is glycolytic even in normal conditions. However, when oxygen is available, lactate production decreases. The uterus must produce powerful, coordinated, and effective contractions for labor to be successful. Every time there are regular labor contractions, the uterine blood flow is reduced and the metabolism briefly goes into anaerobic mode. At the point when the compression subsides, lactate and different metabolites related with hypoxia are removed, and new, oxygenated blood is

shipped to the muscle. Good labor progress and an average level of lactate in the tissue are the results of this process. In contrast, in abnormal, dystocic deliveries, lactate removal appears to deteriorate, and lactate and other metabolites accumulate in the tissue instead of being removed. Recent research has demonstrated that the uterus requires these periods of hypoxia to initiate the subsequent pregnancy. The uterine muscle becomes acidified as a consequence of this. There is still no explanation for this. We know of a few gamble factors related with work dystocia, however not why the course of labor stops. Acidification reduces the amount of Ca²⁺ that reaches muscle cells by blocking the Ca²⁺ channels in the myometrial cells. This weakens the labor regulations and reduces their effectiveness [6, 7, 8].

Although the woman will experience pain, her labor will not progress. Oxytocin's effect has been linked to uterine lactate production, hypoxia, and other factors in recent studies. If labor is to end normally, this interaction is absolutely necessary. In uterine tissue, experimental studies have discovered lactate transport systems. Amniotic fluid appears to be a reservoir for lactate produced by the fetomaternal unit. It appears essential for the uterus to emit lactate in the event of over production (hypoxia) and to withdraw lactate back into the tissue, if necessary, as a form of energy. A group of transporters known as Monocarboxylate Transport (MCT) proteins of the uterus has been studied experimentally in a variety of normal and dystocic labors. Some of these transporters, which belong to a family of proteins, appear to only be activated in the event of acute hypoxia, such as dystocic labor. Others, on the other hand, are basic lactate transporters that operate continuously. Lactate was previously thought to be transported only over concentration gradients [9, 10].

Conclusion

We should find out if we are presently using the right rules to deal with a dystocia work. When dealing with arrested labor, hopefully an understanding of AFL levels can assist us in acquiring new information and a new type of consensus. To deal with a dystocic work, we should determine whether we are currently following the appropriate guidelines. We can hopefully acquire new information and a new kind of consensus when dealing with arrested labor with an understanding of AFL levels.

References

1. Boulvain M, Irion O, Dowswell T. Induction of labour at or near term for suspected fetal macrosomia. *Cochrane Database Syst Rev.* 5, CD000938 (2016).
2. Irion O, Boulvain M. Induction of labour for suspected fetal macrosomia. *Cochrane Database Syst Rev.* 2, CD000938.
3. Grier RM: Elective induction of labor. *Am J Obstet Gynecol.* 54, 511-516 (1947).
4. Dunne C, Silva OD, Schmidt G. Outcomes of elective labour induction and elective caesarean section in low-risk pregnancies between 37 and 41 Weeks' gestation. *J Obstet Gynaecol Can.* 31, 124-1130 (2009).
5. Vardo JH, Thornburg LL, Glantz JC. Maternal and neonatal morbidity among nulliparous women undergoing elective induction of labor. *J Reprod Med.* 56, 25-30 (2011).
6. Osmundson S, Ou Yang RJ, Grobman WA. Elective induction compared with expectant management in nulliparous women with an unfavorable cervix. *Obstet Gynecol.* 117, 583-587 (2011).
7. Stock SJ, Ferguson E, Duffy A Outcomes of elective induction of labour compared with expectant management: Population based study. *BMJ.* 344, e2838 (2012).
8. Cheng YW, Kaimal AJ, Snowden JM. Induction of labor compared to expectant management in low-risk women and associated perinatal outcomes. *Am J Obstet Gynecol.* 207, 502 (2012).
9. Darney BG, Snowden JM, Cheng YW. Elective induction of labor at term compared with expectant management: Maternal and neonatal outcomes. *Obstet Gynecol.* 122, 761-769 (2013).
10. Gibson KS, Waters TP, Bailit JL. Maternal and neonatal outcomes in electively induced low-risk term pregnancies. *Am J Obstet Gynecol.* 211, 249 (2012).