15.0 PEDIATRIC CONSIDERATIONS

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Introduction

Care provided to children experiencing massive hemorrhage needs to be standardized. While 70% of North American Hospitals that provide pediatric healthcare services have a dedicated MHP, there is a significant amount of variability including activation criteria and products administered[1] and many Ontario community/small hospitals lack a hospital approved MHP, particularly pediatric specific protocols.[2] The lack of pediatric specific MHPs is concerning as more than half of these hospitals receive critically injured infants, children and adolescents. As a result, the majority of these patients are transferred from peripheral hospitals to pediatric tertiary care centers rather than being admitted directly through the emergency department.[3] The event rate of large volume transfusions account for approximately 3% of children treated for trauma in a pediatric tertiary care emergency department setting.[3,4,5] For the most part, the epidemiology of massive hemorrhage is similar in adults and children and it is a leading cause of preventable death in both trauma (i.e. motor vehicle collisions, non-accidental physical trauma, falls, homicide and suicide) and elective surgical settings (i.e.. solid organ transplant, tumor resection, congenital heart disease, craniosynostosis and scoliosis repair).[6,7,8] Trauma is the leading cause of death in children > 1 years of age and attributed primarily to traumatic brain injury (TBI), however hemorrhage is the leading cause of preventable death. In the operating room, massive hemorrhage is the leading cause for cardiac arrest in children.[9] Finally, approximately 6 per 10,000 live births result in an emergency blood product request for a neonate experiencing massive hemorrhage in utero or during birth from placental abruption/placenta previa with a mortality rate of 35%.[10] Limited availability/access to a standardized pediatric MHP appears to be an obvious gap in care.

Given the above, the lack of evidence-based guidelines for transfusion during MHP activation in children is concerning.[11] Fortunately, massive hemorrhage is an uncommon event. The current definition is 40 ml/kg of blood products administered over 24 hours,[12] but it does not include the young child in hemorrhagic shock who receives 35 ml/kg of RBCs over an hour in the trauma bay or consider hemostatic resuscitation information within the first three hours of injury, when the majority of deaths occur.[13] Most treatment guidance is based on observational data or extrapolated from the adult literature. Compared to adults, a child's immature body habitus raises issues of scale related to blood product administration (ml/kg vs. units), contributes to mechanistic differences in traumatic injury (higher prevalence of TBI, blunt > penetrating trauma and less exsanguination) and a predisposition to hypothermia. In children, higher "transfusion triggers" may be required that depend on comorbidities (e.g., cyanotic heart disease), age (e.g., premature infant) and the presence of coagulopathy (e.g., after cardiopulmonary bypass for congenital heart disease repair). All of these factors should be accounted for in a pediatric MHP. When utilized in pediatric trauma settings, MHPs have demonstrated improved system resource utilization, speed of blood product delivery to the bedside and decreased patient exposure to blood products,[14,15,16,17,18] however, evidence for outcome benefits in pediatric trauma or surgical settings remain elusive.

The contents of this section of the MHP Toolkit are dedicated to key pediatric MHP domains specific guidance for pediatrics includes MHP activation/termination criteria, damage control resuscitation, laboratory testing, resuscitation targets, hypothermia, hyperkalemia, TXA, fibrinogen and prothrombin complex concentrates, transport and handover and continuing education. Pediatric transfusion literature is less robust compared with similar adult literature. Much of the chapter content is either extrapolated from the adult literature or primarily based on single-center pediatric studies of retrospective or observational design, with varying definitions of massive hemorrhage, non-random treatment allocation and high potential for confounding. Outcomes are clouded by associated traumatic brain injury, inconsistent adherence to transfusion protocols and varying age groups. Massive hemorrhage literature associated with elective surgery is under represented. Please refer to the associated learning aids contained within the appendices (Pediatric Appendices A-G).

