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Peripartum life-threatening haemorrhage



Peripartum life-threatening haemorrhage

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CZECH REPUBLIC
DEVELOPMENT COOPERATION



What is postpartum haemorrhage?

Postpartum haemorrhage (PPH) is the leading cause of maternal death worldwide.

Postpartum haemorrhage (PPH) is commonly defined as a blood loss of 500 ml or more within 24 hours after birth. It affects about 5% of all women giving birth around the world.

Globally, nearly one quarter of all maternal deaths are associated with PPH. In most low-income countries, it is the main cause of maternal mortality.

The majority of PPH-associated deaths could be avoided by the use of prophylactic uterotonics during the third stage of labour and appropriate treatment.

Improving health care for women during childbirth to prevent and treat PPH is a necessary step towards achievement of the health targets of the Sustainable Development Goals (SDGs).

99% of all maternal deaths occur in low- and middle-income countries (LMICs).

section 01



Postpartum vs peripartum haemorrhage

- **Antepartum** (affects 2–5% of all pregnancies)¹
 - Placenta previa
 - Placental abruption
 - Trauma
 - Rupture of the uterus
- **Postpartum** (complicates about 5% of all births)²
 - Uterine atony is responsible for the vast majority of postpartum haemorrhage (up to ~70%)³

1. Walfish M, et al. *Br J Anaesth* 2009;103:i47–56;

2. WHO PPH guidelines 2018. <https://www.who.int/reproductivehealth/uterotonics-for-PPH-prevention-slidedoc.pptx?ua=1>;

3. Evensen A, et al. *Am Fam Physician* 2017;95:442–9

Table 5. Four T's Mnemonic for the Specific Causes of Postpartum Hemorrhage

<i>Pathology</i>	<i>Specific cause</i>	<i>Approximate incidence (%)</i>
Tone	Atonic uterus	70
Trauma	Lacerations, hematomas, inversion, rupture	20
Tissue	Retained tissue, invasive placenta	10
Thrombin	Coagulopathies	1

Management of severe perioperative bleeding: guidelines from the European Society of Anaesthesiology

First update 2016

Sibylle A. Kozek-Langenecker, Aamer B. Ahmed, Asash Alshari, Pierre Albaladejo, Cesar Aldecoa, Guidrius Barauskas, Edoardo De Robertis, David Faraoni, Daniela C. Filipescu, Dietmar Fries, Thorsten Haas, Matthias Jacob, Marcus D. Lancé, Juan V.L. Pitarich, Susan Mallett, Jens Meier, Zsolt L. Molnar, Nils Rahe-Meyer, Charles M. Samama, Jakob Stensballe, Philippe J.F. Van der Linden, Anne J. Willeke, Patrick Wouters, Piet Wyffels and Kai Zacharowski

1.8.3. Obstetric bleeding

We recommend that **peripartum haemorrhage (PPH)** should be managed by a multidisciplinary team. **1C**

We recommended the use of an escalating PPH management protocol including uterotonic drugs, surgical and/or endovascular interventions and procoagulant drugs. **1B**

Risk awareness and early recognition of severe PPH are essential. **C**

DIAGNOSIS AND THERAPY OF LIFE-THREATENING PERIPARTUM HAEMORRHAGE: CZECH-SLOVAK INTERDISCIPLINARY GUIDELINES

A. Parizek^a, T. Binder^{a,b}, J. Blaha^a, J. Blahy^a, M. Bursik^a, J. Feyereis^a, P. Janik^a, Z. Kokedova^a, P. Kreplak^a, J. Kvesnicka^a, M. Lubusky^a, D. Sedkova^a, O. Simetka^a, P. Stourac^a, V. Cerny^{a,b}

Consensus interdisciplinary guidelines for the prevention and treatment of postpartum haemorrhage

Terminology

The term 'peripartum haemorrhage' is widely used in international literature (including World Health Organization guidelines) to describe bleeding conditions related to delivery, and comprises bleeding conditions **before, during and after delivery**. Postpartum haemorrhage, i.e. bleeding after delivery, is the most common form of peripartum haemorrhage.¹ Based on the consensus of the guidelines workgroup, the abbreviation 'PPH' may be used for both 'peripartum haemorrhage' and 'postpartum haemorrhage'. Bleeding conditions during pregnancy and delivery that are life-threatening for the mother are described as 'life-threatening peripartum haemorrhage' (LTPPH).

1. Kozek-Langenecker SA, et al. Eur J Anaesthesiol 2017;34:332-95 2. Palfalk A, et al. Ces Gynecol 2018;80:150-7

What is postpartum haemorrhage?

**Blood loss 500 mL
after birth**

Postpartum haemorrhage vs. **severe** haemorrhage in pregnancy?

**definition of PPH
as blood loss > 500 ml
was used in 29% of studies**

... but also 700 ml, 800 ml, 1000 ml, 1500 ml



A comparison of published studies on PPH outcomes showed that although blood loss ≥ 500 ml was the most common primary inclusion criterion in randomized controlled trials, this definition was used in only 29% of studies [51,54]. However, other randomized trials investigating PPH treatments have used a range of blood loss volumes: 700 ml [55,56], 800 ml [57], 1000 ml [58], or 1500 ml [59]. In contrast, in systematic reviews, blood loss ≥ 1000 ml was the most often preferred definition of PPH, but was reported in only 13% of studies [51]. The preference for 500 ml in trials but 1000 ml in systematic reviews likely reflects the desire of study authors to maximize statistical power versus the preference of reviewers to primarily evaluate clinically relevant outcomes and outcomes associated more closely with maternal morbidity.

**On the contrary, in systematic reviews blood
loss of >1000 ml was most often used, but
even so only in 13% of these reviews!**



Changes in pregnancy

CARDIOVASCULAR SYSTEM EXPANDS

* HIGH VOLUME STATE *

BLOOD VOLUME INCREASES by 30-50%

PHYSIOLOGIC ANEMIA of PREGNANCY

(% of RBCs) HEMATOCRIT GOES DOWN



Wikimedia Commons, Pregnancy, https://commons.wikimedia.org/wiki/File:Pregnancy_vector.svg (Accessed 18 January 2021)



Prevention and Management of Postpartum Haemorrhage

Green-top Guideline No. 52
December 2016

Read the full paper at https://www.rcog.org.uk/~/media/rcogmedia/documents/guidelines/GTG52_PPH.pdf

1. Purpose and scope

Primary postpartum haemorrhage (PPH) is the most common form of major obstetric haemorrhage. The traditional definition of primary PPH is the loss of 500 ml or more of blood from the genital tract within 24 hours of the birth of a baby.¹ PPHs can be minor (500–1000 ml) or major (more than 1000 ml). Major can be further subdivided into moderate (1001–2000 ml) and severe (more than 2000 ml). In women with lower body mass (e.g. less than 60 kg), a lower level of blood loss may be clinically significant. Recommendations in this guideline apply to women experiencing a primary PPH of 500 ml or more.

Secondary PPH is defined as abnormal or excessive bleeding from the genital tract occurring more than 24 hours postnatally.² This guideline also includes recommendations for the management of secondary PPH.

Minor PPH 500–1000 ml
or major PPH >1000 ml
(moderate 1001–2000 ml or
severe >2000 ml)¹

How should PPH be managed?

Identification of the severity of haemorrhage

Clinicians should be aware that the visual estimation of peripartum blood loss is inaccurate and that clinical signs and symptoms should be included in the assessment of PPH. [New 2016]

Communication and multidisciplinary care

Communication with the woman

Communication with the patient and her birthing partner is important, and clear information of what is happening should be given from the outset. [New 2016]

Who should be informed when the woman presents with PPH?

Relevant staff with an appropriate level of expertise should be alerted of PPH. [New 2016]

The midwife in charge and the first-line obstetric and anaesthetic staff should be alerted when women present with minor PPH (blood loss 500–1000 ml) without clinical shock.

A multidisciplinary team involving senior members of staff should be summoned to attend to women with major PPH (blood loss of more than 1000 ml) and ongoing bleeding or clinical shock.

Proper Estimation of Blood Loss on Scene of Trauma: Tool or Tale?

Matthias Frank, MD, Uli Schmucker, MD, Dirk Stengel, MD, PhD, Lutz Fischer, MD, Joern Lange, MD, Rico Grossjohann, Dipl. Phys., Axel Ekkernkamp, MD, PhD, and Gerrit Matthes, MD, PhD

(J Trauma. 2010;69: 1191–1195)

The Journal of TRAUMA® Injury, Infection, and Critical Care • Volume 69, Number 5, November 2010 Proper Estimation of Blood Loss on Scene of Trauma

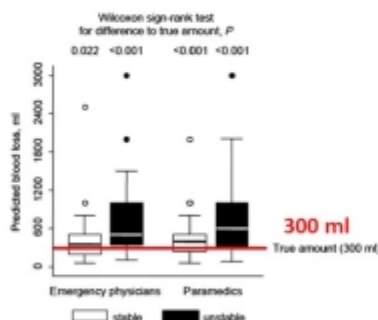


Figure 1. Estimation of blood loss by emergency physicians and paramedics for stable and unstable patients. Given *p* values indicate the statistical significance of the difference to the actual blood loss of 300 mL.

Frank M, et al. J Trauma 2010;69:1191–5

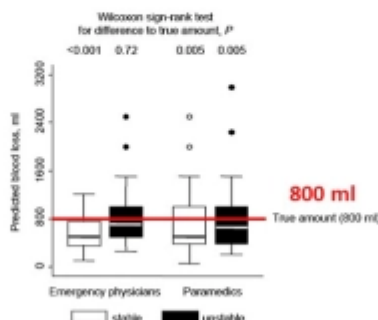


Figure 2. Estimation of blood loss by emergency physicians and paramedics for stable and unstable patients. Given *p* values indicate the statistical significance of the difference to the actual blood loss of 800 mL.

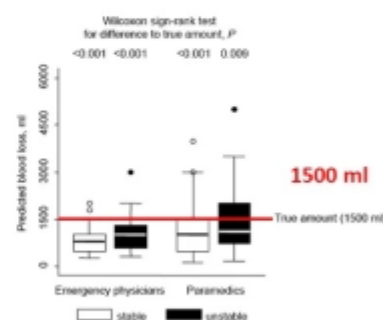


Figure 3. Estimation of blood loss by emergency physicians and paramedics for stable and unstable patients. Given *p* values indicate the statistical significance of the difference to the actual blood loss of 1,500 mL.

International Journal of Gynecology and Obstetrics (2006) 93, 220–224

Drape estimation vs. visual assessment for estimating postpartum hemorrhage

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^d University of Missouri at Kansas City School of Medicine, Kansas City, MO, USA

Abstract

Objective: To compare (1) visual estimation of postpartum blood loss with estimation using a specifically designed blood collection drape and (2) the drape estimate with a measurement of blood loss by photospectrometry. **Methods:** A randomized controlled study was performed with 123 women delivered at the District Hospital, Belgaum, India. The women were randomized to visual or drape estimation of blood loss. A subsample of 10 drape estimates was compared with photospectrometry results. **Results:** The visual estimate of blood loss was 33% less than the drape estimate. The interclass correlation of the drape estimate to photospectrometry measurement was 0.92. **Conclusion:** Drape estimation of blood loss is more accurate than visual estimation and may have particular utility in the developing world. Prompt detection of postpartum hemorrhage may reduce maternal morbidity and mortality in low-resource settings.

