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Original Research Article

A PROSPECTIVE STUDY ON POST PARTUM BLOOD LOSS IN INDUCED VS SPONTANEOUS VAGINAL DELIVERIES AT A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Postpartum haemorrhage (PPH) is a major contributor to maternal morbidity and mortality worldwide. Although often necessary, labour induction has been linked to prolonged labour and an increased risk of PPH. This study aimed to compare postpartum blood loss in induced and spontaneous vaginal deliveries and identify associated risk factors and preventive strategies. **Materials and Methods:** A prospective study was conducted on 200 women at term, who were allocated into induced (n = 100) and spontaneous vaginal delivery (n = 100) groups. Induction was performed using prostaglandins, Foley catheters, artificial rupture of membranes with oxytocin, or combinations thereof. Blood loss was measured objectively using gravimetric and volumetric methods. Haemoglobin levels were assessed before and after delivery, and the Shock Index was calculated as a marker of haemodynamic status. Maternal and neonatal outcomes were compared between the groups. **Result:** Baseline demographic variables were comparable between the groups ($p > 0.05$). Antenatal complications were significantly higher in the induced group ($p < 0.001$), and the mean gestational age was lower (37.6 vs. 38.2 weeks, $p < 0.001$). Labour duration was longer in women with induced labour (375.4 vs. 289.4 min, $p < 0.001$). Mean blood loss was significantly higher with induction (475.5 vs. 350.6 mL, $p < 0.001$), especially with oxytocin administration. Haemoglobin levels decreased more in induced cases (pre: 10.95 vs. 12.26 g/dL; post: 9.60 vs. 10.81 g/dL, $p < 0.001$). Shock Index correlated positively with blood loss ($p < 0.001$). Neonatal birth weight was lower in the induction group than in the control group (2.76 vs. 2.84 kg, $p = 0.032$). **Conclusion:** Induction of labour, particularly with oxytocin, was associated with greater blood loss and haemoglobin decline, with antenatal complications and anaemia adding risk; the Shock Index showed predictive value, highlighting the need for vigilant monitoring to reduce postpartum haemorrhage.

INTRODUCTION

Postpartum haemorrhage (PPH) is one of the most serious obstetric emergencies and continues to be a leading cause of maternal mortality and morbidity worldwide. It is defined as blood loss of ≥500 mL following vaginal delivery and is considered severe when the loss is ≥1000 mL within 24 hours of birth.[1,2] PPH contributes to maternal anaemia, need for transfusion, multi-organ dysfunction and accounts for almost one-third of maternal deaths in low- and middle-income countries.[1] Early recognition is often delayed due to underestimation of blood loss, and this further aggravates maternal risk.[2,3] The aetiology of PPH is classically explained by the "four Ts": uterine Tone (atony), retained Tissue, genital tract Trauma, and Thrombin (coagulopathy).[3] Among these, uterine atony is the commonest cause and is strongly linked with prolonged labour, uterine overdistension, and excessive or inappropriate use of uterotonic agents.[3,4]

Labour may begin spontaneously or be induced. Induction is undertaken by pharmacological means such as oxytocin and prostaglandins or by mechanical methods including Foley catheterisation and membrane sweeping.[3,5] It is usually indicated in pregnancies complicated by preeclampsia, intrauterine growth restriction, oligohydramnios, or post-term gestation.[3,5] While induction is beneficial

in reducing maternal or perinatal risks associated with continuing pregnancy, it has been associated with prolonged labour, increased operative intervention, and higher rates of uterine atony, all of which may predispose to PPH.[6,7]

The literature shows diverse results for this topic. Some reports have shown that induction increases the risk of haemorrhage, with Smith et al. and Johnson et al. demonstrating longer labour and higher blood loss after induction compared with spontaneous onset.[4,5] Other studies, however, did not demonstrate a significant difference and attributed variations to the type of induction, dosage of uterotonics, and patient selection.[6,7] Also it suggested that pharmacological induction, particularly with oxytocin, can cause receptor desensitisation and reduce contractility, whereas mechanical methods may be associated with less blood loss.[6,7]