15.1 Activation and termination

Recognizing significant blood loss in children can be deceiving even for experienced clinicians.[19] Blood volume varies from the premature (90-100 ml/kg) to the older (> 3 months) term infant (70 ml/kg) and in the obese child (65 ml/kg).[20] Adult massive hemorrhage predictions scores are available, but they have limited application in children.[21] Children have robust cardiovascular compensatory responses that delay diagnosis of 30-40% blood volume loss. Subtle changes in the heart rate and extremity perfusion may signal impending cardiorespiratory failure.[22] Hypotension is considered a late finding in pediatric hypovolemic shock and an independent predictor of death when related to trauma.[23] As such, a shock index that is pediatric age-adjusted (SIPA=heart rate/systolic blood pressure) may more expeditiously identify the child at increased risk for emergent intervention[24,25,26] A SIPA > 1 may indicate a need for blood transfusion in children 1-12 years old.[27] Pediatric massive transfusion has been arbitrarily defined in terms of circulating blood volume lost (or blood products transfused) including 50% of total blood volume in 3 hours or 100% of total blood volume in 24 hours.[28] The threshold of 40 mL/kg total blood products administered within a 24 hr time-interval most reliably identify pediatric patients at risk for early or in hospital mortality[12] and has been widely adopted as a pediatric massive transfusion definition. This definition is limited in its ability to trigger activation of the MHP, as both adult[29] and pediatric data suggest that most bleeding requiring MHP activation is intense and occurs in a short time frame (< 3 hours).[23,30] Also, guidelines are now advocating for less aggressive crystalloid resuscitation to avoid a dilutional coagulopathy.

In the absence of defined pediatric MHP triggers, some combination of the following parameters would imply the potential for significant hemorrhage:

- Shock Index Pediatric Age Adjusted (SIPA) 1-12 yrs > 1[25,27]
- Continued hemodynamic instability after two 10 ml/kg boluses of crystalloid[27]
- Continued hemodynamic instability after 20 ml/kg RBC
- Estimated need for more than 40 ml/kg RBC in 3 hours[12,31]
- Penetrating injury[21]
- Glasgow coma scale ≤ 12
- SBP <80 mmHg ≤ 5yrs and <90 mmHg 6-12 yrs

It should be noted that in contrast to adults, it is unknown if the presence of a TBI in children impairs the above shock index performance to classify hypovolemic shock or predict need for blood products.[32] Hemodynamic parameters and the SIPA may not apply in an elective surgical setting. MHP termination criteria may include:[33]

- Bleeding source control has been attained,
- Hemodynamic stability has been achieved,
- Vasopressor requirements have diminished,
- Transfusion rate has slowed such that additional transport personnel are no longer required.

15.2 Damage Control Resuscitation

While the tenents of DCR described previously also apply to children, they have unique MHP considerations in terms of body habitus and physiology which influence the type of injuries experienced and treatment decisions including their consequences, as detailed below.[39,40]

Anatomy and physiology considerations:

Compared with adults, children have less fat, more elastic connective tissue, and a pliable skeleton protecting tightly packed and proportionally larger solid intra-abdominal, abdominal organs and thoracic structures. Similar to adults, large amounts of blood may be lost internally secondary to a long bone fracture, retro-peritoneal bleeding or blunt abdominal trauma. Children suffer relatively more solid organ injury from both blunt and penetrating mechanisms,[22] Intra-abdominal internal organ injury is associated with 30% of major traumas in children, associated skeletal injury is

uncommon and is the most common cause of unrecognized fatal injury in children.[41] However, children have greater hemostasis associated with blunt solid organ injuries; non-operative management is common.[41] When bleeding does occur, children arrest suddenly without hypotensive decline as is seen in adults.[22] A proportionately larger head with reduced cervical muscle control is associated with a higher frequency of traumatic brain injury (TBI), the leading cause of death in pediatric trauma patients. In addition, pediatric head injury predisposes to a proportionately increased blood loss from the scalp or into the cranium, particularly in infants. Finally, children are predisposed to hypothermia during and after massive bleeding.

Coagulopathy:

Children are vulnerable to trauma-induced coagulopathy (TIC) defined as INR prolongation (>1.8) and increased base deficit (>6). They reflect extensive tissue injury and significant hemorrhagic shock, respectively. It is associated with increased morbidity and an elevated mortality rate.[57,58,59] Acidosis and hypothermia associated with major trauma or surgery impair pro-coagulant factors as well as platelets. Platelet numbers decrease earlier in massive hemorrhage related to trauma compared with major elective surgery (e.g., scoliosis) in children.[28] Typically, during massive hemorrhage, fibrinogen levels drop before all other coagulation factors are depleted.[60] Fibrinogen concentrate is an effective treatment for acquired fibrinogen deficiency in children.[61,62,63,64]