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Figure 1. The Blood-Drape, a specially designed blood collection drape with a calibrated collection pouch.

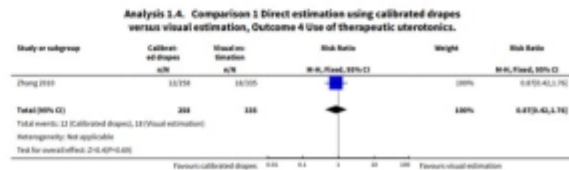
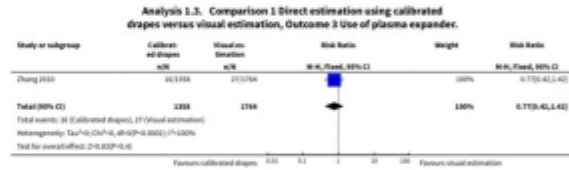
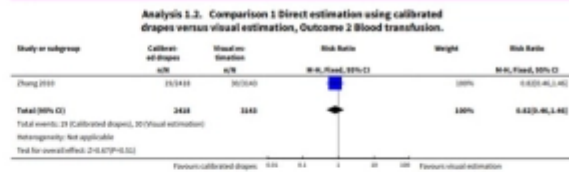
Table 1 Distribution of blood loss between study groups

	Visual group, <i>n</i> = 61	Drape group, <i>n</i> = 62
Blood loss, mean ± S.D. (range) (mL)	203.11 ± 147.49 (50–950)*	302.82 ± 173.28 (50–975)

**P* = 0.0008.

SD, standard deviation

Patel A, et al. Int J Gynecol Obstet 2006;93:220–4



CI, confidence interval; RR, Mantel-Haenszel
Diaz V, et al. Cochrane Database Syst Rev 2018;9:CD010980

Characteristics of life-threatening bleeding

1. Loss of a certain volume of blood per time unit, e.g.:1,2

- I. Loss of whole blood volume within 24 hours (equivalent to about 10 erythrocyte transfusion units in an adult) or
- II. Loss of 50% of blood volume within 3 hours, or
- III. Ongoing blood loss in excess of 150 mL/min

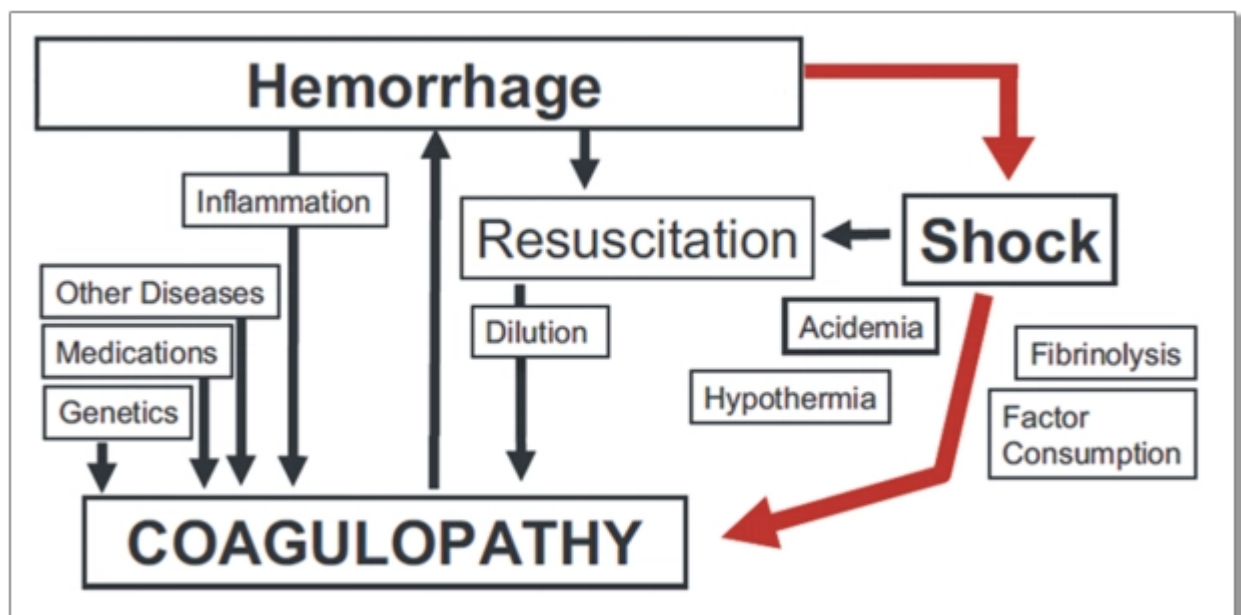
2. Blood loss at the site leading to a threat to vital functions (e.g. bleeding into the CNS)¹

3. Presence of clinical/laboratory signs of tissue hypoperfusion during bleeding²

CNS, central nervous system

1. JPAC Transfusion Handbook. <https://www.transfusionguidelines.org/transfusion-handbook/7-effective-transfusion-in-surgery-and-critical-care/7-3-transfusion-management-of-major-haemorrhage>

2. Kaur P, et al. J Emerg Trauma Shock 2011; 4:103–8



Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial



Published Online
April 26, 2017
[http://dx.doi.org/10.1016/S0140-6736\(17\)30638-4](http://dx.doi.org/10.1016/S0140-6736(17)30638-4)

WOMAN Trial Collaborators*

Summary

Background Post-partum haemorrhage is the leading cause of maternal death worldwide. Early administration of tranexamic acid reduces deaths due to bleeding in trauma patients. We aimed to assess the effects of early administration of tranexamic acid on death, hysterectomy, and other relevant outcomes in women with post-partum haemorrhage.

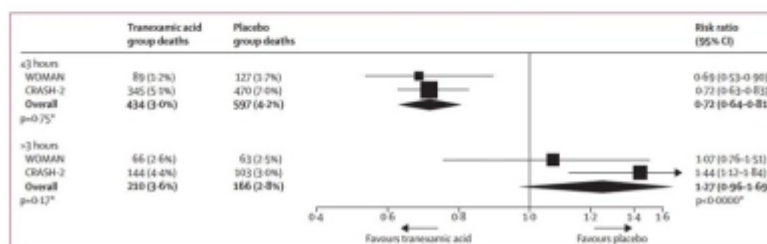


Figure 5: Time to treatment

*Heterogeneity p value.



RESEARCH

Open Access

Estimation of plasma fibrinogen levels based on hemoglobin, base excess and Injury Severity Score upon emergency room admission

Christoph J Schlump¹, Wolfgang Voelckel², Kenji Inaba³, Marc Maegele⁴, Martin Ponschab⁵ and Herbert Schöchl^{1,2*}

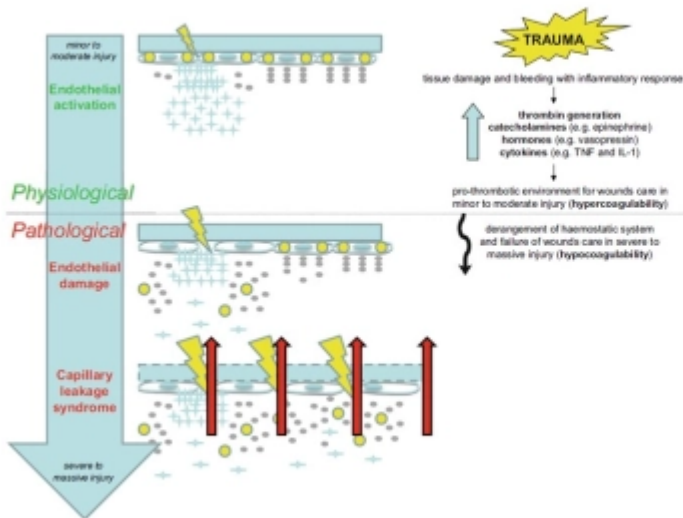
Table 1 Patient demographics stratified according to Injury Severity Score

	ISS 16-24	ISS 25-34	ISS 35-44	ISS 45-54	ISS 55-64	ISS 65-74
Patients n (% of total)	242 (35.9%)	249 (36.9%)	97 (14.6%)	66 (9.9%)	42 (6.3%)	114 (17.3%)
Age (range)	42.5 (26-56)	47 (29.5-60)ns	45 (24-59)ns	45 (24-59)ns	45 (24-59)ns	45 (24-59)ns
Male n (% of ISS group)	200 (82.6%)	195 (78.3%)ns	76 (78.4%)ns	66 (75.9%)ns	42 (48.3%)**	114 (69.1%)ns
Mortality n (% of ISS group)	5 (2.1%)	32 (12.9%)***	27 (27.8%)	27 (40.9%)	27 (64.3%)	27 (23.7%)
FIB mg/dL (IQR)	235 (182.5-286.3)	191.0 (143-240)***	134 (94.5-151)***	114 (69-156)*	114 (69-156)*	114 (69-156)*
Hb g/dL (IQR)	13.0 (11.7-14.2)	12.3 (10.6-13.7)***	10.9 (8.4-13.0)***	9.6 (7.0-11.6)**	9.6 (7.0-11.6)**	9.6 (7.0-11.6)**
BE mmol/L (IQR)	-2.6 (-4.5 to -1.4)	-3.7 (-6.2 to -2.1)***	-5.7 (-8.5 to -2.9)***	-7.1 (-10.9 to -4.6)**	-7.1 (-10.9 to -4.6)**	-7.1 (-10.9 to -4.6)**

ns, not significant; *P < 0.05, **P < 0.01, ***P < 0.001, significance values for comparison with previous hemoglobin; IQR, interquartile range; ISS, Injury Severity Score.

**Hb <100 g/l
= fibrinogen <150 g/l**

**BE >6 mmol/l
= fibrinogen <150 g/l**



The current understanding of trauma-induced coagulopathy (TIC): a focused review on pathophysiology

Nicola Giordano¹, Luca Spies², Elena Campese³, Paolo Stalder⁴
¹Department of Emergency Medicine, University of Medicine, University of Padua, Padua, Italy

Fig. 3 Evolution from progressive endothelial reaction (from physiological activation to pathological damage and capillary leakage) to progressive traumatic damage (from minor to massive injury, indicated with a thunderbolt in the figure). When trauma occurs, tissue damage and bleeding activate the inflammatory response with the generation of greater amounts of thrombin, catecholamines, hormones and cytokines. The aim is to create a pro-thrombotic environment with hypercoagulability capacities that are vital to restore endothelial function after minor-to-moderate injuries. In case of severe-to-massive injuries, a derangement of the haemostatic system takes place resulting in the failure of healing capacities. The final stage is the capillary leakage syndrome with excessive vascular permeability (red arrow in the figure) that leads to edema, hypovolemia and hypotension. DIC disseminated intravascular coagulation, ACoTS acute coagulopathy induced by trauma and shock, FDPs fibrinogen degradation products, TM thrombomodulin, EPCR endothelial protein C receptor, APC activated protein C, EGL endothelial glycocalyx layer, Syn1 syndecan-1, HA hyaluronic acid, HS heparan sulfate, CS chondroitin sulfate, WPIs weibel-palade bodies, tPA tissue plasminogen activator, Ang2 angiotensin-2, PAR1 protease activated receptor 1, TM thrombomodulin, APC activated protein C, NO nitric oxide, PGI₂ prostaglandin I₂, tPA tissue plasminogen activator. → directly leads to, - - - - - inhibits, ~~~~ indirectly leads to and → higher/lower levels of

Therapeutic approach



Figure 3. Therapeutic approach. Patients presenting with severe tissue injury without shock are at high risk of thrombosis and every effort should be made to prevent this serious condition. However, shock and hypoperfusion in patients with severe trauma can induce hypocoagulability and hyperfibrinolysis. This state is associated with very high mortality and needs to be treated urgently with early hemostatic goal-directed resuscitation. Response to trauma is a complex, dynamic process in which risk can shift from bleeding to thrombosis depending on the injury pattern, treatment administered, individual responses, and comorbidities.

