

Guideline for the Management of the Potential Organ Donor

For the most up to date version of this guideline follow this link:
<http://www.gicu.sgul.ac.uk/resources-for-current-staff>

DISCLAIMER

The document is an evolutionary document and reflects the opinions of the authors only.

Organ donor management in ICU / prior to retrieval

Introduction

For any potential organ donor, prolonged (>20 hours) of active management dramatically increases the chances of successful donation, maximises the number of organs they can donate and optimises both graft function and survival [1-5].

IF, following a catastrophic brain injury, the possibility of brainstem death (BSD) is clinically suspected* ☐

* **based upon clinical judgement and the presence of:** Fixed and dilated pupils ☐ GCS 3/15 ☐ No triggering / patient interaction with the ventilator ☐

AND the decision has been made to stop neuroprotection ☐

THEN, BSD should be confirmed (see Appendix) as soon as practical using the Academy of Medical Royal Colleges, 2008 criteria [6] and initial contact should be made with local Transplant Co-ordinator.

In order for the diagnosis of BSD to be made (see Appendix), appropriate cardiovascular, respiratory, temperature and serum electrolyte criteria must be met, together with a clear diagnosis of the mechanism of brain injury and the timely cessation of all sedative and neuromuscular blocking drugs. This guideline should be used as an aid to physiological management prior to the diagnosis of BSD as we feel this represents optimal care regardless of whether or not the patient goes on to donate. Note, it is recommended that apnoea testing be conducted using CPAP [7]. It is widely considered that it is both morally and ethically sound, to manage the patient based upon the assumption that they are a potential organ donor prior to BSD testing and the consent process. If consent or other issues subsequently preclude organ donation then withdrawal of organ support will be undertaken, however, this is often delayed to facilitate the wishes of the family and friends of the deceased.

The donor should be actively managed based on the assumption that they may be able to donate their heart, lungs, kidneys, liver and possibly additional organs / tissues. All of the four major organs require assessment for suitability, whilst simultaneously, optimal perfusion is maintained together with minimal iatrogenic injury. This latter aspect extends to the pre-emptive minimisation of the injury induced by explanted, cold ischaemia.

The physiological changes following brain stem death often result in cardiovascular instability and a multiple organ dysfunction syndrome. This can be prevented and treated thus maximising the number of organs that can be retrieved from an individual, each of which should go on to dramatically improve the life of a recipient. It has been said that following brain stem death, you are no longer treating the donor but instead are treating multiple recipients. In addition, it is reasonable to assume that if organ donation was in accordance with the deceased's wishes then optimising organ retrieval is also in accordance with their wishes.

These guidelines are based on expert opinion as there is limited evidence in this area. We hope they will be viewed as helpful and are not intended to be dogmatic. Local policies may be agreed for specific therapies, and outcomes audited. It is our intention that these guidelines will be regularly reviewed and updated based upon local and national audit data and emerging evidence from clinical trials.

The following general references / links may also be helpful:

Bugge, J. F. (2009). **Brain death and its implications for management of the potential organ donor**. Acta Anaesthesiol Scand 53(10): 1239-1250.

<http://www3.interscience.wiley.com/cgi-bin/fulltext/122545709/PDFSTART>

Mascia, L., I. Mastromauro, et al. (2009). **Management to optimize organ procurement in brain dead donors**. Minerva Anesthesiol 75(3): 125-33.

<http://www.minervamedica.it/en/journals/minerva-anestesiologica/article.php?cod=R02Y2009N03A0125>

Wood, K. E. and J. McCartney (2007). **Management of the potential organ donor**. Transplantation Reviews 21(4): 204-218.

<http://www.sciencedirect.com/science/article/B75B4-4PV1MCN-9/2/1edbb52213982172b7d4ee999909df4d>

Van Raemdonck, D., A. Neyrinck, et al. (2009). **Lung Donor Selection and Management**. Proc Am Thorac Soc 6(1): 28-38.

<http://pats.atsjournals.org/cgi/content/abstract/6/1/28>

Australasian Transplant Coordinators Association Incorporated (2008). **National guidelines for organ and tissue donation, 4th edition**

<http://www.atca.org.au/downloads/ATCA%20National%20Guidelines%20for%20Organ%20&%20Tissue%20Donation.pdf>

Summary / checklist – targets, **tests**, **drugs** and **procedures**

Airway - endotracheal / tracheostomy tube – page 4

Distal tip of the tube is located in the mid / lower trachea ☐ **Recent chest x-ray** ☐ Reported by senior clinician ☐

No cuff leak and cuff pressure is ~32cmH₂O ☐ **Urgent sputum Gram stain & microscopy** ☐ **Continue 6hrly oral chlorhexadine** ☐

Breathing & Blood tests – page 5 & 6

Set (and measured) positive end expiratory pressure (PEEP) 8-10cmH₂O ☐ Perform recruitment manoeuvre (repeat as indicated) ☐

Arterial oxygen tension (PaO₂) 8-14kPa ☐ **and / or** SpO₂ 92-95% ☐ on minimum FiO₂ ☐

Tidal volumes (Vt) 6-8ml/kg (ideal body weight) ☐ Peak (plateau / end inspiratory) pressure ≤30cmH₂O ☐

Arterial carbon dioxide tension (PaCO₂) 5.0-6.5kPa ☐ Respiratory rate and I:E ratio set to achieve target PaCO₂ **and** prevent dynamic hyperinflation ☐

Send blood for: **full blood count** ☐ **clotting screen** ☐ **group and save for transfusion** ☐

renal / liver / bone / cardiac biochemistry ☐ **virology** ☐ **tissue typing** ☐

Circulation and blood Composition– page 7,8 & 9

Rate & rhythm: Sinus rhythm 60-100 bpm ☐ **12 lead ECG** ☐ Reviewed & reported by senior clinician ☐

Preload: stroke volume index (SVI) 33-47 ml/m²/beat ☐ **and / or** stroke volume variation (SVV) <15% ☐ **and / or** CVP 8-12mmHg ☐

Contractility: Cardiac index (CI) ≥2.4 l/min/m² ☐ **Echocardiogram (if possible)** ☐

Afterload: Mean arterial pressure (MAP) 60-80mmHg ☐ **and / or** systemic vascular resistance index (SVRI) 1800-2400 dynes•sec/cm⁵/m² ☐

Global oxygen supply demand balance: Central venous oxygen saturation (ScvO₂) ≥60% ☐ **and / or** mixed venous oxygen saturation (SvO₂) ≥70% ☐ **and / or**

