Management of the Pediatric Organ Donor

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Introduction

The demand for organs and tissues continues to increase despite efforts to increase the donor pool. Historically, patients in need of organs and tissues have died from their organ failure, but these patients are now living longer because of advancements in pharmacologic and medical technology. Advancements in physiologic support mechanisms such as mechanical ventilation, cardiac assist devices, and renal and liver dialysis have extended these patients' lives, increasing the need for donors. Furthermore, the use and refinement of passenger safety restraint systems,[1] helmets, and other safety measures, as well as increased awareness of such precautions and technology,[2,3] have led to a reduction in accident-related fatalities, dramatically reducing the numbers of potential donors; obviously, these advancements are very important in the human condition, but they impose significant pressure to offset the discrepancy between supply and demand in the field of organ donation. The Organ Donation Breakthrough Collaborative efforts to increase organ donation has resulted in a 10.8% increase in the United States during 2004. However, despite these efforts, a large gap continues to exist between the number of donors and recipients.[4]

The ability to recover appropriate and viable organs for the pediatric population comes with a unique set of limitations, making the acquisition of these organs difficult. Size and weight constraints are a major limiting factor, most notably for small children and infants. Furthermore, a greater proportion of potential pediatric organ donors suffer brain injury as result of anoxia and ischemia, making the certainty of the diagnosis of irreversible brain injury more difficult. Therefore, an age-related variation in the timing associated with the confirmation of neurologic death in pediatric donors is suggested. The duration of the waiting period to pronounce neurologic death in children may affect the viability of suitable organs for transplantation by delaying their acquisition. Lastly, the specialized care required for aggressive management of the pediatric organ donor following neurologic death may be lacking at institutions that have limited expertise and support for children.

The management of potential pediatric organ donors requires knowledge of the physiologic derangements associated with this specific patient population. Typical derangements include metabolic and endocrine abnormalities, aberrations in ventilation and oxygenation, hemodynamic instability, and coagulation disturbances. In addition to meticulous management of physiologic derangements, care of the family provided by a team of social workers, chaplains, and other support staff is integral.[5,6] This chapter focuses on the management of potential pediatric organ donors who have progressed to neurologic death. Meticulous care of pediatric organ donors is essential to ensure successful recovery of organs in this selected group of patients.

Donor Suitability

Trauma is the leading cause of neurologic death in both children and adults, although asphyxia and hypoxic-ischemic insults are also significant causes of neurologic devastation and death in children.[7,8] Unique conditions in which nonaccidental trauma has resulted in the death of a child require close

cooperation between forensic investigators, treating physicians, the transplant team, and the organ procurement organization to allow for successful procurement of organs.[9-14] Protocols to facilitate organ recovery in child abuse victims can decrease denials for organ donation from medical examiners.[15,16] In addition, involvement of the district attorney during protocol development should be a consideration.

Pediatric donors become eligible for organ procurement after the determination of neurologic death has been made. Although the vast majority of pediatric donors will be standard criteria donors, donation after cardiac death (DCD), or non-heart-beating organ donors, has the potential to increase organ donation in children.[17] An in-depth discussion about pediatric DCD is beyond the scope of this chapter; however, any child in which a "do not resuscitate" or "withdrawal of care" occurs in the course of management, or any child who expires a nonneurologic death should be considered as a potential DCD donor. The reevaluation of this common means of recovering organs before the development of brain death criteria continues to intensify as attempts are made to meet the demands of a growing national transplant list. In addition, DCD focuses on recovery of the two most commonly needed organs for children, liver and kidney. The most important point is that organ donation should be considered in any patient where end-of-life issues are being discussed.

The Determination of Brain Death

The determination of neurologic death in children remains a clinical diagnosis and is no different than in adults. No unique legal issues exist differentiating declaration of neurologic death in children. However, age-related issues can make confirmation of irreversible injury and declaration of neurologic death more difficult, resulting in age-based recommendations.[18]

The cause of coma and brain injury must be determined to ensure that an irreversible condition has occurred. Duration of observation and the need for ancillary tests should be based on history and clinical examination. Physical examination criteria for neurologic death rely on the coexistence of coma and apnea in a child that is not significantly hypothermic, hypotensive for age, and has not received recent doses of sedative or neuromuscular blocking agents. Absence of brainstem function is defined by the manifestation of all of the following features on physical examination: mid-position or fully dilated nonreactive pupils; absence of spontaneous eye movements induced by oculocephalic or oculovestibular testing; absence of cough, corneal, gag, and rooting reflexes; and absent respiratory effort off ventilator support. The examination results should remain consistent with brain death throughout the observation and testing period.[18]

The apnea test is essential for the confirmation of neurologic death. Testing for apnea must allow adequate time for $PaCO_2$ to increase to levels that would normally stimulate respiration. Apnea testing must be performed while maintaining normal oxygenation and stable hemodynamics.[19-21] Patients should be preoxygenated with 100% oxygen to prevent hypoxia, and mechanical ventilation should be discontinued or changed to continuous positive pressure ventilation while observing the patient for any spontaneous

Table 1. RECOMMENDED OBSERVATION PERIOD AND ANCILLARY TESTING ON THE BASIS OF THE AGE OF THE PATIENT[18]

7 days to 2 months
• Two examinations and EEGs separated by at least 48 hours
2 months to 1 year
• Two examinations and EEGs separated by at least 24 hours; a repeat EEG is not necessary if a cerebral radionuclide scan or cerebral angiography demonstrates no flow or visualization of the cerebral arteries
Older than 1 year
• When an irreversible cause exists, ancillary testing is not required and an observation period of 12 hours is recommended.
• The observation period may be decreased if the EEG demonstrates electrocerebral silence or the cerebral radionuclide or cerebral angiography study demonstrates no flow or visualization of the cerebral vessels.

Abbreviation: EEG, electroencephalogram

respiratory movements over a 5- to 10-minute period. The $PaCO_2$ should be measured and allowed to rise to 60 torr or greater. If no respiratory effort is noted during this time, documentation of the apnea test consistent with neurologic death is noted, and the patient is placed back on mechanical ventilation support until death is confirmed with a repeat clinical examination, or ancillary testing.[22,23] The recommended clinical observation period in children differs from adults, with a greater duration between examinations suggested for younger children. Table 1 lists guidelines recommended from the Special Task Force for Brain Death Guidelines in Children.[18] Observation periods have never been validated and should be used as recommendations only. Many authors agree that except in very immature, preterm newborns, the same criteria to declare brain death can be applied to full-term newborns, infants older than 7 days of age, children, and adults.[8,23-27] The special taskforce guidelines for the determination of brain death provide no guidelines to diagnose cerebral death in infants younger than 7 days of age.[18] Guidelines for this age group were not published in 1987, because there was limited experience, and establishing irreversibility and neurologic death was more difficult to confirm at that time.[19,28] This does not infer that neurologic death does not occur in this patient population. Diagnosing neurologic death can occur in the term infant, even those younger than 7 days of age; however, an observation period of 48 hours has been recommended to confirm the diagnosis.[29] The observation period can be shortened to 24 hours if ancillary studies demonstrate no cerebral blood flow or an isoelectric electroencephalogram (EEG).[28,29] The younger the child, the more cautious determination of neurologic death should be.