Dupuis et al. *Anaesth Analg* 2020;130:684-94

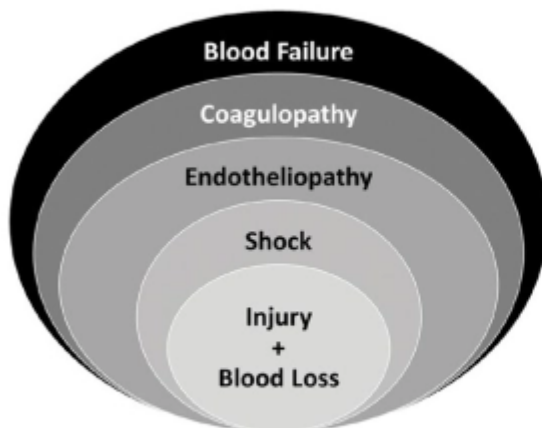


Figure 1. Schematic representing the components of hemorrhagic blood failure.



Figure 2. Schematic of key linkages between oxygen debt, cellular dysfunction, and coagulopathy during hemorrhagic blood failure.

Blood Product	PRBC	Plasma	Cryo	Platelets	Whole Blood
Oxygen Debt (Oxygen Content, Cardiac Output and Delivery)					
Endotheliopathy (Glycocalyx, Proteolysis, Barrier)					
Coagulopathy (Proteolysis, Factors, Clot Formation)					

Figure 3. Schematic summarizing the effects of individual blood products on the three components of hemorrhagic blood failure. PRBC= packed red blood cells, Cryo= cryoprecipitate

Trauma Acute Care Surg. 2017 June; 62(3): 543-548

Factors determining the severity of bleeding^{1,2}

- Rate and/or total amount of blood loss
- Primary cause/source of bleeding
- State of the coagulation system
- Number of units of blood products and/or blood derivatives
- Presence of clinical and/or laboratory signs of tissue hypoperfusion and/or signs of organ dysfunction

1. Pařízek A, et al. *Čes Gynek* 2018;83:150–7;

2. Blatný J, Blaha J, et al. *ANESTEZIOLOGIE A INTENZIVNI MEDICINA* 2017;28(4):103–8

EJA Eur J Anaesthesiol 2017; 34:332–395

GUIDELINES

Management of severe perioperative bleeding: guidelines from the European Society of Anaesthesiology

First update 2016

Sibylle A. Kozek-Langenecker, Aamer B. Ahmed, Arash Afshari, Pierre Albaladejo, Cesar Aldecoa, Guidrius Barauskas, Edoardo De Robertis, David Faraoni, Daniela C. Filipescu, Dietmar Fries, Thorsten Haas, Matthias Jacob, Marcus D. Lancé, Juan V.L. Pitarich, Susan Mallett, Jens Meier, Zsólt L. Molnar, Niels Rahe-Meyer, Charles M. Samama, Jakob Stensballe, Philippe J.F. Van der Linden, Anne J. Wikkelsø, Patrick Wouters, Piet Wyffels and Kai Zacharowski

1.8.3. Obstetric bleeding

We recommend that peripartum haemorrhage (PPH) should be managed by a multidisciplinary team. **1C**

We recommended the use of an escalating PPH management protocol including uterotonic drugs, surgical and/or ~~endovascular interventions and procoagulant drugs.~~ **1B**

Risk awareness and early recognition of severe PPH are essential. **C**

California **M E D I C I N E**

OFFICIAL JOURNAL OF THE CALIFORNIA MEDICAL ASSOCIATION © 1957, by the California Medical Association

Volume 87 OCTOBER 1957 Number 4

Modern Treatment of Abruption Placentae

JAMES A. MERRILL, M.D., San Francisco

HEMORRHAGE CONTINUES to be one of the major causes of maternal death. Deaths due to infection have decreased sharply but there has been much less change in the proportionate number of deaths due to other members of the classical triad—toxemia and hemorrhage. The work of several maternal welfare committees indicates that probably 75 per cent of hemorrhagic deaths are preventable. Therefore, in order to reduce maternal mortality farther, the prevention, control and treatment of hemorrhage must

75% of hemorrhagic deaths are preventable

of abruption placentae that causes death, the mortality rate associated with this grade approximates 10 per cent. Fortunately, the severe form is not common, occurring in about 15 per cent of cases of premature separation. Often the only evidence of premature separation is that found by pathologic examination of the placenta. Moreover, diagnosis and treatment of abruption placentae are frequently linked with another complication of pregnancy—toxemia.

From the Department of Obstetrics and Gynecology, University of California School of Medicine, San Francisco 22.
Presented before the Society on Obstetrics and Gynecology at the 80th Annual Session of the California Medical Association, Los Angeles, April 24 to May 3, 1957.

SYSTEMIC EFFECTS OF ABRUPTIO PLACENTAE

Various lines of investigation have shown the severe grade of premature separation of the placenta to be accompanied by systemic effects, some of which are potentially lethal, and which include:

1. Clinical shock, sometimes out of proportion to blood loss or hypotension.
2. Disseminated deposition of fibrin.
3. An *in vivo* defibrination of the blood with a decrease or absence of fibrinogen, sometimes resulting in incoagulable blood.
4. Ischemia of the renal cortex, leading to varying degrees of necrosis.
5. Activation of a fibrinolysin in the plasma.



the period from 1993 through 2019, the incidence of PPH increased from approximately 8 to 40 per 10 000 deliveries [103]. An increase in high-risk groups such as higher maternal age, obesity, more frequent and serious comorbidities, especially cardiovascular, or increased cesarean section rates cannot fully explain this rise [104,105]. The increasing incidence of PPH suggests an incomplete implementation of guidelines [106–108], resulting in treatment delays or suboptimal care, which are increasingly being reported in 30–90% of PPH cases [109–112]. Moreover, reports from confidential inquiries have shown that as many as 67% of the deaths in the United States and 85% of those in France are avoidable, resulting as they have from either delayed or inadequate treatment [113–115]. Delays in diagnosing and treating PPH are believed to directly affect the severity of bleeding, the development of complications such as coagulopathy, and result in high morbidity and mortality. Delays are reported to be caused by misinterpretation of the extent of blood loss, failure to escalate care, and failure to

treatment delays or suboptimal care in up to 90% of cases

as many as 67% of the deaths in the USA and 85% of those in France are avoidable

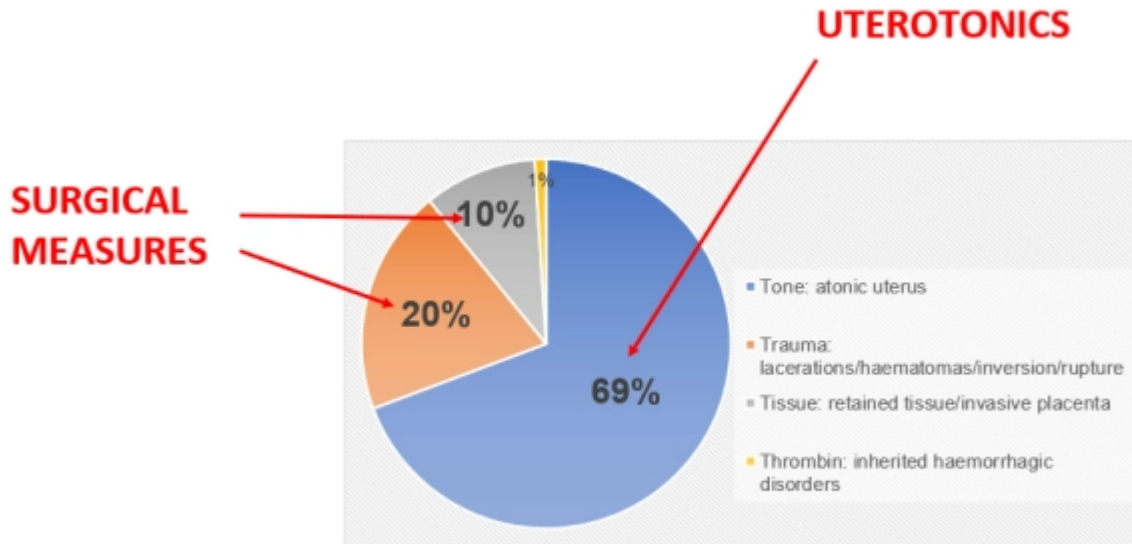
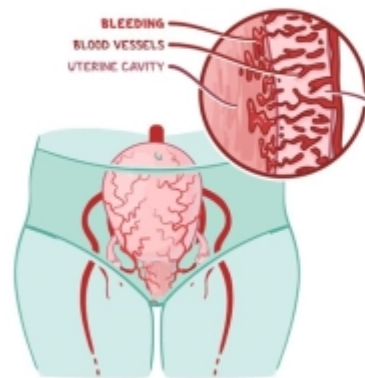


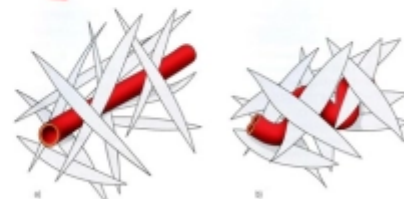
Figure 1. Causes and approximate incidence of PPH



OSMOSIS.org
www.osmosis.org/notes/uterine-artery

BACKGROUND

- FAILURE of the UTERUS to CONTRACT SUFFICIENTLY DURING & AFTER DELIVERY of a BABY
- MYOMETRIUM DOESN'T RESPOND to OXYTOCIN
- MOST COMMON CAUSE of POSTPARTUM HEMORRHAGE (OBSTETRIC EMERGENCY)



myometrial retraction $\Rightarrow$ compression
= tourniquet of the uterine vascular system

Paresh A. Pariparambathil. Obstetrium.org

Received: 5 May 2019 | Revised: 10 September 2019 | Accepted: 9 November 2019 | First published online: 27 December 2019
Wiley Online Library on [behalf of John Wiley & Sons, Inc.] | DOI: 10.1111/1471-2546.15203

Postpartum hemorrhage care bundles to improve adherence to guidelines: A WHO technical consultation¹

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Abstract

Objective: To systematically develop evidence-based bundles for care of postpartum hemorrhage (PPH).