Central venous-to-arterial carbon dioxide difference (Pcv-aCO₂) ≤0.8kPa ☐

Blood composition: [Hb] ≥80g/l ☐ Platelets >50x10⁹/l ☐ INR <2.0 ☐ APTT ratio <1.5 ☐ fibrinogen >2.0g/l ☐

Vascular access: **reliable large bore intravenous access** ☐ **arterial line** ☐ **central venous line** ☐

Dextrose & **drugs** – page 10

Blood glucose 4.0-10.0mmol/l ☐ **Insulin infusion ≥1iu/hr** ☐ **20% dextrose infusion at 25ml/hr (or alternative)** ☐

Give 15mg/kg methylprednisolone IV ☐ **repeat every 24 hours** ☐ – page 6

Electrolytes & environment – page 10

Na⁺ 135-150mmol/l ☐ K⁺ 4.0-5.5mmol/l ☐ Mg²⁺ >0.8mmol/l ☐

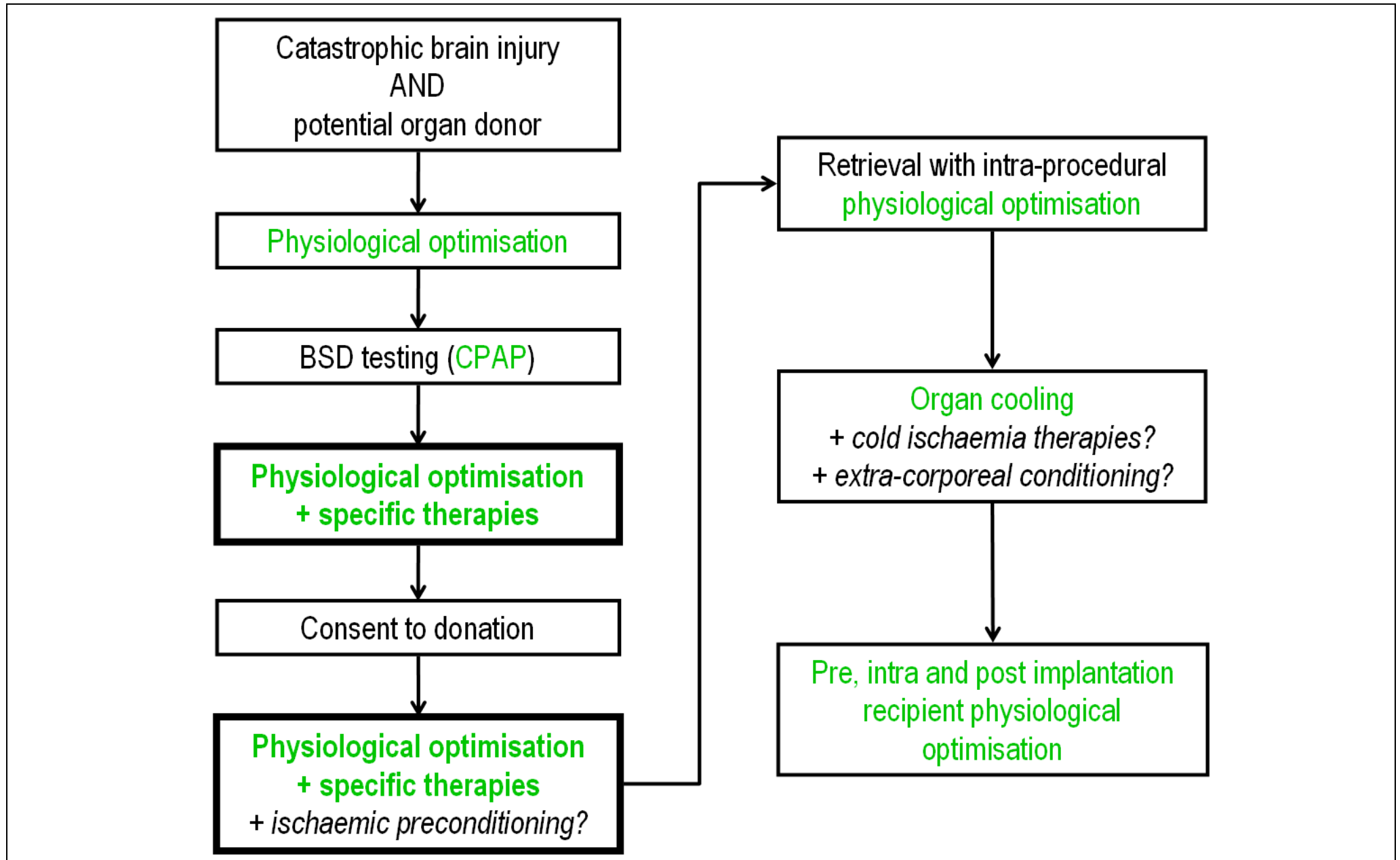
Ca²⁺ 1.0-1.3mmol/l (ionised) ☐ **or** 2.0-2.6mmol/l (total) corrected for albumin ☐ PO₄³⁻ >0.8mmol/l ☐

Core temperature 35.5-37.5°C ☐ Maintain 30-45° head of bed elevation ☐ VTE prophylaxis: **LMWH** ☐ TED stockings ☐ +/- calf compressors ☐

Fluid balance (renal) - page 9

No maintenance fluids ☐ urine output 0.5-2.5ml/kg/hr ☐ (**if less** ⇒ ensure circulation optimised and catheter patent; **if more** ensure adequate replacement / euvolaemia / normonatraemia AND test for +/- treat diabetes insipidus (DI))

