Care of the Potential Organ Donor

MJ SOUTER MB CHB FRCA FNCS

PROFESSOR & CHIEF OF ANESTHESIOLOGY & PAIN MEDICINE,
ADJUNCT PROFESSOR OF NEUROLOGICAL SURGERY,
HARBORVIEW MEDICAL CENTER,
UNIVERSITY OF WASHINGTON, SEATTLE
AIMS

• Define the potential organ donor
• Regulatory Burdens
• Consequences of Brain Death
• Systematic Resuscitation
• Suitable Goals
• Future Options
Confusion

information
SOME DISCLOSURE

• Anesthesiologist / neurointensivist
• Medical Director for Lifecenter Northwest – Organ Procurement Organization serving Washington, Idaho, Alaska & Montana (27% of US Land mass)
• Previous Medical Advisor to AOPO (OPO national organization)
• Council Member – Organ Donation & Transplantation Alliance
• Served on several national committees…..
• … not entirely unbiased…..
WHO’S A POTENTIAL ORGAN DONOR..?

• ...Timing is everything....
  • (78% donor designation in Washington)

• For purposes of discussion
  • consider the potential donor as having suffered devastating brain injury
  • Not been declared dead by neurologic criteria - OR
  • Cannot yet be declared dead by neurologic criteria

• Insulted/dysfunctional homeostasis

• Secondary and tertiary insult
  • Organs at risk
REGULATORY CONSIDERATIONS (1)

• CMS Conditions of participation
  • § 482.45(a) Standard: Organ Procurement Responsibilities
    • A-0370 - The hospital must have written policies and procedures to address its organ procurement responsibilities.
    • A-0371 - Incorporate an agreement with an OPO designated under part 486 of this chapter, under which it must notify, in a timely manner, the OPO or a third party designated by the OPO of individuals whose death is imminent or who have died in the hospital.
REGULATORY CONSIDERATIONS (2)

- At a *minimum*, the written agreement must address the following:
  - The criteria for referral, including the referral of all individuals whose death is imminent or who have died in the hospital;
    - Includes a definition of “imminent death”;
    - Includes a definition of “timely notification”;
  - Addresses the OPO’s responsibility to determine medical suitability for organ donation;
  - The interventions the hospital will utilize to maintain potential organ donor patients so that the patient organs remain viable.
DEFINITIONS - 1

• The definition for “imminent death” might include a patient with severe, acute brain injury who:
  • Requires mechanical ventilation;
  • Is in an intensive care unit (ICU) or emergency department; AND
  • Exhibits clinical findings consistent with a Glasgow Coma Score that is less than or equal to a mutually-agreed-upon threshold; or
  • MD/DOs are evaluating a diagnosis of brain death; or
  • An MD/DO has ordered that life sustaining therapies be withdrawn, pursuant to the family’s decision.
DEFINITIONS - 2

• “Timely notification” is defined as a referral to the OPO

• As soon as it is anticipated that a patient will meet the criteria for imminent death agreed to by the OPO and hospital, or as soon as possible after a patient meets the criteria for imminent death agreed to by the OPO and the hospital (ideally, within one hour);

• AND

• Prior to the withdrawal of any life sustaining therapies (i.e., medical or pharmacological support).
WHAT DOES THAT MEAN?

• Most hospitals have nursing protocols for advising OPO’s.
  • Many OPO’s have in-hospital coordinators for major centers
• This referral allows OPO’s to examine the chart, and talk to the physicians & nursing staff.
  • It does NOT mean they can talk to the family in advance of diagnosis of brain death, or decision to remove life sustaining therapy
• It allows them time to make judgments on suitability and often refer to their medical director
  • E.g. “56 year old man with hx of Vehicular trauma – aiming for brain death testing this afternoon. PMH includes HTN, HLD, and remote tobacco use. Some EtOH. BMI is 34. P/F ratio currently 280, BP Na is 151, Creatinine is 1.4, BUN 27, on norepinephrine at 0.15, 100 mls/hr plasmalyte, with a BP of 124/58 with a heart rate of 98, and making urine at 200 mls/hr. CT shows a liver lac, left renal mass and some basal atelectasis…..” OR
  • “26 year old terminal CF with acute sepsis, creatinine 3.8, BP 90/46 on 0.3 of norepinephrine, and AST of 182, ALT of 138…..
• **A-0373**

• “It is the **responsibility of the OPO** to screen for medical suitability in order to select potential donors. Once the OPO has selected a potential donor, that person’s family must be informed of the family’s donation options.

