Trauma Reports

EVIDENCE-BASED MEDICINE FOR THE ED

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Organ Procurement in Trauma

According to the Organ Procurement and Transplantation Network, there are currently more than 100,000 people in the United States in need of life-saving organ transplants. Unfortunately, this demand far exceeds the number of available organs, and each day an average of 18 people die because of the shortage of organs and organ donors.[1] For this reason, it is vitally important that every potential organ be salvaged and used. This article provides a comprehensive review of organ procurement in trauma.

— The Editor

Introduction

Each year, traumatic injuries account for more than 120,000 deaths, making it the fourth leading cause of mortality in the United States.[2] A recent abstract from a Level I trauma center in Washington, DC found that approximately 50% of trauma death victims at their institution during a four-year period ending December 2006 were appropriate for initiation of organ preservation, and that 70% of those were ultimately suitable donor candidates.[3] Extrapolating these data nationwide suggests that up to 42,000 trauma death victims annually could be suitable organ donor candidates.

Trauma victims represent a potentially large source pool of organs for transplantation, but currently just 30% of all deceased organ donors come from the trauma patient population. Since trauma victims, on average, tend to be younger and healthier than other potential organ donors, such as medical intensive care unit patients, an emphasis needs to be placed on maximizing the life-saving organ donation potential of these patients by increasing recovery rates and by minimizing organ damage after patients arrive in the trauma bay.

As of November 2009, there had been 26,096 solid organ transplants year-to-date, coming from just 13,348 donors (living and cadaveric).[4] As the preceding data suggest, increasing the percentage of organs recovered from trauma victims by just a small number could have a significant impact on survival rates of those in need of an organ transplant.

Historical donor criteria were strict, often limiting donors to the ages of 10 years to 50 years without any co-morbid conditions. Restrictions have been eased significantly due to increasing demand. Generally, there are two absolute contraindications to organ donation: active malignancy and HIV infection with or without AIDS.[5] Exclusion criteria are not universally held. Regional transplant centers have varying absolute and relative exclusion criteria for potential organ donors, and it is important for the treating physicians to refer all potential organ donors and allow their local organ procurement organization (OPO) to evaluate for potential organ donation. *(See Table for example donor criteria used by The New England Organ Bank and California Transplant Donor Network.)*

General Management Considerations

The trauma victim as a potential organ donor represents a unique situation in which maintenance of organ function must be considered even after

Executive Summary

- Trauma victims may be potential organ donors.
- Emphasis on maximizing the life-saving organ donation potential of these patients may increase organ recovery rates.
- Brain death causes a systemic proinflammatory state that results in significant immunological and metabolic derangements.
- Perfusion should be maintained using the minimum required dose of vasopressor and CVP should generally be kept between 8-10 mmHg (slightly lower for potential lung donors).
- Hormone replacement therapy with desmopressin, thyroid hormone, and methylprednisolone should be initiated.
- Careful attention should be paid to electrolytes, particularly sodium, and corrected with dextrose and water or half-normal saline if sodium is greater than 155 mEq/L.

brain death. Unfortunately, 17%-25% of all potential donors are lost due to profound cardiopulmonary and metabolic instability resulting from brainstem herniation and brain death.[6,7] The trauma surgeon and emergency physician must pay particular attention to such factors as blood pressure and ventilator settings, fluid balance, and temperature control during both the resuscitation and after brain death.

Early treatment of the trauma victim should proceed in the usual fashion, with attention to airway, spinal immobilization, ventilation, oxygenation, maintenance of perfusion, and identification of treatable injuries. It should be emphasized that treatment should continue in the manner deemed appropriate for the injuries sustained until the determination of brain death. After this determination, attention is directed toward maintenance of organ function and obtaining familial consent, if needed. The donor should be managed with ICU-level care, as many pathologic states are encountered in the brain-dead patient that could compromise donor organ viability and transplantation success.

During and following brain death, multiple physiological changes occur in virtually all organ systems in response to loss of brain and brainstem function. In the trauma victim, brain death is frequently the result of increased intracranial pressure (ICP) after severe brain injury. This ICP leads to a rostral-caudal pattern of cerebral herniation and brainstem ischemia. Initially, mesencephalic ischemia results in parasympathetic activation which, in turn, leads to sinus bradycardia and hypotension. Subsequent pontine ischemia produces sympathetic stimulation, which leads to hypertension and the characteristic Cushing's response of bradycardia and hypertension.[8] Next, "autonomic storm" develops, characterized by intense sympathetic activity and massive catecholamine release due to medullary ischemia. This dramatically increases myocardial work and jeopardizes end-organ blood flow.[9] This is followed by loss of spinal cord sympathetic function, which results in loss of vasomotor tone with subsequent vasodilation and diminished cardiac output. Finally, damaged and necrotic brain tissue causes a release of plasminogen activator and thromboplastin, which increases the risk of developing disseminated intravascular coagulation (DIC).[10]

