

Physiologically-guided Balanced Resuscitation: An Evidence-based Approach for Acute Fluid Management in Paediatric Major Trauma

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Abstract

Trauma is a major cause of death, and haemorrhage represents an important target for improving outcomes after severe injury. Volume replacement with crystalloids in resuscitation might become harmful in large amounts because of coagulopathy. A fine balance must be achieved between haemodynamic and haemostatic resuscitation. Permissive hypotension refers to permitting some degree of hypotension in such adult patients in an attempt to attain this fine balance. For patients who require a significant volume of blood product resuscitation, the term ‘massive transfusion protocol’ (MTP) is used. There is very little data on transfusion protocols for paediatric trauma patients, and children respond to hypovolemic shock in a different physiological manner compared to adults. Hence, concepts such as permissive hypotension may not be appropriate when treating children involved in major trauma. We recently embarked on a plan to streamline the management of blood transfusion in massive bleeding during paediatric trauma, to reduce the logistical problems associated with the transport of blood products from the blood bank to the patient. From this, we evolved a MTP for paediatric major trauma. Nonetheless, further studies will be needed to see if there is indeed improved outcome after MTP in paediatric major trauma as current evidence is extrapolated from adult studies.

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Key words: Compensated versus decompensated shock, Damage control resuscitation, Massive transfusion protocol, Permissive tachycardia

Introduction

The Burden of Major Trauma and Recent Advances

Trauma is a major cause of death and disability worldwide.^{1,2,3} Since haemorrhage is responsible for more than 40% of all trauma deaths, it is an important target for improving outcomes after severe injury.⁴ With paucity of research in paediatric major trauma, one would be tempted to simply cut and paste existing adult protocols for use in paediatric trauma. However, the management of traumatic haemorrhagic shock in adults and children cannot be the same.

In adults, it is widely recognised that volume replacement in haemorrhagic shock resuscitation might become harmful if a large volume of crystalloids is used in exsanguinating major trauma patients with continuing blood loss. While the volume may be “adequate”, the tonicity and components of the volume infused will be important as large crystalloid

volumes will result in dilutional effects and a whole series of unintended sequelae, including coagulopathy. Therefore, in major trauma, a fine balance must be achieved between haemodynamic/volume and adequate haemostatic resuscitation. When a certain threshold of blood loss is reached, fluid resuscitation consisting of crystalloid administration must be supplemented with the transfusion of blood products, sometimes in large amounts and in preset ratios (massive transfusion).⁵

Massive transfusion has its origins in adult major trauma management⁶ where blood component therapy (packed red blood cells (PRBCs), fresh frozen plasma (FFP), platelets and cryoprecipitates of clotting factors) is common.

Our paediatric emergency department (ED) at KK Women’s and Children’s Hospital (KKH) sees about 178,000 patients a year. According to the KKH Trauma Registry, from 2011 to 2012, we saw a total of 51,001 trauma patients,

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of whom 28 were Tier 1 (Injury Severity Score (ISS) equal to or more than 16), 101 were Tier 2 (ISS between 9 and 15, inclusive) and the rest were Tier 3.

In major paediatric trauma, coagulopathy is prevalent. In fact, in paediatric trauma patients who are ill enough to require transfusion, there is a strong association with mortality.⁷ Massive transfusion protocol (MTP) is recommended to improve outcomes in such ill patients.⁸ Further studies need to be conducted to ascertain if paediatric patients have good outcomes after MTP. Currently the benefits of MTP are largely extrapolated from adult studies.

Key Concept in Adult Major Trauma: The Problem of Coagulopathy

Much has been written about the lethal triad⁹ in trauma: coagulopathy, acidosis and hypothermia. It is estimated that 10% of severe trauma cases are hypocoagulable with the most severely injured being more coagulopathic.⁹ This coagulopathy presents very early after injury.¹⁰ Therefore, coagulopathy is not merely due to excessive crystalloid infusion where at a cellular level, Ringer's lactate and normal saline increase reperfusion injury and leukocyte adhesion.^{11,12}

Coagulopathy in trauma is multifactorial.¹³ Firstly, in haemorrhage where there is volume/haemodynamic resuscitation, dilution and hypothermia cause dilutional coagulopathy and further haemorrhage. In addition, haemorrhage itself causes a stream of effects including shock, acidosis and hypothermia that worsen coagulopathy. To compound the problem, trauma and shock cause acute coagulopathy of trauma-shock which is associated with factor consumption and fibrinolysis. Finally, coagulopathy has also been found to be associated with trauma-induced inflammation.¹³