Ancillary testing can provide data to confirm neurologic death when the clinical examination and apnea test are not feasible or cannot be completed because of undue circumstances. Ancillary tests are not mandatory if clinical brain death determination is feasible; however, they can provide another layer of comfort to the physician who is uncomfortable declaring neurologic death on the basis of clinical examination alone. Ancillary tests may also be used to expedite the diagnosis of brain death by reducing the clinical observation period, potentially increasing viability of transplant tissue. However, if ancillary tests are equivocal or demonstrate blood flow or electrical activity, the patient should be observed according to proposed age-specific guidelines until another clinical examination is performed to confirm neurologic death.

A 4-vessel angiogram evaluating anterior and posterior cerebral circulation remains the gold standard in ancillary testing[30]; however, this test is difficult to perform in small children and requires technical expertise that may not be available in every facility. Furthermore, the requirement for transportation of a potentially unstable patient to the angiography suite complicates this process. Radionuclide flow scan using a portable gamma camera is more easily accomplished at the bedside, without the need for extraordinary technical expertise; therefore, this is one of the most frequently used tests in children.[30,31] In addition, improved radiotracer agents such as Tc-99m hexamethylpropylene-amine oxime have improved the ability to evaluate greater segments of the intracerebral circulation, most notably the posterior fossa.

EEG combined with a neurologic examination remains an accepted means to determine neurologic death in children. EEG testing should not be used alone to determine neurologic death because it does not assess brainstem function, a key in the final determination of brain death. In addition, EEG is influenced by factors such as hypothermia and sedative medications, which can complicate declaration of neurologic death and affect potential organ donation.[32] Doppler ultrasonography and brainstem audio evoked potentials have been used,[33] but have not been validated in children and cannot be relied on as a dependable ancillary study.[24,34] The clinical diagnosis of brain death is highly reliable when made by experienced examiners using established criteria.[35,36] Each state and institution has guidelines for determination of death, but the diagnosis of brain death still requires the thoughtful, mature judgment of a knowledgeable physician who takes all the facts into careful deliberation in each case.[25]

Brain Death Physiology

As neurologic death occurs, multiple physiologic changes become manifest. These derangements develop as a result of the loss of normal central nervous system (CNS) regulation. Endocrine dysfunction occurs because of inhibition or lack of hormonal stimulation from the hypothalamus. This neuroendocrine dysfunction can result in fluid and electrolyte disturbances and cardiovascular instability. Hypothermia

should be an anticipated derangement as a result of hypothalamic dysfunction and increased heat loss from systemic vasodilation as loss of vascular tone occurs. Hypothermia requires aggressive treatment to avoid aggravation of coagulation disturbances and reduction in cardiac output. The brain normally consumes 20% of the cardiac output. Without the metabolic contribution of the brain, glucose needs are reduced and the patient is prone to hyperglycemia that may necessitate the use of an insulin infusion. Furthermore, with such a significant reduction in cerebral metabolism, carbon dioxide production falls and can be observed by a reduction in $PaCO_2$ on serial blood gas sampling.

Once a decision has been made by the family to proceed with organ donation following determination of neurologic death, the focus of care shifts toward the preservation of vital organs. The subsequent care may vary from the management that has occurred up to that point. Previous efforts to reduce intracranial pressure using interventions such as moderate hyperventilation, hypothermia, and sedatives are abandoned and attention moves toward providing ample blood flow and oxygen delivery to prospective transplantable organs. The principles in management are largely the same, including maintenance of adequate oxygen delivery to the tissues through the optimization of cardiac output and oxygen content. Hemodynamics are managed to maintain normal blood pressure for age (Table 2). Reduction in cerebral edema treated with volume restriction and diuretic agents that result in decreased intravascular volume must be corrected. Attention to intravascular volume loss from derangements such as diabetes insipidus (DI) must be anticipated and appropriately addressed. Excessive volume depletion can lead to hemodynamic compromise and end-organ failure secondary to inadequate perfusion if left untreated. Additional management goals include the normalization of $PaCO_2$ via adjustments of mechanical ventilation, normalization of temperature, and addressing neuroendocrine dysfunction with correction of metabolic disturbances. Progression of organ failure following neurologic death results in the loss of 10% to 20% of potential donors; therefore, timely and definitive treatment of the donor is critical.[38,39]

Table 2. NORMAL VITAL SIGNS FOR CHILDREN

Vital signs	Respiratory rate (breaths/min)	Pulse (beats/min)	Systolic blood pressure (mm Hg)*[37]
Infant	30-60	120-160	60-70
Toddler	25-40	90-140	75-90
School age	22-34	80-120	80-100
Adolescent	12-20	60-90	90-120

*Lowest acceptable systolic blood pressure is (2 × age in years) + 70.

General Considerations for Potential Pediatric Organ Donors

Pediatric donor management requires an understanding of the anatomic and physiologic differences in children. The respiratory system of children is much different from that of an adult. The trachea is shorter, which can predispose to misplacement of the endotracheal tube. Smaller airways have increased airway resistance, the chest wall is more compliant, and respiratory muscles are less developed. Cardiovascular dynamics require a higher resting heart rate to maintain cardiac output because the smaller heart of children cannot generate large stroke volumes. Drastic decreases in heart rate result in decreased cardiac output leading to problems with end-organ perfusion. The child also has a larger body surface area and is more prone to develop hypothermia. Radiant warmers, warm blankets and pads, warm intravenous (IV) fluids or a blood warmer for infusion of blood products, increasing the temperature of inspired gas through the humidified ventilator circuit, and environmental warming can be used to maintain the patient in a normothermic state. Limited glycogen stores place infants at greater risk for hypoglycemia and limited catecholamine stores can be depleted leading to hypotension and altered cardiac output. Understanding these anatomic and physiologic differences is important when managing pediatric organ donors.

Vascular access can be challenging in small children. Unlike adults, multilumen pulmonary artery catheters are rarely used in children because of size constraints and potential technical challenges during catheter placement. Double and triple lumen central venous catheters are most commonly used in children.

