How Low Is Too Low? Postpartum Hemorrhage Risk among Women with Thrombocytopenia

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Abstract

Objective To estimate the association between severity of thrombocytopenia and postpartum hemorrhage.

Study Design We performed a secondary analysis of a prospective cohort of women delivering by cesarean or vaginal birth after cesarean conducted by the National Institute of Child Health and Human Development (NICHD) Maternal–Fetal Medicine Unit. Women delivering $\geq$ 20 weeks with platelets $<$ 400,000/mL were included. Thrombocytopenia was defined as predelivery platelets of $<$ 150,000/mL. Primary outcomes were (1) laboratory evidence of hemorrhage, defined as a decrease in hemoglobin $\geq$ 4 mg/dL and (2) clinical evidence of hemorrhage, a composite of atony, transfusion, coagulopathy, hysterectomy, laparotomy, or intensive care unit admission. Odds ratios were calculated for primary outcomes using thrombocytopenia as a dichotomous and ordinal variable.

Results A total of 54,597 women were included; 5,611 (10.3%) had antepartum thrombocytopenia, 1,976 (3.6%) women had laboratory evidence of hemorrhage, and 3,862 (7.1%) had clinical evidence of hemorrhage. Thrombocytopenia was associated with both laboratory evidence of hemorrhage (adjusted odds ratio [aOR]: 1.60, 95% CI: 1.38–1.86) and clinical evidence of hemorrhage (aOR: 1.68, 95% CI: 1.52–1.83). The odds of laboratory and clinical evidence of hemorrhage increased incrementally with severity of thrombocytopenia.

Conclusion Thrombocytopenia is associated with both laboratory and clinical evidence of hemorrhage; risk increases dramatically as platelet count decreases.

Keywords
- postpartum hemorrhage
- platelets
- thrombocytopenia

Postpartum hemorrhage remains a leading cause of maternal morbidity and mortality both in the United States[1] and worldwide.[2] Incidence of postpartum hemorrhage is increasing tremendously.[3–5] Identification of risk factors for postpartum hemorrhage is an important component of safety protocols recommended by the National Partnership for Maternal Safety.[6] Knowledge of maternal risk factors for hemorrhage improves the clinician's ability to prepare for and respond to postpartum hemorrhage.

Thrombocytopenia, defined as a platelet count $<$ 150,000/mL, is common and complicates approximately 12% of all pregnancies.[7,8] The most common etiology of third trimester thrombocytopenia is gestational thrombocytopenia, accounting for 80% of cases;[8] other etiologies include

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preeclampsia and HELLP (hemolysis [H], elevated liver enzymes [EL], and low platelets [LP]) syndrome, infection, and immune etiologies. Thrombocytopenia is a known risk factor for procedural and surgical bleeding complications. In nonobstetrical populations, spontaneous bleeding occurs when platelets fall below 10,000 to 20,000/mL.[9,10] Platelet transfusion is recommended prior to major nonneuraxial surgery for any patient with platelets below 50,000 based on expert consensus.[11,12] While recommendations from the general surgical literature are applied to the pregnant population,[13] the relationship between platelet count and hemorrhage risk has not been clearly illustrated in the obstetric literature. The degree to which mild or severe thrombocytopenia affects the risk for postpartum hemorrhage remains uncertain.

The objective of this study was to estimate the association between severity of antepartum thrombocytopenia and risk of postpartum hemorrhagic morbidity.

Materials and Methods

Study Population

This was a secondary analysis of a prospective, multicenter cohort of women enrolled in the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Maternal–Fetal Medicine Unit's "cesarean registry." This registry was collected prospectively from 1999 to 2002 at 19 academic hospitals. Women with singleton and multiple gestations who delivered by primary or repeat cesarean or by vaginal birth after cesarean section were included in the registry. Demographic, obstetrical, labor and delivery, and postpartum characteristics were collected from medical records by trained researchers in a standardized fashion. The study focused on collecting detailed information from the delivery hospitalization. The methods for this study have been previously described.[14] A deidentified version of this dataset is now publicly available. This secondary analysis was reviewed by the Institutional Review Board (IRB) at the University of North Carolina and considered exempt.