Finding the Balance: Haemostatic Resuscitation in Adults

During shock resuscitation, clots are potentially unstable if the pressure is elevated to a level that is considered normal. A high blood pressure, while allowing better perfusion to the tissues, might contribute to further bleeds and hypocoagulopathy. Therefore, a certain degree of hypotension might be helpful during haemorrhagic shock resuscitation—what is termed “permissive hypotension”—to allow clots to stabilise and prevent clot dislodgement and coagulopathy. A “balanced resuscitation” approach is taken—which incorporates the important concept of haemostatic resuscitation as opposed to the conventional absolute volume and haemodynamic pressor resuscitation alone.

Exceptions to the use of “permissive hypotension” in major adult trauma are when patients have severe head

injuries or are in spinal shock. In such cases, allowing low blood pressure will highly predispose the patient to insufficient cerebral perfusion pressure and hypoxia, and secondary brain injury/damage. Hence, permissive hypotension is not advocated for major adult trauma where there is severe head injury or spinal shock.

It has been shown in various adult studies where patients with severe head injuries are excluded, that a systolic blood pressure (SBP) greater than 80 to 90 or mean arterial pressure (MAP) greater than 60 is associated with a higher risk of rebleeding, with 76% rebleed at pressures greater than 80 mmHg. One centre that has endorsed permissive hypotension in adult polytrauma is the National Institute for Health and Clinical Excellence which advises against fluid administration in patients without head injury if a radial pulse is palpable.¹⁴ However, in the management of polytrauma patients with significant head injuries, as the importance of maintaining cerebral perfusion pressure is well recognised, permissive hypotension is contraindicated.¹⁵

MTP and Damage Control Resuscitation in Adults

Adult MTP originated in the military and has been extrapolated to civilian clinical studies. These data along with recommendations of an international consensus conference on early massive transfusion for trauma,¹¹ make it possible to rapidly identify and treat patients at high risk for coagulopathy on arrival to the resuscitation room. Damage control resuscitation (DCR) is a structured intervention that begins in the ED where targets include normalising the coagulopathy, acidosis and hypothermia, until damage control surgery achieves haemostasis, and is described by Holcomb in this way: Resuscitation is limited to keep systolic blood pressure at 90 mmHg, which prevents rebleeding from recently clotted vessels.^{11,12} Intravascular volume restoration is accomplished with thawed plasma in at least a 1:1 ratio with PRBCs.^{16,17} For casualties who are anticipated to require continued resuscitation, the blood bank is notified to activate the MTP which results in the delivery of 6 units of plasma, 6 units of PRBCs, 6 packs of platelets and 10 units of cryoprecipitate stored in individual coolers.¹⁷ Of note, in the adult military population, for the most severely injured, fresh warm whole blood is used as a primary resuscitative fluid.^{18,19} Crystalloids are only used to keep lines open between blood products.²⁰

The outcome of DCR in adults is positive based on the 10-year institutional experience with DCR described by Frischknecht et al. Patients were categorised as early survivors or early deaths. During the study period, 319 patients underwent DCR. Overall, 52 patients (16.3%) died (early deaths) and 267 patients (83.7%) survived the first 72 hours (early survivors). In the latter group, survival in severely injured trauma patients managed with

DCR exceeded 90%.⁸ Further randomised control trials are required to prove significant survival advantage.

Coagulation in Paediatric Patients

Coagulopathy in paediatric patients is a significant problem, just as it is in adults. Holmes et al reviewed the medical records of all patients younger than 15 years old who were hospitalised at a level 1 trauma centre for either blunt head or torso trauma over a 4-year period. A total of 1082 patients' records were reviewed, and the 830 (77%) patients with coagulation studies obtained composed of the study population. Elevated coagulation studies were detected in 232 (28%) patients, and 49 (6%) of these were found to be markedly elevated. In the multivariate analysis, a Glasgow Coma Scale (GCS) ≤ 13 (odds ratio [OR]: 8.7, 95% confidence interval [CI], 4.3, 17.7), low systolic blood pressure (OR: 4.0, 95% CI, 1.6, 9.9), open/multiple bony fractures (OR: 2.9, 95% CI, 1.4, 6.2), and major tissue wounds (OR: 2.8, 95% CI, 1.4, 5.6) were independently associated with markedly elevated coagulation studies. Their conclusion was that hospitalised paediatric blunt trauma patients frequently have minor elevations in ED coagulation studies. Marked elevations are independently associated with a GCS ≤ 13 , low systolic blood pressure, open/multiple bony fractures, and major tissue wounds.²¹