Small lumen size, predisposing to obstruction, and limiting large volume administration, and compatibility of multiple IV infusions, may require additional vascular access. Although a logical solution would be placement of another multilumen central venous catheter, this is not always possible in a small infant or child. Peripheral IVs are frequently used, but must be checked frequently to ensure patency and infusion of agents into the vascular system. Placement of a peripheral IV in a potential donor can be difficult because of tissue edema, hypothermia, and small, fragile vessels that can be difficult to cannulate.

Donor management goals for children are the same as for adult donors; however, it is important to remember that children are not small adults. There are special considerations when dealing with pediatric donors that require a specialized team of physicians, nurses, respiratory therapists, and social workers, who are all trained in pediatric physiology and the unique needs of the child and the family. The transplant professional should use this team of pediatric specialists as a resource for planning management and intervention of pediatric donors.

Management of Pulmonary Issues for Potential Pediatric Organ Donors

Every donor should be considered and managed as a potential lung donor; therefore, in addition to meticulous care to ensure ventilation and oxygenation, attention should be afforded to prevent barotrauma and any potential further lung injury. This strategy will improve the chances and quality of all potential organs recovered.

Impairments in oxygenation and ventilation can result from lung disease and injury such as pulmonary hemorrhage or contusion, and inhalation or thermal injury. Furthermore, management of pulmonary physiology may be complicated by the development of pulmonary edema either from the progression of brain injury and associated neurogenic pulmonary edema,[40] acute respiratory distress syndrome, or because of volume administration used to correct hemodynamic instability. Infectious etiologies can compound the effects of existing lung disease or injury leading to further impairment in ventilation and oxygenation. In addition, impaired cardiac output, anemia, and inadequate ventilatory support can all contribute to further impairment in oxygen delivery to the tissues. Because of the importance of maintaining oxygenation and ventilation in the potential donor and the myriad of pathologic states that can complicate management, a basic understanding of airway management and oxygenation and ventilation is necessary. This knowledge will equip those caring for these patients to be better able to deal with, and anticipate potential pulmonary problems that may be encountered.

Maintenance and protection of the airway is essential to provide adequate oxygenation and ventilation to potential pediatric organ donors. The endotracheal tube (ETT) used to secure the airway can be cuffed or uncuffed. Cuffed ETTs are used when higher airway pressures are anticipated, for example, in patients with pulmonary edema or underlying respiratory pathology. The appropriate-sized ETT is imperative to providing adequate care for these patients. The size can be estimated on the basis of age ([age in years + 16]/4).[37] Dislodgement of the ETT can easily occur because of the shorter trachea in children. It is important to check the placement of the ETT using radiographic studies. The tip of the ETT should reside between the third and forth vertebral body visualized on an anterior-posterior chest radiograph. A useful formula to assist with the depth of ETT placement is 3 times the size of the ETT,[37] thus, a 4.0 ETT should be approximately 12 cm at the lip. An ETT that is too large will be deeper, and an ETT that is too small will reside higher using this formula. For children 1 to 12 years of age, the formula, "10 + the age in years" can be used to check proper depth of the ETT.[41] Appropriate placement of the ETT is crucial as a 1-cm difference in the depth of the ETT can result in right mainstem bronchus placement or extubation. To prevent the ETT from slipping through the tape from excessive secretions, it should be securely wrapped in a spiral fashion. Maintaining pulmonary toilet is essential in an effort to maintain adequate oxygenation and ventilation.

Basic continuous noninvasive monitoring and serial laboratory testing play a crucial part in the appropriate management of these patients and in detecting physiologic derangements in oxygenation and ventilation. Continuous pulse oximetry and capnography allow monitoring of a patient's oxygen saturation and exhaled carbon dioxide tension, providing measurements of oxygenation and ventilation. Arterial

blood gas analysis provides a direct measure of dissolved oxygen and carbon dioxide concentration, showing efforts at oxygenation and ventilation. Attention to such noninvasive monitoring and blood gas analysis is a cornerstone in patient management.

Acute changes in physiology frequently occur in the management of pediatric organ donors. Acute changes that result in oxygen desaturation require prompt evaluation and intervention that must precede investigation because of the potential consequences to organ viability. Oxygen desaturation must be avoided at all costs. Manual ventilation using a bag valve mask with 100% oxygen should ensue and the cause of the desaturation episode investigated.

Oxygen desaturation can be a result of a dislodged, kinked, or obstructed ETT; bronchospasm; or pneumothorax. A dislodged ETT must be replaced immediately. If the ETT is kinked, examining and repositioning the ETT will restore patency of the airway. Regular pulmonary toilet is essential to clear secretions and alleviate any mucous plugging that can result in obstruction of the airway. This is particularly important with younger children who require a smaller ETT. Bronchospasm can be detected by wheezing or a prolonged expiratory phase and can be more common in predisposed individuals, individuals with pulmonary edema, or those treated with β-blockers for hypertension. The first line therapy for management of bronchospasm is inhaled beta-agonist such as albuterol. Albuterol has also been shown *ex vivo* and in animal studies to augment the clearance of pulmonary edema and may be considered, along with diuretics should this problem occur or contribute to bronchospasm.[42] Corticosteroids can be administered by inhalation or IV for persistent bronchospasm. In addition, corticosteroids may have the added benefit of stabilizing lung function in potential organ donors.[43] In situations in which bronchospasm is unresponsive to inhaled adjunctive agents, IV beta-agonists or IV methylxanthines can be considered.

Sudden oxygen desaturation can also occur from a pneumothorax as a result of high airway pressures used during manual or mechanical ventilation. Needle aspiration of the chest or placement of a chest tube to evacuate the pressurized air in the thorax is imperative to avoid cardiopulmonary collapse.

Understanding oxygenation and ventilation as two separate entities will assist in the treatment of specific pulmonary problems commonly encountered in pediatric organ donors. Oxygen delivery is dependent on hemoglobin concentration, the fraction of inspired oxygen (FiO_2), PaO_2, and cardiac output. Cardiac output is determined by stroke volume and heart rate. Correction of derangements in oxygenation begins with treatment of the underlying cause and in the majority of cases, measures to improve oxygenation require manipulation of the determinants of oxygen delivery, as previously mentioned. The ability to oxygenate can be improved by such maneuvers as increasing the FiO_2, improving cardiac output using volume expansion and/or inotropic support, and increasing the hemoglobin concentration by transfusing red blood cells. Maneuvers to improve PaO_2 can be pursued as well, but discussion of such interventions will be reserved for the portion of this chapter dedicated to mechanical ventilation.

Oxygen saturation is the percentage of hemoglobin that binds oxygen. Although a major determinant of oxygen delivery, it is important to remember that oxygen saturation is not always a reliable indicator of adequate oxygen delivery to the tissues. A child can be well saturated, but still have inadequate oxygen delivery; therefore, the means to detect the adequacy of oxygen delivery is crucial in guiding therapy. Measurements such as PaO_2, lactate, and mixed venous oxygen saturation can be used as additional measures of oxygen delivery.