• Ideally, the OPO and the hospital will decide together how and by whom the family will be approached.”

• Even in the case of seemingly ‘poor standard’ organs - the OPO will work with local transplant groups to determine acuity of need among possible matching recipients

• Effectively – ‘as needs must’
WHO’S RULED OUT..?

Age over 75 years
Tuberculosis
Human Immunodeficiency Virus (HIV) infection
Creutzfeldt-Jakob Disease
Herpetic Septicemia
West Nile Fever
Hepatitis B Surface Antigen and Rabies
All Retro virus Infection
Aspergillus
Malignant Neoplasms, except primary CNS Tumors and Skin Cancer

- Melanoma
- Hodgkin’s Disease, Multiple Myeloma, Leukemia
- Miscellaneous Carcinomas
- Aplastic Anemia
- Agranulocytosis
- Fungal and Viral Meningitis
- Viral Encephalitis
- Gangrene of Bowel
- Extreme Immaturity
- Positive Serological or Viral Culture Findings for Human Immunodeficiency Virus (HIV)
CONSEQUENCES OF PROCESS OF BRAIN DEATH

• Cerebellar herniation
  • Sympathetic storm – tachycardia, hypertension
• Preceded by varying degrees of transtentorial and transfalcine herniation
  • Cerebral and Hypothalamic damage
    • Often protracted over time
    • Vagal discharge alternates with sympathetic activation - excesses of rate and pressure
  • Pontine compression
    • Mixed autonomic effects
    • Respiratory effects
• Inflammatory Cascade
  • Cerebral cytokine storm

Watts et al
J Transplantation (2013), vol. 2013, Article ID 521369
MECHANISMS OF CARDIOVASCULAR INJURY

- Direct Interleukin release from brain (TNF-α, IL6 seen in SAH, TBI)
- Indirect cytokine release
  - Vasoconstriction & Hypoperfusion - anaerobic metabolism inducing NK-κB
  - Flow-induced endothelial shear stress
  - Gut ischemia
- Catecholamine Storm – myocardial injury
- Stress hormone release – inflammation
  - Myocardial ATP depletion
- Neuropeptides – Neuropeptide Y, CGRP – correlates with lung pathology
- Preceding trauma / disease ......PLUS - iatrogenesis
CARDIOVASCULAR CONSEQUENCES

- **Initial:** (minutes)
- Bradycardia and other dysrhythmias
- Increased systemic and pulmonary vascular resistances
- Hypertension
- Elevated pulmonary artery occlusion (wedge) pressure
- Increased cardiac output and left ventricular contractility

- **Secondary:**
- Decreased systemic and pulmonary vascular resistances by 15 minutes
- Decreased left ventricular contractility and blood pressure by 45 minutes
- Reduced right and left ventricular compliance
- Decreased coronary artery perfusion
- Cardiovascular collapse can occur within 5-7 hours

ACCOMPANIMENTS

- Iatrogenic insults
  - Hypernatremia (3% HTS)
  - Hypertension (pressors)
  - Volume depletion (osmotherapy/diuresis)
  - Acid-Base abnormality (HV +/- hypoperfusion)

- Secondary pathophysiological insults
  - Volume depletion (DI)
  - Thermoregulatory dysfunction
  - Endocrine dysfunction
Fig. 1. Possible mechanisms by which brain death can induce a systemic inflammatory response.
END RESULT?