Hendrickson et al looked at paediatric trauma patients requiring blood transfusion within 24 hours of arrival. Coagulation values on ED arrival, clinical details and outcome were analysed. A total of 102 children (mean age, 6 years; mean injury severity score 22, mean Glasgow Coma Scale 7, 80% blunt trauma victims) were studied over a 4-year period. An abnormal prothrombin time was found in 72% of the children, partial thromboplastin time in 38%, fibrinogen in 52%, haemoglobin in 58%, and platelet count in 23%. An abnormal prothrombin time, partial thromboplastin time, and platelet count were strongly associated with mortality ($P = 0.005$, 0.001, and <0.0001 , respectively) and remained significantly associated in multivariate analysis after adjusting for injury severity score. The team concluded that coagulopathy was prevalent in paediatric trauma patients ill enough to require a transfusion and is strongly associated with mortality.⁷

These 2 studies have shown that children with major trauma do experience significant coagulopathy, as do adults.

Physiologically-guided Resuscitation: Adults versus Children

Based on these studies and evolving established principles for the adult polytrauma, can these principles be adopted in the acute management of paediatric polytrauma patients? The key issue is whether children respond to shock, hypoxia

and hypovolaemia in the same way that adults do.

In adults, the first sign of shock is a narrow pulse pressure because of decreased venous return, decreased cardiac output and increased peripheral resistance from neural and hormonal response. Shock usually results in concomitant rise in heart rate and drop in blood pressure.

When monitoring a child's cardiovascular system, we closely monitor the age-adjusted heart rate of the child and the estimated/expected systolic blood pressure which is calculated as $70 + 2 \times (\text{age in years})$, as well as the general clinical condition of the child, rather than just the pulse pressure. In children during compensated shock there is a period of tachycardia without a concomitant drop in blood pressure. With sustained shock, blood pressure eventually drops precipitously as illustrated in Figure 1. In early "compensated" shock, vital organ function is maintained by intrinsic compensatory mechanisms, such as vasoconstriction and peripheral vasoconstriction. Blood pressure is usually maintained, heart rate is increased, pulse pressure is narrowed and signs of peripheral vasoconstriction (cold extremities and prolonged capillary filling time) are present. In decompensated shock, despite intense arteriolar constriction and tachycardia, blood pressure and cardiac output decline. Patients may demonstrate an impairment of major organ perfusion which may manifest as altered mentation, oliguria and myocardial ischaemia. Prolonged shock is characterised by progressive reduction in blood pressure and cardiac output leading to prolonged hypoperfusion of the brain, heart and kidneys which leads to ischaemic cell death. Clinically, this manifests as progressive worsening of coma, renal failure, pulmonary oedema and multi-organ failure.

Therefore, trying to "permit" hypotension in paediatric polytrauma is illogical. By the time the child has documented age-adjusted hypotension, the child is already at death's door. In major paediatric trauma, in the absence of severe

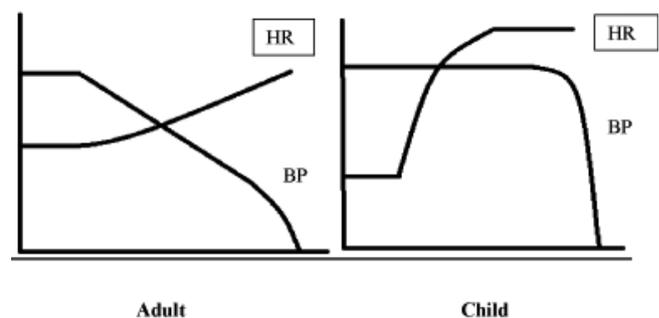


Fig. 1. Adult's versus child's physiological response to shock.*

*Adapted from The Advanced Resuscitation for Children course by the Singapore Paediatric Society

head injury and spinal shock, we can promote a more physiological state by allowing the heart rate to be raised (permissive tachycardia) while the blood pressure is maintained in the normal range (isotension). The optimal range of increased heart rate needs to be determined by prospective randomised controlled study of the treatment outcomes.