Methods: An international technical consultation was conducted in 2017 to develop draft bundles of clinical interventions for PPH taken from the WHO's 2012 and 2017 PPH recommendations and based on the validated "GRADE Evidence-to-Decision" framework. Twenty-three global maternal-health experts participated in the development process, which was informed by a systematic literature search on bundle definitions, designs, and implementation experiences. Over a 6-month period, the expert panel met online and via teleconferences, culminating in a 2-day in-person meeting.

Results: The consultation led to the definition of two care bundles for facility implementation. The "first response to PPH bundle" comprises uterotonics, isotonic crystalloids, tranexamic acid, and uterine massage. The "response to refractory PPH bundle" comprises compressive measures (aortic or binaural uterine compression), the non-pneumatic antishock garment, and intrauterine balloon tamponade (IBT). Advocacy, training, teamwork, communication, and use of best clinical practices were defined as PPH bundle supporting elements.

Conclusion: For the first response bundle, further research should assess its feasibility, acceptability, and effectiveness; and identify optimal implementation strategies. For the response to refractory bundle, further research should address pending controversies, including the operational definition of refractory PPH and effectiveness of IBT devices.

First response PPH bundle

TABLE 2 Description of WHO-recommended clinical interventions for PPH, 2012–2017

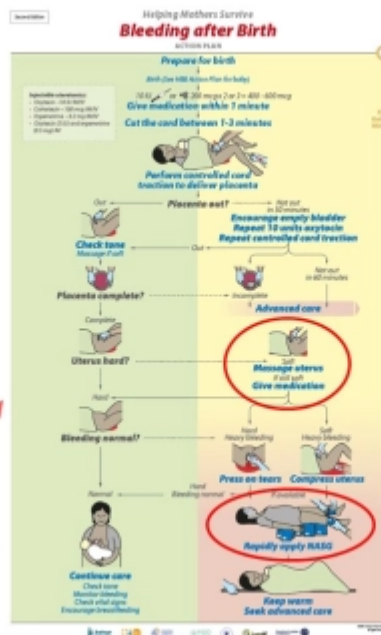
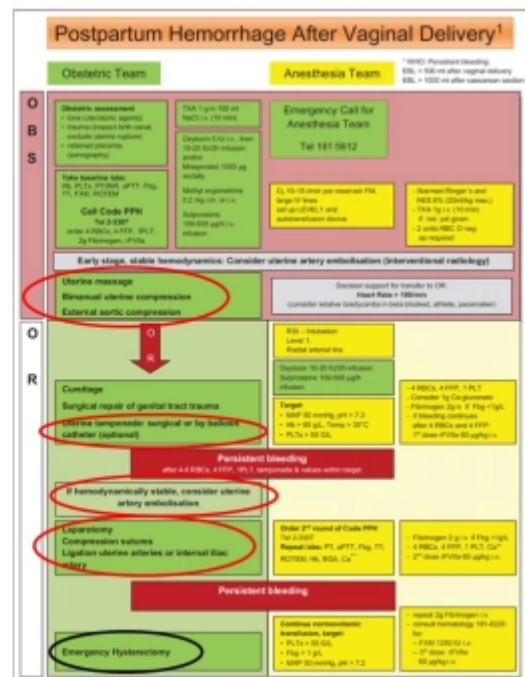
Intervention	Description
Uterotonics	Administration of oxytocin (OTM) ergometrine/methylergometrine or other first-line combination of ergometrine and ergometrine (ME) independent (ind). The preferred drug for prevention of PPH is oxytocin (20 IU/100 mL) if available, give IM ergometrine/methylergometrine or the first-line combination of oxytocin and ergometrine, if not contraindicated. If IM or IV uterotonics are unavailable, give oral misoprostol 800 µg.
Controlled cord traction	After delivery of the newborn and it is assessed that there are no other factors in play, gentle traction is applied to the umbilical cord with one hand, while the other hand applies abdominal counter-pressure on the uterus.
Postpartum aortic/binaural uterine compression	Palpate the uterus to assess uterine firmness/tone. If the uterus is soft or flabby this may indicate uterine atony.
Isotonic crystalloids	Administration of a starting dose 1000 mL of isotonic crystalloids (C) in 30 min and continuing doses of 1000 mL of isotonic crystalloids (C) in 30 min.
Tx	A fixed dose of 1 g of TXA (100 mg/mL IV at 1 mL per min) within 3 h of the time of diagnosis of unknown, time of delivery, a second dose of 1 g can be given if needed 30 min after the first dose.
Uterine massage	Circular rubbing of the uterus achieved by manual squeezing of the abdomen. This is typically sustained until the bleeding stops or the uterus contracts.
Intrauterine balloon tamponade	The procedure entails insertion of a deflated/evacuated balloon into the uterine cavity and then inflating it to achieve a tamponade effect.
Binaural uterine compression	Two-handed one in the anterior vaginal fornix and one behind the uterine fornix, squeezing the uterus between the hands.
External aortic compression	External compression applied with a closed fist at the level of the umbilicus and slightly to the woman's left.
NASC	Used as a temporary measure until source of bleeding found and treated. NASC is a woven body compression device made of stretchy material which is placed tightly with elastic in segments for the ankles, calves, thighs, pelvis, and abdomen and is applied rapidly starting at the ankles.
A single dose of antihemorrhagics	In the context of placental retention, the placenta should be extracted, and a single dose of antihemorrhagics administered.
Uterine artery embolization	If other measures have failed and if the necessary resources are available, the use of uterine artery embolization is recommended as a treatment for PPH due to uterine atony.
Surgical intervention	If bleeding persists despite treatment with uterotonics, drugs and other conservative interventions, surgical intervention should be used without further delay.

Abbreviations: IM, intramuscular; IV, intravenous; NASC, non-pneumatic antishock garment; PPH, postpartum hemorrhage; TXA, tranexamic acid.

Response to refractory PPH bundle

Giuseppe Colucci, MD^{1,2}, Karin Helsing, MD¹,
Franziska Demarmels Biasiutti, MD¹, Luigi Raio, MD³, Pirmin Schmid, MD¹,
Dimitrios A. Tsakiris, MD⁴, Balthasar Eberle, MD⁵, Daniel Surbek, MD³,
Bernhard Lämmle, MD^{1,4}, and Lorenzo Alberio, MD^{1,7}

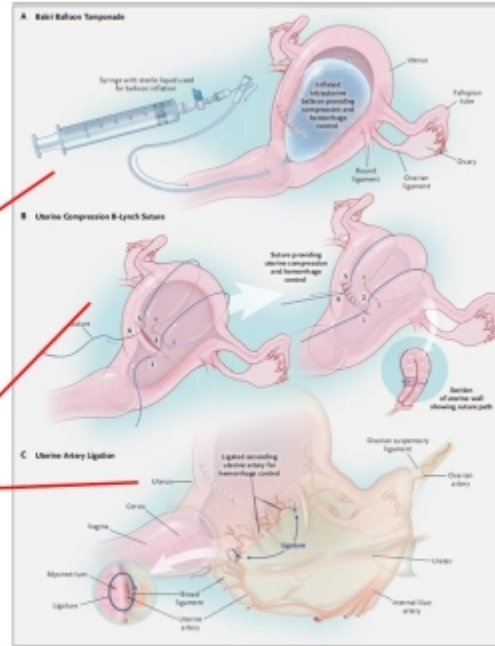
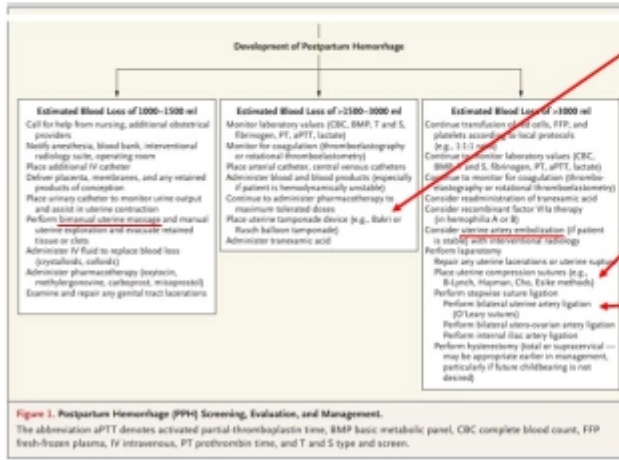
Severe postpartum hemorrhage (PPH) is an obstetric emergency that needs prompt and effective therapy to reduce the risk of complications. In this study, women who developed PPH (study cohort, $n = 27$) were treated according to a standardized management protocol including uterine massage, uterine tamponade, and intravenous administration of uterotonic drugs. The primary outcome was blood loss. Secondary outcome was the need for transfusion. This study was compared to patients treated with different strategies during 2 preceding periods: an in-house guideline applying the management of PPHs (historical cohort 1, $n = 30$) and no specific guideline (historical cohort 2, $n = 27$). The management protocol was used over 33 months. The study was conducted in a tertiary care center. The results showed that the need for transfusion was lower in the study group ($P = .046$), and platelet concentrations ($P = .033$) compared to historical cohort 1 and historical cohort 2, respectively. The necessity of emergency postpartum hysterectomy was lower in the study group ($P = .012$). In conclusion, in patients with severe PPH, the use of a standardized management protocol was associated with a decreased requirement of blood products and lower need to proceed to emergency postpartum hysterectomy.



REVIEW ARTICLE

N Engl J Med 2023;384:1635-45. DOI: 10.1056/NEJMra231247

Postpartum Hemorrhage

Jessica L. Bienstock, M.D., M.P.H., Ahizechukwu C. Eke, M.D., Ph.D.,
and Nancy A. Hueppchen, M.D.

External aortic compression

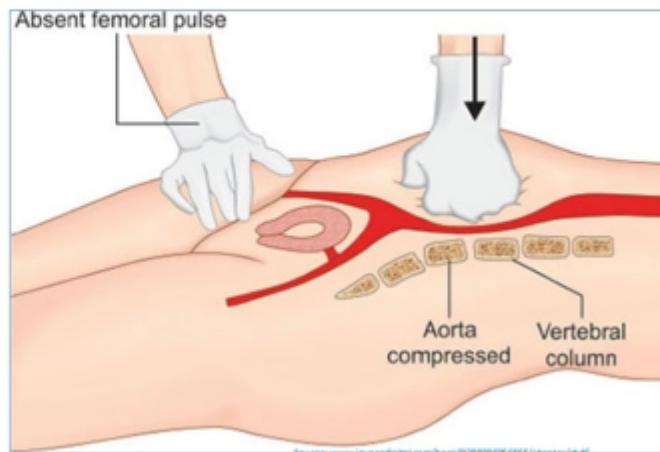
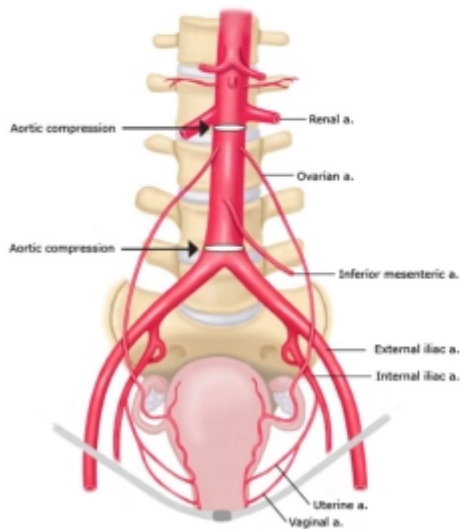
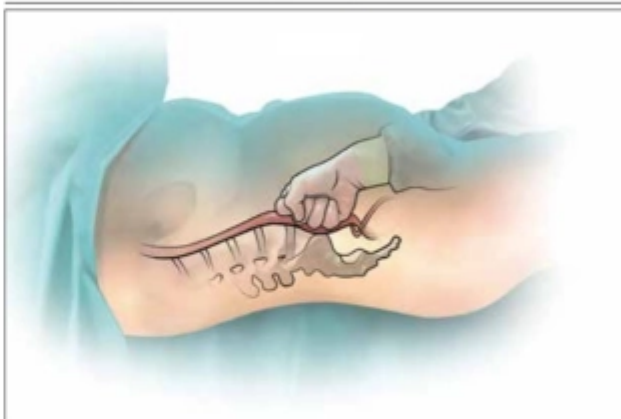




Figure 1. Manual external aortic compression using a knee.