- Volume depletion
- Cardiac stress
- Sympathectomy
- Poikilothermia
- Hypocarbia
- Neuroendocrine shutdown
- Peripheral inflammatory activation

= SHOCK
Cardiac Performance

- Preload
- Afterload
- Contractility
- Left ventricular size
- Myocardial fiber shortening
- Stroke volume
- Heart rate
- Peripheral resistance
- Cardiac output
- Arterial pressure
NORMAL COMPENSATORY MECHANISMS FOR SHOCK

- Goal: maintain normal cellular metabolism via adequate perfusion of body tissues
  1. increase blood pressure via sympathetic nervous system activation
  2. increase blood volume via renal/hormonal mechanisms
- All compensatory mechanisms are effective only in the short term
Figure 3-8. Frank-Starling relationship and the effect of positive and negative inotropic agents.
COMPENSATORY MECHANISMS FOR INCREASING BLOOD PRESSURE

- MAP = HR x SV x SVR (aka TPR, PVR)
  - increase in heart rate, stroke volume or systemic vascular resistance will increase mean arterial pressure
- Sympathetic response
  1. tachycardia (b1 receptor activation)
  2. increased cardiac contractility (b1 receptor)
  3. widespread vasoconstriction (a1 receptor), except cerebral and coronary vessels
Compromised Cardiac Performance

- Preload
- Left ventricular size
- Myocardial fiber shortening
- Stroke volume
- Heart rate
- Peripheral resistance
- Cardiac output
- Arterial pressure

Contractility
Afterload
COMPENSATORY MECHANISMS FOR RESTORING BLOOD VOLUME

- ↑ absorption from fluid from interstitial space in some types of shock
- conservation of salt and water by kidneys
  - reduced glomerular filtration
  - renin-angiotensin -aldosterone activity
  - ADH (vasopressin) activity
- thirst mechanism activated
GOALS OF CARE (EVEN BEFORE BRAIN DEATH....)

• Restore control with aggressive EXTERNAL homeostasis
• Systematic approach to physiological correction
  • Little to no homeoregulation or feedback
• Demands frequent monitoring and attentive care
• Restore volume status
  • What with...? & to what goal....?
• Restore vascular tone
  • What with...? & to what goal....?
TREATMENT OPTIONS

• Fluids
• Hormonal
• Immunological
## INVESTIGATIONAL IMMUNOLOGY

<table>
<thead>
<tr>
<th>Cytokine/chemokine</th>
<th>Potential therapeutic agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>TNF-α</td>
<td>IFN-β, NNZ-2566, etanercept, IFN inhibitors, Hemoadsorption</td>
</tr>
<tr>
<td>IL-1β</td>
<td>IL-1RA, NNZ-2566, Hemoadsorption</td>
</tr>
<tr>
<td>IL-6, IL-8/CXCL-8/MIP-2, IL-10, TGF-β</td>
<td>Hemoadsorption</td>
</tr>
<tr>
<td>ICAM, VCAM</td>
<td>Monoclonal antibodies, IFN-β</td>
</tr>
<tr>
<td>IFN-γ</td>
<td>IFN inhibitors</td>
</tr>
<tr>
<td>COX-2</td>
<td>COX inhibitors</td>
</tr>
</tbody>
</table>
TREATMENT OPTIONS

- Fluids
- Hormonal
- Immunological – (nil as yet....)
FLUIDS

- HES is bad – multiple studies
- Not much difference known in crystalloids
  - Higher chloride associated with RRT need
    - Zhang et al. BMC Nephrology (2013) 14:235
- Albumin – varying evidence in shock states
  - Some concern for increased permeability in transplanted lungs
  - Conflicting data on caveolin mediated transport

HORMONAL - RATIONALE

- HPA function often perturbed (but not invariably...)
  - Up to 90% of patients exhibit dysfunction of osmoregulation provoking diabetes insipidus – hypovolemia & hypernatremia

- Perimortem stress catabolism induces hyperglycemia - compounded by reduced insulin secretion - glycosuria then enhances diuresis and hypovolemia

- Depression of adrenal function as measured on cosyntropin testing in up to 76% of patients after brain death.

- Free triiodothyronine (T3) and free thyroxine (T4) also decrease significantly, with consequent increase in anaerobic metabolism and depletion of mitochondrial ATP regeneration. Loss of high energy phosphates impairs contractility
HORMONAL - TREATMENT

- Use of inotropes associated with increased number of organs/donor, but reduction in pressors also benefits OTPD

- Low dose dopamine administered to donor may improve post-transplant renal function

- Dopamine / epinephrine more arrhythmias than norepinephrine / vasopressin

- Vasopressin sensitizes vasculature to catecholamines - associated with improved function in transplanted hearts, lungs, livers and kidneys.