We included women in this analysis who delivered $\geq$ 20 weeks of gestation and had a documented predelivery platelet count $<$ 400,000/mL during the delivery admission. If more than one platelet count was available, the platelet count closest to the delivery was used. Mode of delivery was analyzed as a dichotomous variable, either cesarean (primary and repeat) or vaginal birth after cesarean (VBAC) section. Women were considered to have labored if they were admitted in active labor or were induced, regardless of ultimate mode of delivery. Thrombocytopenia was defined as a platelet count $<$ 150,000/mL. Women with thrombocytopenia were compared with those with a normal platelet count. Additionally, admission platelet count was examined as an ordinal variable in increments of 20,000/mL, comparing women with platelet count of 130,000 to 149,999/mL, 110,000 to 129,999/mL, 90,000 to 109,999/mL, 70,000 to 89,999/mL, 50,000 to 69,999/mL, and $<$ 50,000/mL to women with a normal platelet count (150,000–400,000/mL).

Outcomes

The two primary outcomes of this study were (1) laboratory evidence of hemorrhage, defined as a decrease in hemoglobin $\geq$ 4 mg/dL from predelivery to postpartum values, and (2) clinical evidence of hemorrhage, a composite outcome, including medical treatment of uterine atony, transfusion of blood products intrapartum or postpartum, coagulopathy, peripartum hysterectomy, postpartum laparotomy, and intensive care unit admission. Medical treatment of uterine atony was defined as an administration of methylergonovine or carboprost tromethamine after delivery. Coagulopathy was defined as decreasing platelets with prolonged partial thromboplastin time greater than 40 seconds or receipt of fresh frozen plasma, cryoprecipitate, or platelets.

Statistical Analysis

Maternal demographic and clinical characteristics were compared between women with and without thrombocytopenia using chi-square and student's *t*-test, as appropriate. Two multivariable logistic regression models were then created, one with laboratory evidence of hemorrhage morbidity as the dependent variable and the other with clinical evidence of hemorrhage as the dependent variable. Initial models included covariates that were statistically significant in the bivariate analysis. Because treatment with magnesium has been associated with an increased risk of postpartum hemorrhage,[15,16] we elected to include magnesium in the models a priori. Both thrombocytopenia and magnesium are highly correlated with preeclampsia; therefore, we did not include preeclampsia as a confounder due to colinearity. If information regarding intrapartum magnesium sulfate treatment was missing, we presumed women to be unexposed. Stepwise regression with backward elimination was used to select an adjusted model. Main effects were retained in the model if *p*-values were $<$ 0.20. Variables were considered statistically significant in the final model only if *p*-values were $<$ 0.05.

Subgroup analyses were performed to assess for persistence of the relationship between thrombocytopenia and hemorrhage in the setting of vaginal delivery, cesarean delivery, and preeclampsia. Statistical significance was defined as $p < 0.05$. All analyses were performed using STATA version 14.2 (College Station, TX).

Results

From the original cesarean section registry of 73,257 women, 54,597 women met the inclusion criteria for this analysis (►**Fig. 1**). Of these, 5,611 women (10.3%) had thrombocytopenia. Among women with thrombocytopenia, the median platelet count was 132 (interquartile range [IQR]: 115–142); 686 women had a platelet count $<$ 100,000/mL, and 64 had a platelet count $<$ 50,000. In contrast, women with a normal platelet count had a median platelet count of 223 (IQR: 191–262).

Women with thrombocytopenia were older; had a lower BMI; were more likely to be nulliparous, have hypertensive disorder of pregnancy, receive magnesium; and have a multiple gestation pregnancy (►**Table 1**). Women with thrombocytopenia were also more likely to deliver preterm ($<$ 37 weeks

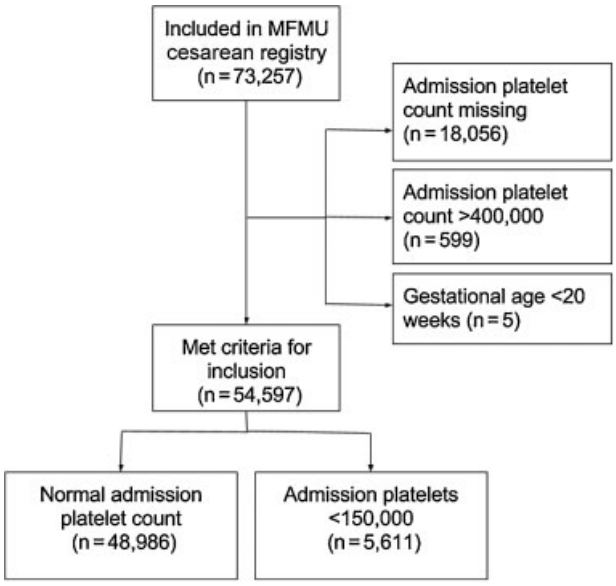

Fig. 1 Flow chart describing study population.

of gestation) via cesarean section, with general anesthesia, and were less likely to have labored compared with women without thrombocytopenia (►**Table 1**).