Summary - Time line flow diagram



Detail: Initial monitoring, physiological targets, investigations and therapies

	Parameter	Method / rationale and notes
Airway	Check endotracheal / tracheostomy tube is patent	If available, check for normal inspiratory and expiratory flow profiles on ventilator
	The distal tip of the tube is located in the lower trachea	Recent CXR. If not available then request a new CXR. Have the film reviewed by a senior clinician for any new lung infiltrates and any other abnormalities / diagnoses.
	No cuff leak and cuff pressure is $\leq 32\text{cmH}_2\text{O}$	Cuff pressure manometer. Cuff leaks result in lung de-recruitment and increase the risk of passive aspiration. High pressures cause local mucosal injury.
	Check for proximal endotracheal secretions	Collect any secretions present and send for urgent microscopy with gram stain. Request culture and sensitivities. Consider antibiotics. If secretions are viscous / difficult to suction consider nebulising hypertonic saline (5%-7%) 5-10mls 2-6hourly [8] and / or bronchoscopy.
Breathing	Perform a recruitment manoeuvre on the ventilator and set optimal level of PEEP to retain the recruited lung (usually 8-10 cmH_2O [7]). Example: Set FiO_2 to achieve SpO_2 of 88-92%. Set PEEP to 10 cmH_2O then perform an inspiratory hold at 35 cmH_2O for 30-60 seconds. Set and maintain Vt target as below. After 15 minutes of steady state SpO_2 , reduce PEEP to 8 cmH_2O . If on reduction of PEEP the SpO_2 falls by >4%, repeat from the beginning but stop at the PEEP level above which the drop in SpO_2 was observed.	This is always needed following apnoea testing. If unfamiliar with recruitment manoeuvres, ask for help and / or refer to [5, 9]. Watch for cardiovascular decompensation. If this occurs consider whether the patient may be hypovolaemic (see circulation section below). Transient desaturation is often seen with recruitment manoeuvres and is of no concern. If recruitment has been successful then there should be an increase in the SpO_2 for the same FiO_2 . In addition, a positive response is indicated by an improvement in dynamic compliance (Cdyn) at the same level of PEEP $\text{Cdyn} = \text{tidal volume} / (\text{end inspiratory pressure} - \text{PEEP})$. In the presence of significant acute lung pathology consider other optimal recruitment / ventilation strategies such as APRV [10].
	Tidal volume (Vt) 6-8ml/kg (ideal body weight (IBW)) [5, 7] Peak / plateau pressures $\leq 30\text{cmH}_2\text{O}$ [5, 7] Respiratory rate and I:E ratio set to achieve target PaCO_2 and prevent dynamic hyperinflation	IBW (kg) for men = $[(\text{height (cm)} - 154) \times 0.9] + 50$ IBW (kg) for women = $[(\text{height (cm)} - 154) \times 0.9] + 45.5$ Online calculator http://www.ukmicentral.nhs.uk/resource/calcs/ibw.asp?group=m Minimise ventilator induced lung injury.
	PaO_2 8-14kPa and / or SpO_2 92-95% on minimum FiO_2	Minimise absorption atelectasis and oxygen toxicity [11]
	PaCO_2 5.0-6.5kPa (or higher as long as $\text{pH} > 7.30$)	Minimise minute ventilation and thereby minimise ventilator induced lung injury. (Mild permissive hypercapnia may be advantageous.)

Detail: Initial monitoring, physiological targets, investigations and therapies - continued

	Parameter	Method / rationale and notes
Ongoing care of lungs	At least 2 hourly clinical assessment of lung recruitability and retention of recruited units. Look for deteriorating saturations / increasing oxygen requirements and deteriorating Cdyn	Set optimal PEEP. Consider recruitment manoeuvre followed by decremental trial to find best PEEP setting. Consider sigh breaths or periodic prolonged inspiratory holds. Only suction if clinically indicated by signs of secretions within the proximal bronchial tree and always consider the need for re-recruitment afterwards. Clinical signs of secretions within the proximal tracheobronchial tree include classic "upper airway secretion" crepitations on auscultation, an irregular or diminished flow profile (expiratory more common than inspiratory) and progressive deterioration in compliance and / or gas exchange. In the latter case, suction prior to the recruitment manoeuvre. Do not disconnect from the ventilator unless absolutely necessary. If unavoidable, perform a recruitment manoeuvre immediately after reconnection.
	Reduce / minimise extravascular lung water	Minimum IV fluids to achieve adequate preload (see below) GIVE 15mg/kg IV methylprednisolone. This has been shown to reduce the accumulation of extravascular lung water and may play an important role as an anti-inflammatory / immunosuppressive agent thereby preventing / minimising lung (and other organ) injury, although a recent trial failed to demonstrate any benefit [12]. If organ donor management continues for >24 hours, consider a second dose of 15mg/kg. If despite PEEP optimisation and recruitment manoeuvres there is a persistent alveolar-arterial oxygen gradient AND there is clinical / radiological evidence of interstitial lung oedema then consider : <ul style="list-style-type: none"> • APRV [10] • Trial of furosemide infusion at 0.5-10mg/hr to achieve 100-200ml/hr negative fluid balance BUT AVOID hypovolaemia

Detail: Initial monitoring, physiological targets, investigations and therapies - continued

	Parameter	Method / rationale and notes
Circulation Monitoring	If not already in place institute continuous 3 lead ECG, invasive arterial pressure and cardiac output monitoring. Please record hourly observations and all therapies on usual ICU chart.	Following brain stem death patients may develop dysrhythmias, hypovolaemia (e.g. diabetes insipidus), cardiac dysfunction (e.g. reversible global myocardial depression), hypertension (e.g. catecholamine storm) and hypotension (e.g. a mixture of the above +/- vasoplegia). Continuous and complete monitoring of the cardiovascular system is essential. Use whichever method of cardiac output monitoring you are most familiar with.
Lines	Ensure that there is reliable large bore intravenous access. If not already in-situ, insert a central venous line. Do not routinely replace lines (peripheral / arterial / central venous) unless not working.	Due to the order of vessel ligation during cardiac retrieval, if possible, when inserting a new "X" use the following sites: <ul style="list-style-type: none"> • peripheral venous access (16 or 14G) in the RIGHT arm. • arterial line use the LEFT radial or brachial arteries. • central venous line use the RIGHT internal jugular or subclavian NOT femoral.
Cardiac assessment	Perform a 12 lead ECG	To fully assess cardiac rhythm and diagnose any abnormality. If available, compare to a recent ECG performed prior to brain stem testing. Look for conduction abnormalities, signs of hypertrophy and acute or previous ischaemia / infarction. Acute strain patterns may represent reversible pathology.
	If available, request a echocardiogram	This is to exclude any structural heart disease including, valves, hypertrophic / thinned myocardium, regional wall abnormalities etc. Global myocardial depression, if seen, MAY be entirely reversible and should not preclude attempts to optimise function and reassess for donation.