- Higher doses raise concern for intestinal ischemia
Methylprednisolone + Vasopressin + (T4/T3) = ‘Triple Cocktail’
This combination associated with best results.
Single/dual combinations did not have same effect.
### RESULTS OF AGGRESSIVE THERAPY

<table>
<thead>
<tr>
<th>Age</th>
<th>Non-Hormonal Resuscitation (10,292)</th>
<th>Hormonal Resuscitation (701)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;= 40</td>
<td>3.8 organ/donor</td>
<td>4.2 organ/donor*</td>
</tr>
<tr>
<td>&gt; 40</td>
<td>2.5 organ/donor</td>
<td>3.1 organ/donor*</td>
</tr>
</tbody>
</table>

Younger
Less CVA death
Less
Diabetes/Hypertension
Lower Creatinine

Rosendale Transplantation, 2003; 75: 482-487
WHICH COMPONENTS?

• Steroids
  • proposed as inhibiting pro-inflammatory effects
  • more likely function as gate-keepers of catecholamine efficacy
    • Nicolas-Robin et al. Anesthesiology. 2010 May;112(5):1204-10.

• Vasopressin
  • fluid regulation, blood pressure control - established

• Thyroid hormone
  • RCT’s provoke doubts on T3
  • Is T4 different...?
STIRRED, NOT SHAKEN...

- Methylprednisolone 15 mg/kg bolus
- Triiodothyronine (T3) 4 mcg bolus, 3 mcg/hr
- Thyroxin (T4) 20 mcg bolus, 10 mcg/hr
- Arginine vasopressin 1 unit bolus, 0.5-4 units/hr (titrate SVR 800 – 1200)
- Insulin infusion 1 unit/hr titrate blood glucose to 120 – 180 mg/dl)
CLINICAL ALGORITHM – MANAGEMENT OF ADULT PATIENTS WITH DEVASTATING BRAIN INJURIES

GOAL
To maintain hemodynamic stability in patients with devastating brain injuries

Patient evaluated in ED or ICU with devastating brain injury

Pénétrant and/or blunt trauma to the brain or devastating stroke
Evaluates by Neurosurgery and/or Neurology
Deemed to be non-survivable with no benefits from neurosurgery intervention
Still requires resuscitation by Trauma/Critical Care Services consult to be obtained during course of treatment
Patient should weigh greater than or equal to 100 lbs

Note: All patients with devastating brain injury have the potential to be organ donors. However, donation should not be discussed with the family unless directed by the attending M.D.

Initiate Steps of Management

1. ABG/Seum lactate
2. CBC w/plt, PT/PTT, Electrolytes, Hepatic Function Panel
3. Type & Crossmatch & PRBC, Transfuse to maintain HCT=30, INR < 1.4, Patients = 100, fibrinogen = 100
4. Bours 1 liter Normal saline
5. Protect from hypothermia
6. Central line (large lumen & arterial line placement)
7. Control active bleedings

Maintain MAP >70 with fluid bous
If CVP <8 add DOPamine 3-5mg/kg/min or if tachyphylaxis develops switch to noradrenaline drip @3mcg/kg/min MAP >70
Consider placement of PA catheter
If UOP >200ml/hour serum osmolarity, urine osmolarity, and urine specific gravity

All patient’s requires 4-6 hour central or mixed venous blood gases, ABGs, CPA, CBC, CIC follow up, serum lactate, and carciomx output if available seven after declaration of brain death unless care is withdrawn

Consider Social Work and Palliative Care team consultation

Hormone Replacement Protocol (to be initiated only after primary attending approval)
Goal: To maintain hemodynamic stability in patients with devastating brain injuries

Precautions:
1. Continue resuscitation to minimum CVP of 7 mmHg
2. Transfuse to achieve an HCT > 30
3. Maintain K, Ca++, Mg++, and Phosphate within normal limits

Indications:
Patient requiring a corticosteroid in the dosage greater than 15 mg/d (IV alternate every 6-12 hours or 200 mg every 12 hours) to maintain a systolic pressure of 105 after resuscitation is complete