The Need For A Paediatric-centred MTP

In adult literature, 3 definitions of MTP are commonly used:²² Transfusion of 10 or more units of red blood cells (RBC) within 24 hours, transfusion of 4 or more units of RBC within 1 hour with anticipation of continued need, and replacement by transfusion of 50% total blood volume (TBV) in 3 hours.

There are several reasons why we cannot apply these definitions for adult MTP indiscriminately to paediatric MTP. Firstly, the TBV, along with volume loss and volume replacement, in neonates and children vary considerably according to age.²³ Hence, any definition of MTP in paediatric patients must be relative to the TBV of each individual patient, or at least likely weight groups. Diab YA et al²⁴ suggested that paediatric MTP may be defined as transfusion of >50% TBV in 3 hours, transfusion of >100% TBV in 24 hours, or transfusion to replace ongoing blood loss of >10% TBV per minute.

Studies in adult trauma have consistently shown that trauma-associated coagulopathy is common and associated with adverse outcomes.^{10,25-27} Likewise, coagulopathy in paediatric trauma patients is prevalent and strongly associated with mortality. These associations have been observed in paediatric trauma patients suffering from blunt or penetrating injuries, with or without head involvement. When it comes to predicting mortality in paediatric trauma patients, the BIG score which incorporates coagulation data (BIG score: base deficit + [2.5 x international normalised ratio] + [15 – Glasgow Coma Scale score]) may be a more accurate scoring system.^{7,21,28-31}

Despite an increasing realisation of the prevalence of paediatric trauma and the importance of coagulopathy, the implementation of MTP for paediatric patients has lagged behind that of adults.^{32,33} This may be due to the different mechanisms of injuries in children compared to adults (with less penetrating trauma and exsanguination) as well as difficulties in adapting a universal protocol for children of all ages and weights.

Finally and more importantly, the child responds in a very different manner to hypovolaemia as compared to the adult. While hypotension is a relatively late sign in adults compared to the narrow pulse pressure and tachycardia, children have such resilient cardiovascular reserves that frank hypotension

indicates a point of physiological derangement when severe morbidity is likely despite resuscitation. Therefore, in paediatric MTP, as more extensively explained above, one should consider permissive tachycardia rather than the adult MTP concept of permissive hypotension.

International Experience with Paediatric MTP

There have been 2 significant single-centre studies that describe their experience with MTP in paediatric patients. Hendrickson et al conducted a retrospective study on 53 children after implementing paediatric MTP that utilised FFP and red blood cells in a 1:1 ratio of FFP:RBC and alternating weight-adjusted amounts of aphaeresis platelets or cryoprecipitate units. These data were then compared with data from historical controls. Although there was no improvement in outcome with MTP, there were 2 important conclusions that arose: (1) majority of patients had coagulopathy at presentation; and (2) implementation of paediatric MTP with increased FFP:RBC ratios and early plasma transfusion to coagulopathic children is feasible.³²

Chidester et al conducted a prospective cohort study of 55 children, of whom 22 received transfusions according to their MTP and 33 received transfusions at physician's discretion. Although there was no improvement in mortality with MTP, there was 1 interesting finding: thromboembolic events were observed exclusively in the non-MTP group, possibly related to under-transfusion in these patients.³⁴

In both studies, there was a delay in transfusing plasma due to additional time required for thawing, so neither study was able to reach the protocol's goal for 1:1 ratio for FFP/RBC transfusions.

Local Experience: Setting up MTP in KKH

Members of our paediatric trauma committee as well as other important stakeholders undertook an extensive literature review and used an evidence-based approach to develop our own in-house paediatric MTP protocol (see Appendices 1 and 2).

Massive transfusion is defined in our hospital as transfusion of >40 mL/kg (blood volume in children over the age of 1 month is approximately 80 mL/kg). Indications for the use of MTP include: (1) primary cause of shock is haemorrhagic shock from uncontrolled bleeding; or (2) the child who has been given more than 40 mL/kg of non-blood products or more than 20 mL/kg of blood products. This is given for haemodynamic stabilisation and the aim is to limit crystalloid resuscitation with use of blood products for haemodynamic resuscitation. The rate of blood products to be given will be determined acutely by the haemodynamic status of the paediatric patient. Having MTP protocols would allow blood products in the appropriate 1PCT:1FFP:1platelet ratio to

be continuously made available as required during trauma resuscitation and limit waiting time usually required for ad hoc requests for such blood products. The MTP activation process is summarised in Appendix 1.