Brain-dead donors are also particularly vulnerable to diabetes insipidus due to pituitary gland ischemia, fluid and electrolyte imbalance, hypothermia, hypotension, endocrine dysfunction, and oxygenation challenges.[11] Furthermore, recent literature suggest that a brain-death-related systemic inflammatory response could be an important cause of lower recipient survival after cadaveric compared to living donor organ transplants.[12,13] As a result of these changes, only a minority of victims are able to maintain hemodynamic stability without intensive therapy.

Cardiovascular Considerations. Maintenance of cardiovascular stability during both the initial trauma resuscitation and following brain death until organ procurement is of vital importance. Unfortunately, this process can be complicated in the trauma victim, as hemodynamic instability may be the result of not only acute blood loss but as a consequence of brain death itself. The goals of a potential donor's hemodynamic management are to establish normovolemia, to maintain adequate blood pressure, and to optimize cardiac output so as to ensure end-organ perfusion pressure with the least support of vasoactive drugs, which could potentially induce organ ischemia. Adequate management requires, at a minimum, central venous pressure (CVP) monitoring. Insertion of a pulmonary artery catheter (PAC) should be considered if hemodynamic dysfunction persists despite adequate fluid resuscitation and/or if vasopressors are required.

Hemodynamic instability from brain death occurs in two phases. In the first phase, brain death leads to a massive sympathetic nervous system response as a result of a large release of catecholamine stores, causing tachycardia, hypertension, and increased systemic vascular resistance (SVR).[6] The consequences of this autonomic storm include increases

in cardiac workload and myocardial oxygen demand that can cause cardiac myocytolysis and necrosis.[14] Recent studies suggest that treating autonomic storm may reduce myocardial dysfunction and improve the success rate of cardiac transplantations.[8] Audibert and colleagues studied a cohort of 46 potential cardiac donors in whom autonomic storm was ultimately diagnosed in 29 patients. Of these 29 patients, 12 patients were treated (six with esmolol, five with urapidil, and one with nicardipine). Treatment of autonomic storm was associated with significantly higher LVEF and increased probability of successful cardiac transplantation.[15]

This period of intense catecholamine release is typically short-lived and followed by a second phase of brain-death-related hemodynamic instability resulting from autonomic collapse due to depletion of available catecholamine stores. This second phase of hemodynamic dysfunction is characterized by diminished sympathetic activity, reduced vascular tone, decreased peripheral arterial and venous resistance, and impaired cardiac output.[6] Concurrent traumatic blood loss may further complicate these conditions.

This hypovolemic and distributive vascular state requires careful hemodynamic monitoring. Mean arterial blood pressure should be maintained between 60 mmHg and 70 mmHg and central venous pressure between 8 mmHg and 10 mmHg.[16] Aggressive fluid resuscitation and traumatic blood loss may result in anemia and blood products may be required to keep the hemoglobin at least 10 g/dL.[17] If a PAC is placed, pulmonary capillary wedge pressure (PCWP) should be kept at 6-10 mmHg, cardiac index (CI) > 2.4 L/min/m^2, and SVR 800-1200 dynes/s/cm.[18]

Dopamine has historically been the vasopressor of choice, with a target dose of less than 10 mcg/kg/min.[13] However, recent studies have failed to show a beneficial effect on renal and hepatic circulation. Furthermore, doses greater than 10 mcg/kg/min, especially if donor systolic blood pressure is consistently less than 80-90 mmHg, have been shown to be associated with a greater incidence of acute tubular necrosis.[16] Additionally, dopamine may suppress the function of the anterior pituitary hormones.[19,20] Therefore, some sources have advocated a move toward earlier use of vasopressin, and this is currently the recommended initial therapy by the American College of Cardiology. *(See section on "Endocrine Considerations" below for dosing guidelines.)*[21]

Endocrine Considerations. Brain death results in profound metabolic and endocrine derangements that can have serious implications for potential organ donors. Dysfunction of the hypothalamic-pituitary axis is one of the most important conditions to recognize, as diabetes insipidus due to loss of anti-diuretic hormone (ADH or vasopressin) leads to severe volume and electrolyte disturbances. In fact, levels of ADH are undetectable in 75%-90% of all brain-dead organ donors.[22] Diabetes insipidus leads to polyuria which, in turn, causes volume loss, hypernatremia, and hemodynamic instability.[23] Other electrolyte abnormalities include hypokalemia, hypomagnesemia, hypocalcemia, and hypophosphatemia.