Hormone Replacement Protocol
1. Administer 1 vial of the following in the rapid succession
   1 amp of 50% Dextrose
   2 vials of Methylprednisolone
   20 units Regular Insulin
   Insulin drip to maintain glucose between 150-180mg/dl.
   20 mcg Levothyroxline (thyroid hormone)
   (do not give unless serum T3 > 3.5 X)
2. Start a drip of 200 mg thyroxline in 500 ml NS (0.4 ng/ml)
   Administer at 25 ml (10 mg per hour)
   Reduce doses of other pressors as much as possible and then adjust thyroxline as necessary to maintain desired pressure per M.D. order
3. Monitor X-rays calcium. The only perceived consolation of the hormone replacement protocol identified to this point is an unusually high X requirement (hypercalcaemia) in some cases
4. Maintain CVP at desired level by replacing urine output if over 1200mL/hour
   * Note: thyroxline may lead to tachyphylaxis and hypocalcaemia within 30 min of initiation

Common Problems and Special Considerations

DIF: If a patient has clinical signs of DIC, transfuse immediately with 4-6 units of PPP. Delaying transfusion while waiting for lab results with uncontrolled hemorrhage is not indicated. Monitor Hb > 90 g/l with APBC

DI: If patient is normotensive, serum sodium >150 and UOP > 600ml/hr, give 2-3 mg/kg/min or 2-4 mg/kg/min
4-5 hour of UOP >200 ml/hour for UOP <200 ml/hour for UOP >100 ml/hr
6-8 hour of UOP >100 ml/hr
If patient is hypotensive, use vasopressin as described in above protocol

Common error: Assuming high UOP is due to DI but is really from ED lactic acidosis and renal failure.

Refer to Hormone Replacement protocol see next page

Yes

CVP >10 and/or PAOP (wedge) >17

Continue fluid resuscitation with 1% albumin (if serum albumin <2.0), Blood products (if indicated) and or Normal saline until MAP >70
Continue dopamine to max 20 mg/kg/min or noradrenaline to max of 30 mcg/kg/min 5 minutes until MAP >70
If require >10 mcg/kg/min Dopamine or 10 mcg/kg/min noradrenaline, add vasopressin at 24 hours

Refer to Hormone Replacement protocol see next page

Yes

Cardiac index <2.5 add Dobutamine
20 mcg/kg/min and titrate to an index of 3.5
Cardiac index <4 add phenylephrine
(20 to 200 mcg/min) or noradrenaline (1 to 20 mcg/min)
Cardiac index >2.5 add
neopropine (1 to 20 mcg/min) or noradrenaline drobe MAP >70

Start vasopressin at 2-4 hours, and replace UOP >200 ml/hour with 300 ml NS for ml every hour

Yes

Cardiac index <3 add Dobutamine
20 mcg/kg/min and titrate to an index of 3.5
Cardiac index >4 add phenylephrine
(20 to 200 mcg/min) or noradrenaline (1 to 20 mcg/min)
Cardiac index >2.5 add
neopropine (1 to 20 mcg/min) or noradrenaline drobe MAP >70

Lactate and symptoms suggestive of Diabetic ketoacidosis:
- UOP > 600ml/hr
- serum sodium >150 (units)
- urine osmolarity > serum osmolarity

If patient’s neurologic exam has deteriorated and brain death is suspected based on the clinical evaluation, please refer to the Declaration of Brain Death Guidelines and Form and contact the attending physician immediately.