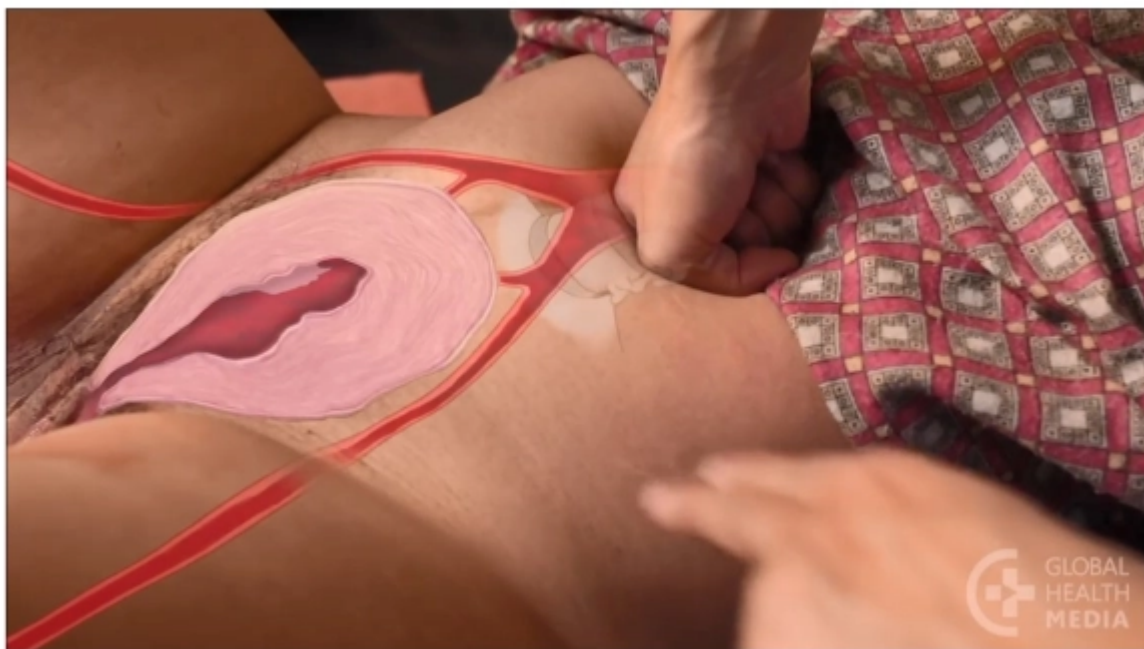
Source: Soepfand et al. *Ann Emerg Med*. 2012 Mar;79(3):287-292

Aortic compression through an open abdominal incision



During cesarean delivery through a low transverse abdominal incision, the surgeon directly applies pressure to the aorta just above the lumbar sacral promontory

Source: Barbieri RJ. *Obstet Manag*. 2016 October;30(3):33-37, 14



Prehospital assessment and management of postpartum haemorrhage- healthcare personnel's experiences and perspectives

Ann-Christin Linqvist Leonardsen^{1,2*}, Ann Karin Helgesen¹, Linn Ulvey^{1,3} and Vigdis Abrahamson Grandahl¹

Abstract

Background: Postpartum hemorrhage (PPH) is a serious obstetric emergency, and one of the top five causes of maternal mortality globally. The most common causes of PPH include uterine atony, placental disorders, birth trauma and coagulation defects. Timely diagnosis and early management are critical to reduce morbidity, the need for blood transfusion or even mortality. External, manual aortic compression (AC) has been suggested as an intervention that reduce PPH and extend time for control of bleeding or resuscitation. This procedure is not commonly utilized by healthcare personnel. The incidence of home-births is increasing, and competence in PPH assessment and management is essential in prehospital personnel. The objective was to explore prehospital personnel's competence in PPH and AC, utilizing different tools.

Methods: The study was conducted in a county in South-eastern Norway, including five ambulance stations. All prehospital personnel ($n = 250$) were invited to participate in a questionnaire study. The questionnaire included the PPH self-efficacy (PPHSE) and PPH collective efficacy (PPHCE) tools, as well as tool developed utilizing the Delphi technique. Descriptive statistics were used to analyze the quantitative data, while qualitative content analysis was used to analyse free-text responses.

Results: A total of 87 prehospital personnel responded to the questionnaire, 57.5% male, mean age 37.9 years. In total, 80.4% were ambulance workers and/or paramedics, and 96.6 and 97.7% respectively reported to need more education or training in PPH. Moreover, 82.8% reported having managed patient(s) with PPH, but only 2.9% had performed AC. Prehospital personnel's responses varied extensively regarding knowledge about what PPH is, how to estimate and handle PPH, and how to perform AC. Mean self-efficacy varied from 3.3 to 5.6, while collective efficacy varied from 1.9 to 3.8.

Conclusions: This study indicates that prehospital personnel lack knowledge about PPH and AC, due to various responses to the developed questionnaire. Even though AC is an acknowledged intervention in PPH, few participants reported that this was utilized. Our findings emphasize the need for education and training in PPH and PPH handling generally, and in AC specifically.

Experience

Prehospital personnel's experience with PPH and AC is shown in Table 4.

Reasons for not using AC were 'lack of education' (74.7%), 'lack of training' (10.3%), 'feel unsecure on the procedure' (10.3%), and 'difficult to cause the patient pain' (4.6%) (fixed response alternatives).

Our findings indicate that prehospital personnel lack knowledge about postpartum hemorrhage (PPH) and manual aortic compression (AC). As much as 82.8% had experienced PPH, but only 2.3% had utilized AC.

Table 4 Experiences with PPH and AC ($n = 87$)

	n (%)
Have experience with PPH	72 (82.8)
Have used AC	2 (2.3)
Have considered using AC	5 (5.7)
Had patients where AC may have been appropriate	
Yes	6 (6.9)
No	70 (79.3)
Undecided	11 (13.8)

AC = external, manual, aortic compression

Injury, Int. J. Care Injured 48 (2017) 26–31

Bi-manual proximal external aortic compression after major abdominal-pelvic trauma and during ambulance transfer: A simulation study

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ABSTRACT

Background: Applying manual pressure after hemorrhage is intuitive, cost-free, and logistically-simple. When direct abdominal-pelvic compression fails, clinicians can attempt indirect proximal-external-aortic-compression (PEAC), while expediting transfer and definitive rescue. This study quantifies the sustainability of simulated bi-manual PEAC both immediately on scene and during subsequent ambulance transfer. The goal is to understand when bi-manual PEAC might be clinically-useful, and when to prioritize compression-devices or endovascular-occlusion.

Methods: We developed a simulated central vessel compression model utilizing a digital scale and Malfman intra-abdominal pressure monitor inside a cardiopulmonary resuscitation mannequin. Twenty prehospital health-care professionals (HCPs) performed simulated bi-manual PEAC (i) while stationary and (ii) inside an 80km/h ambulance on a closed driving track. Participants compressed at "the maximal effort they could maintain for 20 min". Results were measured in monthly applied-pressure and kilograms compression-weight. The Borg scale of perceived exertion was used to assess sustainability, with < 16 regarded as acceptable.

Results: While stationary all participants could maintain 20 min of compressive pressure/weight: within five-persons of their starting effort, and with a Borg-score < 16. Participants applied 88–306 monthly compression pressure: (mean 180mmHg), 16–55 kg compression-weight (mean 33 kg), and 37–68% of their bodyweight (mean 43%). In contrast, participants could not apply consistent or sustained compression in a moving ambulance: Borg Score exceeded 16 in all cases.

Conclusions: Survival following major abdominal-pelvic hemorrhage requires expedited operative/interventional rescue. Firstly, however, we must temporize pre-hospital exsanguination both on scene and during transfer. Despite limitations, our work suggests PEAC is feasible while waiting for, but not during, ambulance-transfer. Accordingly, we propose a chain-of-survival that cautions against over-reliance on manual PEAC, while supporting pre-hospital devices, endovascular occlusion, and expeditious but safe hospital-transfer.

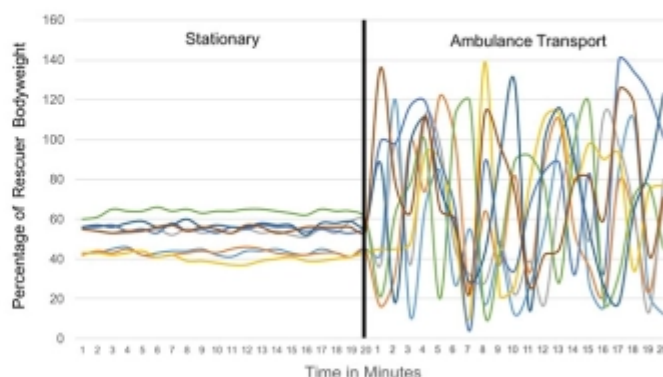


Fig. 3. Combined Stationary and Ambulance Transport Compression.

Resuscitative endovascular balloon occlusion of the aorta: the postpartum haemorrhage perspective

Jostein Radseth Bredt^{1,2,3,4*}, Edmund Savik⁵ and Marius Rehn^{2,6,7}

Keywords: REBOA, Postpartum haemorrhage, PPH, Aortic occlusion

Not only in trauma centres, but also in hospitals with obstetric departments, REBOA should be considered an emergency procedure to be immediately available 24/7 by physicians trained in ultrasound-guided and fluoroscopy-free Seldinger technique. Local considerations will decide whether the REBOA is placed by an emergency physician, anaesthesiologist, obstetricians, interventional radiologist or the general surgeon.

Conclusions

REBOA carries more indications than trauma and should be increasingly considered and evaluated in management of PPH. REBOA may not only save a life, it might also save a uterus.