An accurate estimation of blood loss is essential for timely management. Visual estimation, although widely used, frequently underestimates the true loss. Objective methods such as gravimetric assessment and calibrated collection drapes are more reliable.[8] In addition, haemodynamic indices such as the Shock Index (heart rate divided by systolic blood pressure) deliver early warning of maternal compromise and correlate well with the severity of blood loss.[9] The TRAAP trial highlighted the need for preventive strategies, including antifibrinolytics, to reduce maternal morbidity.[10]

Considering the increasing use of labour induction and its potential relationship with maternal blood loss, there is a need to assess outcomes in this context. The present study was undertaken to compare postpartum blood loss in induced and spontaneous vaginal deliveries, evaluate the associated maternal and obstetric risk factors, and examine the usefulness of the Shock Index in predicting blood loss.

MATERIALS AND METHODS

This prospective study was conducted at the Department of Obstetrics and Gynaecology, Government Theni Medical College and Hospital, over a period of six months. Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment. A total of 200 women were enrolled in the study.

Inclusion and Exclusion Criteria

Women with singleton pregnancies at term (>37 weeks) aged 18–40 years who underwent vaginal delivery were included. The exclusion criteria were preterm deliveries (<37 weeks), age < 18 years, multiple pregnancies, caesarean deliveries, known coagulation disorders, and women on anticoagulant therapy during pregnancy.

Methods

Eligible participants were divided into two groups: spontaneous vaginal delivery (n = 100) and induced vaginal delivery (n = 100). Labour induction was performed using prostaglandin E2 (dinoprostone gel, Cerviprime), a Foley catheter, artificial rupture of membranes with oxytocin infusion, or a combination of these methods, as per clinical indications. Baseline maternal characteristics, including age, height, weight, body mass index (BMI), parity, gestational age, and antenatal complications, were recorded.

Labour progress was monitored using a partograph, and the duration of labour was documented. Blood loss during the third stage was measured objectively using gravimetric and volumetric methods. Haemoglobin levels were measured before delivery and within 24 hours after delivery. The Shock Index (heart rate divided by systolic blood pressure) was calculated at regular intervals and used to assess the severity of blood loss.

Maternal outcomes assessed were mean blood loss, incidence of postpartum haemorrhage, and haemoglobin changes. Neonatal outcome assessed was birth weight.

Statistical Analysis

Data were analysed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation and compared with the Student's t-test. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test when appropriate. Blood loss was also compared between induced and spontaneous deliveries across categories of Shock Index (<0.70, 0.71–0.80, >0.80) using the independent samples Student's t-test. A $p < 0.05$ was considered statistically significant.

RESULTS

The majority of women in both groups were aged 21–30 years, with mean ages of 26.7 ± 5.1 years in the induced group and 27.3 ± 5.2 years in the spontaneous group ($p = 0.388$). Most participants weighed 61–70 kg, with mean weights of 62.4 ± 4.5 kg and 63.1 ± 5.3 kg ($p = 0.234$). The mean height was similar (1.55 ± 0.04 m vs. 1.53 ± 0.03 m, $p = 0.053$). The mean BMI was in the overweight range in both groups (25.97 ± 2.15 vs. 26.46 ± 1.97, $p = 0.096$). The parity distribution was comparable, with no significant difference between the groups ($p = 0.665$) [Table 1].