Replacement of vasopressin to treat diabetes insipidus is critical. Vasopressin mediates it effects through three receptor subtypes: V1, V2, and V3. V1 receptors are located on the vascular endothelium, and their stimulation is responsible for the vasopressor effect of vasopressin. V2 receptors are found within the renal collecting ducts and mediate the antidiuretic effect, while V3 receptors stimulate adrenocorticotropin release. Desmopressin (DDAVP or 1-desamino-8-D-arginine vasopressin) is a vasopressin analogue that is highly selective for the V2 receptor subtype. It has no significant vasopressor activity, has a longer half-life than vasopressin, and is the drug of choice for replacement therapy.[8,16,24] Begin with a loading dose of 8 ng/kg followed by an infusion of 4 ng/kg/hr and titrate as needed.[6]

In addition to diabetes insipidus caused by low levels of vasopressin, there are also significant decreases in

Table. Organ Donor Exclusion Criteria: New England Organ Bank, California Transplant Donor Network

Absolute Contraindications

- Age older than 80
- HIV infection
- Active metastatic cancer
- Prolonged hypotension or hypothermia
- Disseminated intravascular coagulation
- Sickle cell anemia or other hemoglobinopathy

Relative Contraindications

- Malignancy other than the central nervous system or skin that is in remission
- Hypertension
- Diabetes
- Hepatitis B or C
- History of smoking

circulating levels of adrenocorticotropic hormone (ACTH), cortisol, tri-iodothyronine (T3), thyroxine, and insulin. Decreased T3 levels result in a switch from aerobic to anaerobic metabolism, which disrupts cellular function. A bolus of 4 mcg of T3 can be administered, followed by an infusion of 3 mcg/hr. The use of supplemental T3 has been shown to reverse myocardial dysfunction and decrease inotropic requirements.[25,26] For example, Jeevanandam and colleagues report that T3 replacement at a maximal dose of 0.6 mcg/kg resulted in lower ventricular filling pressures, hemodynamic stabilization, and decreased pressor requirements.[26] Salim and colleagues have also shown that supplemental T4, when given to hemodynamically unstable organ donors requiring significant vasopressor support, resulted in more donated organs than in those patients that did not receive T4.[27]

Methylprednisolone should be given to offset the cortisol deficiency caused by brain death. It has been shown to improve outcomes in lung and liver transplantation. Kotsch and colleagues performed a prospective randomized study with 100 deceased liver donors. Donor treatment consisted of 250 mg of methylprednisolone at the time of consent for organ donation followed by an infusion of 100 mg/hr until organ recovery.[28] Their study showed a significant decrease in circulating inflammatory mediators, significantly ameliorated ischemia/reperfusion injury, and decreased the incidence of acute graft rejection. Follette and colleagues performed a retrospective analysis designed to determine the effect of high-dose steroid administration on oxygenation and donor lung recovery after brain death.[29] They examined 118 consecutive organ donors, 80 of whom received methylprednisolone (mean 14.5 ± 0.06 mg/kg) after the OPO began management of the donor, and 38 donors who received no steroids. There were no differences in hemodynamic, clinical, or demographic variables between groups. However, the steroid-treated donors had a progressive increase in oxygenation before aortic cross-clamping, while the steroid-free group had progressive decrease (mean change in PaO_2/FiO_2 16 ± 14 vs -34.2 ± 14; $p = 0.01$). Additionally, the number of procured lungs was significantly greater in the steroid-treated group (25/80 vs 3/38; $p < 0.01$).

Taken together, the practice of administering thyroid hormone (either T3 or T4), steroids, and desmopressin is known as hormonal replacement therapy (HRT) or a "Papworth cocktail." The practice was pioneered in the late 1980s and has been used routinely since.[30] Several recent studies have investigated the role of HRT in organ donor management. A retrospective study by Salim and colleagues looked at an aggressive donor management (ADM) strategy consisting of 2 grams of solumedrol, 20 units of regular insulin, and 20 mcg of T4 followed by a continuous T4 infusion. The study compared two cohorts of patients, one cohort before the institution of ADM and the other cohort after an ADM strategy. Those patients from the post-ADM period had an 87% decrease in the number of donors or organs lost to hemodynamic instability.[31] Another study by Rosendale and colleagues retrospectively compared brain-dead donors receiving HRT to those that did not. HRT in this study consisted of methylprednisolone, vasopressin, and either T3 or L-thyroxine. The number of organs from the HRT donors was 22.5% greater than those from the non-HRT group.[25]

Finally, hyperglycemia should be treated with an initial insulin infusion of 1 unit/hr, which may be titrated to achieve a blood glucose level of 120-180 mg/dL.[6]

Temperature. Thermoregulation after brain death results from multiple pathophysiologic mechanisms. Factors contributing to hypothermia include cerebral herniation, leading to impairment of hypothalamic thermoregulation; dysfunction of sympathetic tone, which results in peripheral vasodilation with subsequent heat emission; and finally, reduced metabolic activity.[15] Hypothermia, in turn, results in a myriad of adverse effects, such as cardiac dysfunction and arrhythmias, impaired microcirculation and oxygen consumption, coagulopathy, and cold-induced diuresis. For these reasons, core body temperature should be maintained at > 35 °C by using warming blankets and warmed fluids.