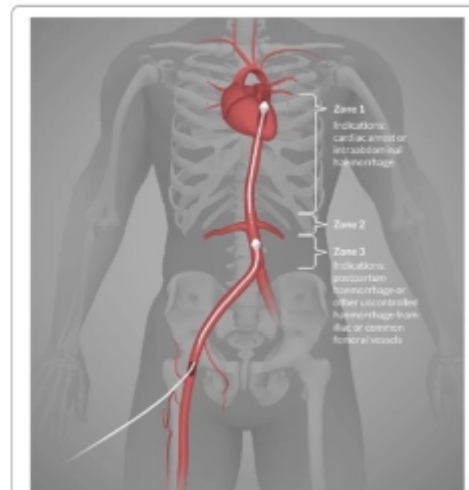
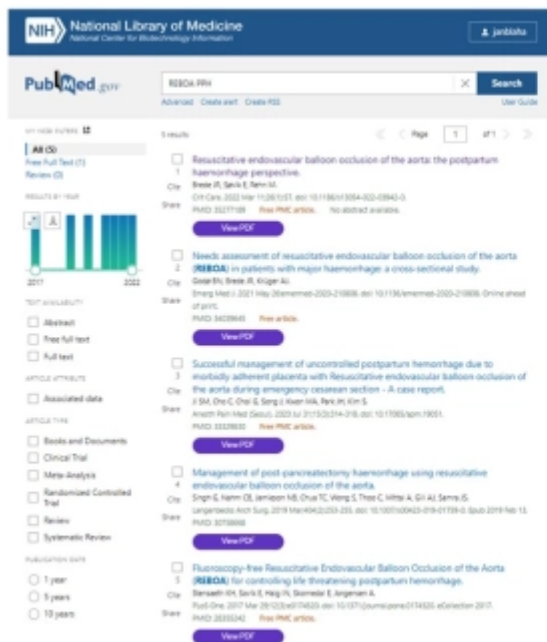


Fig. 1 Aortic zones and indications for occlusion



A systematic review and meta-analysis of the use of resuscitative endovascular balloon occlusion of the aorta in the management of major exsanguination

R. L. S. Berger van der Burg¹, Thijs T. C. F. van Dongen^{1,2}, J. J. Morrison³, P. P. A. Hodeman Joosten³, J. J. DuBois⁴, T. M. Höner⁵, R. Hoencamp^{1,6}

Abstract

Background: Circulatory collapse is a leading cause of mortality among traumatic major exsanguination and in ruptured aortic aneurysm patients. Approximately 40% of patients die before hemorrhage control is achieved. Resuscitative endovascular balloon occlusion of the aorta (REBOA) is an adjunct designed to sustain the circulation until definitive surgical or endovascular repair. A systematic review was conducted for the current clinical use of REBOA in patients with hemorrhagic instability and to discuss its potential role in improving prehospital and in-hospital outcome.

Methods: Systematic review and meta-analysis (1900–2017) using MEDLINE, Cochrane, EMBASE, Web of Science and Central and Embase using the keywords “aortic balloon occlusion”, “aortic balloon tamponade”, “REBOA”, and “Resuscitative Endovascular Balloon Occlusion” in combination with hemorrhage control, hemorrhage, resuscitation, shock, ruptured abdominal or thoracic aortic, endovascular repair, and open repair. Original published studies on human subjects were considered.

Results: A total of 490 studies were identified; 99 met criteria for inclusion. Of the 1436 patients, overall reported mortality was 49.2% (813/1248) with significant differences ($p < 0.001$) between clinical indications. Hemodynamic shock was evident in 79.3%, values between clinical indications showed significant difference ($p < 0.001$). REBOA was favored as treatment in trauma patients in terms of mortality. Pooled analysis demonstrated an increase in mean systolic pressure by almost 50 mmHg following REBOA use.

Conclusion: REBOA has been used in trauma patients and ruptured aortic aneurysm patients with improvement of hemodynamic parameters and outcomes for several decades. Formal, prospective study is warranted to clarify the role of this adjunct in all hemorrhagic unstable patients.

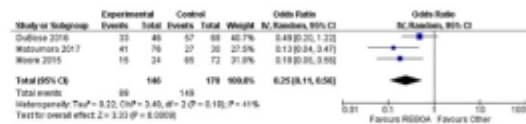


Fig. 2 Meta-analysis of mortality after use of REBOA in trauma. REBOA indicates resuscitative endovascular balloon occlusion of the aorta, IV inverse variance, Random random effect, CI confidence interval, df degrees of freedom, P p value

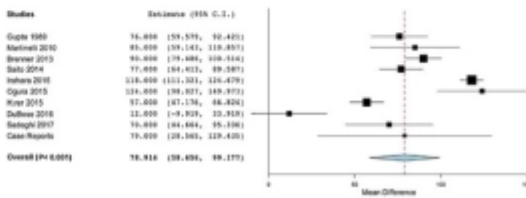


Fig. 3 Meta-analysis of rise in SBP after REBOA use in trauma. SBP indicates systolic blood pressure in mmHg, REBOA resuscitative endovascular balloon occlusion of the aorta, CI confidence interval, P p value

Prevention and Management of Postpartum Haemorrhage

Green-top Guideline No. 52 December 2016

Please cite the paper as: Pheasant E, Allard S, Chandrasekaran E, Collier P, Green L, Hunt BJ, Rios S, Thomson AJ on behalf of the Royal College of Obstetricians and Gynaecologists. Prevention and management of postpartum haemorrhage. BJOG 2016;124:e106–e168.

5.6.2 What surgical treatments can be employed to arrest the bleeding?

If pharmacological measures fail to control the haemorrhage, surgical interventions should be initiated sooner rather than later. D

Intrauterine balloon tamponade is an appropriate first-line 'surgical' intervention for most women where uterine atony is the only or main cause of haemorrhage. C

Conservative surgical interventions may be attempted as second line depending on clinical circumstances and available expertise. C

It is recommended that a laminated diagram of the brace suture technique be kept in theatre. ✓

Resort to hysterectomy sooner rather than later (especially in cases of placenta accreta or uterine rupture). C

Ideally and when feasible, a second experienced clinician should be involved in the decision for hysterectomy. ✓

The use of pharmacological agents other than those detailed should not delay recourse to surgery. Once the decision is made to embark on surgical haemostasis, the most appropriate choice of procedure will depend, in part, on the experience and expertise of available staff.

Compression of the aorta may be a temporary but effective measure to allow time for resuscitation to catch up with the volume replacement and the appropriate surgical support to arrive. The judgement of senior clinicians, taking into account the individual woman's future reproductive aspirations, is required in deciding the appropriate sequence of interventions.

Haemostatic support in postpartum haemorrhage

A review of the literature and expert opinion

Stefan Hofer, Jan Blaha, Peter W. Collins, Anne-Sophie Ducloy-Bouthors, Emilia Guasch, Francesco Labate, Filipe Langa, Lil Trine Nyflet, Kostja Steiner and Marc Van de Velde

Postpartum haemorrhage (PPH) remains the leading cause of pregnancy-related deaths worldwide. Typically, bleeding is controlled by timely obstetric measures in parallel with resuscitation and treatment of coagulopathy. Early recognition of abnormal coagulation is crucial and haemostatic support should be considered simultaneously with other strategies as coagulopathies contribute to the progression to massive haemorrhage. However, there is lack of agreement on important topics in the current guidelines for management of PPH. A clinical definition of PPH is paramount to understand the situation to which the treatment recommendations relate; however, reaching a consensus has previously proven difficult. Traditional definitions are based on volume of blood loss, which is difficult to monitor, can be misleading and leads to treatment delay. A multidisciplinary approach to define PPH considering vital signs, clinical symptoms, coagulation and haemodynamic changes is needed. Moreover,

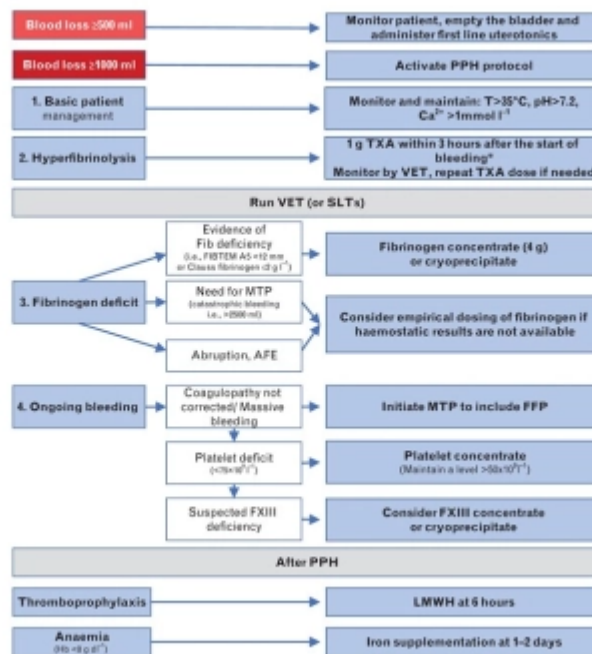
standardised algorithms or massive haemorrhage protocols should be developed to reduce the risk of morbidity and mortality and improve overall clinical outcomes in PPH. If available, point-of-care testing should be used to guide goal-directed haemostatic treatment. Tranexamic acid should be administered as soon as abnormal bleeding is recognised. Fibrinogen concentrate rather than fresh frozen plasma should be administered to restore haemostasis where there is elevated risk of fibrinogen deficiency (e.g., in catastrophic bleeding or in cases of abruption or amniotic fluid embolism) as it is a more concentrated source of fibrinogen. Lastly, organisational considerations are equally as important as clinical interventions in the management of PPH and have the potential to improve patient outcomes.

Published 2022

Haemostatic support in postpartum haemorrhage

A review of the literature and expert opinion

Stefan Hofer, Jan Blaha, Peter W. Collins, Anne-Sophie Ducloy-Bouthors, Emilia Guasch, Francesco Labate, Filipe Langa, Lil Trine Nyflet, Kostja Steiner and Marc Van de Velde





Column 1	Column 2	Column 3	Column 4	Column 5	Column 6	Column 7	Column 8	Column 9	Column 10
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41	42	43	44	45	46	47	48	49	50
51	52	53	54	55	56	57	58	59	60
61	62	63	64	65	66	67	68	69	70
71	72	73	74	75	76	77	78	79	80
81	82	83	84	85	86	87	88	89	90
91	92	93	94	95	96	97	98	99	100



First aid—Medical care in Okinawa Beach, June 1944. Note the absence of a drip.



Vietnam, 1968



Iraq, 2007

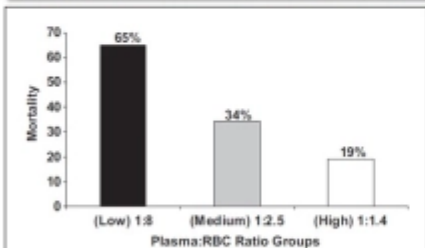
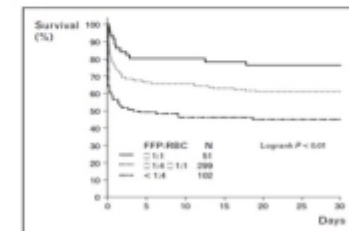


Fig. 1. Percentage mortality associated with low, medium, and high plasma to RBC ratios transfused at admission. Ratios are median ratios per group and include units of fresh whole blood counted both as plasma and RBCs.