Detail: Initial monitoring, physiological targets, investigations and therapies - continued

	Parameter	Method / rationale and notes
Circulation Ideal targets	Rate & rhythm: <ul style="list-style-type: none"> Sinus rhythm 60-100 bpm 	<p>In the event of haemodynamically significant bradycardia consider short acting positive chronotrope e.g. isoprenaline, dobutamine, etc.</p> <p>For sinus tachycardia consider the differential diagnosis and treat the cause. If associated with hypertension consider short acting β-blockade with esmolol. If associated with hypotension consider hypovolaemia, myocardial depression and vasoplegia.</p>
	Preload: <ul style="list-style-type: none"> stroke volume index (SVI) 33-47 ml/m²/beat stroke volume variation (SVV) <15% CVP <12mmHg <p>Note – CVP may be a poor measure of preload and an unreliable predictor of volume responsiveness. For a detailed review on preload assessment see [13]</p>	<p>If the patient has a MAP of 60-80mmHg with no vasoactive drug support then monitor preload parameters for trend information.</p> <p>If the patient has a MAP of <60mmHg or is requiring vasopressors to achieve a MAP of 60-80mmHg AND 1 (or more) preload parameters indicates hypovolaemia then administer a 3-5ml/kg fluid bolus (balanced crystalloid) as rapidly as possible. Record the pre and post fluid bolus stroke volume / cardiac output. An increase of $\geq 15\%$ indicates a positive response to the fluid challenge. If positive, consider whether to repeat the challenge and when to review this decision. If <15% then consider +/- treat for myocardial depression and / or vasoplegia.</p>
	Contractility: <ul style="list-style-type: none"> Cardiac index (CI) ≥ 2.4 l/min/m² <p>Note: Myocardial stunning is a common observation following BSD BUT may reverse with optimal supportive therapy, albeit over a 24-48 hour period [14, 15].</p>	<p>If the CI is low AND there is no response to a fluid bolus (see above) then:</p> <ol style="list-style-type: none"> Commence dobutamine at 2.5μg/kg/min Increase dobutamine to 5μg/kg/min Commence T₃ (tri-iodothyronine) 4μg bolus then 3μg/hr infusion or T₄ (thyroxine) 20μg bolus then 10μg/hr infusion Increase dobutamine to 10μg/kg/min Seek expert advice <p>Notes</p> <ul style="list-style-type: none"> Low dose dobutamine (2.5μg/kg/min) may be beneficial in ameliorating ex-vivo, cold ischaemic injury [16] but high doses may deplete high energy phosphates. Some cardiothoracic transplant units encourage the use of thyroid replacement therapy for all donors. Trial evidence [17] suggests it may add little to active donor management and is not necessary routinely. Audit of use and outcomes is in progress, and will inform future guidelines. It may be valuable as a second line positive inotrope in patients with myocardial depression [18].

Detail: Initial monitoring, physiological targets, investigations and therapies - continued

	Parameter	Method / rationale and notes
Circulation Ideal targets	<p><i>Afterload / perfusion pressure:</i></p> <ul style="list-style-type: none"> Mean arterial pressure (MAP) 60-80mmHg SVRI 1800-2400 dynes•sec/cm⁵/m² <p>* This can be simplified to 2mg made up to 48mls. Give 2 ml bolus then infuse @ 2mls per hour. Doses up to 5.2µg/kg/hr can be considered (equivalent to 2mg 6 hourly - used in variceal haemorrhage and hepato-renal failure).</p> <p>** If the patient appears to exhibit noradrenaline "resistance", defined as a dose >0.2 µg/kg/hr then a trial dose of hydrocortisone 50-100mg (in addition to the methylprednisolone already administered) may reverse a state of functional hypoadrenalism. The rationale for this approach is based on the mineralocorticoid effects of hydrocortisone [19] (methylprednisolone has no mineralocorticoid action). If successful in facilitating a ≥15% dose reduction in noradrenaline consider either a hydrocortisone infusion at 10mg/kr or repeated 50mg boluses at 4-6 hourly intervals.</p>	<p>If MAP <60mmHg and preload AND myocardial contractility have been optimised / treated then</p> <ol style="list-style-type: none"> Commence vasopressin 1iu bolus (repeated if necessary) then 0.5-4iu/hr or terlipressin 1.3µg/kg bolus then 1.3µg/kg/hr* [20, 21] Commence noradrenaline (norepinephrine) 0.05-0.20µg/kg/min Give hydrocortisone 50mg bolus** Increase noradrenaline up to 1.0µg/kg/min Review preload and myocardial contractility Seek expert advice <p>Notes</p> <ul style="list-style-type: none"> Vasopressin / terlipressin need not be used routinely if goals are achieved and maintained easily. They are however the first line vasopressors or should be initiated promptly following the diagnosis of diabetes insipidus (DI). <p>If MAP >80mmHg then consider:</p> <ul style="list-style-type: none"> Esmolol (loading dose then infusion) up to 200µg/kg/min AND / OR Glycerine trinitrate up to 40mg/hr [22]. <p>The use of very short acting drugs is recommended as there is a significant risk of brain stem dead patients rapidly developing a rebound hypotensive state.</p>
	<p><i>Global oxygen supply demand balance:</i></p> <ul style="list-style-type: none"> Central venous oxygen saturation (ScvO₂) ≥70% Central venous-to-arterial carbon dioxide difference (Pcv-aCO₂) ≤0.8kPa [23] <p>Arterial / central venous lactate measurements may be elevated despite optimal oxygen delivery as high levels of catecholamines, endogenous or exogenous (especially β₂ agonists) increase production beyond the elimination threshold. In addition, the injured brain can respond by producing lactate.</p>	<p>Having optimised / treated heart rate / rhythm, preload, contractility and afterload, a measure of global oxygen delivery - consumption balance can be made by monitoring the ScvO₂. If this is low, it suggests that either delivery is suboptimal and / or consumption is high. Re-consider the 4 cardiovascular variables above AND consider haemoglobin concentration. If [Hb] is ≤8.0g/dL then consider packed RBC transfusion. It may be of value to increase [Hb] above this threshold to allow for blood loss during organ retrieval. Please discuss this with the retrieval teams as there maybe negative consequences of blood transfusion on graft function.</p> <p>A useful additional marker is Pcv-aCO₂. For move information regarding this and other pitfalls of ScvO₂ see [23, 24].</p>

Detail: Initial monitoring, physiological targets, investigations and therapies - continued