Oxygen saturation can also be improved by altering pulmonary blood flow, which can affect ventilation/perfusion matching. Increasing pulmonary blood flow can be achieved by altering pH and using mechanical ventilation and pharmacologic agents.

The use of alkalosis, either by respiratory or metabolic therapies, can reduce pulmonary vascular resistance (PVR), thereby increasing pulmonary blood flow. Alkalosis can reduce the ability of oxygen unloading from hemoglobin at the tissue level as the hemoglobin/oxygen dissociation curve shifts leftward, thus potentially reducing tissue levels of oxygen. In contrast, a respiratory or metabolic acidosis will increase PVR, thus decreasing blood flow to the lungs. Acidosis enhances the unloading of oxygen to the tissues. The manipulation of PVR by altering pH to improve oxygenation, although appealing, does not come without consequence; therefore, if manipulation of PVR is desired, use of a selective pulmonary vasodilator such as inhaled nitric oxide is worthy of consideration.[44-47] Maintaining arterial pH in the

normal range, between 7.35 and 7.45, in addition to keeping the $PaCO_2$ between 35 and 40 mm Hg, thereby maximizing oxygen unloading to the tissues to maintain end organ viability, is preferable. In addition, the PaO_2 should be kept greater than 100 mm Hg.

Ventilatory requirements may become minimal in the organ donor as neurologic death occurs with loss of respiratory regulation. Decreased glucose metabolism, oxygen consumption, and a respiratory alkalosis occur as metabolic production of carbon dioxide from the brain ceases and compliance of the chest wall changes. The attainment of normocarbia is important because of previously discussed effects on the unloading characteristics of oxygen from hemoglobin, with obvious implications on availability of oxygen to the tissues. Decreasing ventilation parameters is not uncommon to restore normocarbia with a goal of 35 to 40 mm Hg in the child who is progressing or has achieved neurologic death. Minute ventilation, which affects $PaCO_2$, is determined by respiratory rate and tidal volume. Adjusting either of these parameters will alter exchange of carbon dioxide. Further discussion regarding adjustment of ventilation parameters is discussed in the next section.

Mechanical Ventilation

Ventilator parameters should be adjusted on an individual basis. Variables that require adjustment for mechanical ventilation in children include tidal volume, peak inspiratory pressure, positive end-expiratory pressure (PEEP), inspiratory time, rate, and FiO_2. Although manipulation of ventilator parameters is required to control oxygenation and ventilation, decisions regarding ventilation strategies are best left to the pediatric intensivist or anesthesiologist, who routinely provides assisted ventilation to children. PEEP should be provided for all children who are receiving mechanical ventilation to maintain alveolar inflation. Inspiratory time is typically set to provide an inspiratory-expiratory ratio of 1:3. Normal inspiratory time is 0.6 to 0.75 seconds for infants and smaller children respectively; an inspiratory time of 1 second is appropriate for older children. Improvement in oxygenation can be achieved by increasing the inspiratory time with a net effect of increasing mean airway pressure. When adjusting inspiratory time, it is important to consider the effect on the expiratory time. If the inspiratory time is increased, there will be an obligatory decrease in the expiratory time. To avoid stacking of breaths leading to carbon dioxide retention, the inspiratory-expiratory ratio should not exceed 1:1.

To maintain adequate oxygen saturation in children receiving mechanical ventilation, the FiO_2 can be increased. High concentrations of oxygen should be used only as needed. Hyperoxia can result in toxicity to the pneumocytes and interfere with surfactant production predisposing to altered physiology and pathology such as atelectasis and scar formation. Although the lowest concentration of inspired oxygen that results in appropriate oxygen delivery and saturations should be used, 0.4 FiO_2 for the patient requiring less oxygen provides a buffer should the ETT become obstructed or dislodged, leading to desaturation. If further improvement in oxygenation is needed beyond that which can be achieved by raising FiO_2, increasing the inspiratory time or increasing PEEP, to recruit and maintain alveolar inflation resulting in an improvement in functional residual capacity, are reasonable considerations. PEEP can also assist in decreasing pulmonary edema. The benefit associated with the use of PEEP must be balanced against the risk of potential barotrauma and effects on preload, which can potentially decrease cardiac output in donors with cardiac dysfunction. Cardiovascular effects can be minimized if adequate preload is ensured before the escalation of PEEP.

Minute ventilation is determined by respiratory rate and tidal volume. Adjustment of tidal volume can be made either by direct manipulation of measured volume if ventilating in a volume-based ventilation mode or adjusting peak inspiratory pressure when ventilating in a pressure-based ventilation mode. The magnitude of an adequate tidal breath, which can be determined by the volume of air required to expand the chest adequately by direct observation, usually corresponds to 8 to 10 mL/kg. This value allows for compensation of the ventilator circuit, tubing compliance, and gas compressibility. The lowest possible tidal volume that promotes chest rise is recommended to avoid excessive hyperventilation. Evidence suggests that high volumes potentiate barotrauma.[48] Synchronous modes of ventilation are not indicated in this particular patient population, because neurologic death has occurred and spontaneous respiratory effort is absent.

Treatment of Hemodynamic Instability

Cardiac dysfunction is the greatest limiting factor to successful organ procurement. Of all the physiologic derangements encountered in prospective organ donors, the cardiovascular system is fraught with the greatest complexity and variation. This variation is a reflection of the powerful neuroendocrine changes that occur during progression to neurologic death. The efforts to regulate cerebral perfusion pressure, hemodynamic manifestations of herniation, and, ultimately, the physiology of an absent CNS, all contribute to the unstable physiology that commonly occurs in the prospective organ donor.

Management goals for pediatric organ donors is directed at achieving and maintaining adequate circulating blood volume, optimizing cardiac output and oxygen delivery to the tissues, and maintaining normal blood pressure for age. Inotropic agents such as dopamine, dobutamine, and epinephrine can be titrated to effect; however, establishing appropriate circulating volume is essential before using inotropic support. Central venous pressure (CVP) monitoring, and clinical indicators such as perfusion and urine output are used in determining adequate intravascular volume, but must be evaluated in the context of the child with profound CNS alterations. Serial lactate levels serve as a guide of tissue perfusion. Elevations in serum lactate and the development of a metabolic acidosis provide evidence of tissue ischemia and should prompt immediate attention. As the patient with severe intracranial pathology progresses toward cerebral death, the associated neuroendocrine dysfunction will result in tremendous variations in physiology that require the application and adjustment of specific interventions to restore normal physiologic parameters.