Bugman et al. / Trauma 2007;63:805-813



Figure 1 Survival curves for each category of fresh frozen plasma:packed red blood cell ratio



The lowest mortality was observed in the category of patients who received a ratio of fresh frozen plasma:packed red blood cells (FFP:PRBCs) equal to or greater than 1:1. Most of the separation of the survival curves occurred immediately after the injury. Data from [16].

Colfax et al. Current Opinion in Anaesthesiology 2005;23:243-248

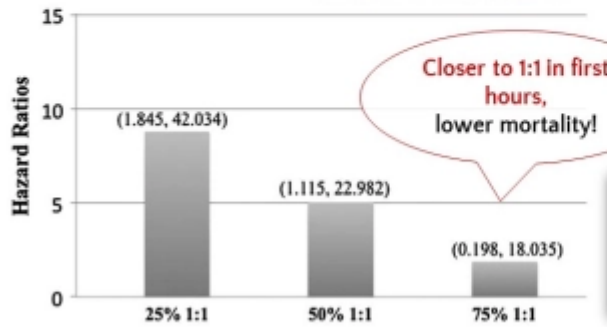
Time matters in 1:1 resuscitations: Concurrent administration of blood:plasma and risk of death

Stephanie A. Savage, MD, Ben L. Zarzaur, MD, Martin A. Croce, MD, and Timothy C. Fabian, MD, Memphis, Tennessee

J Trauma Acute Care Surg 2014; Volume 77, Number 6

Hazard Ratios for Mortality in CAT+ Patients

Critical Administration Threshold = 3 TU of blood/hour



Transfusion Medicine Reviews 10 (2000) 14-15

Optimal Dose, Timing and Ratio of Blood Products in Massive Transfusion: Results from a Systematic Review

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ARTICLE INFO

Available online 6 July 2017

Keywords:

Massive transfusion

Transfusion

Plasma

Red blood cells

Platelets

Thrombocytopenia

Transfusion

ABSTRACT

Optimal dose, timing and ratio of red blood cells (RBC) of blood component therapy (fresh frozen plasma [FFP], platelets, cryoprecipitate or fibrinogen concentrate) to reduce mortality and mortality in critically bleeding patients requiring massive transfusion is unknown. We performed a systematic review for randomized controlled trials (RCT) in MEDLINE, the Cochrane Library, Embase, CINAHL, PubMed, the Transfusion Evidence Library and using multiple clinical trials registries to 21 February 2017. Studies RCTs were identified, all completed (one in adult trauma patients, one pediatric trauma patients) and two ongoing trials. Of the completed trials, three were feasibility trials, comparing a FFP, platelets and RBC ratio of 1:1:1 to laboratory-guided transfusion practice (n = 105), early cryoprecipitate compared to standard practice (n = 45), and early fibrinogen concentrate compared to placebo (n = 45). One trial compared the effect of FFP, platelets and RBC ratio of 1:1:1 with 1:1:2 on 24-hour and 30-day mortality (n = 180); one compared whole blood to blood component therapy on 24-hour blood use (n = 187); one compared a FFP to RBC ratio of 1:1 with 1:1 (n = 14). Data from two trials were pooled in a meta-analysis for 24-hour mortality because the transfusion ratio achieved were similar. Results from these two trials suggest higher transfusion ratios were associated with modification of more FFP and platelets without evidence of significant difference with respect to mortality or morbidity. On the limited evidence available, there is insufficient evidence to recommend a 1:1:1 over a 1:1:2 ratio or standard care for adult patients with critical bleeding requiring massive transfusion.

Study or Subgroup	Experimental	Control	Risk Ratio	M-H, Random, 95% CI
Roberts 2015	75	138	0.89 (0.65, 1.11)	
Tasmanian 2015	13	48	2.27 (0.30, 17.4)	
Total (95% CI)	219	377	1.28 (0.48, 3.22)	
Total events	85	94		
Heterogeneity: Tau ² = 0.36; Chi ² = 3.95, df = 1, P = 0.046; I ² = 75%				
Test for overall effect: Z = 0.47 (P = 0.64)				

Fig. 2. Forest plot for 24-hour mortality.





35
minutes



8
minutes

East of England Trauma Network
East of England Regional Transfusion Committee

Massive blood loss in adults

≥ 40% loss of total blood volume
4 litres in 24 hours 2 litres in 3 hours > 150ml/min

Get senior help

Contact transfusion laboratory
Transfusion Lab (Ext 54711)
Stop 1111 end of hour

Check patient identification
Check patient name, date of birth, blood group, and Rh status
Check patient allergies, especially to latex, antibiotics, and contrast media
Check patient current and previous blood group and Rh status
Check patient current and previous coagulation status

Assess ABC

IV access

Check patient identification
Check patient name, date of birth, blood group, and Rh status
Check patient allergies, especially to latex, antibiotics, and contrast media
Check patient current and previous blood group and Rh status
Check patient current and previous coagulation status

Resuscitate

Give 1 litre of crystalloid or colloid
Give oxygen

Give blood

Give 1 unit of blood
Give 1 unit of platelets
Give 1 unit of cryoprecipitate
Give 1 unit of plasma
Give 1 unit of whole blood
Give 1 unit of cryoprecipitate
Give 1 unit of plasma
Give 1 unit of whole blood

Prevent coagulopathy

Give 1 unit of cryoprecipitate
Give 1 unit of plasma
Give 1 unit of whole blood
Give 1 unit of cryoprecipitate
Give 1 unit of plasma
Give 1 unit of whole blood

Get help to stop bleeding

50 ŽIVOT OHROŽUJÍCÍ KRVÁCENÍ (ŽOK)

Zajištění perfuzního tlaku

- systolický TK 80–90 mmHg, MAP 60–70 mmHg – permissivní hypotenze, vysoký krevní tlak zhoršuje krvácení
- **volumeterapie** – vše ohříte, spíše restriktivní, řízená odpovědí organismu
 - koloidní/krytaloidní roztoky 1:1
 - objemové výjevy u kardiálně kompromitovaných
 - transfuzní přípravky podat co nejdříve – EBR O Rh⁺, dále dle krevní skupiny
 - po každých 2 jednotkách EBR i FFP podle 1 amp. CaCl₂
 - NE dohazovat celé krevní ztráty náhradními roztoky
- **farmaka – vazopresory**
 - noradrenalin (5 mg/50 ml) – rychlost dle tlaku

Dodávka kyslíku

- cílem SpO₂ 90–95 %
- cílový hemoglobin 70–90 g/l
- 1 jednotka EBR zvyšuje hladinu hemoglobinu o cca 10 g/l
- dostatečný srdeční výdej (vazopresory, event. inotropika)

Podpora koagulace

- fibrinogen v úvodní dávce 25–50 mg/kg – dále cílené dávky dle ROTEM
- mražená plazma (FFP) – 15–20 ml/kg, poměr EBR:FFP 2:1/1:1
- krevní destičky – udržení hladiny > 50–10⁹/l, při nezářitelném krvácení je cílem hladina 100–10⁹/l
- Exacel (lys. tranexamsy) – antifibrinolytikum, 2 g ihřad
- koncentrát protrombinového komplexu (faktory II, V, VII, IX a X)

Legend: Data are expressed as mean $\pm$ S.E.M. (n=8). *: P<0.05; **: P<0.001 vs baseline.

Initial assessment on the impact of crystalloids versus colloids during damage control resuscitation

Chrissy Guidry, DO,^{a,b} Elizabeth Gleeson, MD, MPH,^{a,c} Eric R. Simms, MD,^a Lance Stuke, MD, MPH,^d Peter Meade, MD, MPH,^a Norman E. McSwain Jr, MD,^a and Juan C. Duchesne, MD^{a,*}

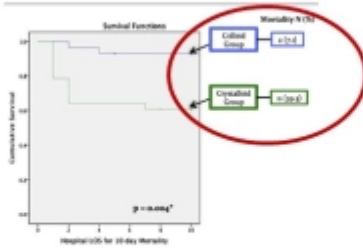


Fig. 1 - KM survival curve for matched case control.

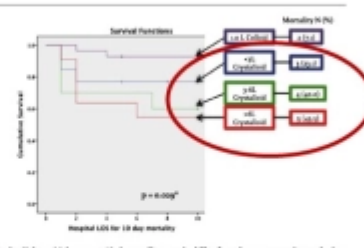


Fig. 2 - Kaplan-Meier curves, 30-d mortality, survival for subgroup quantity analysis.

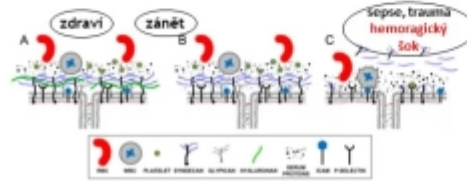


Figure 1. A) An intact endothelial glycocalyx provides a barrier between the plasma compartment and the cell membrane and limits RBC, WBC, and plasmas from contacting the cell surface. The glycocalyx and associated immobile proteins layer overlies the cell, providing contributing to endothelial barrier properties for both water and protein flux. B) During mild to moderate inflammation, shedding and proteolytic cleavage of the glycocalyx (in this case removal of hyaluronan) increases the permeability of the glycocalyx. C) During severe inflammation and trauma, breakdown of the glycocalyx exposes ICAM and P-selectin resulting in increased WBC and plasmas adhesion, respectively, and propagation of the inflammatory response. Note the presence of shed glycocalyx and heparan sulfate in the plasma that are hypothesized to contribute to auto-hemagglutination and the coagulopathy of trauma (see text for details).

Chagwale, Shock. 2018 April; 43(4): 338-348

In massive bleeding, fibrinogen is the first factor to reach critically low levels!

Brenni M, et al. *Acta Anaesthesiol Scand*. 2010;54:111-117

Injury, Int. J. Care Injured 48 (2017) 1074–1081

Fibrinogen is an independent predictor of mortality in major trauma patients: A five-year statewide cohort study

Zoe K. McQuilten^{a,b,*}, Erica M. Wood^b, Michael Bailey^a, Peter A. Cameron^c, David J. Cooper^d

Z.K. McQuilten et al. / *Injury, Int. J. Care Injured* 48 (2017) 1074–1081

Table 2

Patient outcomes according to fibrinogen level on admission.

Outcome	less 1 g/L	1–1.5 g/L	1.6–2.0 g/L	2.1–4.0 g/L	Greater than 4 g/L	p-value
Massive transfusion	63 (55.3%)	93 (32.9%)	104 (36.9%)	124 (4.1%)	12 (1.6%)	<0.01
ICU LOS days ^a , mean (95% CI)	8 (6, 11)	7.5 (6, 9)	6 (5, 7)	5 (4, 5)	5 (4, 5)	<0.01
Hospital LOS days ^a , mean (95% CI)	21 (17, 26)	17 (15, 19)	12 (11, 13)	8 (8, 9)	9 (8, 10)	<0.01
24-h mortality	36 (31.6%)	29 (10.7%)	24 (8.9%)	44 (1.5%)	3 (0.4%)	<0.01
In-hospital mortality	54 (47.4%)	71 (25.1%)	77 (26.5%)	186 (6.2%)	53 (7.2%)	<0.01

ICU – intensive care unit; LOS – length of stay.