A total of 3,862 women (7.1% of all women) experienced clinical evidence of hemorrhage, and a total of 1,976 women (3.6% of all women) met criteria for laboratory evidence of hemorrhage. Also, 754 women (1.4% of all women) met criteria for both clinical and laboratory evidence of hemorrhage. Women with thrombocytopenia were more likely to experience both laboratory and clinical evidence of hemorrhage; among women with thrombocytopenia, 11.7% had clinical evidence of hemorrhage, 8.3% had laboratory evidence of hemorrhage, and 2.9% had both laboratory and clinical evidence of hemorrhage as compared with 6.5, 3.3, and 1.2%, respectively, to those without thrombocytopenia ($p < 0.001$ for all comparisons, ►**Table 2**). The incidence of both laboratory and clinical evidence of postpartum hemorrhage increased with the severity of thrombocytopenia (►**Fig. 2**).

In multivariate models, women with thrombocytopenia had increased odds of both clinical evidence of hemorrhage (adjusted odds ratio [aOR]: 1.68, 95% confidence interval [CI]: 1.52–1.83) and laboratory evidence of hemorrhage (aOR: 1.60, 95% CI: 1.38–1.86) compared with women without thrombocytopenia. When further classifying by the severity of thrombocytopenia, odds of both clinical and laboratory evidence of hemorrhage increased with the degree of thrombocytopenia, considering those with normal platelets as the reference group (►**Table 3**). Women with platelets < 50,000/mL had ninefold odds of clinical evidence of hemorrhage compared with women without thrombocytopenia and over fourfold odds of laboratory evidence of hemorrhage.

Table 1 Maternal demographic, clinical, and delivery characteristics by thrombocytopenia in the Maternal–Fetal Medicine Unit Cesarean Registry (1999–2002, $n = 54,597$)

Variable	Thrombocytopenia ($n = 5,611$) n (%)	Normal platelets ($n = 48,986$) n (%)	*p*-Value
Maternal age (SD)	29.2 (6.1)	28.5 (6.1)	<0.001
Race/ethnicity			
White	2,650 (47.2)	21,021 (42.9)	<0.001
Black	1,523 (27.1)	14,774 (30.1)	
Hispanic	1,153 (20.6)	10,879 (22.2)	
Delivery BMI (kg/m^2) (SD)	31.5 (5.2)	33.0 (7.2)	<0.001
Preeclampsia, eclampsia, or HELLP syndrome	1,142 (20.4)	4,724 (9.6)	<0.001
Tobacco use	624 (11.1)	7,332 (15.0)	<0.001
Magnesium sulfate treatment	1,111 (19.8)	4,639 (9.5)	<0.001
Nulliparous	1,442 (25.8)	11,122 (22.8)	<0.001
Multiple gestation	3,548 (7.2)	757 (13.5)	<0.001
Prior cesarean delivery	3,475 (62.2)	32,472 (66.5)	<0.001
Mean gestational age at delivery (SD)	37.5 (3.6)	37.9 (3.4)	<0.001
Delivery at < 37 wk	1,609 (28.7)	11,072 (22.6)	<0.001
Delivery at < 32 wk	510 (9.1)	3,459 (7.2)	<0.001
Labored	2,908 (51.8)	27,196 (55.5)	<0.001
Cesarean delivery	4,680 (83.4)	39,928 (81.5)	<0.001
General anesthesia	572 (10.5)	3,008 (6.3)	<0.001

Abbreviations: BMI, body mass index; SD, standard deviation.