Intracranial hypertension with cerebral ischemia leads to massive sympathetic discharge, "sympathetic or autonomic storm." Organs are exposed to extreme sympathetic stimulation either from direct neural stimulation or from endogenous catecholamines, resulting in systemic hypertension and tachycardia.[49] The local effects of sympathetic stimulation result in increased vascular tone, effectively reducing blood flow and potentially causing ischemia to end organs. This autonomic storm also has direct effects to the myocardium, as the surge of catecholamines increases systemic vascular resistance, thus increasing myocardial work and oxygen consumption. Myocardial injury can occur as the left ventricle is exposed to a significant increase in afterload, which reduces cardiac output. Ischemic changes have been reported as a result of this imbalance between myocardial oxygen supply and demand.[49,50]

As left ventricular afterload increases, impairment of cardiac output may result with consequential dysfunction to organ systems. Left ventricular end diastolic pressure rises, which results in increased left atrial pressure. As left atrial pressure exceeds pulmonary artery pressure, a hydrostatically induced extrusion of fluid into the interstitial space of the lungs occurs, resulting in pulmonary edema. This condition is further exacerbated by the increase in venous return as a result of catecholamine-mediated systemic vasoconstriction. The increased volume load to the right heart not only exacerbates the hydrostatic pressure in the pulmonary vasculature, but also displaces the ventricular septum into the left ventricle, further impairing left ventricular preload and, therefore, cardiac output through a mechanism called ventricular interdependence.[51]

The sympathetic storm with associated hypertension is a predictably transient phenomenon. Although end-organ ischemia can transiently occur, treatment with antihypertensive agents may not be warranted and could create additional problems with perfusion when this phase of sympathetic outflow has passed. If hypertension is severe and treatment is felt to be indicated, IV infusions of ultra short-acting antihypertensive agents such as nitroprusside sodium (Nipride) or esmolol hydrochloride (Brevibloc) can be titrated to effect. β-Blockers may aggravate a low cardiac output state, in addition to promoting bronchospasm in predisposed individuals, and should be used with caution. Longer-acting agents require close observation to avoid hypotension. Intermittent doses of IV hydralazine hydrochloride (Apresoline) or labetalol (Trandate, Normodyne) can also be used to control hypertension.

As neurologic death occurs, sympathetic outflow is reduced. This results in a loss of autonomic tone, leading to vasodilation with potential impairment in cardiac output and hypotension. Vasodilation results in decreased circulating volume, which can be compounded by excessive urine output because of hormonal imbalances leading to DI and hyperglycemia. These derangements warrant the use of volume as the intervention of choice. Inadequate intravascular volume will be signified by a drop in CVP and narrowed pulse pressure with a reduction in blood pressure and perfusion. Urine output may not be a

good clinical indicator if DI is present or being aggressively managed using pharmacologic agents. Loss of beat-to-beat variation and a fixed heart rate are also common as brainstem death occurs; therefore, heart rate will also not be a reliable sign of intravascular volume status. In addition, perfusion may be altered if the child is hypothermic. The goal during this phase of patient management is restoration of circulating volume using blood pressure, perfusion, and CVP monitoring as a guide. Aggressive volume resuscitation must occur to restore and maintain adequate perfusion to vital tissues for possible transplantation.

The use of isotonic fluids for volume resuscitation is principle, and fluids containing dextrose should never be used as a volume expander. Bolus infusions of 10 to 20 mL/kg of crystalloid, such as isotonic sodium chloride solution or lactated Ringer's solution, or colloid such as 5% albumin should be used and repeated as needed to support the blood pressure with a goal appropriate for the age of the child. Table 2 shows normal vital signs on the basis of the age of the child. The normal blood pressure for any child older than 1 year of age is 2 times the age in years plus 80, with the lowest acceptable blood pressure equaling 2 times the age in years plus 70. [37] For infants younger than 30 days of age, a systolic blood pressure of 60 mm Hg is acceptable, and for children 1 month to 1 year of age, a systolic blood pressure of 70 mm Hg is appropriate. [37] Infants are proportionately more dependent on circulating free calcium for cardiac function, which must be considered when administering 5% albumin in this age group. The sarcoplasmic reticulum of the infant myocardium is underdeveloped and therefore cannot store reserves of calcium as effectively as older children and adults; therefore, infants are particularly vulnerable to administration of large amounts of albumin solution, which can bind free calcium resulting in hypocalcemia and hypotension.[52,53] Intermittent or continuous IV administration of calcium chloride or calcium gluconate to maintain the ionized calcium levels greater than 1.0 mmol/dL can reverse this effect. Artificial plasma expanders such as Hespan or Dextran should be avoided because large volumes can alter coagulation parameters.[54,55] Blood products such as packed red blood cells for significant blood loss or anemia can be administered in aliquots of 10 to 15 mL/kg administered over 2 to 4 hours, or faster, if hemodynamic instability with ongoing blood loss requires more aggressive resuscitative measures. Blood can be warmed to help maintain or restore thermoregulatory stability in potential donors, if needed.

If volume loading with 60 to 80 mL/kg of IV fluids does not restore normal blood pressure, the use of inotropic agents such as dopamine or dobutamine, administered through a central venous catheter, should be considered. Both these agents help improve cardiac function and increase blood pressure. Patients who have an inadequate response to these inotropic agents may require additional vasopressors such as epinephrine, norepinephrine, or phenylephrine to help support their blood pressure. Although necessary, the administration of vasoactive agents can be associated with reduced perfusion to donor organs, potentially jeopardizing their viability upon acquisition. Hormonal replacement therapy using thyroid hormone and steroids should be considered in children who are refractory to high-dose inotropic infusions. Ideally, a management strategy to maintain blood pressure, normovolemia, and optimization of cardiac output, with the least amount of vasoactive agents, has been adopted in many centers that are involved in donor management and organ procurement.

Arrhythmias can occur for many reasons during the progression toward and following the achievement of neurologic death. The catecholamine storm resulting in increased adrenergic stimulation can promote rhythm disturbances and myocardial ischemia. Hypotension secondary to hypovolemia with resultant myocardial ischemia; acidosis secondary to poor cardiac output; hypoxemia secondary to pulmonary insufficiency; hypothermia; cardiac trauma; proarrhythmic properties of inotropes; and electrolyte and metabolic disturbances such as hypomagnesemia, hypocalcemia, and hypokalemia that occur with DI can also precipitate rhythm disturbances. Identification and correction of the underlying cause for the arrhythmia are essential to minimize or eradicate rhythm disturbances. Replacement of deficient electrolytes such as potassium, calcium, or magnesium can reduce or eliminate ventricular rhythm disturbances and improve blood pressure.[56] Hypotension should be treated with volume resuscitation and inotropic support. Reducing the amount of inotrope, provided that the blood pressure will tolerate this maneuver, may be beneficial in decreasing or eliminating rhythm abnormalities. Sodium bicarbonate or tromethamine (THAM) can be used to correct metabolic acidosis and ventilator adjustments to improve minute ventilation may be used to correct a respiratory acidosis. Hypoxemia can be corrected by adjusting ventilator parameters, and cardiac output and oxygen delivery can be improved by achieving volume and/

or inotropic support, and transfusion of red blood cells. Hypothermia can be corrected by active warming measures. Intravenous lidocaine or amiodarone (Cordarone) can be used to treat ventricular arrhythmias once metabolic disturbances have been corrected, whereas IV adenosine (Adenocard IV) can be used to treat supraventricular arrhythmias such as supraventricular tachycardia (SVT). IV amiodarone can be used as well, if recurrent SVT becomes problematic. For hemodynamically unstable SVT, synchronized electrical cardioversion is considered first-line therapy.[37]