	Parameter	Method / rationale and notes
Renal	Urine output (UO) 0.5-2.5 ml/kg/hr	<p>Cardiovascular optimisation should achieve this.</p> <p>Watch for polyuria / diabetes insipidus (DI). Suspect DI if UO suddenly increases or exceeds 2.5ml/kg/hr for >2hours. If present, first ensure adequate intravascular volume replacement (with 5% dextrose). Confirmatory tests include a rapidly rising blood Na⁺, a urine specific gravity of <1.005, a urine osmolality <200mOsm/kg in the presence of plasma osmolality >295mOsm/kg. Definitive treatment options included vasopressin / terlipressin [25] infusion (see page 8) and / or DDAVP therapy (0.5-1.0mcg bolus repeated as necessary). Monitor and normalise blood Na⁺.</p> <p>Low dose dobutamine 2.5 µg/kg/min [16] OR dopamine 4 µg/kg/min [26] may be useful in mitigating against cold ischaemic injury.</p>
Liver	Normal bilirubin / ALT / ALP / γGT / pT (INR) / fibrinogen / lactate / glucose	<p>Cardiovascular optimisation should achieve this.</p> <p>If available consider measuring indocyanine green (ICG) clearance [27]</p>
LUNG assessment	<p>FIRST Optimise cardiovascular system and ventilator settings</p> <p>Diffusion tests:</p> <p>PaO₂ after 15 minutes of 100% O₂</p> <p>OR PaO₂/FiO₂ ratio</p> <p>OR oxygenation index [28]</p> <p>OR Alveolar-arterial oxygen gradient</p> <p>Compliance / ventilatory efficiency tests:</p> <p>Calculate C_{dyn}</p> <p>Calculate ventilatory ratio [29]</p> <p>Estimate ventilatory dead-space fraction</p>	<p>To give accurate assessment of best physiological performance</p> <p>Suitable lungs should achieve a PaO₂ >40kPa (300mmHg)</p> <p>Suitable lungs should achieve a ratio >40kPa (300mmHg)</p> <p>= mean airway pressure x FiO₂ x 100 / PaO₂. Expected value <50cmH₂O/kPa</p> <p>= [(FiO₂) x (Atmospheric Pressure - H₂O Pressure) - (PaCO₂/0.8)] - PaO₂. Expected value = (Age/4) + 4.</p> <p>Online calculator @ http://www.mdcalc.com/a-a-o2-gradient</p> <p>= tidal volume / (end inspiratory pressure – PEEP). Expected value @ PEEP 5cmH₂O & Vt 8ml/kg IBW is ≥28ml/cmH₂O</p> <p>= minute volume (ml/min) x PaCO₂ (kPa) / 100 x IBW (kg) x 5 value <1.00</p> <p>See [30]</p>

Detail: Initial monitoring, physiological targets, investigations and therapies - continued

	Parameter	Method / rationale and notes
Temperature	Maintain temperature at 35.5-37.5°C	Hypothermia secondary to brain stem death is more common than hyperthermia. Actively warm / cool as appropriate.
Fluid balance	Neutral to negative (unless grossly hypovolaemic)	Optimal intravascular volume for perfusion vs. minimisation of extravascular lung water. Avoid maintenance fluids. Consider balanced crystalloid replacement of last hour's urine output.
Glycaemic control	Blood sugar 4.0-10.0mmol/l Measure at least 2 hourly In addition, insulin may play a role in cellular protection and therefore is recommended (even in those donors with normoglycaemia) at an infusion rate of 1iu/hr.	To avoid hypoglycaemia give a continuous infusion of 20% or 50% dextrose (25 or 10mls/hr respectively). Consider instigating (or continuing) low dose / low volume enteral feeding. This may have multiple potential benefits including sodium and water balance, splanchnic perfusion, glycaemic control etc.
Electrolytes	Na ⁺ 135-150mmol/l K ⁺ 4.0-5.5mmol/l Mg ²⁺ >0.8mmol/l Ca ²⁺ 1.0-1.3mmol/l (ionised) or 2.0-2.6mmol/l (total) corrected for albumin PO ₄ ³⁻ >0.8mmol/l	Actively manage as necessary. Of note Na ⁺ >150 mmol/l is associated with significant hepatic graft dysfunction. Insulin, hypothermia & polyuria can all cause hypokalaemia If you are needing to give K ⁺ replacement give MgSO ₄ 20mmol over 4 hours
Haematology	[Hb] ≥80g/l Platelets >50x10 ⁹ /l INR <2.0 / APTT ratio <1.5 / fibrinogen >2.0g/l	To mitigate against potential blood loss during organ retrieval
Patient position	Maintain 30-45° head of bed elevation. Consider side-back-side rotation.	To reduce passive aspiration and increase FRC.
DVT prophylaxis	In not already in place fit TED stockings +/- calf compressors Continue prophylactic dose heparin / LMWH	
Review medications	Stop all unnecessary drugs	Consider starting / continuing chlorhexidine 2% mouthwash / gel (to minimise bacterial oropharyngeal colonisation and passive aspiration) and simple eye ointment (especially if corneal donation is being considered)

Transfer to operating theatre / intra-operative management

Introduction

- In most centres the organ donor management described above takes place in Intensive Care.
- As the anaesthetist managing the donor during multi-organ retrieval, you will be providing ongoing critical care support / physiological stability with the aim of facilitating the unhurried removal of organs in optimum condition. Your role is vital. Please don't hesitate to request senior anaesthetic / critical care support if needed.
- The monitoring, physiological targets and suggested interventions are identical to those described above.
- **Essentially this is a laparotomy extended by median sternotomy (even if thoracic organs are not to be retrieved) which may take up to 3 hours of 'anaesthesia' time.**
- Please ensure that a record is kept of the donor's physiology during the procedure, either on the ICU chart or a separate anaesthetic chart. This may help you identify trends during the procedure and will be needed by the transplant coordinator. A short note in the patient notes is also usual.
- The surgeons involved are excellent, and blood loss is usually minimal. You should however **be prepared to manage sudden and significant blood loss** if it occurs, including transfusion if indicated.

Preparation

- **Please refer to the checklist on page 3 to assess completed and outstanding issues.** In unfamiliar with these guidelines, please take a moment to review them as there are subtle but important differences between routine critical care and the management of brain dead organ donors.
- Before going to theatre it will usually be decided which organs are to be retrieved. A specialist nurse for organ donation (SNOD) will be present, who will tell you which organs are likely to be retrieved and any specific requirements.
- The donor should be maintained on the lowest possible inspired oxygen but transfer to the operating theatre will generally be done with raised inspired oxygen to allow for disconnections and transfers.
- As for any unstable or potentially unstable critically ill patient, **before leaving ICU** check that you have reliable access and monitoring, and that the donor is cardiovascularly stable with the rapid availability of fluids / blood / preferred vasoactive drugs.

