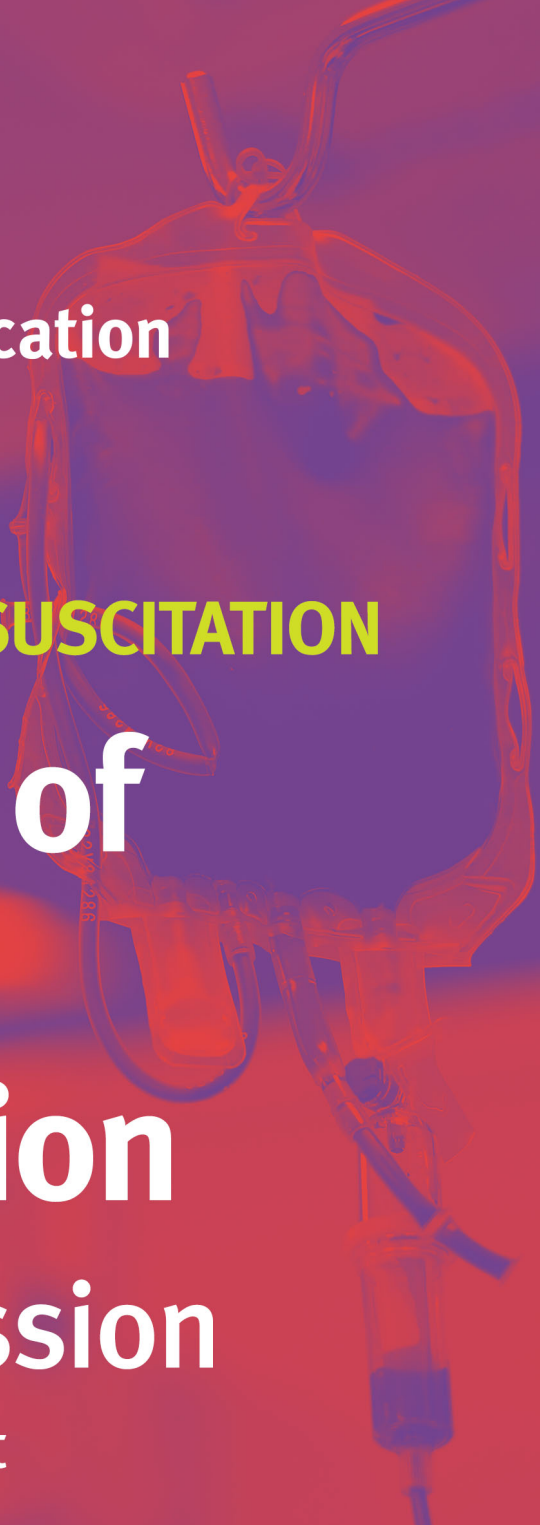
Queensland
Trauma Education

HAEMOSTATIC RESUSCITATION

Delivery of massive transfusion

Case discussion

Facilitator resource kit



CSDS
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Queensland Trauma Education

The resources developed for Queensland Trauma Education are designed for use in any Queensland Health facility that cares for patients who have been injured as a result of trauma. Each resource can be modified by the facilitator and scaled to the learners needs as well as the environment in which the education is being delivered, from tertiary to rural and remote facilities.

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Queensland Trauma Education
Haemostatic resuscitation - Delivery of massive transfusion: Case discussion – Facilitator resource kit
Version 1.0

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About this training resource kit

This resource kit provides the learner with the knowledge in the management of a patient who requires haemostatic resuscitation.

National Safety and Quality Health Service (NSQHS) Standards

Target audience

Medical, nursing, and allied health clinicians

Duration

30 minutes

Group size

Suited to small group participation

Learning objectives

By the end of this session the participant will be able to:

- Understand haemostatic resuscitation in the trauma patient
- Describe the factors important in haemostatic resuscitation
- Identify the parameters for targeted transfusion

Facilitation guide

1. Use clinical scenario for discussion focus for haemostatic transfusion targets
2. Utilise local guidelines and processes to embed clinical care

Supporting resources

- Critical Bleeding algorithm RBWH (Royal Brisbane and Women s Hospital)
- Massive transfusion protocol Queensland Health
- Lethal triad
- Trauma-associated coagulopathy

Overview of transfusion targets

This resource can be used to facilitate discussion around transfusion targets in the care of the bleeding trauma patient. Local guidelines should be used to provide local context.