In children, a clear definition of TIC (comprised of acute traumatic coagulopathy [ATC] and iatrogenic coagulopathy [IC]) is lacking[133] and a similar mechanism of ATC has not been fully elucidated.[134,135] In the presence of severe TBI children may experience fibrinolytic dysregulation leading to either hyper-fibrinolysis or associated fibrinolysis shut-down.[51,52,53,83,136] Early or premature platelet and plasma administration may exacerbate the latter condition.[48,50,53] In pediatric literature there is clinical equipoise regarding the mortality benefit of high plasma to RBC ratios in pediatric trauma[14,42,43,137,138,139,175] and increased morbidity and mortality have been associated with plasma transfusions in critically ill children.[53,140] Similar to adults,[35,36,37] mortality benefit from platelets has not been reported in pediatric combat and civilian trauma treated for massive hemorrhage[42,43,138,139] and prophylactic platelet transfusion has been associated with increased major bleeding events and mortality with higher compared with lower transfusion thresholds in neonates.[47] A 2% increase in mortality for every additional standard dose of platelets (10 ml/kg) administered has been reported in a pediatric critical care setting.[141]

Avoiding secondary injury:

Damage control surgery

In order to manage pediatric patients with massive injuries associated with severe bleeding, damage control resuscitation often involves surgical intervention. The goal is to prevent secondary injuries and damage control surgeries such as a damage control laparotomy (DCL) is implemented quickly to attain hemostasis. DCL is relatively understudied in children.[65,66] It has an incidence of approximately 12% in children with abdominal trauma and is associated with hypothermia and multi-system trauma.[41] Perioperative transfusion is often encountered and intra-abdominal hypertension and abdominal compartment syndrome are associated with excessive fluid resuscitation, shock[67] and a high mortality rate,[65] but are under recognized despite their common occurrence (0.6-13% incidence). Liver hypo-perfusion (e.g., aortic cross-clamp above hepatic artery to maintain blood pressure) may result in citrate toxicity, causing life-threatening hypocalcemia and progressive coagulopathy or ischemia leading to hyperkalemia.[39,68] Massive transfusion related electrolyte disturbances including hypomagnesemia are relatively common and thus close monitoring is required.[15]

Fluid management

There are negative consequences of fluid over resuscitation in children. Both morbidity and mortality are directly related to increasing volumes of crystalloid, which should be limited to 20 ml/kg in the hemorrhaging pediatric patient prior to moving to blood, as recommended by the 10th edition of the Advanced Trauma Life Support (ATLS) manual.[40,69] Large volume resuscitation has been linked to increased intensive care unit length of stay, ascites, pleural effusions, and sequential organ failure.[15,40] Permissive hypotension to a low targeted blood pressure has not gained wide traction in pediatric trauma due to a high prevalence of associated TBI, a contraindication to permissive hypotension.[39,70] Prolonged transport times in the Canadian pediatric trauma population are not conducive to extended periods of permissive

hypotension and should be limited to the adolescent population with exclusively penetrating trauma and short transport times. It should only be viewed as a temporary strategy to allow for definitive surgical management of bleeding.[45,71] Information regarding recommended BP targets and the appropriate use crystalloids or vasopressors in children experiencing massive hemorrhage is limited.

Traumatic brain injury

Care delivery in the massively bleeding pediatric trauma patient is complicated by the presence of a TBI. There is a higher incidence of blunt trauma and associated TBI in children compared with adults. In TBI, permissive hypotension is contraindicated. Vigilant hemodynamic control is required and a cerebral perfusion pressure (Mean arterial minus the greater of either intracranial or central venous pressure) target between 40 (infants) and 50 (adolescents) mm Hg is recommended; and an intracranial pressure < 20 mm Hg is suggested.[72] Excessive crystalloid may exacerbate cerebral edema and associated mortality with TBI.[73,74] Severity and duration of hyperglycemia after TBI is associated with worse outcomes,[75,76] however, there is no benefit to target glucose concentrations lower than 8.3-10 mmol/L (150-180 mg/dl) in critically ill children.[77,78] Therapeutic hypothermia has not been shown to improve outcomes in pediatric patients with TBI.[79,77] Patients with blunt injury including TBI are at increased risk of trauma induced coagulopathy (TIC) thus making active warming to 36°C a priority.[58,57] Coagulopathy is commonly reported in children with isolated TBI as a dysregulation of fibrinolysis with aspects of disseminated intravascular coagulation.[80,81,82] It should be noted that the clinical presentation of TIC can be complicated when TBI is combined with poly-trauma.[52,53,59,83] The role of TXA in pediatric TBI is unclear and is reviewed in the associated section.