Coagulation. Due to the cascade of pathophysiological effects described previously, brain death is associated with an increased risk of DIC.[32] The goals of therapy are to maintain an international normalize ratio (INR) of < 2.0 and a platelet count of greater than 80,000/mm^3. Some authors advocate replacement of blood products, including fresh frozen plasma and platelets, to approximate these goals.[58] It should be mentioned, though, that the concept of blood-product replacement is not universally held out of concern of "feeding" a low-grade DIC.

Electrolytes and Acid–Base Balance. Disturbances in electrolytes, most importantly hypernatremia and hypokalemia, are common after brain death. Multiple mechanisms, both physiologic and as a result of resuscitation, are responsible for these derangements and it is important for the treating physician to be aware of how therapeutic interventions may affect electrolyte balance. As mentioned previously, brain death leads to diabetes insipidus, with resultant water loss. Additionally, excessive fluid replacement in an attempt to maintain hemodynamic stability as well as osmotic diuresis with mannitol for the treatment of increased intracranial pressure both lead to the exacerbation of hypernatremia. Unfortunately, high donor sodium concentrations have been associated with higher rates of primary hepatic graft failure by promoting the accumulation of osmoles within hepatocytes. Subsequent transplantation into recipients with relatively normal sodium concentrations then

results in intracellular water accumulation, cell lysis, and death. Keeping donor serum sodium below 155 by infusing 5% dextrose in water or half-normal saline (0.45%) has been shown to decrease the incidence of liver allograft failure.[33] Totsuka and colleagues looked at 181 consecutive orthotopic liver transplants. The cases were divided into three groups based on donor serum sodium concentration: group A, serum sodium < 155 mEq/L before organ procurement (n = 118); group B, peak sodium > 155 mEq/L and final sodium < 155 mEq/L (n = 36); and group C, final sodium > 155 mEq/L (n = 27). There were no significant differences in donor or recipient variables among the three groups. The frequencies of graft loss were 15/118 (12.7%) in group A, 4/36 (11.1%) in group B, and 9/27 (33.3%) in group C ($p < 0.05$).[32]

Finally, the clinician should pay consideration to acid-base balance during the peri-donation period and how therapeutic interventions may be affecting this balance. For example, hyperventilation for the treatment of increased intracranial pressure will result in a respiratory alkalosis which may impair tissue oxygenation.

Fluid Therapy. Competing requirements for organ perfusion may result in antagonistic strategies for fluid replacement. A minimally positive fluid balance is associated with higher rates of successful lung procurement,[34] but aggressive volume replacement is often necessary to maintain renal perfusion and increase the chances of kidney survival after transplant.

The solution to this problem is the early assessment of the suitability of the potential donor organs and the institution of a focused medical management strategy when one or more organs are clearly not suitable for transplant. For example, in the trauma victim who has suffered a penetrating thoracic injury rendering the lungs unusable, the team caring for the donor needs to quickly recognize that a more aggressive fluid resuscitation strategy, with emphasis on adequate liver and renal perfusion, is appropriate.

Organ-Specific Considerations

The Lung. Once a trauma victim has been identified as a potential lung donor (age < 55 years, clear chest x-ray, absence of chest trauma, smoking history < 20 pack-years) pulmonary preservation techniques should be initiated. Unfortunately, successful lung procurement is very challenging, with more than 30% of all lungs that are theoretically suitable for donation not being recovered.[35] The reason is that brain death is associated with neurogenic pulmonary edema and an intense inflammatory response.[6,8,16] Pulmonary function also frequently deteriorates due to aspiration, pneumonia, mucous plugging, low tidal volumes, and lack of positive end expiratory pressure (PEEP). Finally, the antagonistic competing organ interests related to fluid resuscitation further exacerbate pulmonary edema. For example, a minimal-volume strategy limits the development of extravascular lung water in the setting of brain-death-related pulmonary edema, but this tactic could potentially have deleterious effects on the preservation of other organs, such as the kidney.