Table 2 Clinical and laboratory evidence of hemorrhage by presence of thrombocytopenia in the Maternal–Fetal Medicine Unit Cesarean Registry (1999–2002, $n = 54,597$)

Characteristics	Thrombocytopenia ($n = 5,611$) n (%)	Normal platelets ($n = 48,986$) n (%)	*p*-Value
Clinical evidence of hemorrhage	654 (11.7)	3,208 (6.6)	<0.001
Medical treatment of atony	325 (5.8)	1,963 (4.0)	<0.001
Blood transfusion	262 (4.7)	1,160 (2.4)	<0.001
Coagulopathy	149 (2.7)	171 (0.4)	<0.001
Repeat laparotomy	24 (0.4)	117 (0.2)	0.008
Peripartum hysterectomy	33 (0.6)	234 (0.5)	0.261
Intensive care unit admission	96 (1.7)	313 (0.64)	<0.001
Laboratory evidence of hemorrhage	352(8.3)	1,624 (4.6)	<0.001

Subgroup analyses were then performed to evaluate the persistence of relationship between thrombocytopenia and hemorrhage in the presence ($n = 5,866$) and absence of preeclampsia ($n = 48,731$) in women delivering vaginally ($n = 9,989$) and by cesarean section ($n = 44,608$). The relationship between increasing risk of hemorrhage and worsening severity of thrombocytopenia persisted in these analyses (►**Table 3**).

Comment

We found that thrombocytopenia—regardless of severity—conferred a significant increased risk of postpartum hemorrhage. Results were consistent regardless of whether we used a clinical definition or an objective, laboratory-derived definition. Additionally, we found that the risk of hemorrhage increased as the platelet count decreased with the most dramatic odds occurring with platelets < 50,000/mL. These findings persisted despite controlling for confounders.

The threshold at which the risk of hemorrhagic morbidity increases with thrombocytopenia is not well established in the obstetric literature. A small retrospective study previously illustrated increased postpartum hemorrhage with platelets < 40,000/mL in the setting of HELLP syndrome,[17] but hemorrhage risk with milder thrombocytopenia remained unclear. Another small retrospective study of patients with immune thrombocytopenic purpura did not illustrate an increased risk of hemorrhage with worsening thrombocytopenia.[18] Notably, both of these studies were small, and neither was adequately powered to detect differences in hemorrhage risk of the magnitude found in this study. Traditional teaching is that thrombocytopenia should not increase hemorrhage risk as long as platelets are above 50,000/mL, and guidelines for management of women with immune thrombocytopenic purpura state that a platelet count of ≥ 50,000/mL is adequate for cesarean delivery.[19] The findings of this study are therefore notable; our analysis confirms that risk for hemorrhagic morbidity is greatly increased in women with platelets < 50,000/mL, and that even

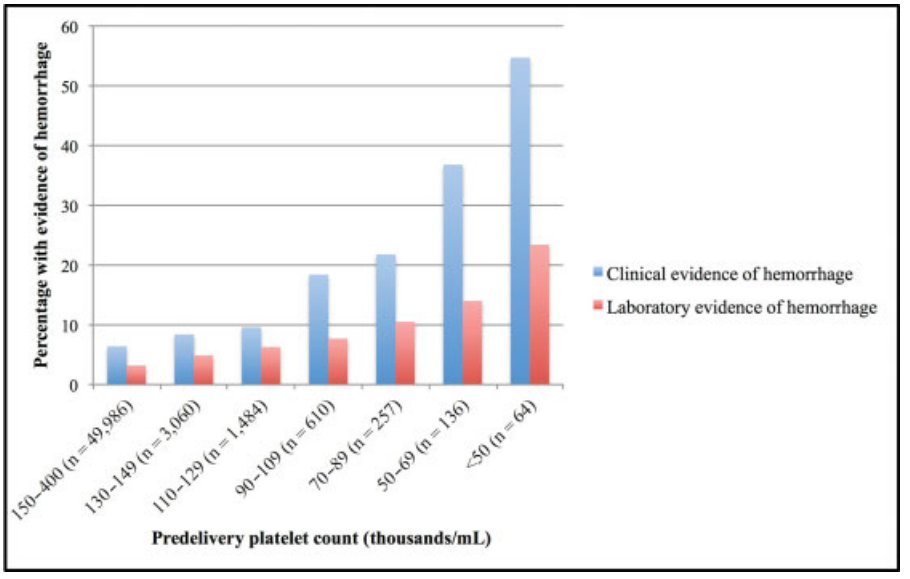

Fig. 2 Percentage of patients with laboratory or clinical evidence of hemorrhage by antepartum platelet count.