15.3 Laboratory tests

There are unique considerations in the drawing, processing and type of blood samples required in pediatric patients, which should be grouped together when possible to simplify collection and minimize the volume and number of samples. Intraosseous blood sampling for laboratory testing, with exception of blood group typing, is not recommended in children or adults if other access can be obtained.[84]

Typically, sites with smaller red blood cell inventories will be able to manage an MHP activation in young children with their on-site Group O units; however, any delay in switching to group specific RBCs in older children/adolescents can quickly deplete the O unit inventory. Beyond the more common blood gas, electrolytes and biochemistry testing, blood glucose is recommended as an additional minimum lab test to be measured early on in an MHP activation, and monitored closely throughout. Both hypo- and hyperglycemia can occur in pediatric trauma patients and is of particular concern in the presence of severe TBI. Magnesium deficiency should be anticipated in the child who has lost 1.5-2 blood volumes, particularly in the setting of arrhythmias refractory to calcium supplementation.[28]

15.4 Resuscitation Targets

As there is a significant risk of over transfusion in the pediatric patient, they must be transfused based on per kg dosing for blood products, fibrinogen concentrate and PCCs as outlined in Appendix B. Total volume of RBCs and plasma transfused need to be tracked in order to maintain an appropriate ratio of product administered. While ideally blood product transfusion should be laboratory-guided, rapid exsanguination may require a formula-based approach (10-20: 10 ml/kg RBC to plasma ratio). Inadequate platelet counts can also be expected earlier in the resuscitation of a trauma compared with an elective surgical patient.[20,28] Importantly, pediatric patients who are adult-sized (> 40 kg) and > 12 years of age should be managed using the adult-directed algorithm.

Thresholds for blood product administration in children should consider developmental changes in the hematologic and coagulation systems throughout childhood. The hemoglobin concentration approaches adult levels by 12 weeks of age. In children, the medical literature recommends hemoglobin should be maintained between 70-100 g/L.[86,87,88,89] A hemoglobin transfusion threshold of 80 g/L accounts for unpredictable blood loss and hemodynamic instability during massive hemorrhage. Certain pediatric populations, such as neonates, patients with congenital heart disease, those receiving extracorporeal life support, or are in severe respiratory distress, may, however, require higher thresholds for RBC transfusion.

Unlike RBCs, the coagulation system undergoes significant changes during childhood, most marked in the first six months of life. Although platelet numbers are equivalent to adults at birth, platelet function and both pro-coagulants (with the exception of fibrinogen and factors V and VIII) and most of the natural anticoagulants (e.g., protein C) concentrations are reduced.[90,91,92] Most anticoagulant concentrations achieve adult levels by the age of 5 years, however, coagulation factor level concentrations vary through childhood and adolescence, achieving adult levels in the mid-teens. Only the upper limit of the reference value for PTT in infants is higher than in older children and adults.[93,94] Age-appropriate reference intervals should be applied when evaluating laboratory coagulation data, particularly in neonates.

There is limited data to suggest laboratory investigations of coagulation are useful to diagnose a clinical coagulopathy or guide hemostatic therapy during MHP activation.[55,83,95] A threshold platelet count of 50 x 10^9/L requiring treatment is not evidenced based, but is routinely recommended.[35,43,46] There is growing interest in the pediatric surgical and trauma literature to use viscoelastic technology; however, it has yet been proven to improve outcomes and its role in hemostatic resuscitation remains unclear.[15,96,97,98] In Ontario, viscoelastic goal-directed management of coagulopathy is uncommon in children, even when available to providers.[96]

15.5 Hypothermia

Pediatric patients have physiological differences from adults (e.g., less body fat, higher body surface area to volume ratio) which increase their susceptibility to hypothermia.[99] Hypothermia is an independent risk factor for coagulopathy, arrhythmias, acidosis, transfusion and mortality in pediatric trauma.[100,101,102] Considering the morbidity and mortality associated with hypothermia in massively bleeding children, all patients should receive interventions aimed to prevent hypothermia (goal >36°C).[103] Heat loss occurs through conduction, convection, radiant and evaporative mechanisms.[103] Multiple passive and active warming strategies targeting the patient and environment should be utilized simultaneously to prevent and/or treat hypothermia. These include warming the external environment, removal of wet clothing/blankets, applying a clear plastic cover sheet or attaching a heat and moisture exchanger to an endotracheal tube.[104] Active rewarming using convective air blankets (e.g., Bair Hugger™ designed for over or under patient placement), overhead radiant heaters or hospital-grade exothermic chemical pads placed under the child should be initiated with a core temperature of <36°C. Intravenous fluids can be warmed in-line or placed in a warming cupboard prior to administration. Multiple modalities are available to measure temperature (e.g., esophageal, nasopharyngeal and bladder); rectal temperature has been shown to have high correlation to brain temperature in children with TBI in the normal temperature range.[105] Please refer to appendices C and D for pediatric MHP approaches to blood product administration and temperature preservation, respectively.