ADDITIONAL VALUES

ABG = Arterial Blood gas
CBC = Complete Blood count
CVP = Central venous pressure
DI = Diabetes insipidus
DIC = Disseminated Intravascular coagulation
FFP = Fresh frozen plasma
HCT = Hematocrit
Hgb = Hemoglobin
HFP = Hepatic function panel
LVEF = Left ventricular ejection fraction
NPI = Normal saline
PA = Pulmonary artery
PAOP = Pulmonary arterial occlusion pressure
PP = Pulmonary capillary wedge pressure
PVR = Pulmonary vascular resistance
SBP = Systolic blood pressure
UO = urine output
### Table 1: UNOS Region 5 donor management goals

<table>
<thead>
<tr>
<th>Donor management goals</th>
<th>Parameters</th>
<th>% of Renal grafts meeting specific DMG at</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Time of consent (n = 722)</td>
</tr>
<tr>
<td>1. Mean arterial pressure</td>
<td>60–100 mmHg</td>
<td>78%</td>
</tr>
<tr>
<td>2. Central venous pressure</td>
<td>4–10 mmHg</td>
<td>32%</td>
</tr>
<tr>
<td>3. Ejection fraction</td>
<td>≥50%</td>
<td>16%</td>
</tr>
<tr>
<td>4. Vasopressors</td>
<td>≤1 and low dose&lt;sup&gt;1&lt;/sup&gt;</td>
<td>59%</td>
</tr>
<tr>
<td>5. Arterial blood gas pH</td>
<td>7.3–7.45</td>
<td>69%</td>
</tr>
<tr>
<td>6. PaO&lt;sub&gt;2&lt;/sub&gt;:FiO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>≥300</td>
<td>39%</td>
</tr>
<tr>
<td>7. Serum sodium</td>
<td>135–155 mEq/L</td>
<td>69%</td>
</tr>
<tr>
<td>8. Blood glucose</td>
<td>≤150 mg/dL</td>
<td>53%</td>
</tr>
<tr>
<td>9. Urine output</td>
<td>0.5–3 cc/kg/h over 4 h</td>
<td>64%</td>
</tr>
</tbody>
</table>

<sup>1</sup>Low dose of vasopressors was defined as dopamine ≤ 10 mcg/kg/min, neosynephrine ≤ 60 mcg/kg/min and norepinephrine ≤ 10 mcg/kg/min.
REGION 5
DONOR MANAGEMENT GOALS

<table>
<thead>
<tr>
<th>Critical Care Endpoint</th>
<th>DMG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mean Arterial Pressure (MAP)</td>
<td>60 – 110 mmHg</td>
</tr>
<tr>
<td>2. Central Venous Pressure (CVP)</td>
<td>4 – 12 mmHg</td>
</tr>
<tr>
<td>3. Ejection Fraction (EF)</td>
<td>≥ 50%</td>
</tr>
<tr>
<td>4. Vasopressor use</td>
<td>≤ 1 and low dose</td>
</tr>
<tr>
<td>5. Arterial Blood Gas pH</td>
<td>7.3 – 7.5</td>
</tr>
<tr>
<td>6. PaO2:FiO2 (P:F)</td>
<td>≥ 300 on PEEP = 5</td>
</tr>
<tr>
<td>7. Serum Na</td>
<td>≤155 mEq/L</td>
</tr>
<tr>
<td>8. Blood Glucose</td>
<td>≤ 150 mg/dL</td>
</tr>
<tr>
<td>9. Urine Output (averaged over 4 hours)</td>
<td>≥0.5 cc/kg/hr</td>
</tr>
</tbody>
</table>
REGION 5
DONOR MANAGEMENT GOALS

- Donor Management Goals met at consent improve outcomes
  - 90% increase in the chance of achieving 4 organs transplanted per donor
  - 50% decrease in the chance of developing recipient renal delayed graft function
  - 90% greater chance of achieving 3 organs per donor in extended criteria patients

Malinoski et al. J Trauma. 2011;71: 990–996
Patel et al. JAMA Surg. 2014;149(9):969-975
Fig. 1 – OTPD by groups of DMPs met at each time point.