Table 3 Adjusted odds of clinical hemorrhagic morbidity and laboratory evidence of hemorrhage by severity of thrombocytopenia in the Maternal–Fetal Medicine Unit Cesarean Registry (1999–2002, $n = 54{,}597$)

Platelets (thousands/mL)	150–400 ($n = 49{,}986$, 91.6%)	130–149 ($n = 3{,}060$, 5.6%)	110–129 ($n = 1{,}484$, 2.7%)	90–109 ($n = 610$, 1.1%)	70–89 ($n = 257$, 0.5%)	50–69 ($n = 136$, 0.2%)	<50 ($n = 64$, 0.1%)
All women							
Clinical evidence of Hemorrhage,[a] aOR (95% CI)	Creanga et al[1]	1.25 (1.09–1.42)	1.39 (1.16–1.66)	2.69 (2.17–3.33)	2.92 (2.15–3.98)	5.44 (3.78–7.82)	9.31 (5.60–15.47)
Laboratory evidence of hemorrhage,[b] aOR (95% CI)	Creanga et al[1]	1.39 (1.13–1.71)	1.56 (1.19–2.05)	1.62 (1.09–2.41)	2.49 (1.50–4.14)	3.31 (1.79–6.10)	4.46 (2.06–9.65)
Women without preeclampsia, eclampsia, or HELLP syndrome							
Clinical evidence of Hemorrhage,[a] aOR (95% CI)	Creanga et al[1]	1.23 (1.06–1.44)	1.36 (1.10–1.68)	2.66 (2.02–3.50)	2.47 (1.57–3.87)	8.42 (4.75–14.93)	8.49 (2.82–25.54)
Laboratory evidence of hemorrhage,[b] aOR (95% CI)	Creanga et al[1]	1.45 (1.14–1.83)	1.65 (1.20–2.26)	1.82 (1.12–2.99)	0.81 (0.25–2.58)	3.55 (1.22–10.31)	***
Women with preeclampsia, eclampsia, or HELLP syndrome							
Clinical evidence of hemorrhage,[a] aOR (95% CI)	Creanga et al[1]	1.35 (1.03-1.78)	1.52 (1.09–2.12)	2.91 (2.06–4.12)	3.70 (2.40–5.70)	4.69 (2.96–7.45)	10.21 (5.72–18.22)
Laboratory evidence of hemorrhage,[b] aOR (95% CI)	Creanga et al[1]	1.19 (0.76–1.88)	1.34 (0.79-2.27)	1.29 (0.65–2.56)	4.04 (2.19–7.47)	2.85 (1.34–6.03)	4.67 (2.09–10.43)
Women delivering by cesarean section							
Clinical evidence of hemorrhage,[a] aOR (95% CI)	Creanga et al[1]	1.22 (1.05–1.41)	1.39 (1.15–1.68)	2.74 (2.19–3.43)	3.03 (2.20–4.16)	5.01 (3.44–7.29)	8.90, (5.32–14.88)
Laboratory evidence of hemorrhage,[b] aOR (95% CI)	Creanga et al[1]	1.40 (1.12–1.74)	1.57 (1.18–2.09)	1.67 (1.10–2.54)	2.56 (1.52–4.33)	3.13 (1.66–5.90)	4.42 (2.04–9.57)
Women delivering by vaginal birth after cesarean							
Clinical evidence of hemorrhage,[a] aOR (95% CI)	Creanga et al[1]	1.54 (1.07–2.21)	1.43 (0.83–2.44)	2.19 (1.04–4.61)	2.19 (0.66–7.31)	21.87 (5.39–88.73)	NA[c]
Laboratory evidence of hemorrhage,[b] aOR (95% CI)	Creanga et al[1]	1.35 (0.69–2.67)	1.43 (0.56-3.67)	1.14 (0.27–4.86)	1.52 (0.19–12.08)	8.20 (0.71–94.33)	NA[c]

Abbreviations: aOR, adjusted odds ratio; CI, confidence interval.

[a]Adjusted odds of clinical evidence of hemorrhage, controlling for labored status, preterm delivery, race, history of prior cesarean, maternal age, multiple gestation, and magnesium exposure.

[b]Adjusted odds of laboratory evidence of hemorrhage, controlling for labored status, preterm delivery, race, smoking, obesity, history of prior cesarean, maternal age, multiple gestation, nulliparity, and magnesium exposure.