Further reading

Massive Transfusion Protocol (2)	
Publication	Life in the Fast Lane
Link	https://bit.ly/3QBi3Kt

Blood transfusion, NICE Guideline	
Organisation	National Institute for Health and Care Excellence
Link	https://bit.ly/3bP53Cg

Patient Blood Management Guidelines: Module 4 Critical Care	
Organisation	National Blood Authority Australia
Link	https://bit.ly/3BXylth

Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial	
Authors	CRASH-2 trial collaborators
Link	https://bit.ly/3JPsfgf

Case discussion

Case study

A 63-year-old man is brought to hospital after a high-speed RTC.

He has been assessed and has sustained a compound tibial fracture, a lateral compression pelvic fracture and a splenic injury with a large hemoperitoneum suspected by a FAST scan demonstrating free fluid.

He has a pelvic binder insitu, large bore cannula access and pathology is pending.

His heart rate is 110, blood pressure 80 systolic, respiratory rate 28, temperature 35 degrees C, SpO2 90% on 15L O2 NRB. He is cool and clammy peripherally.

Question and answer guide

1. What is the most common cause of shock following blunt trauma?

Hypovolaemic shock, from haemorrhage. Hypovolaemic shock occurs when there is decreased intravascular volume to the point of cardiovascular compromise.

Other forms of shock in trauma include cardiogenic (secondary to blunt cardiac injury or precipitation of cardiac ischaemia), obstructive shock (tension pneumothorax or haemothorax and cardiac tamponade) and distributive (neurogenic shock secondary to spinal cord injury or severe traumatic brain injury).

2. What are your immediate concerns and red flags with this patient?

Clinical signs of shock including:

- Tachycardia
- Hypotension
- Poor peripheral perfusion

Radiological findings:

- Positive FAST
- Pelvic fracture

Compound Tibia fracture

3. What is the 'lethal triad' in haemorrhagic shock?

- Acidosis (pH <7.35. This is often occurring due to a lactic acidosis from tissue hypoxia because of hypoperfusion from haemorrhage)
- Hypothermia (Temp < 35 degrees Celsius is an independent predictor of mortality in trauma. Hypothermia has been shown to affect coagulation efficacy and cardiovascular function worsening the traumatic injury)
- Coagulopathy (Trauma Induced Coagulopathy (TIC), involves coagulation activation, hyperfibrinolysis and a consumptive coagulopathy- this has been associated with multiple factors and the presence of TIC has been shown to have significant effects on outcome following trauma)

4. How does acidosis affect bleeding in haemorrhagic shock?

Hypovolaemia and peripheral vasoconstriction lead to anabolic respiration and acidosis. Acidosis affects the coagulation cascade with function reduced by up to 70% when the pH is < 7.0.

The acidosis may also occur secondary to the administration of volume replacement with crystalloid solutions, adding to the support for minimisation of these fluids in trauma resuscitation.

5. How does hypothermia affect bleeding in haemorrhagic shock?

At low temperatures, the normal haemostatic enzymes in the body do not work. Therefore, cardiac function is affected, and clotting is reduced therefore worsening coagulopathy.

6. What is the impact on coagulation factors in haemorrhagic shock?

Coagulation abnormalities following traumatic injury have been described as Trauma Induced and Trauma Associated Coagulopathy. These terms have been used interchangeably, however also delineate differing processes. TAC is the term to describe the overall process with the effect of acidosis, hypothermia, and dilution in conjunction with the direct effect of trauma, named trauma induced coagulopathy. This TIC process results from direct tissue trauma and combines the activation of coagulation, hyperfibrinolysis and consumption of coagulation factors. In the acute coagulopathy of trauma shock (ACoTS) theory, the injury to the tissues causes release of mediators which activates the coagulation cascade, thereby activating the process.

When compared to DIC, another coagulation abnormality occurring in the shocked state- there is a higher incidence of fibrinolysis leading to massive bleeding not seen with DIC.