Neurogenic pulmonary edema is thought to result from severe vasoconstriction and elevated SVR secondary to catecholamine surge, which, in turn, causes large fluid volume shifts from the periphery to pulmonary circulation.[36] Altered pulmonary capillary permeability may be an additional cause.[37]

Inflammation also appears to play a role in lung injury in the brain-dead patient.[38] In fact, several studies have looked at attenuation of the inflammatory response with steroids after brain death, and have demonstrated increased lung donor utilization by limiting cytokine-mediated cellular injury.[39-41] For example, Venkateswaran and colleagues performed a randomized trial involving 60 lung donors and examined the effect of steroids on the extravascular lung water index (EVLWI). They showed that the administration of 1 gram of methylprednisolone as soon as possible after consent and initial assessment attenuated the increase in the EVLWI, reducing progressive lung water accumulation.[40]

Appropriate management of the potential lung donor requires optimization of ventilation, airway pressures, and fluid balance. Ventilation can be assisted by aggressive pulmonary care including chest physiotherapy and management of secretions by proper suctioning and repositioning. The FiO_2 should be kept as low as possible while still keeping the paO_2 greater than 100 mmHg. Careful use of crystalloids and colloids, with a CVP goal of 6-8 mmHg and a wedge pressure of 8-12 mmHg, should be sought to minimize the risk of pulmonary edema.[42] A PEEP of 5 cm H_2O and a tidal volume of 10-12 mL/kg to keep $PaCO_2$ between 30-35 mmHg is recommended.[35] The American College of Cardiology recommends a systolic blood pressure of 90-140 mmHg.

The two most common causes of hypoxia preventing recovery of lungs for transplantation are atelectasis and excessive fluid replacement. Unfortunately, fluid replacement is a necessary evil in most cases to maintain cardiovascular stability and adequate tissue perfusion. In potential lung donors, colloid solutions are recommended to sustain oxygenation and minimize the accumulation of pulmonary edema.[38] To prevent atelectasis as well as to remove secretions and foreign bodies, bronchoscopy should be performed on all potential lung donors. Doing so also permits sampling of secretions and isolation of pathogens, which helps to optimize antibiotic therapy in both donor and recipient, a measure of extreme importance as pneumonia is a chief cause of lung rejection.[43]

Despite advances in respiratory care and lung transplant organ allocation algorithms, waiting lists for lung transplants continue to grow. Unfortunately, attempts at

increasing organ donation rates have generally had little impact on the number of transplants. Therefore, there has recently been an emphasis on taking greater advantage of the existing pool of cadaveric organ donors. To this end, the once-strict lung donor selection criteria have been relaxed, and several reports have shown that recipient outcomes using less-than-ideal lungs have outcomes equivalent to those using ideal lungs.[44] It should be emphasized that aggressive pulmonary management, including strict regulation of ventilatory parameters, moderate use of PEEP, frequent suctioning, inhalation of albuterol, use of bronchoscopy, a minimally positive fluid balance, and use of appropriate antibiotics were critical to successful transplantation using marginal donor lungs.[44]

The Liver. Appropriate donor selection is crucial to a successful liver transplantation. Evaluation criteria include donor age, body mass index, blood type, and medical history such as hepatobiliary disease, infection, malignancy, and donor use of drugs and/or alcohol. Also, cause of death and hemodynamic stability, potentially important considerations in the trauma victim, are taken into account. The ideal donor is younger than 50 years of age, has no hepatobiliary disease, is hemodynamically stable, and has no severe abdominal trauma, systemic infection, or cancer. Unfortunately, as with lungs, the paucity of donors has challenged traditional notions of what makes an ideal liver donor and has necessitated the use of extended donor criteria. Extended donor criteria have not been formalized, but donors are generally considered to be marginal if they are older than age 65, have a peak serum sodium of greater than 155 mEq/L, have been on high-dose or multiple pressors, or had a prolonged interval between brain death and procurement. However, as with most organ transplant donors, absolute contraindications to liver donation are the presence of transmissible agents that may lead to death or severe disease of the recipient. Such contraindications include Creutzfeldt-Jakob, HIV, and active malignancy.[45]

Fortunately, the liver has several inherent attributes that generally improve its candidacy as a transplantable organ, including a weak immunologic response and its ability to tolerate long periods of hypoperfusion due to its large physiological reserve. Nevertheless, the inflammatory processes related to brain death and reperfusion injury take their toll on liver allografts. ABO incompatibility is independently associated with an increased rate of recipient death and retransplantation.