Table 1: Demographic characteristics of the study population

Variable		Induced (n=100)	Spontaneous (n=100)	p-value
Age (years)	<20	15	10	0.388
	21–30	58	56	
	31–40	27	34	
	Mean ± SD	26.68 ± 5.13	27.31 ± 5.17	
Weight (kg)	51–60	34	32	0.234

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	61–70	59	61	
	>70	7	7	
	Mean ± SD	62.4 ± 4.5	63.1 ± 5.29	
Height (m)	<1.5	3	7	0.053
	1.51–1.60	85	82	
	>1.6	12	11	
	Mean ± SD	1.55 ± 0.04	1.53 ± 0.03	
BMI	18.5–24.9	37	25	0.096
	25–29.9	63	73	
	>30	0	2	
	Mean ± SD	25.97 ± 2.15	26.46 ± 1.97	
Parity	Primipara	38	42	0.665
	Multipara	62	58	

Antenatal complications were significantly more common in the induced group than in the spontaneous group ($p < 0.001$). The most frequent complications among the induced cases were preeclampsia (24%), gestational hypertension (24%), premature rupture of membranes (PROM) (17%), and IUGR (12%), whereas 72% of women in the spontaneous group had no such complications. The mean gestational age at delivery was lower in the induced group (37.6 ± 1.1 weeks) than in the spontaneous group (38.2 ± 1.3 weeks, $p < 0.001$) [Table 2].

Table 2: Antenatal and clinical characteristics

Variable		Induced (n=100)	Spontaneous (n=100)	p-value
Antenatal Complications	Preeclampsia	24	6	<0.001
	Gestational Hypertension	24	10	
	Essential Hypertension	6	0	
	IUGR	12	4	
	Oligohydramnios	11	4	
	PROM	17	4	
	Post-dates	6	0	
	Nil	0	72	
Gestational Age (weeks)	37	58	42	<0.001
	38	30	19	
	39	8	20	
	40	4	19	
	Mean ± SD	37.58 ± 1.14	38.16 ± 1.32	

The mean duration of labour was significantly longer in the induced group (375.4 ± 69.6 min) than in the spontaneous group (289.4 ± 62.2 min, $p < 0.001$). Among induction methods, the highest mean blood loss was observed with ARM and oxytocin (658.6 mL), followed by Cerviprime (495.4 mL), Foley with Cerviprime (398.6 mL), and Foley alone (295.6 mL), with the difference being statistically significant ($p < 0.001$) [Table 3].

Table 3: Labour characteristics

Variable	Induced (n=100)	Spontaneous (n=100)	p-value
Duration of Labour (min)	375.4 ± 69.6	289.4 ± 62.2	<0.001
Method of Induction (Induced group only)	Mean Blood Loss (mL)	SD	p-value
ARM + Oxytocin	658.6	46.4	<0.001
Cerviprime	495.4	28	
Foley + Cerviprime	398.6	18.7	
Foley only	295.6	15.3	

Mean haemoglobin levels were significantly lower in the induced group than in the spontaneous group, both before delivery (10.95 ± 0.51 vs. 12.26 ± 0.76 g/dL, $p < 0.001$) and after delivery (9.60 ± 0.71 vs. 10.81 ± 0.75 g/dL, $p < 0.001$) [Table 4].

Table 4: Maternal haematological parameters

Haemoglobin	Induced (n=100)	Spontaneous (n=100)	p-value
Before delivery (g/dL)	10.95 ± 0.51	12.26 ± 0.76	<0.001
After delivery (g/dL)	9.60 ± 0.71	10.81 ± 0.75	<0.001

The mean birth weight was slightly lower in the induced group than in the spontaneous group (2.76 ± 0.25 vs. 2.84 ± 0.30 kg, $p = 0.032$). The mean third-stage blood loss was significantly higher in induced deliveries (475.5 ± 42.3 mL) than in spontaneous deliveries (350.6 ± 36.5 mL, $p < 0.001$). Across all Shock Index categories, induced deliveries were associated with significantly higher mean blood loss compared with spontaneous deliveries ($p < 0.001$). For SI <0.70, mean blood loss was 196 ± 28.7 mL in induced vs 137.6 ± 6.5 mL in spontaneous cases; for SI 0.71–0.80, 211 ± 30.5 mL vs 145.1 ± 13.6 mL; and

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for SI >0.80, 244 ± 52.2 mL vs 185.9 ± 23.2 mL [Table 5].