Concurrently, the lethal triad of hypothermia, coagulopathy and acidosis must be avoided and corrected with close monitoring upon activation of MTP.

Other Interventions for Coagulopathy in Trauma

Early Intervention with Tranexamic Acid

As time is of the essence in managing haemorrhagic shock, early intervention is key. One such intervention that could potentially be started in the prehospital setting is tranexamic acid (TXA). TXA minimises blood loss by inhibiting lysine binding sites on plasminogen, thereby preventing the conversion of plasminogen to plasmin. This inhibits fibrinolysis and reduces clot breakdown, resulting in a reduction in bleeding,³⁵ and has been used to minimise blood loss associated with surgery.

The CRASH-2 randomised controlled trial was the first extensive multicentre trial to evaluate the use of TXA in civilian trauma care.^{36,37} CRASH-2 studied the early administration of TXA in adult trauma patients in 274 hospitals in 40 countries. Patients ($n=20,211$) were enrolled if the treating physician judged them to have or be at risk for significant haemorrhage and were randomised to either TXA or placebo. Within 8 hours of injury, participants received a 1 g intravenous (IV) loading dose of either TXA or placebo over 10 minutes; a 1 g infusion over 8 hours followed. Patients and study staff were blinded to the treatment groups. The primary outcome was overall mortality in the 4 weeks after injury. Secondary outcomes included vascular occlusive events, major surgical intervention, quantity of blood transfusion (if any), and cause of death (bleeding, vascular occlusion, multi-organ failure, head injury, or other cause).

TXA reduced all-cause mortality in the first month after trauma (relative risk [RR] = 0.91; 95% confidence interval [CI], 0.85 to 0.97; $P = .0035$; number needed to treat [NNT] = 68). There were 3076 deaths from all causes in both groups, 35% of which were the result of bleeding. Among patients who received TXA, the overall risk of death due to bleeding was 4.9%, vs 5.4% in the placebo group (RR = 0.85; 95% CI, 0.76 to 0.96; $P = 0.0077$; NNT = 119). For those treated with TXA within the first hour of injury, the risk of death due to bleeding was 5.3%, vs 7.7% for the placebo group (RR = 0.68; 95% CI, 0.57 to 0.82; $P < 0.0001$; NNT = 41). Giving TXA between one and 3 hours of injury also reduced the risk of death due to bleeding, to 4.8%, vs 6.1% for the placebo group (RR = 0.79; 95% CI, 0.64 to

0.97; $P = 0.03$; NNT = 77).

However, TXA administered more than 3 hours after injury, appeared to increase the risk of death due to bleeding, to 4.4% compared with 3.1% for the placebo group (RR = 1.44; 95% CI, 1.12 to 1.84; $P = 0.004$; number needed to harm = 77). Therefore, the earlier it is given after injury, the better the outcome.

TXA is easy to store and does not require refrigeration or reconstitution prior to administration. TXA has been included in both the United States and British army trauma protocols. In addition, TXA is used by National Health Service ambulances in the United Kingdom, and given to all adults and teenagers who incur major traumatic injury.³⁸ Given the short time window for its benefit, TXA may be most appropriate in the prehospital setting.

At present, there is no published data on TXA in paediatric major trauma. Prior use of TXA in paediatrics include the field of scoliosis³⁹ and cardiac surgery.^{40,41} The Royal College of Paediatrics and Child Health working group recommendations recognise that the adverse effects of TXA are rare and there are no thromboembolic effects noted from other uses.

Use of Recombinant Factor VIIa in Severe Bleeding in Major Trauma and MTP

Traditionally, recombinant activated factor VII has been successfully used in the management of bleeding paediatric patients with haemophilia and high anticoagulation factor antibodies titres. However, there were increasing off-label reports of its use for bleeding post major surgeries and in major trauma. Recent high quality systematic reviews of the use of recombinant factor VIIa for the treatment of bleeding in adult patients without haemophilia had not shown to offer any significant therapeutic advantage.^{42,43} Observational studies for its routine use during MTP protocols had also not shown any significant long-term survival benefits.⁴⁴⁻⁴⁶

Specifically for the paediatric population, a review of its use in the management of bleeding in the paediatric population without haemophilia showed that the lowest rates of complete response were seen in younger patients and patients with trauma. Reported decreased in the requirement of blood product transfusion after the use of factor VIIa (recombinant) versus standard therapies or placebo was commonly observed but was not statistically significant in many cases.⁴⁷

Given the lack of well-designed controlled studies and cost-effectiveness of its use, the current evidence is inconclusive regarding the safety and efficacy of factor VIIa (recombinant) for unlabeled indications in paediatric patients with severe trauma and during MTP.