As previously mentioned, the liver is particularly sensitive to donor electrolyte disturbances, namely hypernatremia (sodium concentration > 155 mEq/L). High sodium concentrations result in the accumulation of osmoles within the liver cells which, after transplantation into a recipient with normal sodium levels, promote intracellular water absorption leading to cell lysis and death. For this reason, maintaining eunatremia in the cadaveric liver donor during the peri-procurement period is critical. Keeping the donor plasma sodium below 155 mEq/L, maintaining CVP between 8-10 mmHg, using a low PEEP to prevent hepatic congestion, and restoring liver glycogen reserves with adequate nutrition may help decrease the incidence of allograft failure.[45]

The Kidney. As discussed above, brain death results in significant hemodynamic changes that may damage the kidneys, and it is imperative that the trauma surgeon, intensivist, and emergency physician be aware of these changes and their effects, as well as the implications of their corrective action, on the chances of allograft survival.

Vasopressors are frequently required in the brain-dead trauma victim to support blood pressure. As discussed previously, dopamine has historically been the pressor of choice. However, high doses of dopamine (> 10 mcg/kg/min) may compromise renal perfusion and increase the incidence of acute tubular necrosis and allograft failure. Conversely, epinephrine improves systemic hemodynamic function while maintaining renal perfusion. Furthermore, combination therapy with multiple catecholamines has been associated with a reduction in the rates of acute rejection and with improved graft survival; unfortunately, there is no consensus on the specific combination of catecholamines.[42]

Because glomerular filtration declines below a systolic blood pressure of 80-90 mmHg, prompt hemodynamic support of potential organ donors is important to maintain renal perfusion and diuresis. If urine output drops below 1 mL/kg/hr despite optimal hemodynamic management, diuretics such as furosemide and mannitol should be considered.[42]

Declaration of Death

In most cases, recovering organs from a potential donor comes after the declaration of brain death. Unfortunately, the definition of death itself has long been a matter of debate and has evolved as medical technology and knowledge have advanced. Importantly, the development of the mechanical ventilator in the 1950s and 1960s challenged the idea of what death meant, as we were now able to artificially keep bodies alive despite cessation of brain function. Initial attempts at defining death in the post-ventilator era began in 1968 with the development of the Harvard criteria.[48] The Harvard committee consisted of a multidisciplinary group of scholars, and the final report was not evidence-based but, rather, based on the experience of the committee members. The criteria required that the patient be unreceptive and unresponsive, demonstrate no movement or breathing, have no reflexes, and have a "flat EEG."

In 1995, the Quality Standards Subcommittee of the American Academy of Neurology reevaluated the clinical criteria for brain death and the procedures of testing for

adults.[49] According to this document, four prerequisites must be met before any testing for brain death may be done:

1. There must be clinical or neuroimaging evidence of a CNS catastrophe;
2. Complicating medical conditions that may confound clinical assessment such as severe electrolyte, acid-base, or endocrine abnormalities must be corrected;
3. The patient must be free of drug intoxication or poisoning; and
4. Core temperature must be greater than 90 °F or 32 °C.

After these prerequisites have been met, three cardinal findings must be present:

1. Coma or unresponsiveness measured by the absence of motor response to pain in all extremities;
2. Absence of brainstem reflexes (no pupillary response to intense light, loss of oculocephalic reflex, loss of cold calorics, loss of corneal reflex, and absent gag reflex);
3. Apnea testing defined as the absence of respiratory movements with a pCO_2 > 60 mmHg or an increase of 20 mmHg pCO_2 over the patient's normal baseline. Apnea may be confirmed by visual observation or by application of EMG leads testing appropriate respiratory muscles. Hypotension is common during apnea testing and is typically avoided by adequately preoxygenating with 100% FiO_2. There is a theoretical concern that any hypotension and/or hypoxemia caused by this test may have negative consequences on the viability of potential donor organs, but this has not been verified in clinical practice.[50]

If any portion of the clinical bedside examination cannot be done, then a confirmatory test such as conventional cerebral angiography or magnetic resonance angiography, scintigraphy or nuclear angiography, or transcranial Doppler (TCD) ultrasonography should be performed. These tests are used to demonstrate a lack of cerebral blood flow and confirm the diagnosis of brain death. Of note, TCD measures cerebral blood flow in the vessels above the tentorium and may not be accurate in patients that have suffered a devastating brainstem lesion in that it may show cerebral blood flow despite clinical evidence of brain death.

Although EEG testing was a historical component of brain death determination as outlined by the Harvard criteria, it has since been abandoned as requirement. In fact, the ICU can be a technically difficult environment for obtaining an EEG due to the high amount artifact produced by mechanical and electronic equipment.[51]

Finally, adequate documentation in the medical record must be emphasized. Details should include the etiology and irreversibility of the condition, a complete neurological exam including brainstem reflexes, motor response to pain, and absence of respirations. If a confirmatory test is done, the results must be documented.