In theatre

"Surgical pause"	An appropriate 'surgical pause' should be undertaken to allow team introductions, plan of procedure, identification of donor and review of brainstem death documentation and allergy history.
Position and Access	The donor is supine, with arms by the side to facilitate surgical access. Ensure your large bore access is patent and reliable. A large bore extension set and three-way tap is often useful if arm veins are used. Check for availability of intravenous fluid warming equipment if haemorrhage should occur, although this is uncommon.
Ventilation	Perform a recruitment manoeuvre on the ventilator and set optimal level of PEEP (usually 8-10cmH ₂ O) to retain the recruited lung. Tidal volume (Vt) 6-8ml/kg (ideal body weight (IBW)) Peak / plateau pressures ≤30cmH ₂ O Respiratory rate and I:E ratio set to achieve target PaCO ₂ and prevent dynamic hyperinflation PaO ₂ 8-14kPa and / or SpO ₂ 92-95% on minimum FiO ₂ PaCO ₂ 5.0-6.5kPa (or higher as long as pH>7.30)
Haemodynamics	Sinus rhythm 60-100 bpm Stroke volume index (SVI) 33-47 ml/m ² /beat and / or stroke volume variation (SVV) <15% and/ or CVP 8-12mmHg Cardiac index (CI) ≥2.4 l/min/m ² Mean arterial pressure (MAP) 60-80mmHg and / or systemic vascular resistance index (SVRI) 1800-2400 dynes•sec/cm ⁵ /m ² Central venous oxygen saturation (ScvO ₂) ≥60% and / or mixed venous oxygen saturation (SvO ₂) ≥70%
Anaesthesia	Although there is no need for anaesthesia, donors who are tending towards hypertension are well managed by the introduction of low concentrations of volatile anaesthetic agents such as isoflurane. There is some data to suggest that such agents provide a preconditioning effect and reduce ischaemia-reperfusion injury. Spinal reflexes will still be present and can be marked, and it is important to ensure full paralysis. Rocuronium 100 mg, or equivalent, will usually suffice for the entire procedure.
Steroids	If not already given, administer 15mg/kg of methylprednisolone intravenously.
Antibiotics	It is routine practice to give a single prophylactic dose of broad spectrum antibiotics. This is usually provided by the retrieval team. Check for allergies before administration.

Surgical procedure

- Exploratory laparotomy. The purpose of this is to ensure no obvious intra-abdominal contra-indication to proceeding with the operation. Generally there are few cardiovascular changes at this time providing intravascular volume is well maintained.
- After an assessment of the abdominal organs and some initial dissection the team will proceed to perform a median sternotomy using a powered or Gigli saw. During the incision of skin and dissection round the sternum **it is worthwhile increasing the inspired oxygen to 100% to allow for the disconnection during the actual sternotomy. To facilitate sternotomy it is ideal to have “table down, lungs down”**. The operating table is lowered to its maximum extent and the ventilator is disconnected to allow lungs to deflate. At the conclusion of the sternotomy inspired oxygen can be reduced again to the minimum necessary to maintain good saturations, with a recruitment manoeuvre if required.
- The pericardium is opened at which point you will be able to assess cardiac contractility and atrial size as an index of filling.
- The abdominal organ team will continue with dissection now aided by the median sternotomy and insertion of a sternal retractor. This affords good surgical access for dissection of the abdominal organs. This can take some time particularly when pancreatic retrieval is proposed.
- If cardiothoracic retrieval is to be performed they will usually arrive in theatre at around this time. **Initially they will perform a bronchoscopy** (raise inspired oxygen to 100% and ensure a catheter mount with a bronchoscopic port is used.). After ensuring that the airways are clear they will scrub and further assess the heart and lungs. A repeat recruitment manoeuvre may be required.

Assessment of heart & lungs

- The cardiac surgeon will examine the heart externally for function and evidence of coronary artery disease. It may be at this stage that unsuspected coronary artery disease is revealed or regional wall motion abnormality. These may influence the decision as to whether or not cardiac retrieval is to proceed. The cardiac team should communicate well with you at this stage.
- Assessment of the lungs will often involve taking differential blood gases from right and left superior and inferior pulmonary veins. **The patient should be on 100% oxygen for five minutes beforehand** and four syringes should be available which are compatible with your local blood gas analyser. It is important that the syringes are able to be aspirated rather than self-filling syringes because of the low pressures in the systems. **Manipulation of the heart during sampling may reduce cardiac output and blood pressure dramatically**. The cardiac surgeons will be aware of this but you should keep them informed if blood pressure does not recover rapidly when the heart is returned to its normal position.
- **Marked cardiac irritability should prompt you to check potassium which may be markedly low if diabetes insipidus is present. Magnesium supplementation is also sometimes required.**
- Following assessment of the blood gases **inspired oxygen can be reduced again** to the lowest level compatible with good saturations. A repeat recruitment manoeuvre may be required.

Abdominal dissection

- Abdominal dissection will be continued and cannulae inserted for abdominal organ perfusion. The cardiac team will re-scrub and insert their cannulae for cardioplegia and pulmonoplegia. At this stage there will be discussion between the cardiac and abdominal retrieval teams as to timing of heparin administration.
- When abdominal and cardiac teams are happy, **ensure that 20,000 units of heparin** (or other dose as discussed with retrieval teams) **is given intravenously and well mixed in the circulation**. This will vary depending on whether or not thoracic organs are to be retrieved. In all cases the heparin will have been injected, and time allowed for circulation. The abdominal aorta is ligated below the renal arteries and if a femoral arterial line is being used, the waveform will be lost.

Perfusion and organ retrieval

- If thoracic organs are not to be retrieved, the aorta is cross-clamped and perfusion commenced via the abdominal aorta below the cross-clamp. Ventilation can cease immediately and the heart will rapidly arrest.
- If cardiothoracic organs are to be retrieved the heart will be arrested using 1L of cold cardioplegia solution. . This will be run through a standard blood giving set (ensuring that no air is present) and is pressurised to 150mm Hg. Communicate with the cardiac team as to who will have control of turning the cardioplegia solution on.
- 3.8L cold pulmonary perfusion solution (Perfadex) is provided and is run through a large bore set (again ensuring no air bubbles) and is raised on a drip stand.
- Occasionally the cardiac team will inject prostacyclin just before cardioplegia (blood pressure will fall but there is no need to treat as cardioplegia is imminent).
- Cardiac team will clamp the aorta and ask you to turn on the cardioplegia solution. Maintain the bag pressure at least 150 mmHg. The heart should rapidly stop in asystole. If the heart continues to beat, quickly check that there is no clamp on the cardioplegia line or if pressure has fallen in the pressure bag.
- Shortly after cardiac arrest the cardiac team will ask for the Perfadex to be run for lung preservation, this should be opened and checked that it is running well. Usually open the one litre bag first to ensure that it is emptying well.
- Ventilation should continue throughout the sequence if lung retrieval is contemplated.
- Dissection will continue and at a later stage the surgeon will ask for the lungs to be maximally inflated and the endotracheal tube withdrawn. At this time they will staple across the trachea ensuring the lungs are well inflated.