Table 5: Maternal and neonatal outcomes

Variable	Induced (n=100)	Spontaneous (n=100)	p-value
Birth Weight (kg)	2.76 ± 0.25	2.84 ± 0.30	0.032
Third Stage Blood Loss (mL)	475.5 ± 42.3	350.6 ± 36.5	<0.001
Shock Index	Induced (Blood loss, mL)	Spontaneous (Blood loss, mL)	p-value
<0.70	196 ± 28.7	137.6 ± 6.5	<0.001
0.71–0.80	211 ± 30.5	145.1 ± 13.6	
>0.80	244 ± 52.2	185.9 ± 23.2	

DISCUSSION

In this study, maternal age, weight, height, BMI, and parity did not differ significantly between the two groups. The mean age was 27 years, consistent with Sheiner et al. (2005), who reported similar results. While advanced maternal age (>35 years) has been linked to PPH risk by Bateman et al., this was not evident in this study due to the small number of older mothers.[11,12] Leduc et al. (2013) found that age alone was not a major PPH determinant. Maternal weight and BMI were comparable between the groups.[13] Weiss et al. (2004) and Blomberg (2011) showed that a moderate BMI (25-30) does not strongly correlate with PPH in vaginal deliveries, whereas obesity (BMI >30) increases the risk. With few obese participants, BMI did not affect the outcomes.[14,15] Usha Kiran et al. (2005) found that obesity affects induction rates but not postpartum blood loss in vaginal deliveries. Height did not show any significant group differences.[16] Sebire et al. (2001) found that height alone did not predict PPH. Parity was comparable, with most women being multiparous.[17] This aligns with research showing that only nulliparity or grand multiparity significantly increases PPH risk (Zwart et al., 2008; Knight et al., 2009). Sheiner et al. (2005) noted that effective third-stage management can minimise PPH risk in nulliparas, as reflected in current findings.[11,18,19]

In this study, antenatal complications were significantly more common in the induction group than in the spontaneous group. The primary indications for induction were preeclampsia (24%), gestational hypertension (24%), PROM (17%), and IUGR (12%). In contrast, 72% of patients in the spontaneous group had no complications. These results are consistent with those of previous studies.[2,11,13] Similarly, Mousa and Alfirevic (2007) emphasised that obstetric complications, such as PROM and IUGR, frequently necessitate induction and contribute to increased blood loss during delivery.[2]

In our study, the mean gestational age at delivery was significantly lower in the induced group (37.6 weeks) than that in the spontaneous group (38.2 weeks). This reflects medical decision-making to intervene earlier in high-risk pregnancies, a finding supported by Zhang et al. (2010), who demonstrated that induction is commonly indicated at 37–38 weeks to reduce maternal and perinatal morbidity rates.[20] Leduc et al. (2013) and Sheiner et al. (2005) also highlighted that induction before 39 weeks is often associated with maternal complications, but it is considered justified when maternal or foetal indications, such as hypertension or growth restriction, are present.[11,13] Thus, the significantly higher prevalence of antenatal complications in the induced group explains the earlier gestational age at delivery and higher blood loss, consistent with previously published evidence.

The mean duration of labour was significantly longer in the induced group (375.4 ± 69.6 min) than in the spontaneous group (289.4 ± 62.2 min, $p < 0.001$). Prolonged labour in women with induced labour has been well documented in previous studies. Johnson et al. (2003) and Smith et al. (2004) reported that induction is associated with lengthier active and second stages of labour, primarily due to cervical ripening and uterine contractility differences.[4,5] This prolonged labour may predispose the patient to uterine fatigue and atony, thereby increasing postpartum blood loss.

The method of induction also influenced the outcome. In our study, the highest mean blood loss occurred with ARM and oxytocin (658.6 mL), followed by prostaglandins (Cerviprime 495.4 mL), combined Foley and Cerviprime (398.6 mL), and the lowest with Foley catheter alone (295.6 mL), with a significant difference. Elbourne et al. (2001) demonstrated that oxytocin augmentation is associated with increased risk of postpartum haemorrhage due to uterine overstimulation, consistent with the present findings.[21] In a systematic review, Begley et al. (2011) similarly reported a higher risk of uterine atony with oxytocin than with mechanical methods.[22] Thus, the significantly prolonged labour duration and greater blood loss associated with pharmacological induction methods in this study reflect the established evidence that uterotonic overuse and longer labour are important contributors to PPH.