Donation after Cardiac or Circulatory Death

The continually increasing demand for organs and supply-demand mismatch has led to the introduction of the principle of Donation after Cardiac or Circulatory Death (DCD) or "non-heart-beating" organ donation, which may be further classified as controlled or uncontrolled. In controlled DCD, circulatory arrest follows consented termination of life-support measures. Uncontrolled DCD (uDCD), however, involves procuring organs from patients in whom unsuccessful resuscitation efforts are terminated in the field or emergency department; as of the time of this writing, uDCD is still experimental. DCD in any form is contentious. However, it represents a significant source of potential organs by opening the donor pool to those patients who are facing or who have faced cardiac death but may be neurologically intact and may not fulfill the criteria for brain death.

DCD is controversial because there are ethical, religious, legal, and even scientific uncertainties about whether the patient is actually dead. Organ procurement is only permissible when 1) the donor is dead (the "dead donor rule"), and 2) the recovery of organs cannot be a contributing factor in the death of the donor. This begs the question, "How do we define death?" In most circumstances, this question is answered using the criteria for brain death outlined previously. Unfortunately, in the event of cardiopulmonary arrest, the non-heart-beating donor can be neurologically intact and may not fulfill brain death criteria immediately after the cessation of cardiac activity. To complicate matters further, because it is not clear if termination of cardiopulmonary function is irreversible after only a short time, the Institute of Medicine (IOM) has recommended a waiting period of 5 minutes before allowing organ recovery.[52] Waiting longer than 5 minutes will prolong warm ischemia time and may compromise the viability of the procured organs. There have been case reports of autoresuscitation with return of neurologic function, the so-called "Lazarus phenomenon," after 10 minutes of cardiac asystole.[53,54]

While an in-depth analysis of the DCD dilemma is beyond the scope of this report, several excellent review articles are available.[55-57]

Organ Allocation System

The U.S. Department of Health and Human Services contracts with the United Network for Organ Sharing (UNOS)[4] to maintain a centralized database of potential organ recipients. The procurement of organs is accomplished regionally and managed by local OPOs. The OPOs are responsible for overseeing the identification, evaluation,

and management of potential organs and organ donors, while UNOS is responsible for matching recipients to those organs.

The Centers for Medicare & Medicaid Services and The Joint Commission require the timely reporting by hospitals of all deaths or impending deaths to their local OPO. Ideally, this reporting should be done before brain death occurs or before the withdrawal of life support so that the suitability of the potential donor can be determined.[58] The OPO is the only organization that may determine if an individual is medically acceptable for donation. If the donor is appropriate, only a specially trained representative may approach the family to discuss donation or explain the donor's registry status.

UNOS maintains a centralized computer network linking all OPOs and transplant centers. At each transplant center, a potential recipient is carefully evaluated, including his her mental and physical condition. If the transplant center feels that a patient is a potential transplant candidate, then the patient will be added to the national transplant waiting list maintained by UNOS. At this point, this is not a ranked list, but rather simply a pool of national transplant candidates.

Once an organ has been identified by an OPO, a transplant coordinator from that OPO accesses the UNOS network. It is at this point that a ranked list is actually generated, as the computer matches each patient in the pool against the characteristics of the donor organ. The order of the generated list is determined by multiple factors. The most important factor once blood type and body size have been matched is severity of illness of potential recipients; other factors include length of time on the waiting list and distance between the potential recipient and the donor.

After the rank list has been generated, the organ is offered to the transplant team of the first patient on the list. However, the first patient may not get the organ on the list for several reasons, including recipient condition or unavailability of the recipient. When the patient has been selected, the patient must be immediately available and willing to be transplanted. He or she must also be healthy enough to undergo major surgery at the moment the organ becomes available.

Conclusions

Trauma patients are a large source of potential organs, but successfully recovering and transplanting these organs requires careful attention to the donor even after the trauma resuscitation and declaration of brain death. The initial management of the potential post-traumatic organ donor should proceed in the usual fashion, with attention to the airway, breathing, circulation, and identification and treatment of injuries.

Brain death causes a systemic pro-inflammatory state which results in significant immunological and metabolic derangements.

Different organs have competing hemodynamic interests (for example, the lung and kidney). If possible, the organs intended for recovery, as well as those that are unsuitable, should be identified as early as possible so that donor stabilization can be optimized for the organs of interest.

Particular attention should be paid to the cardiovascular, pulmonary, and endocrine systems of potential donors. Perfusion should be maintained using the minimum required dose of vasopressor and CVP should generally be kept between 8-10 mmHg (slightly lower for potential lung donors). Hormone replacement therapy with desmopressin, thyroid hormone, and methylprednisolone should be initiated. Careful attention should be paid to electrolytes, particularly sodium, and corrected with dextrose and water or half-normal saline if sodium is greater than 155 mEq/L.