References

1. Hagan ME, McClean D, Falcone CA, Arrington J, Matthews D, Summe C: **Attaining specific donor management goals increases number of organs transplanted per donor: a quality improvement project.** *Prog Transplant* 2009, **19**:227-231. <http://www.ncbi.nlm.nih.gov/pubmed/19813484>
2. Franklin GA, Santos AP, Smith JW, Galbraith S, Harbrecht BG, Garrison RN: **Optimization of donor management goals yields increased organ use.** *Am Surg* 2010, **76**:587-594. <http://www.ncbi.nlm.nih.gov/pubmed/20583513>
3. Malinoski DJ, Patel MS, Daly MC, Oley-Graybill C, Salim A: **The impact of meeting donor management goals on the number of organs transplanted per donor: Results from the United Network for Organ Sharing Region 5 prospective donor management goals study.** *Crit Care Med* 2012. <http://www.ncbi.nlm.nih.gov/pubmed/22846779>
4. Malinoski DJ, Patel MS, Ahmed O, Daly MC, Mooney S, Graybill CO, Foster CE, Salim A: **The Impact of Meeting Donor Management Goals on the Development of Delayed Graft Function in Kidney Transplant Recipients.** *Am J Transplant* 2013. <http://www.ncbi.nlm.nih.gov/pubmed/23406284>
5. Minambres E, Ballesteros MA, Rodrigo E, Garcia-Migueluez A, Llorca J, Ruiz JC, Arias M: **Aggressive lung donor management increases graft procurement without increasing renal graft loss after transplantation.** *Clin Transplant* 2013, **27**:52-59. <http://www.ncbi.nlm.nih.gov/pubmed/22897405>
6. **A code of practice for the diagnosis and confirmation of death** [http://www.aomrc.org.uk/publications/statements/doc_view/42-a-code-of-practice-for-the-diagnosis-and-confirmation-of-death.html]
7. Mascia L, Pasero D, Slutsky AS, Arguis MJ, Berardino M, Grasso S, Munari M, Boifava S, Cornara G, Della Corte F, et al: **Effect of a Lung Protective Strategy for Organ Donors on Eligibility and Availability of Lungs for Transplantation.** *JAMA: The Journal of the American Medical Association* 2010, **304**:2620-2627. <http://jama.ama-assn.org/content/304/23/2620.abstract>
8. Rogers DF: **Mucoactive agents for airway mucus hypersecretory diseases.** *Respir Care* 2007, **52**:1176-1193; discussion 1193-1177. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=17716385
9. Jauncey-Cooke JI, Bogossian F, East CE: **Lung recruitment--A guide for clinicians.** *Australian Critical Care* 2009, **22**:155-162. <http://www.sciencedirect.com/science/article/B8CX6-4X07754-1/2/5c8100f7c6ad25e0a76ca3b2e7eb7af4>
10. Hanna K, Seder CW, Weinberger JB, Sills PA, Hagan M, Janczyk RJ: **Airway Pressure Release Ventilation and Successful Lung Donation.** *Arch Surg* 2011, **146**:325-328. <http://archsurg.ama-assn.org/cgi/content/abstract/146/3/325>
11. Kallet RH, Matthay MA: **Hyperoxic acute lung injury.** *Respir Care* 2013, **58**:123-141.
12. Kainz A, Wilflingseder J, Mitterbauer C, Haller M, Burghuber C, Perco P, Langer RM, Heinze G, Oberbauer R: **Steroid Pretreatment of Organ Donors to Prevent Postischemic Renal Allograft Failure.** *Annals of Internal Medicine* 2010, **153**:222-230. <http://www.annals.org/content/153/4/222.abstract>
13. Durairaj L, Schmidt GA: **Fluid Therapy in Resuscitated Sepsis: Less Is More.** *Chest* 2008, **133**:252-263. <http://www.ncbi.nlm.nih.gov/pubmed/18187750>
14. Casartelli M, Bombardini T, Simion D, Gaspari MG, Procaccio F: **Wait, treat and see: echocardiographic monitoring of brain-dead potential donors with stunned heart.** *Cardiovasc Ultrasound* 2012, **10**:25. <http://www.ncbi.nlm.nih.gov/pubmed/22721412>
15. Christmas AB, Bogart TA, Etson KE, Fair BA, Howe HR, Jacobs DG, Sing RF: **The reward is worth the wait: a prospective analysis of 100 consecutive organ donors.** *Am Surg* 2012, **78**:296-299. <http://www.ncbi.nlm.nih.gov/pubmed/22524766>
16. Benito Y, Grietje B, Peter S, Claude B, Meike S, Mathias B, Uwe G, Yang X, Annette B, Silke W, et al: **Prevention of Cold-Preservation Injury of Cultured Endothelial Cells by Catecholamines and Related Compounds.** *American Journal of Transplantation* 2004, **4**:22-30. <http://dx.doi.org/10.1046/j.1600-6143.2003.00268.x>
17. Macdonald PS, Aneman A, Bhonagiri D, Jones D, O'Callaghan G, Silvester W, Watson A, Dobb G: **A systematic review and meta-analysis of clinical trials of thyroid hormone administration to brain dead potential organ donors.** *Critical Care Medicine* 2012, **40**:1635-1644. <http://www.ncbi.nlm.nih.gov/pubmed/22511141>
18. Ranasinghe AM, Bonser RS: **Thyroid hormone in cardiac surgery.** *Vascular Pharmacology* 2010, **52**:131-137. <http://www.sciencedirect.com/science/article/B6X3P-4XT3HPT-1/2/046f3ad676fabd10eb956053aaf351c6>
19. Druce LA, Thorpe CM, Wilton A: **Mineralocorticoid effects due to cortisol inactivation overload explain the beneficial use of hydrocortisone in septic shock.** *Medical Hypotheses* 2008, **70**:56-60. <http://www.sciencedirect.com/science/article/B6WN2-4P77GDS-1/1/a7e388ad0bea5e95064c2cbcad8e449f>
20. Morelli A, Ertmer C, Rehberg S, Lange M, Orecchioni A, Cecchini V, Bachetoni A, D'Alessandro M, Van Aken H, Pietropaoli P, Westphal M: **Continuous terlipressin versus vasopressin infusion in septic shock (TERLIVAP): a randomized, controlled pilot study.** *Critical Care* 2009, **13**:R130. <http://ccforum.com/content/13/4/R130>
21. Maybauer M, Maybauer D: **Vasopressin analogues and V1a receptor agonists in septic shock.** *Inflammation Research* 2011, **60**:425-427. <http://dx.doi.org/10.1007/s00011-011-0314-9>
22. Levy P, Compton S, Welch R, Delgado G, Jennett A, Penugonda N, Dunne R, Zalenski R: **Treatment of Severe Decompensated Heart Failure With High-Dose Intravenous Nitroglycerin: A Feasibility and Outcome Analysis.** *Annals of Emergency Medicine* 2007, **50**:144-152. <http://www.sciencedirect.com/science/article/pii/S0196064407002831>
23. Vallee F, Vallet B, Mathe O, Parraguet J, Mari A, Silva S, Samii K, Fourcade O, Genestal M: **Central venous-to-arterial carbon dioxide difference: an additional target for goal-directed therapy in septic shock?** *Intensive Care Med* 2008, **34**:2218-2225. <http://www.ncbi.nlm.nih.gov/pubmed/18607565>