Thromboelastometry and Goal-Directed Therapy in Haemostatic Resuscitation

Thromboelastometry® (TEM®, previously named rotational thromb-elasto-metry/-graphy) is an established viscoelastic method for haemostatic testing in whole blood. TEM® essentially analyses the viscoelastic properties of clot formation and the established point-of-care (POC) equipment currently in the market are ROTEM® and Thromboelastography® (TEG®).⁴⁸⁻⁵⁵

TEM® investigates the interaction of coagulation factors, their inhibitors, anticoagulant drugs, blood cells, specifically platelets, during clotting and subsequent fibrinolysis. The rheological conditions mimic the sluggish flow of blood in veins. TEM® can be performed with the ROTEM® or TEG® whole blood analysers and is an enhancement of thrombelastography, originally described by Hartert in 1948.⁵⁶

Rapid POC is to manage coagulopathy in major trauma and calibrate correction of coagulopathy in major trauma, and it is a much faster way to anticipate coagulopathy not just in polytrauma, but in ECMO and major surgery as well. TEM® also can sensitively identify patients with postinjury hypercoagulability. However, the use of TEM® to compare the degree of hypercoagulability and fibrinolysis after different types of trauma has not been described.⁵⁷

As in the standard prothrombin time/activated partial thromboplastin time (PT/APTT) standards, adjustments and calibrations must be made for the paediatric community. Oswald et al⁵⁸ from Austria analysed 407 children (American Society of Anaesthesiology Classification, ASA I and II) undergoing elective surgery in a prospective, 2-centre, observational study. Subjects were grouped into 0 to 3, 4 to 12, 13 to 24 months, 2 to 5, 6 to 10, and 11 to 16 years. Study objectives were to establish age-dependent reference ranges for ROTEM® assays, analyse age dependence of parameters, and compare ROTEM® data with standard coagulation tests.

Data from 359 subjects remained for final analysis. Except for extrinsically activated clot strength and lysis, parameters for ROTEM® assays were significantly different among all age groups. The most striking finding was that subjects aged 0 to 3 months exhibited accelerated initiation and propagation of coagulation.

We should therefore move towards adopting TEM® for acute use in managing coagulopathy in paediatric polytrauma so that in addition to MTP for paediatrics, we can institute a more goal-directed management plan in correcting and managing coagulopathy.

Conclusion

In conclusion, the management of haemorrhagic shock in major trauma for children needs to be refined. In major trauma, there must be a focus not just on volume/haemodynamic resuscitation but also haemostatic resuscitation to address coagulopathies rather than volume alone.

For volume replacement, blood should be used when the threshold is reached. While there is a role of permissive hypotension in adult polytrauma sans severe head injury/spinal shock—there is no role for this for paediatric major trauma. One should instead consider permissive tachycardia with a normal blood pressure measurement in such cases without severe head injury/spinal shock as children do not respond in the same physiological manner as in adults during shock.

One should also move towards more defined and refined goal-directed approaches to correcting the coagulopathies in polytrauma through use of POC tests like ROTEM® or TEM®. Finally, robust studies in the paediatric population need to be done to ascertain the use and timing of prohemostatic agents like recombinant factor VIIa.