PERSONAL BIAS

- CVP sucks....!
  - As a resuscitation goal...
A + C = Volume e.g. 500ml
B = Patient is preload responsive
D = Patient not preload responsive
Figure 1. Receiver operating characteristic analysis for the stroke volume variation (SVV), central venous pressure (CVP), heart rate (HR), and systolic blood pressure (SBP) as predictors of increase in stroke volume by more than 5% after volume loading step. The area under the curve for SVV (0.870, 95% CI: 0.809 to 0.903) was statistically more than those for CVP (0.493, 95% CI: 0.397 to 0.590, P = 7 × 10^{-10}), HR (0.593, 95% CI: 0.443 to 0.635, P = 5.7 × 10^{-10}), and SBP (0.729, 95% CI: 0.645 to 0.813, P = 4.3 × 10^{-5}). CI = confidence interval.
Figure 2. ROC curves comparing the ability of ΔPp, ΔPs, Pra, and Ppao to discriminate responder (CI increase ≥ 15%) and nonresponder patients to VE. The area under the ROC curve for ΔPp was greater than for ΔPs, Pra, and Ppao (p < 0.01).
EARLY GOAL DIRECTED DONOR MANAGEMENT ALGORITHM

1. PPV > 15% CI < 2.7
   - MAP < 60
   - 60 < MAP < 70
   - MAP ≥ 70

2. PPV > 15% CI ≥ 2.7
   - MAP < 60
   - 60 < MAP < 70
   - MAP ≥ 70

3. PPV < 15% CI < 2.7
   - MAP < 60
   - 60 < MAP < 70
   - MAP ≥ 70

4. PPV < 15% CI ≥ 2.7
   - MAP < 60
   - 60 < MAP < 70
   - MAP ≥ 70

5. MAP < 60
6. MAP ≥ 70
7. 250 - 500 mL Fluid Bolus
8. Observe (consider reducing vasopressors)
9. Reduce Vasopressors
10. Norepinephrine or Neosynephrine
11. Observe
12. Reduce Vasopressors
13. Consider Inotrope
14. Consider Anti-Hypertensive
15. Consider Anti-Hypertensive
16. Consider Anti-Hypertensive
17. Consider Anti-Hypertensive

TREATMENT PLAN

• Correct hypovolemia
• Reappraise...
• Correct for pressor requirement
• Reappraise...
• Titrate both to need
• Check lung function
• Reappraise & balance

• “External Homeostasis”
EVEN WITH THAT...

30% Dysfunctional Hearts

38% Dysfunctional Hearts

Fig. 1 Change in Ejection Fraction from TTE 1 to TTE 2 in procured (a) and non-procured (b) subsets in donors with cardiac dysfunction (CD) receiving serial echocardiograms. The box represents the median and interquartile range, and the whiskers represent the range.

Figure 1. Ejection fraction (EF; %) versus time (hr) in the donor subgroup with serial transthoracic echocardiograms (TTEs) (n = 11).
OPTIMAL RESUSCITATION?

• Retrospective analysis of OPTN database between 1994-2006
  • Effect of brain death duration on DGF, acute rejection and graft failure.
  • Longer duration decreased the risk of DGF at 1 and three years.
  • In donors younger than 55, each hour of brain death duration associated with decreased risk of DGF.
• Multicenter RCT of algorithmic approach to fluid and inotropic resuscitation
  • No effect on number of organs procured
  • Correcting to PPV goal had no adverse effect on lungs
FUTURE

• Resuscitate Donor Versus Get The Organ in a Box...?
• Kidneys versus lungs – outcomes
  • Types of fluid
• Neuroendocrine replacement
  • Feeding, glycemic control, vasopressin
  • ‘Dissecting the bundle’?
• Cardiac Markers
• Inflammatory Response/modulation
• MULTICENTER STUDIES...
KIDNEYS IN A BOX
LUNG IN A BOX
OCS HEART

NEAR-PHYSIOLOGIC PRESERVATION

- Control perfusion pressure (AOP)
- Optimize coronary flow
- Normothermic
- Maintain critical substrates, electrolytes and hormones
FUTURE

• Resuscitate Donor Versus Organ in a Box..?
• Kidneys versus lungs – outcomes
  • Types of fluid
• Neuroendocrine replacement
  • Feeding, glycemic control, vasopressin
  • ‘Dissecting the bundle’?
• Cardiac Markers
• Inflammatory Response/modulation
• MULTICENTER STUDIES...
“It keeps me from looking at my phone every two seconds.”
QUESTIONS?

• “I know that you believe you understand what you think I said, but I'm not sure you realize that what you heard is not what I meant.”
  
  • Robert McCloskey, White House Spokesman 1967

• “If I seem unduly clear to you, you must have misunderstood what I said.”
  