Organ Donor Management: What to Expect After Brain Death

Heather Angell, RN, MSN
Organ Procurement Coordinator
Pathophysiology

- Loss of brain stem function results in systemic physiologic instability:
  - Loss of vasomotor control leads to a hyperdynamic state
  - Cardiac arrhythmias
  - Loss of respiratory function
  - Loss of temperature regulation → hypothermia
  - Hormonal imbalance → DI, hypothyroidism
Effective Donor Management

- Stabilize the Donor
  - Facilitate brain death exam or DCD tool
- Manage the Donor
  - To optimize the function and viability of all transplantable organs
- Evaluate each organ
  - Bedside liver biopsy, ECHO, bronchoscopy

21 people die each day awaiting a life-saving organ transplant
Effective Donor Management

- Requires clinical expertise, vigilance, flexibility, and the ability to address multiple complex clinical issues simultaneously and effectively.

- Requires collaboration among UW OTD, donor hospital critical care staff and consultants, and transplant program staff.
Effective Donor Management

- Donor care is not usually assumed until after consent for donation has been obtained.

- It is appropriate to collaborate prior to brain death, consent, etc., to prevent death and keep the option of organ donation open.
Effective Donor Management

- Revision of existing orders or placement of new medical orders is intended to:
  - D/C medications no longer needed or appropriate (e.g., anticonvulsants, Mannitol, sedatives, antipyretics)
  - Continue needed medications or therapy (e.g., vasoactive drug infusions, IV fluids, vent settings)
  - Create “call orders” that inform bedside personnel of the goals for physiologic parameters and alert OPC of changes in donor status
Following the Diagnosis of Brain Death

- Therapy shifts in emphasis from cerebral resuscitation to optimizing organ function for subsequent transplantation.
- The normal sequelae of brain death results in cardiovascular instability & poor organ perfusion.
- Medical staff must focus on:
  - providing hemodynamic stabilization.
  - support of body homeostasis.
  - maintenance of adequate cellular oxygenation and donor organ perfusion.
- Without appropriate intervention, brain death is followed by severe injury to most other organ systems. Circulatory collapse will usually occur within 48 hrs.
Autonomic/Sympathetic Storm

- Release of catecholamines from adrenals (Epinephrine and Norepinephrine) results in a hyper‐dynamic state:
  - Tachycardia
  - Elevated C.O.
  - Vasoconstriction
  - Hypertension
Failure of the Hypothalamus Results in:

- Impaired temperature regulation - hypothermia or hyperthermia
- Leads to vasodilation without the ability to vasoconstrict or shiver (loss of vasomotor tone)
- Leads to problems with the pituitary
Normal Pituitary Gland

- Controlled by the hypothalamus
- Releases ADH to conserve water
- Stimulates the release of thyroid hormone
Pituitary Failure

- Pituitary Failure Results in:
  - ADH ceases to be produced = Diabetes Insipidus
  - Can lead to hypovolemia and electrolyte imbalances
  - Leads to problems with the thyroid gland
Normal Thyroid Gland

- Produces hormones that increase the metabolic rate and sensitivity of the cardiovascular system
  - Levothyroxine (T₄)
  - Triiodothyronine (T₃)
Thyroid Failure

- Thyroid failure leads to:
  - Cardiac instability
  - Labile blood pressure
  - Potential coagulation problems
Cardiovascular System

Intensive Care Management

- “Rules of 100’s”
  - Maintain SBP > 100mmHg
  - HR < 100 BPM
  - UOP > 100ml/hr
  - PaO2 > 100mmHg
- Aggressive fluid resuscitative therapy directed at restoring and maintaining intravascular volume
  - SBP > 90mmHg (MAP > 60mmHg) or CVP ~ 10 mmHg
Neurogenic Pulmonary Edema

- Brain death is associated with numerous pulmonary problems
- The lungs are highly susceptible to injury resulting from the rapid changes that occur during the catecholamine storm
- Left-sided heart pressures exceed pulmonary pressure, temporarily halting pulmonary blood flow
- The exposed lung tissue is severely injured, resulting in interstitial edema and alveolar hemorrhage, a state commonly referred to as neurogenic pulmonary edema
Release of Plasminogen Activator $\rightarrow$ DIC

- Results from the passage of necrotic brain tissue into the circulation
- Leads to coagulopathy and sometimes progresses further to DIC
- DIC may persist despite factor replacement requiring early organ recover

(Also affected by hypothermia, release of catecholamines & hemodilution as a result of fluid resuscitation)
Organ Donor Management (In a Nut Shell)

- Hypertension → Hypotension
- Excessive Urinary Output
- Impaired Gas Exchange
- Electrolyte Imbalances
- Hypothermia
Hypotension Management

- Fluid Bolus – NS or LR (Followed by MIVF NS or .45 NS)
- Consider colloids (seriously)
- Thyroxine (T₄ protocol)
- Vasopressin
- Dopamine
- Neosynephrine
T₄ Protocol