24. Barbee RW, Reynolds PS, Ward KR: **Assessing shock resuscitation strategies by oxygen debt repayment.** *Shock* 2010, **33**:113-122.
<http://www.ncbi.nlm.nih.gov/pubmed/20081495>
25. Krag A, Bendtsen F, Pedersen EB, Holstein-Rathlou NH, Moller S: **Effects of terlipressin on the aquaretic system: evidence of antidiuretic effects.** *Am J Physiol Renal Physiol* 2008, **295**:F1295-1300. <http://www.ncbi.nlm.nih.gov/pubmed/20695722>
26. Schnuelle P, Gottmann U, Hoeger S, Boesebeck D, Lauchart W, Weiss C, Fischereder M, Jauch K-W, Heemann U, Zeier M, et al: **Effects of Donor Pretreatment With Dopamine on Graft Function After Kidney Transplantation: A Randomized Controlled Trial.** *Jama* 2009, **302**:1067-1075. <http://jama.ama-assn.org/cgi/content/abstract/302/10/1067>
27. Faybik P, Hetz H: **Plasma Disappearance Rate of Indocyanine Green in Liver Dysfunction.** *Transplantation Proceedings* 2006, **38**:801-802.
<http://www.sciencedirect.com/science/article/B6VJ0-4JT975Y-1H/1/aa9c88a570fb99199f3534992bbad042>
28. Seeley E, McAuley DF, Eisner M, Miletin M, Matthay MA, Kallet RH: **Predictors of mortality in acute lung injury during the era of lung protective ventilation.** *Thorax* 2008, **63**:994-998. <http://thorax.bmj.com/cgi/content/abstract/63/11/994>
29. Sinha P, Fauvel NJ, Singh S, Soni N: **Ventilatory ratio: a simple bedside measure of ventilation.** *Br J Anaesth* 2009, **102**:692-697.
<http://bj.oxfordjournals.org/cgi/content/abstract/102/5/692>
30. Siddiki H, Kojicic M, Li G, Yilmaz M, Thompson T, Hubmayr R, Gajic O: **Bedside quantification of dead-space fraction using routine clinical data in patients with acute lung injury: secondary analysis of two prospective trials.** *Critical Care*, **14**:R141. <http://ccforum.com/content/14/4/R141>

Appendix - BSD testing record form

The Diagnosis of Death Following Irreversible Cessation of Brain-Stem Function [6]		
Adults The diagnosis of death by brain-stem testing should be made by at least two medical practitioners who have been registered for more than five years and are competent in the conduct and interpretation of brain-stem testing. At least one of the doctors must be a consultant. Testing should be performed completely and successfully on two occasions with both doctors present.		
Patient Name		Hospital Number
Doctor One		Doctor Two
Name	Name	
Grade	Grade	
Signature	Signature	
Preconditions		
Unresponsive Coma <input type="checkbox"/> Apnoea <input type="checkbox"/> Date and time of onset Evidence for Irreversible Brain Damage of known aetiology (please define):		
Exclusion of Potentially Reversible Causes		
	<i>Dr One</i>	<i>Dr Two</i>
1. Is the coma due to depressant drugs? State drug levels (if taken):	Y / N	Y / N
2. Is the patient's body temperature $\leq 34^{\circ}\text{C}$?	Y / N	Y / N
3. Is the coma due to a circulatory, metabolic or endocrine disorder?	Y / N	Y / N
4. Is the respiratory failure due to neuromuscular blocking agents, other drugs or potentially reversible causes of apnoea (eg. cervical injury, profound neuromuscular weakness)?	Y / N	Y / N
5. Sodium levels at time of coma onset (<i>must be</i> 115-160mmol/L)	Level	
6. Sodium levels at time of 1 st TEST. (<i>must be</i> 115-160mmol/L AND not have changed by > 0.5mmol/L per hour between time of coma onset and 1 st TEST.)	Level	
7. Potassium levels at time of 1 st TEST (<i>must be</i> > 2mmol/L)	Level	
8. Phosphate levels at time of 1 st TEST (<i>must be</i> 0.5-3.0mmol/L)	Level	
9. Magnesium levels at time of 1 st TEST (<i>must be</i> 0.5-3.0mmol/L)	Level	
10. Glucose levels at time of 1 st TEST (<i>must be</i> 3.0-20.0mmol/L)	Level	

Tests for Absence of Brain-Stem Function				
	1 st TEST		2 nd TEST	
	Dr One	Dr Two	Dr One	Dr Two
1. Do the pupils react to light?	Y / N	Y / N	Y / N	Y / N
2. Is there any eye movement when each cornea is touched in turn?	Y / N	Y / N	Y / N	Y / N
3. Is there nystagmus or any eye movement present when each ear is instilled with 50mls ice cold water?	Y / N	Y / N	Y / N	Y / N
4a. Is the gag reflex present?	Y / N	Y / N	Y / N	Y / N
4b. Is the cough reflex response present when a suction catheter is passed down the trachea?	Y / N	Y / N	Y / N	Y / N
5. Is there any motor response when supraorbital pressure is applied?	Y / N	Y / N	Y / N	Y / N
6a. Apnoea Test - pre-conditions				
PaCO ₂ 6.0-8.0kPa AND	Level		Level	
arterial pH 7.20-7.40	Level		Level	
6b. Apnoea Test - After 5 minutes				
Was there any spontaneous respiration?	Y / N	Y / N	Y / N	Y / N
PaCO ₂ (must have increased by > 0.5kPa)	Level		Level	
PaO ₂ (must be > 5kPa)	Level		Level	
Was the MAP ≥ 60mmHg throughout?	Y / N	Y / N	Y / N	Y / N
Ancillary Investigations Used To Confirm the Diagnosis				
Is there a need for ancillary investigations to confirm the diagnosis?			Y / N	
If yes, please outline the results of these investigations:				
Completion of Diagnosis				
Are you satisfied that death has been confirmed following the irreversible cessation of brain-stem-function?	Y / N		Y / N	
Legal time of death is when the 1 st TEST indicates death due to the absence of brain-stem reflexes. Death is confirmed following the 2 nd TEST.	Date: Time:		Date: Time::	
	Dr One Initials		Dr One Initials	
	Dr Two Initials		Dr Two Initials	