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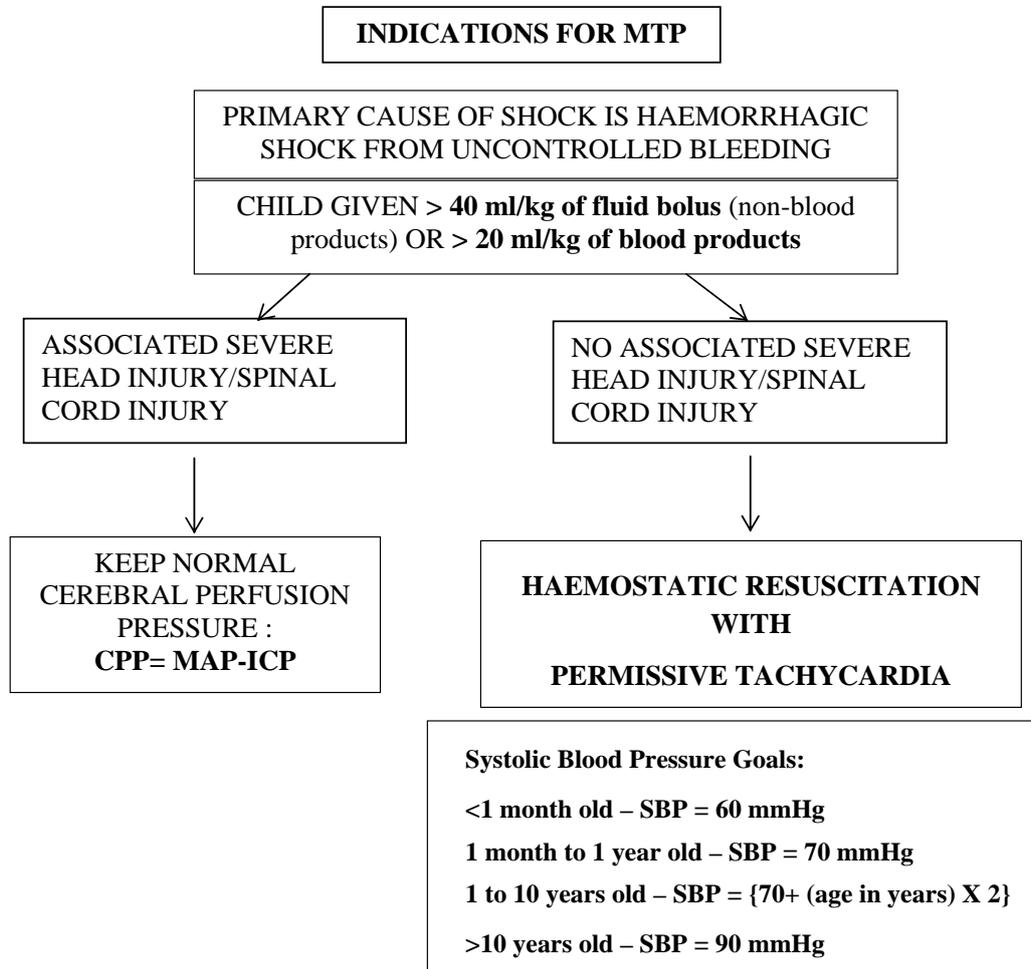
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Appendix 1
KK Women’s and Children’s Hospital’s MTP for Paediatrics