15.6 Hyperkalemia

Hyperkalemia due to red blood cell transfusion is second only to hypovolemia from blood loss as the most common causes of cardiac arrest in the pediatric perioperative environment.[9] Ease of rapid administration of RBCs into a volume contracted and acidotic small child are key risk factors. Non-irradiated red cell units should be provided, especially in younger children who are at higher risk of fatal hyperkalemia.[106] To reduce the risk, vigilant hemodynamic management, avoidance of transfusion through central lines, use larger bore (>23G) and shorter peripheral IV catheters (to reduce red cell shear/hemolysis), as well as frequently monitoring for ECG and electrolyte abnormalities frequently (e.g., hypocalcemia and acidosis) including vigilant ECG monitoring is recommended.

15.7 Tranexamic Acid

TXA administration is not yet standard practice for use in pediatric trauma patients, but is often used in children and adolescents requiring transfusion within the same time parameters as adults. Prospective evidence of benefit or harm is not yet available for TXA in children with trauma, but clinical trials are in progress and an on-going pediatric civilian trauma study (Tic-Toc trial) is evaluating TXA both in thoraco-abdominal and TBI hemorrhage as a feasibility study in preparation for a definitive large multi-center prospective trial.[108,109] In pediatric combat casualties, TXA has been shown to be independently associated with decreased mortality and improved discharge neurologic status.[110] For pediatric

patients the initial bolus of TXA can be dosed at 15 mg/kg up to a maximum of 1 gram and additional doses/infusion based on local policy with empiric dosing at 2-5 mg/kg/hr until the bleeding stops. Pharmacokinetic simulation data suggest initial doses between 10-30 mg/kg and infusion rates of 5-10 mg/kg/hr may be most effective.[111] Reports suggest a reduction in blood loss and transfusion requirements in children receiving TXA for cardiac surgery, craniosynostosis surgery and spinal fusion for scoliosis repair.[111,112] Administration should be over 10 minutes either in 100 ml of fluid or as a slow IV push. While TXA may be associated with seizures particularly with high bolus doses (100 mg/kg) or in susceptible surgical populations (e.g., cardiac or trauma)[113], TXA is not currently contraindicated in children with an underlying seizure disorder.[111] However, renal impairment/dysfunction requires a dose adjustment because of the increased risk of drug accumulation.[111] A recent report of survivor benefit in adults with isolated mild to moderate TBI receiving TXA, has not been extrapolated to adopt its routine use in Canadian children with similar clinical findings.[114]

15.8 Fibrinogen and Prothrombin Complex Concentrates

In Canada, fibrinogen concentrate is indicated for coagulation support in children. In adults it has been shown that early use of fibrinogen concentrates can increase survival, reduce bleeding and transfusion requirements.[142,143,144] However, the best approach to incorporate fibrinogen concentrate in hemostatic resuscitation in children and adults is unknown. Although the use of fibrinogen concentrates appears to be safe and effective in the treatment of acute bleeding, there is a paucity of evidence directly supporting the use of fibrinogen concentrates in pediatric massive hemorrhage protocols.[145,146] Nonetheless, the perceived risk of thromboembolic events is low and reports in adult and pediatric surgical patients suggest no difference in thromboembolic complications when fibrinogen concentrate was compared with placebo/comparator.[147,148] Considering the effectiveness and safety reported in adults to treat bleeding and emerging evidence of off-label use in children and neonates we recommend the administration of fibrinogen concentrates as part of our massive hemorrhage protocol.[63,97,145,149,150] We recommend administering a dose of 50 mg/kg (maximum single dose 4 gms, 2 gms if < 30 kg) to maintain fibrinogen level > 1.5 g/L (> 2 g/L may be required with critical bleeding) or as guided by viscoelastic point of care testing. For sites that do not stock frozen plasma or cryoprecipitate, fibrinogen concentrate should be used as a fibrinogen substitute. For sites that have the option to carry either frozen plasma or cryoprecipitate, fibrinogen should be considered as first line for fibrinogen replacement. If fibrinogen concentrate is unavailable, cryoprecipitate 5 ml/kg should be considered an equivalent dose for fibrinogen replacement.