^a Restricted to patients who survived until hospital discharge.

A new global assay of coagulation and fibrinolysis

Neil A. Goldenberg^{A,*,B}, William E. Hathaway^{A,B}, Linda Jacobson^B, Marilyn J. Manco-Johnson^{A,B}

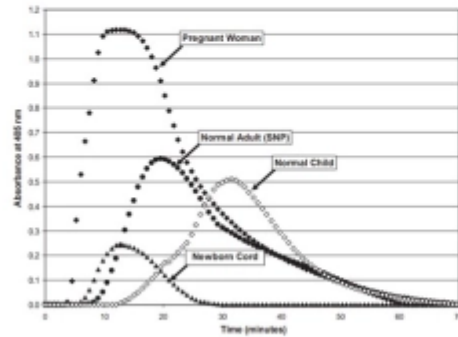


Figure 2 Representative Coflat curves from a healthy adult and child, newborn infant, and pregnant woman. BNP=standard normal pooled adult plasma.

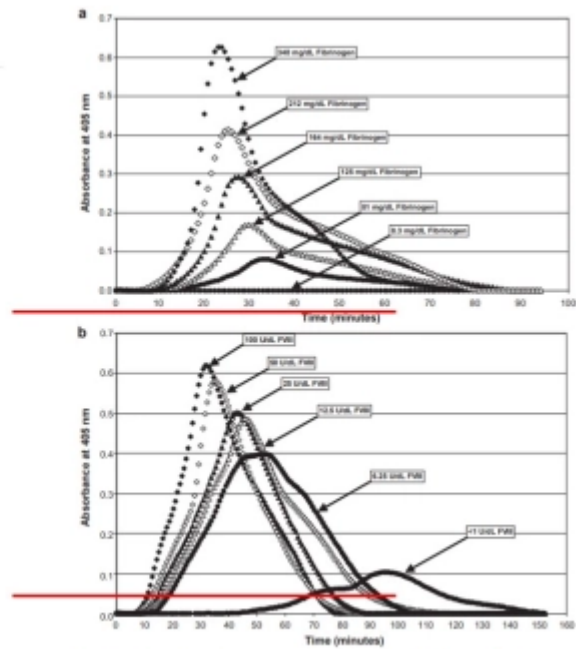
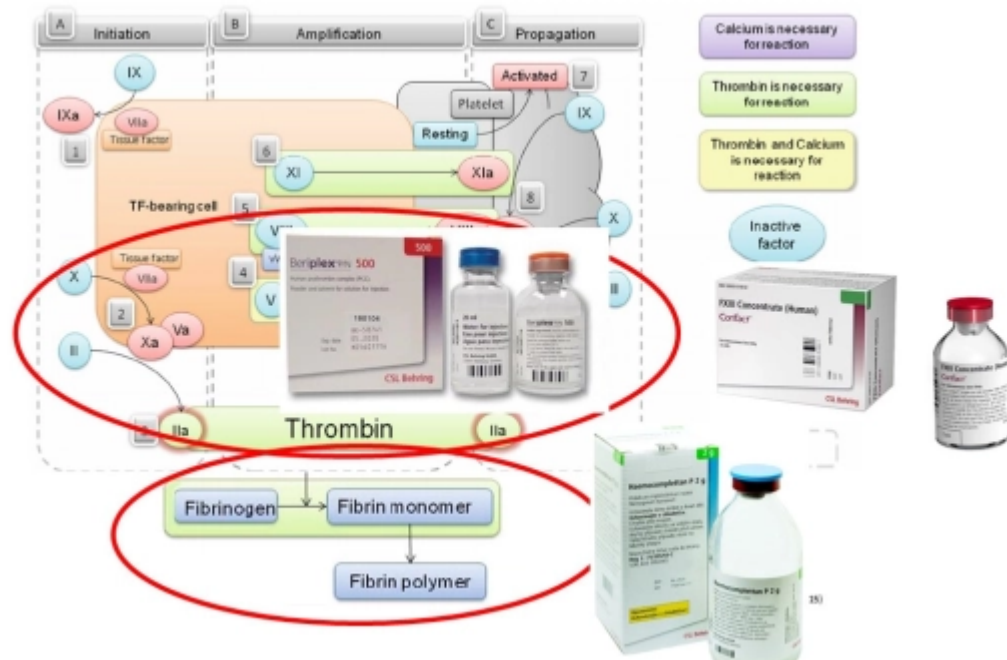
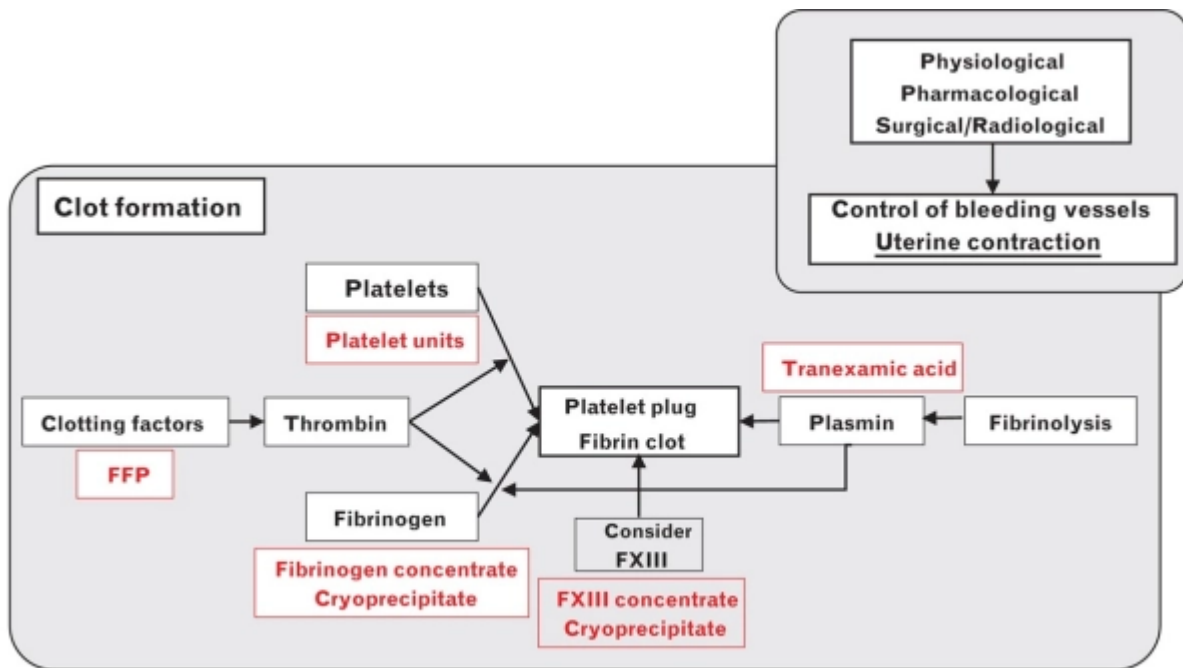


Figure 3 Influence of (a) fibrinogen concentration and (b) factor VIII activity upon the Coflat curve.





Eur J Anaesthesiol 2022; **37**:1 – 10



Fibrinogen concentrate vs. fresh frozen plasma for the management of coagulopathy during thoraco-abdominal aortic aneurysm surgery: a pilot randomised controlled trial

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Summary

Major vascular surgery is frequently associated with significant blood loss and coagulopathy. Existing evidence suggests hypofibrinogenemia develops earlier than other haemostatic deficiencies during major blood loss. The purpose of this study was to evaluate the use of an infusion of fibrinogen concentrate versus plasma and fresh frozen plasma during surgery. Twenty patients undergoing elective repair of thoraco-abdominal aortic aneurysm were randomly allocated to receive either fresh frozen plasma or fibrinogen concentrate to treat hypofibrinogenemia during surgery. Complications were assessed during and after surgery by means of a standardised scoring system, and treatment was guided by pre-defined transfusion triggers. Despite blood losses of up to 11,000 ml in the patients who received the fibrinogen concentrate, none required fresh frozen plasma during surgery, and only two required plasma transfusion. The median (SD) length of stay (days) in patients allocated to fresh frozen plasma vs. fibrinogen concentrate was 11.5 (3.4) vs. 10.5 (3.4), respectively ($p = 0.01$). All patients in both groups were assessed for haemostatic abnormalities at the end of surgery. Mean (SD) postoperative fibrinogen (g/L) was significantly higher in patients allocated to fresh frozen plasma and fibrinogen concentrate (7.4 (2.1) vs. 6.8 (2.1), respectively) ($p = 0.001$). Fibrinogen concentrate may be used as an alternative to fresh frozen plasma for the management of coagulopathy during thoraco-abdominal aortic aneurysm repair.

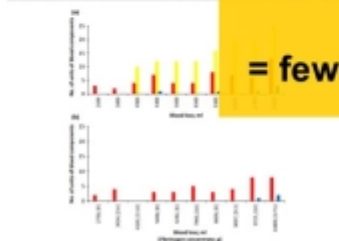


Figure 2: Blood coagulation abnormalities in patients allocated to fibrinogen concentrate (n=10) and fresh frozen plasma (n=10) during surgery for elective repair of thoraco-abdominal aortic aneurysm. Blood loss and fibrinogen concentrate administration during surgery in all patients.

Reversal of trauma-induced coagulopathy using first-line coagulation factor concentrates or fresh frozen plasma (RETEC): a single-centre, parallel-group, open-label, randomised trial

doi:10.1055/s-0010-16000

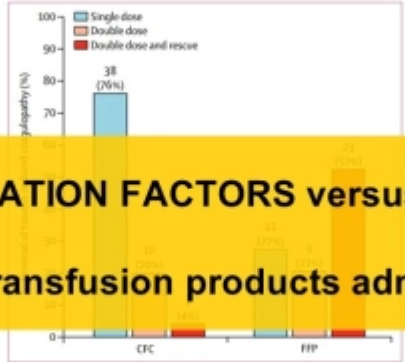


Figure 3: Percentage of patients with reversal of coagulopathy after either single-dose or double-dose study drug administration during the first therapy loop, and percentage of patients needing double-dose and rescue medication during the first 24 h in the intention-to-treat population. CFC=coagulation factor concentrates, FFP=fresh frozen plasma.

The Clinical Efficacy of Fibrinogen Concentrate in Massive Obstetric Haemorrhage with Hypofibrinogenaemia

doi:10.1055/s-0010-16000

	Desigton F + F (n=10)	Desigton F (n=10)	P-value
AP (g/dL)	7.27 ± 1.39	7.00 ± 1.81	>0.05
PTs	18.5 ± 3.6	18.5 ± 3.6	>0.05
Fibrinogen (mg/dL)	614 ± 219	62.5 ± 110	>0.05
Estimated blood loss (mL)	2194 ± 1196	2681 ± 1182	>0.05
FFP	6.88 ± 2.19	19.0 ± 9.40	0.0012
FFP	2.96 ± 1.60	0.0007	

Desigton F: patients before treatment and Desigton F: patients after treatment. AP: activated partial thromboplastin time, PTs: prothrombin time, FFP: fresh frozen plasma, FFP: fibrinogen concentrate, F: fresh frozen plasma.