- Background
  - Brain death leads to sudden reduction in circulating pituitary hormones
  - May be responsible for impairment in myocardial cell metabolism and contractility which leads to myocardial dysfunction
  - Severe dysfunction may lead to extreme hypotension and loss of organs for transplant
  - ECHO after 6 hours of starting T₄
  - Heart cath
**T₄ Protocol**

- **Bolus**
  - 15 mg/kg Methylpred
  - 20 mcg T₄ (Levothyroxine)
  - 20 units of Regular Insulin
  - 1 amp D50W

- **Infusion**
  - 200 mcg T₄ in 500 ml NS
  - Run at 25 ml/hr (10 mcg/hr)
  - Titrate to keep SBP > 100

Monitor potassium levels closely!
Vasopressin (AVP, Pitressin)

- Low dose shown to reduce inotrope use
- Plays a critical role in restoring vasomotor tone

**Vasopressin Protocol**
- 4 unit bolus
- 1-4 u/hour – titrate to keep SBP >100 or MAP >60
Diabetes Insipidus Management

- Treatment is aimed at correcting hypovolemia

- Desmopressin (DDAVP) 1 mcg IV, may repeat x 1 after 1 hour

- Replace hourly urine output on a volume per volume basis with MIVF to avoid volume depletion

- Leads to electrolyte depletion/instability monitor closely to avoid hypernatremia and hypokalemia
Diabetes Insipidus

- Goal is UOP 1-3 ml/kg/hr
- Rule of thumb – 500 ml UOP per hour x 2 hours is DI
- Severe cases – Notify OPC, assess clinical situation
Impaired Gas Exchange Management

- Maintain PaO2 of >100 and a saturation >95%
- Monitor ABG’s q2h or as requested by OPO
- PEEP 5 cm, HOB up 30°
- Increase ET cuff pressure immediately after BD declaration
- Aggressive pulmonary toilet (keep suctioning & turning q2h)
- CXR (radiologist to provide measurements & interpretation)
- OPO may request bronchoscopy
- CT of chest requested in some cases
Correct Impaired Gas Exchange and Maximize Oxygenation!

- **Most organ donors are referred with:**
  - Chest trauma
  - Aspiration
  - Long hospitalization with bed rest resulting in atelectasis or pneumonia
  - Impending Neurogenic Pulmonary Edema

**Brain death contributes to and complicates all of these conditions.**
Impaired Gas Exchange

Goals...

- Goals are to maintain health of lungs for transplant while **optimizing oxygen delivery to other transplantable organs**

- Avoid over-hydration

- Ventilatory strategies aimed to protect the lung

- Avoid oxygen toxicity by limiting Fi02 to achieve a Pa02 100mmHg & PIP < 30mmHg
Electrolyte Imbalance Management

**Hypokalemia**
If $K^+ < 3.4$ – Add KCl to MIVF
(anticipate low $K^+$ with DI & $T_4$ administration)

**Hypernatremia**
If $Na^+ > 155$ – Change MIVF to include more free $H_2O$, Free $H_2O$ boluses down NG tube (this is often the result of dehydration, $Na^+$ administration, and free $H_2O$ loss $2^\circ$ to diuretics or DI)

**Calcium, Magnesium, and Phosphorus**
Deficiencies here common...often related to polyuria associated with osmotic diuresis, diuretics & DI.
Hypothermia Management

- Monitor temperature continuously
- NO tympanic, axillary or oral temperatures. Central only.
- Place patient on hypothermia blanket to maintain normal body temperature
- In severe cases (<95 degrees F) consider:
  - warming lights
  - covering patient’s head with blankets
  - hot packs in the axilla
  - warmed IV fluids
  - warm inspired gas
Anemia

- Hematocrit < 24% must be treated
- Transfuse 2 units PRBC’s immediately
- Reassess 1o after completion of 2nd unit and repeat infusion of 2 units if HCT remains below 24%
- Assess for source of blood loss and treat accordingly
Incidence of Pathophysiologic Changes Following Brain Death

<table>
<thead>
<tr>
<th>Condition</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>81%</td>
</tr>
<tr>
<td>Diabetes Insipidus</td>
<td>65%</td>
</tr>
<tr>
<td>DIC</td>
<td>28%</td>
</tr>
<tr>
<td>Cardiac arrhythmias</td>
<td>25%</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>18%</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>11%</td>
</tr>
</tbody>
</table>

Physiologic changes During Brain Stem Death
- Lessons for Management of the Organ Donor.
The Journal of Heart & Lung Transplantation Sept 2004 (suppl)
Why We Do This!

From an organ donor family member:

We are forever grateful that the doctors asked if we would consider organ donation. It was like a ray of sunshine in the void. We couldn’t ask for a better gift than to have something positive come from our tragedy. A part of Rob would now live through others.