[c]Too few women were available to calculate aOR.

milder degrees of thrombocytopenia appear to be associated with a clinically meaningful increased risk of peripartum hemorrhage. Nonetheless, we emphasize that though the risk of hemorrhage is elevated for women with platelet counts $<$ 150,000 mL, these data alone are insufficient to change routine clinical practice. For example, we do not recommend a routine platelet transfusion prior to delivery for women with platelets $<$ 150,000 on the basis of these results but would recommend an increased clinical vigilance for postpartum hemorrhage.

In this study, we examined two separate but complementary outcomes. We chose to examine laboratory evidence of hemorrhage to identify women with clinically significant decreases in hemoglobin that would be consistent with true postpartum hemorrhage. Many women who meet the criteria for postpartum hemorrhage may not experience a decrease in hemoglobin as profound as a drop of 4 points or receive blood transfusion prior to such a decrease and would not therefore show laboratory evidence of hemorrhage. Further, a decrease in hemoglobin of 4 mg/dL may also be attributed to a dilutional effect in some individuals, which may have produced misclassification bias. As such, we also chose to examine clinical evidence of hemorrhage as a composite outcome of clinically meaningful outcomes associated with maternal morbidity. There is also a small risk that neither hemorrhage definition would capture a minority of cases (e.g., postpartum hemorrhages that did not require transfusion and were not secondary to uterine atony).

Our study has several strengths. The study population was large and derived from multiple centers across the United States, increasing heterogeneity and generalizability. Data collection was prospective and uniform by trained research staff. Our hemorrhagic outcomes were objective and did not use estimated blood loss, a subjective measure with traditionally very poor inter-rater reliability and equally poor sensitivity for postpartum hemorrhage.[20–23] In other words, the outcomes used in this study were measurable, reproducible and thus, clinically meaningful.

Our study also has several limitations. Despite the large population, relatively few patients had thrombocytopenia $<$ 100,000/mL. However, this likely represented the uncommon distribution of severe thrombocytopenia in the obstetric population. While we were able to compare the incidence of hypertensive disorders of pregnancy between women with and without thrombocytopenia, other common etiologies of thrombocytopenia (e.g., gestational thrombocytopenia, immune-mediated thrombocytopenia) were not documented. Thus, we were unable to control for etiology of thrombocytopenia in this analysis. Additionally, we did not have information regarding platelet trends; it is possible that acutely falling platelets may confer higher risk compared with platelets that are low, but stable. Finally, though baseline rates of hemorrhage in this cohort were similar to what we expected, the a priori risk for hemorrhage was likely to be higher in this population of women with a prior cesarean or planning to undergo cesarean, limiting the ability to generalize these results to women planning vaginal delivery without history of prior cesarean. As described above, we are unable to recommend antepartum or preoperative platelet transfusion based on the findings of this study.

Existing data are sparse surrounding hemorrhage risk associated with thrombocytopenia, particularly among women with mild and moderate thrombocytopenia. Based on our findings, clinicians should consider women with any degree of thrombocytopenia to be at an increased risk for peripartum hemorrhage during the antepartum risk assessment, and should have a low threshold to initiate hemorrhage management as clinically indicated. Thresholds for platelet transfusion in the obstetric setting should be addressed in future studies.

Condensation

The risk of postpartum hemorrhage is directly proportional to the severity of thrombocytopenia.

Note

This study was presented, in part, as a poster at the 37th Annual Pregnancy Meeting of the Society for Maternal–Fetal Medicine from January 22 to 28, 2017 at Las Vegas, NV (final abstract no. 644).

Funding

None.

Conflict of Interest

None.