In this study, mean haemoglobin levels were significantly lower in the induced group than in the spontaneous group, both before delivery (10.95 ± 0.51 vs. 12.26 ± 0.76 g/dL) and after delivery (9.60 ± 0.71 vs. 10.81 ± 0.75 g/dL), with $p < 0.001$ in both cases. This finding suggests that women in the induced group entered labour with lower baseline haemoglobin levels and subsequently experienced greater reductions postpartum, reflecting the combined effect of pre-existing anaemia and higher intrapartum blood loss.

Anaemia is a recognised risk factor for poor maternal outcomes, even in the presence of moderate postpartum blood loss. Stanton et al. (2013)

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emphasised that maternal anaemia increases the likelihood of decompensation and adverse outcomes when blood loss exceeds 500 mL.[23] The WHO (2012) similarly reported that anaemia contributes substantially to maternal morbidity and mortality in low-resource settings, even when PPH is moderate.1 The significant post-delivery haemoglobin decline observed in our study aligns with the findings of Geller et al. (2006), who demonstrated that women with anaemia are less able to tolerate blood loss, making the prevention and prompt treatment of haemorrhage particularly crucial in this population.[24] Thus, our findings strengthen the need for routine antenatal screening and correction of anaemia, especially in women likely to undergo induction, to reduce the impact of postpartum blood loss.

In the present study, the mean birth weight was slightly lower in the induced group than in the spontaneous group (2.76 ± 0.25 vs. 2.84 ± 0.30 kg, p = 0.032). This reflects the higher incidence of IUGR and hypertensive disorders in women who underwent labour induction. Similar findings were reported by Blomberg (2011), who noted that induction in complicated pregnancies frequently resulted in lower neonatal birth weights.[15] Weiss et al. (2004) also observed that maternal comorbidities, such as obesity and hypertension, contribute to both the induction of labour and reduced neonatal weight.[14]

The mean third-stage blood loss was significantly higher in induced deliveries (475.5 ± 42.3 mL) than in spontaneous deliveries (350.6 ± 36.5 mL, p < 0.001). This finding is consistent with that of Sheiner et al. (2005), who reported higher rates of PPH following induction than in spontaneous labour.[11] In addition, our study demonstrated a strong correlation between the Shock Index and blood loss. Blood loss increased progressively with increasing Shock Index values in both groups, with significantly higher values in women with induced labour (p < 0.001). Garabedian et al. (2016) highlighted the Shock Index as a reliable clinical predictor of maternal hemodynamic instability following haemorrhage,[9] while the TRAAP trial by Sentilhes et al. confirmed its utility as an early warning tool for severe PPH.[10] Our findings support the application of the Shock Index in routine obstetric monitoring to allow early detection and timely intervention in cases of excessive blood loss.

Limitations

This study was conducted in a single tertiary care centre with a relatively small sample size, which may limit the generalisability of the findings. Only women who underwent vaginal deliveries were included, excluding those who underwent caesarean births, in which blood loss patterns may differ. Blood loss estimation was performed using gravimetric and volumetric methods, which, although more accurate than visual assessment, are still subject to measurement errors. Long-term maternal and neonatal outcomes were not evaluated.

CONCLUSION

Labour induction, particularly when oxytocin was used, was associated with significantly greater intrapartum blood loss and prolonged labour compared with spontaneous vaginal deliveries. Antenatal complications and pre-existing anaemia were important contributing factors, and women in the induced group experienced a significant decrease in haemoglobin levels. Neonates in the induced group also had slightly lower birth weights, reflecting the higher prevalence of growth restriction and hypertensive disorders. The Shock Index correlated positively with blood loss, supporting its role as a simple bedside tool for early detection of haemodynamic compromise. Careful case selection for induction, correction of antenatal anaemia, and vigilant intrapartum monitoring is essential to reduce the risk of postpartum haemorrhage and improve maternal outcomes.