Hormonal Replacement Therapy

The use of hormonal replacement therapy has been controversial in the adult literature.[43,50,51] Thyroid and cortisol depletion may contribute to the hemodynamic instability encountered in patients who have progressed to neurologic death, although no studies have firmly established this fact. Furthermore, correlations between hormone usage, cardiac function, and clinical outcome measures have been disappointing.[57-61] However, hormonal replacement therapy has shown promise in reducing requirements for vasoactive agents in 100% of unstable donors and abolished the need in 53% of such donors in 1 adult series,[62] whereas other retrospective series have demonstrated that hormone replacement therapy was associated with a significant increase in the number of organs transplanted from donors.[63] This benefit has been noted by Zuppa and associates,[64] who observed decreased inotropic requirements in children who received levothyroxine. Given these observations, many organ procurement teams have adopted the use of hormone replacement therapy as a routine part of organ donor management. Furthermore, it is reasonable to consider these agents in situations in which the hemodynamic status of the child is refractory to conventional therapy with fluid and inotropic administration.[51,64,65] Thyroid hormone has also been associated with an increase in transplanted organs from donors receiving hormone replacement therapy.[63,66,67] Commonly used agents and doses for hormonal resuscitation in children are listed in Table 3.

Levothyroxine (Synthroid) and triiodothyronine (T3) are the two IV thyroid agents available for administration. Dosing of thyroid hormone for pediatric organ donors is not well established. Levothroxine dosing, like many other pharmacologic agents used for this patient population, is based on the weight of the child. One retrospective study[64] provided younger children with a larger bolus and infusion dose compared with older children, and demonstrated an ability to wean inotropic support in children who progressed to neurologic death. Administration of T3 is used in some centers for hormonal replacement therapy; however, its cost may be prohibitive and the benefits in this patient population are controversial. Novitsky et al[61] has reported beneficial hemodynamic effects in brain-dead patients receiving T3

Table 3. PHARMACOLOGIC AGENTS USED FOR HORMONAL RESUSCITATION IN CHILDREN[68]

Drug	Dose	Route	Comments
Desmopressin (DDAVP)	0.5 mcg/h	IV	Terminal half life, 75 minutes (range 0.4-4 hours) Titrate to effect to control urine
Vasopressin (Pitressin)	0.5 mU/kg/h	IV	Half life, 10-35 minutes Titrate to effect to control urine output Hypertension can occur
Levothyroxine (Synthroid)	0.8-1.4 mcg/kg/h	IV	Titrate to effect Bolus dose 1-5 mcg/kg can be administered; smaller infants and children require a higher bolus and infusion dose
Triiodothyronine (T3)	0.05-0.2 mcg/kg/h	IV	Titrate to effect
Hydrocortisone (Solucortef)	1 mg/kg	IV	Fluid retention and glucose intolerance
Insulin	0.05-0.1 U/kg/h	IV	Titrate to effect to control blood glucose levels Monitor for hypoglycemia

Abbreviations: IV, intravenous

administration, whereas others studies[69,70] have shown no benefit with this agent. The effects of thyroid hormone on myocardial contractility are complex and can be immediate or delayed. The acute inotropic properties of T3 may occur as a result of β-adrenoreceptor sensitization or may be completely independent of β-adrenergic receptors.[68-72] Furthermore, T3 administration may play an important role in maintaining aerobic metabolism at the tissue level after brain death has occurred.[73]

Steroids, such as hydrocortisone, are another adjunct used by many centers to assist with hemodynamic support; however, there is even less data attesting to its benefits in potential pediatric donors.[51] The potential benefit of hydrocortisone and other steroids may lie in its ability to alter adrenergic receptors and regulate vascular tone by increasing sensitivity to catecholamines; but to date the clinical benefit remains untested in this population.[74-76]

Application of hormonal replacement therapy in potential pediatric organ donors has generated some support through scientific studies.[60,61,64] Although hormonal replacement therapy is controversial; it is widely practiced despite lack of convincing evidence. The combination of thyroid hormone and steroids may be used as pharmacologic adjuncts to reduce dosing of vasoactive agents in children requiring high-dose inotropic support and this application may indeed improve success in organ procurement for children; however, further studies are clearly warranted.

Fluid and Electrolyte Disturbances

The disturbances in the management of fluids and electrolytes in pediatric donors are the result of physiologic derangements, as well as iatrogenesis. Derangements commonly encountered include dehydration, hyperglycemia, sodium and potassium derangements, and hypocalcemia. Meticulous management of fluids and electrolytes is necessary because metabolic swings associated with neurologic death can adversely affect organ viability.

Addressing basic fluid management in pediatric patients requires an understanding of the normal physiologic needs of these patients. Fluid requirements can be determined on the basis of weight for infants and small children who require a proportionately greater amount of fluids compared to their older counterparts, in whom fluids can also be calculated on the basis of body surface area (m^2). Standard calculations for estimated maintenance fluids can be found in Table 4. Another important consideration in children is their reduced need for sodium and greater glucose requirement. Because of their standard daily sodium needs, infants up to 1 year of age require one fourth or one third normal saline solution as their maintenance IV fluids. For a child older than 1 year, one third to one half normal saline solution is appropriate because of their greater maintenance sodium requirement. Limited glycogen stores in young infants make them particularly vulnerable to hypoglycemia. For these infants, fluids containing a 10% dextrose concentration and frequent reassessment of their blood glucose levels are imperative to prevent hypoglycemia. After 6 months to 1 year of age, glycogen storage matures and solutions containing 5% dextrose can be used. Age-appropriate fluid management remains important for children who become candidates for organ donation.