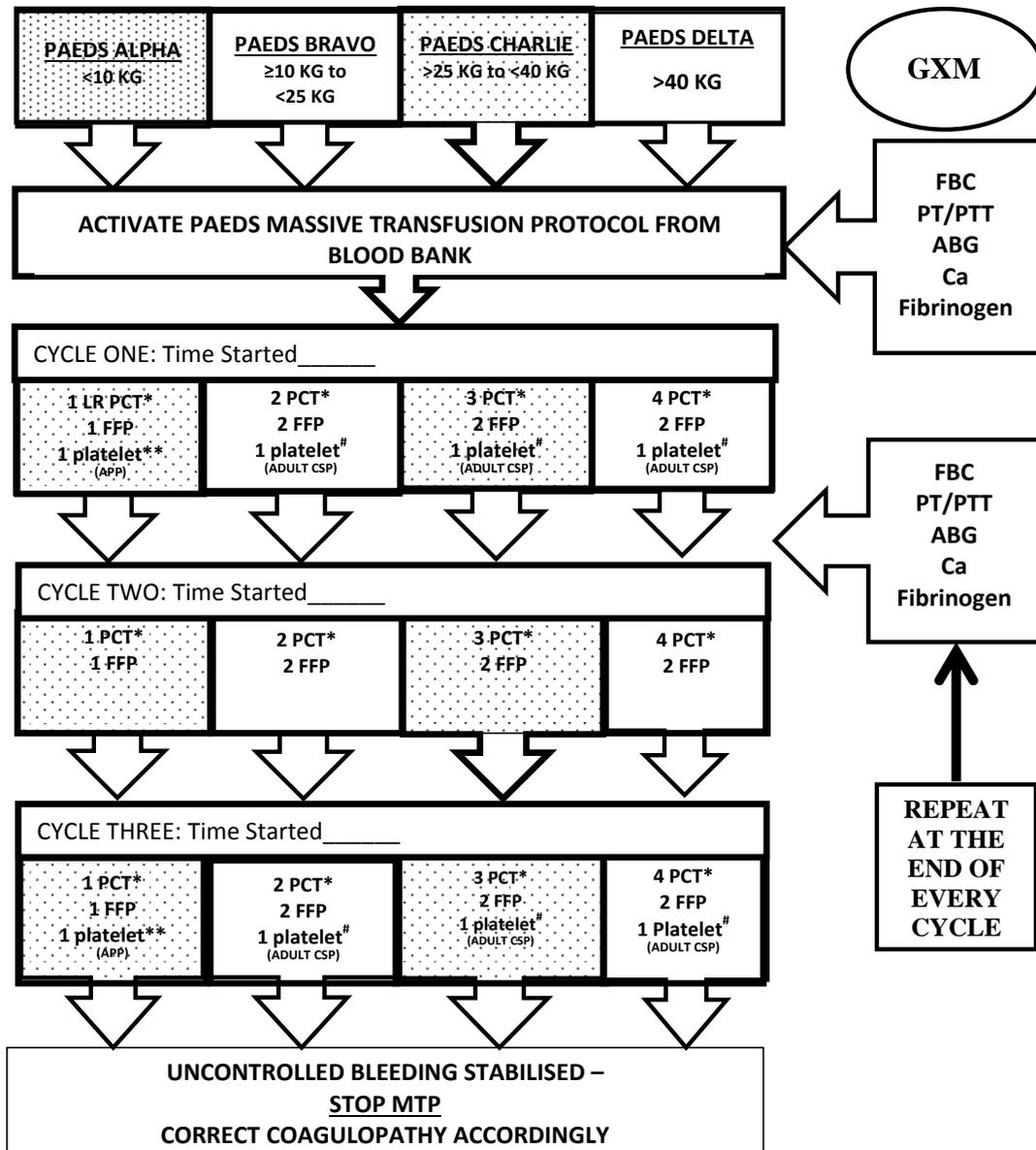
Massive transfusion is defined* in adults as replacement of >1 blood volume in 24 hours or >50% of blood volume in 4 hours (adult blood volume is approximately 70 mL/kg). In children, it is defined as transfusion of >40 mL/kg (blood volume in children over 1 month old is approximately 80 mL/kg).
 *As defined by the Australian Red Cross Blood Service



- MONITOR FOR:**
- Temperature >35⁰ C and treat hyperthermia aggressively
 - Treat hypotension aggressively in patients with head or spinal cord injury
 - Full blood count
 - Platelets $\geq 50 \times 10^9 /L$ if no significant head injury
 - Platelets $\geq 100 \times 10^9 /L$ if significant head injury present
 - Blood gases
 - pH >7.2
 - Base excess < -6
 - Ca²⁺ >1.1 mmol/L
 - K⁺ : monitor for hyper K⁺
 - Coagulation profile
 - PT/APTT <1.5 × normal
 - INR ≤ 1.5
 - Fibrinogen (if done) >1.0 g/L
 - Lactate (if done) <3 mmol/L

Appendix 2

MTP for Paediatric Trauma in KK Women’s and Children’s Hospital



Notes

PCT*: Packed cells are O+ for MTP (should the patient be known to be O-, further follow-up and counselling will be undertaken following subsequent patient stabilisation)
 LR PCT: Leukocyte-reduced packed cells
 Platelets** (for less than 10 kg): APP (Apheresed platelets)
 Platelets# (For >10 kg): Adult platelets (CSP or pooled platelets)
 If ionised calcium <1 mmol/L, give 0.1 ml/kg 10% calcium gluconate
 Fibrinogen levels are not processed in-house
 Haemostatic resuscitation
 If ongoing haemorrhage after 1st Box 3 - if PT/PTT prolonged, consider additional 20 ml/kg of FFP
 If fibrinogen <1 g/L, consider additional 5 ml/kg cryoppt and IV tranexamic acid 10 mg/kg (max 1 g) over 10 minutes and 8 H/prn (NB: cryoppt will not be available stat)
 Keep platelets >50, KIV consider additional 10 ml/kg platelets if lower