Changes identified in coagulation testing with TIC will include:
1) Coagulation activation- procoagulants in the systemic circulation, impairment of endogenous anticoagulant activity, thrombin generation in the systemic circulation.
2) Hyperfibrinolysis- acute release of t-PA and hyperfibrinolysis, coagulation activation induced hyperfibrinolysis.
3) Consumption coagulopathy.

7. What end point targets are measured and then managed in the bleeding trauma patient?

Transfusion targets include both clinical state and laboratory values to direct the ongoing and cessation of transfusion process once bleeding has been controlled, either through surgical damage control techniques or the use of angioembolisation. [(1)]

Specific targets include the following:

- Haemoglobin > 80g/dL- this absolute cut off level may alter depending on the individual patient vital signs, organ perfusion and the co-morbidities
- Ionised calcium > 1.1mmol/L- calcium is usually required after 4-6 units of blood product replacement. Many prehospital protocols now given calcium empirically, resulting in near normal calcium levels despite prehospital blood administration
- pH >7.2- to avoid acidosis, corrected with ventilation strategies
- Lactate <4- another marker of acidosis, in particular the metabolic component
- Platelets > 50 x 10^9/L (>100 if concurrent severe TBI (Traumatic Brain Injury))

- PT/aPTT <1.5 x normal
- INR< 1.5
- Fibrinogen > 2.5g/L
- Temperature >35 deg C- managed using warmed blood products and external warming blankets/warm trauma rooms and theatre, plus covering the patient once the primary and secondary survey has been completed

8. What is haemostatic resuscitation?

Haemostatic resuscitation is the term given to describe the overall process in which the lethal triad is addressed, a balanced (or targeted) resuscitation strategy is employed with the aim to 'turn off the tap,' allowing time to injury identification and definitive haemorrhage control.

In addition, the use of external splints/haemostatic dressings, haemostatic adjuncts (like TXA), and targeted blood pressure are all used to reduce further bleeding until haemostasis is achieved.

9. The use of tranexamic acid is often used in haemostatic resuscitation. What is TXA and how does it work?

Tranexamic acid (TXA) has been demonstrated to be of benefit in the bleeding patient in the large, multicentre Crash2 trial.

TXA works by contributing to the clot strength by reducing fibrinolysis that occurs following trauma as part of the TIC process.

TXA is administered as an initial 1g IV bolus over 10 minutes, then as a second 1g IV infusion over 8 hours.

The administration of TXA within 3 hours following injury resulted in reduction in all-cause mortality and death due to bleeding in the treatment arms.

(See other reading for Crash2 trial)

10. What is typically in a massive haemorrhage protocol pack?

The Queensland Massive Haemorrhage Protocol (attached) can be used to guide the management of haemorrhagic shock. Many institutions have adapted this protocol to suit their local environment.

Regardless of the local protocol adjustments, a MHP pack usually contains:

- Packed red blood cells- which are used to carry oxygen to the tissues. With large volume replacement not only is hypothermia a risk due to the cold storage process, but in addition this process results in lower levels of 2,3DPG in the red cells thereby affecting the oxygen storage capacity.

- Fresh Frozen Plasma (FFP or extended life plasma ELP)- which include the clotting factors (II, VII, IX, and X)
- Platelets- which combine with fibrin to form the initial clot plug

In addition, blood bank may have available:

- Cryoprecipitate- a fibrinogen replacement, this forms the fibrin mesh
- Fibrinogen (Riastap) - dry powder storage of fibrinogen, reconstituted with sterile water

11. Can haemostatic resuscitation be implemented without activating an MHP?

Yes! (At the start)

By activating an MHP, the blood bank services will provide a 'balanced' product delivery in a standardised ratio. This makes it much easier to ensure a balanced ratio will be delivered to the patient. The blood bank should be notified as soon as possible that a massive transfusion of blood and blood products is expected, to ensure the required products are available when needed.

12. At what temperature should the products be delivered?

Ideally warmed to body temperature to improve clotting function and avoid hypothermia. This can be done using external fluid warmers.