In children with traumatic brain injury that has progressed to neurologic death, intravascular volume depletion is frequently encountered because of fluid restriction and treatment with hypertonic solutions and osmotic diuretics used in the management of cerebral edema. Additional contributors to intravascular volume depletion are hyperglycemia, as a result of steroid and catecholamine use, and the increased availability of glucose because of loss of cerebral metabolism. Furthermore, DI compounds sodium and

Table 4. STANDARD CALCULATIONS FOR MAINTENANCE INTRAVENOUS FLUIDS

Patient weight (kg)	Hourly fluid rate (mL/h)
1st 10 kg	4 mL/kg
2nd 10 kg	2 mL/kg
>20 kg	Weight (kg) + 40

Example: 16 kg child: 1st 10 kg x 4 mL/kg = 40 mL + 6 kg x 2 mL/kg = 12 mL: Total hourly fluids = 52 mL

water balance if left untreated. As previously discussed, potential pediatric organ donors must be adequately volume resuscitated, guided by CVP, perfusion, and potentially serial lactate measurements. The choice of fluid for volume resuscitation is isotonic crystalloid in doses of 10 to 20 mL/kg. Restoring intravascular volume is the mainstay of securing organ viability.

Diabetes Insipidus

In potential pediatric donors who develop DI, there is excessive free water loss resulting in fluid and electrolyte disturbances. DI is characterized by hypernatremia and polyuria, with elevated serum osmolarity and urine specific gravity of <1.002. In this condition, loss of antidiuretic hormone produced in the CNS allows unrestricted free water loss without regard to intravascular volume. Hypernatremia, with a serum sodium level greater than 150 mg/dL, is commonly encountered in DI and can be detrimental to end organs. Hypernatremia has been associated with graft failure after liver transplantation[77,78]; thus meticulous and definitive management is imperative to preserve organ function.

The management of DI requires supplementation of antidiuretic hormone to restrict free water loss, while replacing free water to avoid significant dehydration. Hormonal replacement therapy with pharmacologic agents such as vasopressin or desmopressin (DDAVP) can be used to control urine output. Each agent has specific indications and side effects that must be considered when contemplating its use in pediatric patients.

Vasopressin is a polypeptide hormone secreted by the hypothalamus and stored in the posterior pituitary. Vasopressin acts on the V_1 and V_2 vasopressin receptors and stimulates contraction of vascular smooth muscle resulting in vasoconstriction. It has a short half life of 10 to 20 minutes, and unlike Desmopressin has no effect upon platelets.[79] Vasopressin can be administered by bolus or continuous IV infusion. The most desirable features of this agent are its titratability to control urine output and, when no longer required, its effects are short lived. Vasopressin is administered at doses of 0.5 milliunits/kg/h and can be titrated to control urine output to 2 to 4 mL/kg/h.[79] By titrating to this degree, renal function can continue to be preserved, and volume overload and metabolic derangements such as hyperkalemia can be avoided. Vasoconstrictive effects of vasopressin in high doses may reduce splanchnic perfusion, which can be detrimental to hepatic and pancreatic blood flow, and increased smooth muscle contractility may affect coronary and pulmonary blood flow.[50] Excessive dosing of vasopressin should be avoided to preserve end-organ function. Vasoconstriction may result in hypertension, which may facilitate weaning of other inotropic agents for blood pressure support.

Desmopressin is a synthetic polypeptide structurally related to vasopressin and has a more potent antidiuretic effect. This agent lacks smooth muscle contractile properties and is more specific for the V_2-vasopressin receptor.[80] Desmopressin enhances platelet aggregation and it has a longer half life of 6 to 20 hours when administered as a single IV dose.[81-83] The lack of hemodynamic side effects may make Desmopressin a better agent for the correction of hypernatremia in the hemodynamically stable donor. It may also be preferred because of its ability to enhance platelet function in potential donors with an existing coagulopathy. Desmopressin can be administered by continuous infusion at 0.5 mcg/h and titrated to control urine output. Intramuscular and intranasal administration can result in erratic absorption and should be avoided. The terminal half life of Desmopressin administered by continuous IV infusion is 75 minutes, with a range of 0.4 to 4 hours.[84] The longer half life makes Desmopressin a less desirable agent to some transplant surgeons who prefer the shorter half life of vasopressin. One approach in dealing with this situation is to discontinue Desmopressin therapy 4 to 5 hours before organ recovery and administer fluid replacement for excessive urine output as needed over the following hours.

The replacement of ongoing volume loss in DI is imperative to preserve organ function for transplantation. The use of hypotonic solutions such as one half or one fourth normal saline solution to provide more free water will help correct hypernatremia and maintain euvolemia. Excessive uncontrolled urine output can result in dehydration and hypovolemic shock. Urine output over 3 to 4 mL/kg/h should be aggressively replaced 1:1 or milliliter for milliliter with one fourth or one half normal saline solution on an hourly basis to prevent intravascular volume depletion. Measuring the urine sodium concentration can be

used as a guide to facilitate sodium replacement in patients with DI. The concentration of isotonic sodium chloride solution is 154 mEq/dL; therefore, if the measured urine sodium content were approximately 70 mEq/dL, one half normal saline solution would be an appropriate replacement fluid. If urine sodium were closer to 40 mEq/dL, one fourth normal saline solution would be a reasonable choice for fluid replacement. Lastly, the use of enteral water supplementation administered through a nasogastric tube can be considered for the correction of severe hypernatremia. Rapid osmolar shifts during correction of hypernatremia are inconsequential because the child has already progressed to neurologic death.

Oliguria

Oliguria, although less common, can be secondary to volume depletion, acute renal insufficiency or failure, and iatrogenesis from overly aggressive DI management. If urine output falls to less than 1 mL/kg/h and iatrogenesis has been ruled out, the patient's intravascular volume status must be evaluated. If the patient's intravascular volume appears depleted, a bolus of 10 to 20 mL/kg of isotonic sodium chloride or lactated Ringer's solution, or colloid is indicated. If urine output remains low, additional administration of isotonic IV fluids may be repeated. The patency of the Foley catheter must be checked to ensure that obstruction of the catheter is not the primary cause for decreased urine output. If several boluses of crystalloid result in no improvement in urine output, colloid for volume expansion or vasopressor support, if poor perfusion is present, may assist in improving urine output. Furosemide (Lasix) or mannitol can be used to stimulate urine output in patients with adequate intravascular volume status.

Glucose Derangements

Hyperglycemia, as a result of steroid and catecholamine use and the increased availability of glucose because of the loss of cerebral metabolism, can lead to an osmolar diuresis, exacerbating an already depleted volume status. Hyperglycemia can be avoided by frequently assessing blood glucose levels and making appropriate adjustments in the dextrose concentration in the maintenance fluids. If these maneuvers are unsuccessful in controlling blood glucose levels, an insulin infusion should be instituted to maintain glucose levels between 80 to 150 mg/dL. Insulin infusion at a starting dose of 0.05 to 0.1 U/kg/h can be titrated to effect. It is important to follow serum glucose levels closely to avoid hypoglycemia.