Similar to fibrinogen concentrates, prothrombin complex concentrates (PCCs) are indicated for coagulation support in Canadian children. However, they should be considered as a third line hemostatic treatment in massive hemorrhage where coagulopathy is a contributing factor because pediatric trial-related efficacy, dosing and safety data are unavailable.[31,151] In contrast to early depletion of fibrinogen during massive hemorrhage, in adults thrombin generation in the early stages of trauma-related bleeding is often increased suggesting reduced thrombin generation is a late finding in massive hemorrhage related to trauma or surgery.[133,152,153,154,155] It is unclear if a similar pattern of a delayed reduction in thrombin generation during trauma or surgery occurs in children.[134,156] Evidence of early reduced thrombin generation is associated with increased mortality.[152,155] PCC can be considered a “plasma substitute”, and it benefits from many of the same advantages as fibrinogen concentrate (e.g. low risk of pathogen transmission and transfusion reactions) and most importantly, the volume administered is inconsequential. Concerns about TACO and worsening of coagulopathy through plasma-related dilution of RBCs, platelets and coagulation factors are avoided with the use of PCCs, but there is a theoretical increased risk for venous thromboembolic (VTE) events up to 3-4 days after administration.[155]

Pediatric PCC data is relegated to case reports in severely injured children or case series of neonates to adolescents utilizing 4-factor PCCs to treat perioperative bleeding or provide rapid VKA reversal in a cardiac surgery setting.[31,157,158] Reported complications were rare (limited to a single DVT) and children may have a lower risk for VTE, in part due to their quantitative and qualitative differences in hemostasis and reported lower thrombin generation (reduced by as much as 26%) in older children[90,134,159] and appears to be reflected in a lower incidence of VTE in children (0.02-1.2%) compared with adults (20-58%) in the absence of thrombo-prophylaxis.[135,160] It is possible VTE incidence is under reported in children.[160,161] Nonetheless, VTE risk continues to be perceived as low in children as routine VTE prophylaxis is not recommended for age <12 years old, unless there is a history of VTE or presence of a femoral central venous line.[162,163,164] Also, only four-factor PCCs (compared to 3-factor) are licensed in Canada and are associated with fewer thromboembolic events[165] and do not increase the thromboembolic risk over plasma for VKA reversal in

adults.[166,167,168,169,170] In addition, increased thromboembolic events have been reported in animal studies at higher PCC doses (>34 IU/kg).[171,172] Considering the data available, we recommend PCCs combined with vitamin K as described in the associated dosing table to rapidly reverse VKAs with the goal to reduce the INR<1.8.[157] We also recommend a moderate dose of PCC (25 IU/kg) as a third line treatment after tranexamic acid and fibrinogen concentrate, but it should only be administered with ongoing massive bleeding where coagulopathy is a contributing factor despite treatment for other causes (e.g. hyper-fibrinolysis, low fibrinogen or platelets or platelet dysfunction).[173] A low risk of a thrombotic complication should be weighed against the need for rapid and effective correction of a potentially fatal coagulopathy. For sites that do not carry frozen plasma or cryoprecipitate, PCC should be used as a frozen plasma substitute, while sites that carry frozen plasma or cryoprecipitate, frozen plasma should be considered first line for coagulation factor replacement. No recommendation can currently be provided on the need for thromboprophylactic measures in children treated with PCC.

15.9 Transport and handover

Infants and children (< 13 years old) subject to or are at risk of MHP activation should be cared for in a pediatric definitive-care hospital setting to receive specialized care.[115,116] Children cared for in a non-definitive care hospital setting, particularly in rural communities, should trigger early/urgent consultation with a specialized service using an MHP-associated standardized hand-over tool to facilitate patient care and transfer to a pediatric definitive care setting (see Pediatric Appendix E for an example of a patient hand-over tool).

15.10 Education

Care of children undergoing an MHP needs to be incorporated in training materials and simulations/drills. Steps for MHP adoption, implementation and compliance in a clinical setting are described elsewhere. A pediatric MHP has the added challenge of being a rare event with several unique considerations compared with adults as previously discussed. As such, annual review of a pediatric specific MHP using in-situ simulation (see Appendix G for script).[117,118,119,120,121,122] Please refer to Appendices (A-G) for examples of pediatric cognitive aids including an MHP algorithm, medication dosing table and related infographics. A pediatric MHP bag or cart containing equipment and drugs with attached cognitive aids in a location known to all providers is a simple first step to reduce provider anxiety and variability in pediatric care. Expired equipment can be used for training scenarios.[123]

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