13. What complications may occur following large volume transfusion? [(2)]

- Volume overload
- Over transfusion with haemoglobin supratherapeutic
- Hypothermia
- Dilutional coagulopathy
- Transfusion related lung injury
- Excessive citrate (metabolic alkalosis and hypocalcaemia)
- Hyperkalaemia
- Disease transmission (very low risk)

14. When does Rh O negative blood need to be used for un-cross matched blood transfusion? (3)

- Rh negative patients may make anti-D when exposed to the D-antigen with transfusion of Rh-positive blood. This immunological response may occur after 3-4 months post transfusion, and the patient is not at immediate risk of haemolysis.
- As O negative blood is often in limited supply a strategy to manage low risk patients includes reserving O negative transfusions for females of childbearing age

to avoid alloimmunisation and prevent haemolysis of the newborn (in general women < 55 years).

- O positive transfusions may be given to women over childbearing age or males who require urgent transfusion as the preference remains to utilise group and type specific blood products as soon as possible.

Supporting resources

The following supporting documents are provided for this case discussion:

1. Critical Bleeding algorithm, RBWH
2. Massive transfusion protocol Queensland Health
3. Lethal triad
4. Trauma-associated coagulopathy

RBWH MAJOR HAEMORRHAGE TRANSFUSION PROTOCOL

ACTIVATION CRITERIA

ONGOING BLEEDING and SBP < 90MMHG and ANTICIPATED TRANSFUSION > 4U RBC IN AN HOUR

- Ring Blood Bank 67188 to notify
- Runner to take samples & request form to Blood Bank [coag tube + cross match tube] & return with products
- Transfuse 2-4 units O NEG or O POS RBC [including prehospital products] and reassess
- TRANEXAMIC ACID [if within 3 hours of injury] 1g over 10min, then 1g over 8 hours
- PACK ZERO Only for critical patient with no ROTEM available
  4 RBC, 2 FFP, 4G FIBRINOGEN CONCENTRATE
  Check Calcium: keep iCa > 1mmol/L
- If ongoing transfusion requirements continue with 4 RBC + ROTEM guided products - repeating as required

The ROTEM test guiding product use must have been taken within the previous 30 minutes

ROTEM STEPS

Follow the TEMguide app

Repeat ROTEM 10 mins after any step actioned

Flat line trace? GIve 4G Fibrinogen Conc & 1g TXA

Fibrinogen Replace if FIBTEM A5 < 10mm
Use Fibrinogen Concentrate only if time critical & FIBTEM A5 < 8mm
Otherwise use Cryoprecipitate 10-20 units Then repeat ROTEM

OBSTETRIC HAEMORRHAGE	Target 2mm higher in obstetric haemorrhage Treat if FIBTEM A5 < 12mm [use FC if < 10mm]

Platelets Replace if EXTEM A5 < 35mm AND
FIBTEM A5 > 10mm [normal]
Give 1 dose platelets Then repeat ROTEM

Plasma Replace if EXTEM CT > 90s AND
EXTEM A5 > 35mm [normal]
Give 2-4U FFP Then repeat ROTEM

Tranexamic Acid Replace if EXTEM ML > 15%
Give 1g TXA Then repeat ROTEM

TARGETS Temp > 36°C, iCa > 1mmol/L, Hb > 70 g/L, Platelets > 75 x 10^9/L, Fibrinogen > 2g/L 3g/L in OBSTETRICS

ETC Cons 75900
ICU Cons 75946
TRAUMA Service CNC 0418723231

L4
Duty ANAES 75922
ANAES Reg [after hours] 75924
Theatre Shift Co-ord 75915
Cell Salvage 74670