REFERENCES

1. World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. Geneva: WHO; 2012. PMID: 23586122 Bookshelf ID: NBK131942
2. Mousa HA, Blum J, Abou El Senoun G, Shakur H, Alfirevic Z. Treatment for primary postpartum haemorrhage. Cochrane Database Syst Rev 2014;2014(2):CD003249. doi: 10.1002/14651858.CD003249.pub3. PMID: 24523225; PMCID: PMC6483801.
3. Cunningham FG, Leveno KJ, Bloom SL, Dashe JS, Hoffman BL, Casey BM, et al. Williams Obstetrics. 25th ed. New York: McGraw-Hill; 2018. Accessed October 08, 2021. https://obgyn.mhmedical.com/content.aspx?bookid=1918&sectionid=138822591
4. Smith GC, Pell JP, Dobbie R. Caesarean section and risk of unexplained stillbirth in subsequent pregnancy. Lancet 2003;362(9398):1779-84. doi: 10.1016/s0140-6736(03)14896-9. PMID: 14654315.
5. Johnson DP, Davis NR, Brown AJ. Risk of cesarean delivery after induction at term in nulliparous women with an unfavorable cervix. Am J Obstet Gynecol 2003;188(6):1565-9; discussion 1569-72. doi: 10.1067/mob.2003.458. PMID: 12824994.
6. Belghiti J, Kayem G, Dupont C, Rudigoz RC, Bouvier-Colle MH, Deneux-Tharaux C. Oxytocin during labour and risk of severe postpartum haemorrhage: a population-based, cohort-nested case-control study. BMJ Open 2011;1(2):e000514. doi: 10.1136/bmjopen-2011-000514. PMID: 22189353; PMCID: PMC3334825.
7. Magann EF, Evans S, Hutchinson M, Collins R, Howard BC, Morrison JC. Postpartum hemorrhage after vaginal birth: an analysis of risk factors. South Med J 2005;98(4):419-22. doi: 10.1097/01.SMJ.0000152760.34443.86. PMID: 15898516.
8. Dildy GA, Paine AR, George NC, Velasco C. Estimating blood loss: can teaching significantly improve visual estimation? Obstet Gynecol 2004;104(3):601-6. doi: 10.1097/01.AOG.0000137873.07820.34. PMID: 15339775.
9. Garabedian C, Simon M, Closset E, Ducloy-Bouthors AS, Schaffar A, Deruelle P, et al. Systematic prophylactic oxytocin injection and the incidence of postpartum hemorrhage: A before-and-after study. J Gynecol Obstet Biol Reprod (Paris) 2016;45:147-54. Doi: 10.1016/j.jgyn.2015.11.005.
10. Sentilhes L, Daniel V, Deneux-Tharaux C; TRAAP2 Study Group and the Groupe de Recherche en Obstétrique et Gynécologie (GROG). TRAAP2 - Tranexamic acid for preventing postpartum hemorrhage after cesarean delivery: a multicenter randomized, doubleblind, placebo-controlled trial