References

1 Creanga AA, Berg CJ, Syverson C, Seed K, Bruce FC, Callaghan WM. Pregnancy-related mortality in the United States, 2006-2010. Obstet Gynecol 2015;125(01):5–12
2 American College of Obstetricians and Gynecologists. ACOG practice bulletin: clinical management guidelines for obstetrician-gynecologists number 76, October 2006: postpartum hemorrhage. Obstet Gynecol 2006;108(04):1039–1047
3 Kramer MS, Berg C, Abenhaim H, et al. Incidence, risk factors, and temporal trends in severe postpartum hemorrhage. Am J Obstet Gynecol 2013;209(05):449.e1–449.e7
4 Mehrabadi A, Hutcheon JA, Liu S, et al; Maternal Health Study Group of Canadian Perinatal Surveillance System (Public Health Agency of Canada). Contribution of placenta accreta to the incidence of postpartum hemorrhage and severe postpartum hemorrhage. Obstet Gynecol 2015;125(04):814–821
5 Knight M, Callaghan WM, Berg C, 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
6 Main EK, Goffman D, Scavone BM, et al; National Partnership for Maternal Safety; Council on Patient Safety in Women's Health Care. National partnership for maternal safety: consensus bundle on obstetric hemorrhage. Obstet Gynecol 2015;126(01):155–162
7 Boehlen F, Hohlfeld P, Extermann P, Perneger TV, de Moerloose P. Platelet count at term pregnancy: a reappraisal of the threshold. Obstet Gynecol 2000;95(01):29–33
8 American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice bulletin no.166: thrombocytopenia in pregnancy. Obstet Gynecol 2016;128(03):e43–e53
9 Gaydos LA, Freireich EJ, Mantel N. The quantitative relation between platelet count and hemorrhage in patients with acute leukemia. N Engl J Med 1962;266:905–909

10 Heckman KD, Weiner GJ, Davis CS, Strauss RG, Jones MP, Burns CP. Randomized study of prophylactic platelet transfusion threshold during induction therapy for adult acute leukemia: 10,000/microL versus 20,000/microL. J Clin Oncol 1997;15(03):1143–1149
11 Wall MH, Prielipp RC. Transfusion in the operating room and the intensive care unit: current practice and future directions. Int Anesthesiol Clin 2000;38(04):149–169
12 Kaufman RM, Djulbegovic B, Gernsheimer T, et al; AABB. Platelet transfusion: a clinical practice guideline from the AABB. Ann Intern Med 2015;162(03):205–213
13 Dasenbrock HH, Devine CA, Liu KX, et al. Thrombocytopenia and craniotomy for tumor: a national surgical quality improvement program analysis. Cancer 2016;122(11):1708–1717
14 Landon MB, Hauth JC, Leveno KJ, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal and perinatal outcomes associated with a trial of labor after prior cesarean delivery. N Engl J Med 2004;351(25):2581–2589
15 Wetta LA, Szychowski JM, Seals S, Mancuso MS, Biggio JR, Tita AT. Risk factors for uterine atony/postpartum hemorrhage requiring treatment after vaginal delivery. Am J Obstet Gynecol 2013;209(01):51.e1–51.e6
16 Witlin AG, Friedman SA, Sibai BM. The effect of magnesium sulfate therapy on the duration of labor in women with mild preeclampsia at term: a randomized, double-blind, placebo-controlled trial. Am J Obstet Gynecol 1997;176(03):623–627
17 Roberts WE, Perry KG Jr, Woods JB, Files JC, Blake PG, Martin JN Jr. The intrapartum platelet count in patients with HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome: is it predictive of later hemorrhagic complications? Am J Obstet Gynecol 1994;171(03):799–804
18 Webert KE, Mittal R, Sigouin C, Heddle NM, Kelton JG. A retrospective 11-year analysis of obstetric patients with idiopathic thrombocytopenic purpura. Blood 2003;102(13):4306–4311
19 British Committee for Standards in Haematology General Haematology Task Force. Guidelines for the investigation and management of idiopathic thrombocytopenic purpura in adults, children and in pregnancy. Br J Haematol 2003;120(04):574–596
20 Prasertcharoensuk W, Swadpanich U, Lumbiganon P. Accuracy of the blood loss estimation in the third stage of labor. Int J Gynaecol Obstet 2000;71(01):69–70
21 Bose P, Regan F, Paterson-Brown S. Improving the accuracy of estimated blood loss at obstetric haemorrhage using clinical reconstructions. BJOG 2006;113(08):919–924
22 Toledo P, McCarthy RJ, Hewlett BJ, Fitzgerald PC, Wong CA. The accuracy of blood loss estimation after simulated vaginal delivery. Anesth Analg 2007;105(06):1736–1740
23 Patel A, Goudar SS, Geller SE, et al. Drape estimation vs. visual assessment for estimating postpartum hemorrhage. Int J Gynaecol Obstet 2006;93(03):220–224