Neonates and infants have a continuously high glucose need with limited glycogen stores, as previously noted. Hypoglycemia, although less common, can develop rapidly and result in end-organ damage if left untreated. In addition, hypoglycemia can present with poor cardiac output and signs of decreased perfusion. Furthermore, hypoglycemia may also be an indication of sepsis. If glucose levels fall below 60 mg/dL, a bolus of 2 to 4 mL/kg of 10% dextrose IV should be administered to increase the serum glucose level. Tight glycemic control may help restore normal energy metabolism to tissues and enhance energy delivery, thus acting as a positive inotrope.[85]

Potassium Derangements

Potassium derangements can result from diuresis, renal insufficiency or failure, and steroid administration. Potassium can be supplemented if hypokalemia becomes a significant problem. However, the adverse effects of hyperkalemia are clearly more detrimental than hypokalemia. If treatment of hypokalemia is pursued, potassium replacement should be undertaken in the face of a normal serum pH, because serum potassium levels may be falsely reduced in the presence of an alkaline pH. The opposite is true if the patient is acidotic.

Potassium supplementation can be administered using potassium chloride or potassium acetate. Potassium acetate can be used to buffer a metabolic acidosis secondary to renal disease, increased lactate production, or hyperchloremia. The adverse effects of hypokalemia most likely to affect potential donors are arrhythmias.

Calcium Derangements

Hypocalcemia typically occurs secondary to large volume replacement with colloids such as albumin,[53] massive blood transfusions that result in large amounts of citrate reducing free calcium concentrations, and sepsis. Calcium is important for muscle contraction and acts as an inotrope to support blood pressure. The use of calcium chloride is preferred because this agent is not dependent on hepatic activation unlike calcium gluconate. Calcium chloride should be administered through a central venous catheter because extravasation of this agent into the skin and soft tissues can cause profound tissue necrosis. A continuous infusion of calcium chloride or calcium gluconate can be used to treat hypocalcemia or augment blood pressure support in donors with persistent hypocalcemia. The use of calcium supplementation should be guided by ionized calcium levels.

Metabolic Acidosis

There are several causes for metabolic acidosis. Decreased cardiac output results in impaired oxygen delivery to the tissues resulting in a lactic acidosis. Excessive bicarbonate losses from the gastrointestinal tract, renal wasting of bicarbonate secondary to renal insufficiency or failure, and increased chloride in IV fluids can result in a hyperchloremic metabolic acidosis. It is important to define and treat the underlying cause of the metabolic acidosis, such as increased chloride, ongoing bicarbonate losses, or restoration of normal cardiac output to improve oxygen delivery to the tissues. Agents such as sodium bicarbonate or buffers such as tromethamine can be used to correct a metabolic acidosis. Excessive doses of sodium bicarbonate may exacerbate an existing hyperosmolar state and increase production of carbon dioxide, which must be compensated for by increasing minute ventilation to avoid carbon dioxide retention and a resultant respiratory acidosis. Tromethamine should be avoided in patients with renal insufficiency. Tromethamine administered in high doses may cause hypoglycemia, and can exacerbate an existing coagulopathy.[86]

Coagulation Abnormalities

Coagulation abnormalities can occur secondary to the release of tissue thromboplastin and cerebral gangliosides from injured brain.[87,88] Thrombocytopenia and platelet dysfunction can be induced by common drugs such as heparin, antibiotics, β-blockers, calcium channel blockers, and hespan.[89] Patients with liver disease can have reduced synthesis of vitamin K-dependent clotting factors. Interestingly, coagulopathy may be related to the catecholamine surge associated with traumatic brain injury as well.[51,90] A dilutional coagulopathy can also occur from massive transfusions without replenishing coagulation factors. Correction of the coagulopathy can be treated using fresh frozen plasma, platelets, and cryoprecipitate. The goal of blood product replacement for coagulopathy should be tailored on the basis of the derangements encountered.

Platelets should be used for inadequate circulating platelets and clinically significant bleeding, with a reasonable goal being greater than 50 000/mm^3. If surgery is anticipated, a platelet count greater than 75 000/mm^3 is preferable. Fresh frozen plasma is a rich source of coagulation factors and can be used to keep the prothrombin time less than 25 and partial thromboplastin time less than 40 seconds. Hypofibrinogenemia can be treated with cryoprecipitate for fibrinogen levels less than 100 mg/dL.

Vitamin K is another adjunct in the management of coagulopathy, which can be administered intramuscularly or intravenously. IV administration of vitamin K is preferred because intramuscular injections can result in a depot effect with erratic uptake into the systemic circulation. The effects of vitamin K are slower compared to fresh frozen plasma, platelet, and cryoprecipitate administration. The use of aminocaproic acid (Amicar), an antifibrinolytic agent, and other similar hemostatic agents is not recommended because microvascular thrombosis may be induced in donor organs.[50]

Coagulation abnormalities can also be influenced by hypothermia; therefore, it is essential to keep pediatric organ donors normothermic. Vasodilation with an inability to compensate for heat loss by shivering or vasoconstriction is a common cause of thermoregulatory instability following neurologic

death. In addition, infusion of large volumes of IV fluids at room temperature to treat DI and volume depletion can contribute to hypothermia. Hypothermia can promote cardiac dysfunction, arrhythmias, coagulopathy, a cold-induced diuresis secondary to decreased renal tubular concentration gradient, and a leftward shift of the oxyhemoglobin dissociation curve resulting in decreased oxygen delivery to the tissues.[91] Radiant warmers, warm blankets, thermal mattresses, warm IV fluids or a blood warmer for infusion of blood products, and environmental warming will help maintain body temperature. In addition, the temperature of the heated inspired gases can be adjusted to help control body temperature. Prevention of hypothermia is essential to prevent deterioration of potential organ donors.

Summary

Care of pediatric organ donors requires a skilled team of specialists who deal not only with the deceased child, but also the family, and multiple potential recipients. Early involvement of the organ procurement organization and coordination with physicians, social workers, chaplains, and family support services enhances the chance for the family to understand and agree to organ donation. The option to donate should be available to every family and it should be the expectation that the family will be approached in a professional, compassionate manner that allows for open discussion during the most difficult, agonizing time in their lives. Care of pediatric organ donors is a natural extension of care for a critically ill and injured child. Early recognition of brain death and shifting the focus of care to preservation of organs for transplantation is essential for positive outcomes and can greatly facilitate the management of this selected group of patients. This continuum of care and anticipation and timely intervention are essential in well-managed pediatric donors to preserve organ function. Meticulous care of potential donors, combined with teamwork, will result in more transplantable organs with better function that can mean the difference between life and death for another person waiting for a needed organ. The best outcomes occur as pediatric intensive care specialists work together with a dedicated team of professionals to provide specialized care to a very limited group of potential donors.[92]

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