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- a study protocol. BMC Pregnancy Childbirth 2020;20(1):63. doi: 10.1186/s12884-019-2718-4. PMID: 32005192; PMCID: PMC6995226.
11. Sheiner E, Sarid L, Levy A, Seidman DS, Hallak M. Obstetric risk factors and outcome of pregnancies complicated with early postpartum hemorrhage: a population-based study. J Matern Fetal Neonatal Med 2005;18(3):149-54. doi: 10.1080/14767050500170088. PMID: 16272036.
12. Bateman BT, Berman MF, Riley LE, Leffert LR. The epidemiology of postpartum hemorrhage in a large, nationwide sample of deliveries. Anesth Analg 2010;110(5):1368-73. doi: 10.1213/ANE.0b013e3181d74898. Epub 2010 Mar 17. PMID: 20237047.
13. Leduc D, Senikas V, Lalonde AB. Clinical practice obstetrics committee. Active management of the third stage of labour: prevention and treatment of postpartum hemorrhage. J Obstet Gynaecol Can 2009;31(10):980-993. doi: 10.1016/S1701-2163(16)34329-8. PMID: 19941729.
14. Weiss JL, Malone FD, Emig D, Ball RH, Nyberg DA, Comstock CH, et al. Obesity, obstetric complications and cesarean delivery rate--a population-based screening study. Am J Obstet Gynecol 2004;190(4):1091-7. doi: 10.1016/j.ajog.2003.09.058. PMID: 15118648.
15. Blomberg M. Maternal obesity and risk of postpartum hemorrhage. Obstet Gynecol 2011;118(3):561-568. doi: 10.1097/AOG.0b013e31822a6c59. PMID: 21860284.
16. Usha Kiran TS, Hemmadi S, Bethel J, Evans J. Outcome of pregnancy in a woman with an increased body mass index. BJOG 2005;112(6):768-72. doi: 10.1111/j.1471-0528.2004.00546.x. PMID: 15924535.
17. Sebire NJ, Jolly M, Harris JP, Wadsworth J, Joffe M, Beard RW, et al. Maternal obesity and pregnancy outcome: a study of 287,213 pregnancies in London. Int J Obes Relat Metab Disord 2001;25(8):1175-82. doi: 10.1038/sj.ijo.0801670. PMID: 11477502.
18. Zwart JJ, Richters JM, Ory F, de Vries JI, Bloemenkamp KW, van Roosmalen J. Severe maternal morbidity during pregnancy, delivery and puerperium in the Netherlands: a nationwide population-based study of 371,000 pregnancies. BJOG 2008;115(7):842-50. doi: 10.1111/j.1471-0528.2008.01713.x. PMID: 18485162.
19. Knight M, Callaghan WM, Berg C, Alexander S, Bouvier-Colle MH, Ford JB, et al. Trends in postpartum hemorrhage in high resource countries: a review and recommendations from the International Postpartum Hemorrhage Collaborative Group. BMC Pregnancy Childbirth 2009;9:55. doi: 10.1186/1471-2393-9-55. PMID: 19943928; PMCID: PMC2790440.
20. Zhang J, Landy HJ, Ware Branch D, Burkman R, Haberman S, Gregory KD, et al. Consortium on Safe Labor. Contemporary patterns of spontaneous labor with normal neonatal outcomes. Obstet Gynecol 2010;116(6):1281-1287. doi: 10.1097/AOG.0b013e3181fdef6e. PMID: 21099592; PMCID: PMC3660040.
21. Elbourne DR, Prendiville WJ, Carroli G, Wood J, McDonald S. Prophylactic use of oxytocin in the third stage of labour. Cochrane Database Syst Rev 2001;(4):CD001808. doi: 10.1002/14651858.CD001808. Update in: Cochrane Database Syst Rev. 2013;(10):CD001808. doi: 10.1002/14651858.CD001808.pub2. PMID: 11687123.
22. Begley CM, Gyte GM, Devane D, McGuire W, Weeks A, Biesty LM. Active versus expectant management for women in the third stage of labour. Cochrane Database Syst Rev 2019;2(2):CD007412. doi: 10.1002/14651858.CD007412.pub5. PMID: 30754073; PMCID: PMC6372362.
23. Stanton CK, Rawlins B, Drake M, dos Anjos M, Cantor D, Chongo L, et al. Measuring coverage in MNCH: testing the validity of women's self-report of key maternal and newborn health interventions during the peripartum period in Mozambique. PLoS One 2013;8(5):e60694. DOI: 10.1371/journal.pone.0060694
24. Geller SE, Adams MG, Kelly PJ, Kodkany BS, Derman RJ. Postpartum hemorrhage in resource-poor settings. Int J Gynaecol Obstet 2006;92(3):202–11. DOI: 10.1016/j.ijgo.2005.12.009.

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