L5
ANAES Reg 75925
Women's Theatre
Shift Co-ord 65969

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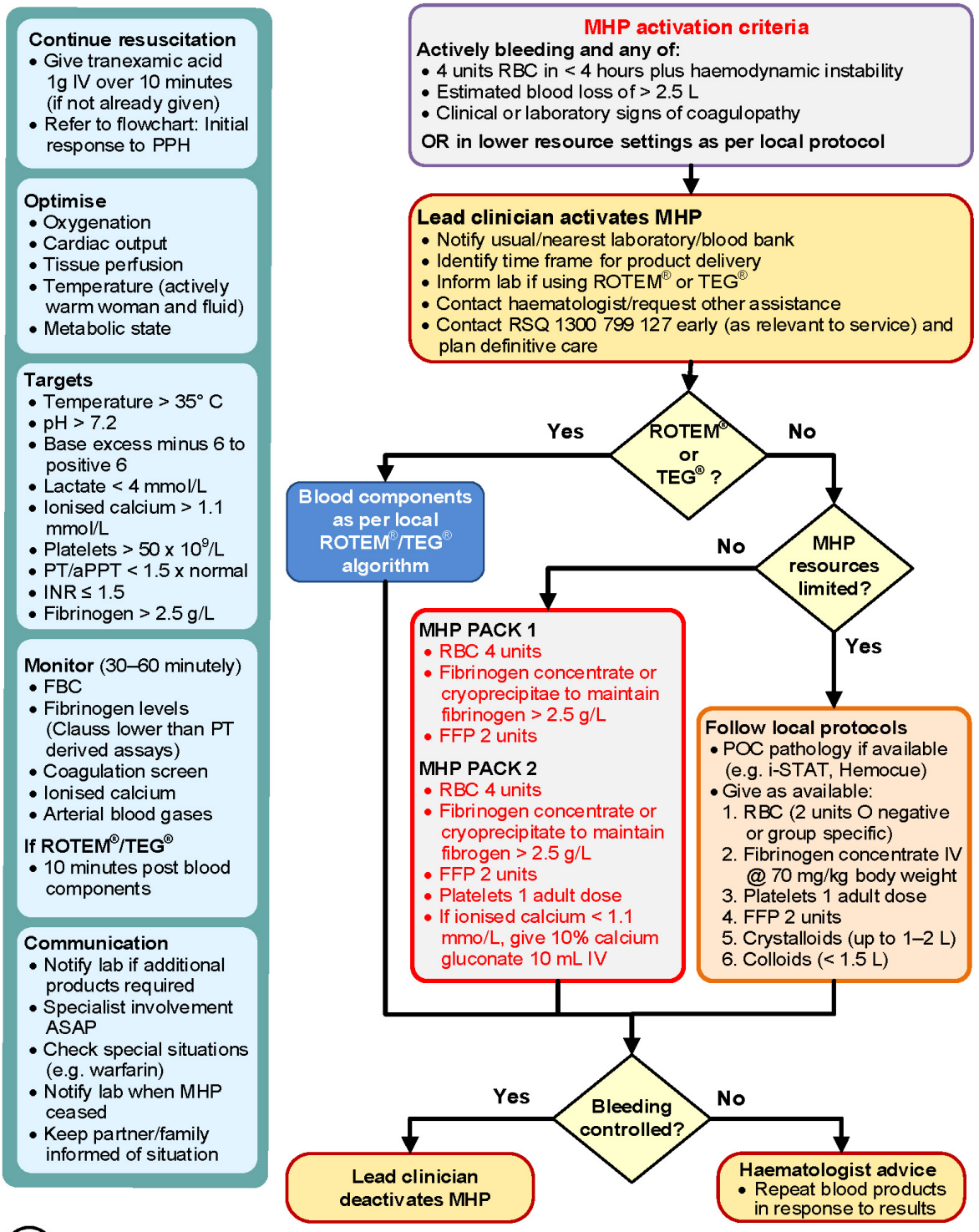
Version 5: Effective date Febrary 2020, Revision due February 2022

Massive haemorrhage protocol (MHP)