UNOS maintains a national database of transplant candidates and works with OPOs to ensure equitable organ distribution. Unfortunately, the demand for donated organs far exceeds the supply. This discrepancy is only likely to get worse as medical technology continues to improve, keeping a greater number of potential recipients alive longer while waiting for a transplant.

Organs should be procured after declaring brain death according to the recommendations of the Quality Standards Subcommittee of the American Academy of Neurology. However, there is a movement toward non-heart-beating organ donation and practitioners should be familiar with the guidelines of his or her institution.

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CNE / CME Questions

1. Which of the following is one of the multiple pathophysiologic changes that occur following brain death?
   A. Decreased circulating levels of plasminogen activator and thromboplastin
   B. Hyperthermia
   C. Diabetes insipidus
   D. Decreased catecholamine release

2. Which of the following is an electrolyte disturbance commonly seen after brain death?
   A. Hypernatremia
   B. Hyperkalemia
   C. Hypermagnesemia
   D. Hypercalcemia

3. The use of supplemental T3 has been shown to:
   A. increase the rate of successful lung procurement.
   B. decrease incidence of donor hyperglycemia.
   C. decrease donor vasopressor requirements.
   D. increase donor anaerobic metabolism.

4. Which of the following has been shown to be an effect of supplemental methylprednisolone on the brain dead organ donor?
   A. Hyperkalemia
   B. Decreased circulating inflammatory mediators
   C. Hypoglycemia
   D. Increased extravascular lung water

5. Which of the following is *not* a component of hormonal replacement therapy in the brain-dead organ donor?
   A. Steroids
   B. Desmopressin
   C. Thyroid hormone
   D. ACTH

Trauma Reports CNE / CME Objectives

To help physicians:
a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CNE / CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

6. Donor hypernatremia (serum sodium > 155 mEq/L) is a potentially preventable cause of liver graft failure.
   A. True
   B. False

7. According to the most recent guidelines from the American Academy of Neurology, EEG testing is required to confirm brain death.
   A. True
   B. False

8. Which of the following is true about controlled Donation after Cardiac Death (cDCD)?
   A. Circulatory arrest follows consented termination of life-support.
   B. cDCD involves procuring organs from patients in whom unsuccessful resuscitation efforts are terminated in the field.
   C. It represents only a small potential source of organs.
   D. cDCD patients are not neurologically intact.

9. Which of the following is a role of the United Network for Organ Sharing (UNOS)?
   A. Responsible for evaluation of potential donor organs
   B. Responsible for matching recipients to donor organs
   C. Oversees the identification of potential donor organs
   D. Oversees management of potential organs and organ donors

10. Which of the following factors is *not* considered when matching an organ to a potential donor?
   A. Donor and recipient blood type
   B. Recipient body size
   C. Recipient age
   D. Severity of recipient illness

Answers: 1. C, 2. A, 3. C, 4. B, 5. D, 6. A, 7. B, 8. A, 9. B, 10. C

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Please take a moment to answer the following questions to let us know your thoughts on the CNE/CME program. Fill in the appropriate space and return this page in the envelope provided. **You must return this evaluation to receive your letter of credit. ACEP members — Please see reverse side for option to mail in answers.** Thank you.

CORRECT ● INCORRECT ⊘ ✓ ✗ ⚠

1. In which program do you participate? ○ CNE ○ CME
2. If you are claiming physician credits, please indicate the appropriate credential: ○ MD ○ DO ○ Other_____
3. If claiming nursing contact hours, please indicate your highest credential: ○ RN ○ NP ○ Other_____

	Strongly Disagree	Disagree	Slightly Disagree	Slightly Agree	Agree	Strongly Agree
After participating in this program, I am able to:						
4. Discuss conditions that should increase suspicion for traumatic injuries.	○	○	○	○	○	○
5. Describe the various modalities used to identify different traumatic conditions.	○	○	○	○	○	○
6. Cite methods of quickly stabilizing and managing patients.	○	○	○	○	○	○
7. Identify possible complications that may occur with traumatic injuries.	○	○	○	○	○	○
8. The test questions were clear and appropriate.	○	○	○	○	○	○
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11. This activity reaffirmed my clinical practice.	○	○	○	○	○	○
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1. A
   B
   C
   D

2. A
   B
   C
   D

3. A
   B
   C
   D

4. A
   B
   C
   D

5. A
   B
   C
   D

6. A
   B

7. A
   B

8. A
   B
   C
   D

9. A
   B
   C
   D

10. A
    B
    C
    D