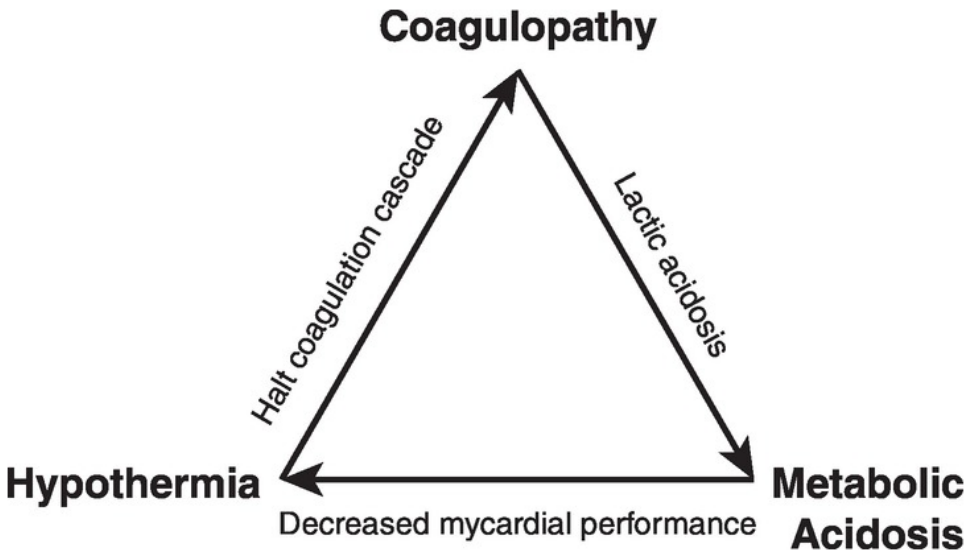


Medical Officers (call senior asap)	LAB	Theatre

Queensland Clinical Guidelines: Massive haemorrhage protocol (MHP). Flowchart version: F18.1-2-V4-R23

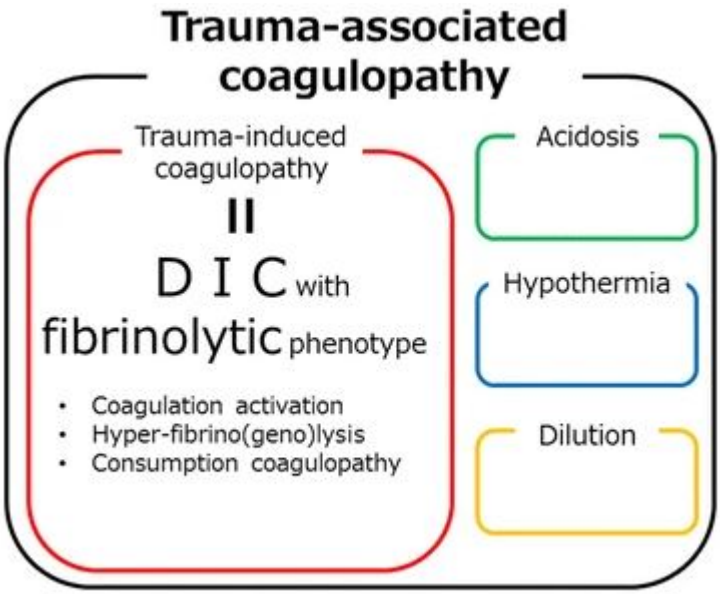
APPT: activated partial thromboplastin time, **ASAP**: as soon as possible, **FBC**: full blood count, **FFP**: fresh frozen plasma, **INR**: international normalised ratio, **IV**: intravenous, **MHP**: massive haemorrhage protocol, **POC:** point of care, **PPH:** postpartum haemorrhage **PT**: prothrombin time, **RBC**: red blood cells, **ROTEM®/TEG®**: types of blood clotting analysers, **<**: less than, **>**: greater than

Lethal triad



Reference. Transfusion in trauma (chapter 3)- The Emergency Medicine Trauma Handbook. Cambridge University Press.

Trauma-associated coagulopathy



Reference: Hayakawa, M. Pathophysiology of trauma-induced coagulopathy: disseminated intravascular coagulation with the fibrinolytic phenotype. *j intensive care* **5,** 14 (2017). https://doi.org/10.1186/s40560-016-0200-1

Acronyms and abbreviations

Term	Definition
RTC	Road traffic collision
NRB	Non-rebreather Bag
TIC	Trauma induced coagulopathy
TAC	Trauma associated coagulopathy
TXA	Tranexamic acid
MHP	Massive haemorrhage protocol
PT	Prothrombin time
aPTT	Activated partial thromboplastin time
DIC	Disseminated intravascular coagulation

References

1. ACS TQIP Massive Transfusion in Trauma guidelines. October 2014. https://bit.ly/3qcy6mO
2. Nickson, C. (2014). *Massive Transfusion Protocol,* Life in the Fast Lane. Accessed from https://bit.ly/3BfxzH3
3. Flommersfeld S, Mand C, Kühne C, A, Bein G, Ruchholtz S, Sachs U, J: (2018). *Unmatched Type O RhD+ Red Blood Cells in Multiple Injured Patients.* Transfus Med Hemother; 45:158-161. Accessed from https://bit.ly/3qeNPCc

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