Guideline

Management of the Potential Organ and Tissue Donor following Neurological Determination of Death

Summary Reference for managing and delivering standardised clinical therapies to potential organ and tissue donors following neurological death.

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NSW Health

GUIDELINE

Management of the Potential Organ and Tissue Donor following Neurological Determination of Death

GUIDELINE SUMMARY

This Guideline provides recommendations for managing and delivering standardised clinical therapies to potential organ and tissue donors following neurological death. The goal is to support and optimise organ and tissue function and improve organ retrieval for transplantation.

The Guideline must be read in conjunction with the State Form SMR010517 Neurological Determination of Death (also known as Brain Dead) and the NSW Health Policy Directive *Organ and Tissue Donation, Use and Retention* (PD2022_035).

KEY PRINCIPLES

The criteria for neurological determination of death are established by Australian and New Zealand Intensive Care Society (ANZICS) and set out in section 1.2 Neurological determination of death in the Australian and New Zealand Intensive Care Society (ANZICS) *The Statement on Death and Organ Donation* edition 4.1 (2021) (ANZICS Statement).

The management of the potential organ and tissue donor after neurological determination of death aims to support organ function and optimise the number of organs retrieved for transplantation.

This includes frequent clinical assessment of organ function and response to interventions as well as ensuring that the time from determination of death to retrieval surgery is as short as possible.

The recommendations are largely based on physiological rationale, consensus statements and limited clinical research with a non-negligible risk for bias.

Consent is another key principle of all donations as outlined in the NSW Health Policy Directive *Organ and Tissue Donation, Use and Retention* (PD2022_035).

A valid consent is essential for any donation, refer to the NSW Health *Consent to Medical and Healthcare Treatment Manual* (The Consent Manual). The hospital's Designated Officer must also have granted authorisation to remove the organ/s and/or tissue.

In NSW the Organ and Tissue Authority's Best Practice Guideline *for Offering Organ and Tissue Donation in Australia* (Best Practice Guideline) is used to support families make an informed decision about donation and ensures that a Donation Specialist participates in the Family Donation Conversation.

Consent must be in writing or by other manner prescribed as per the NSW Health Policy Directive *Organ and Tissue Donation, Use and Retention* (PD2022_035) and the NSW Health Consent to Medical and Healthcare Treatment Manual (The Consent Manual).

REVISION HISTORY

Version	Approved By	Amendment Notes
GL2023_013 April-2023	Deputy Secretary, Population and Public Health & Chief Health Officer	Updated terminology, including paediatric references and ensure reflection of best practice.
GL2016_008 March-2016	Deputy Secretary, Population and Public Health & Chief Health Officer	New Guideline

CONTENTS

1. BACKGROUND

The clinical management of the potential organ and tissue donor (donor) who has died by neurological criteria follows generic intensive care principles to optimise organ function. Potential paediatric donors have specific requirements that need to be considered. However, the overall physiological management delivered to a critically ill child is similar to that of an adult and will be included in this document.[1]

Herniation of the brain stem triggers a multitude of autonomic, endocrine and inflammatory changes that may make management of the donor a clinical challenge. Hypotension (>80% of donors), central diabetes insipidus (>60% of donors), hypothermia and hypernatraemia are common issues. Disseminated intravascular coagulation, cardiac arrhythmias, pulmonary oedema and metabolic acidosis are less frequently encountered problems.

Donor management aims to support donor organ function and has shown to improve the number and function of organs retrieved for transplantation, particularly for the heart and lungs.[2,3] While several donor management guidelines and reviews have been published,[4-12] including a literature review in 2015,[13] there is still little evidence to guide clinical practice. The recommendations are largely based on physiological rationale, consensus statements and limited clinical research with a non-negligible risk for bias.

Cadaveric organ donation is a difficult area of research. However, an ongoing multicentre, randomised, double-blind, placebo-controlled trial of nebulised salbutamol to improve the ratio of partial pressure of oxygen (PaO_2) to the fraction of inspired oxygen (FiO_2) in intended lung donors demonstrated that large scale clinical trials are still feasible.[14]

The following practice points are useful:

- Early notification to the organ and tissue donation team will assist in donor management
- The time from neurological determination of death to retrieval surgery should be kept as short as possible.
- The goal of donor management is to optimise organ function to maximise the number of transplantable organs and the transplantation outcome for the recipient.
- The key to optimal donor management is frequent clinical assessment of organ function and response to interventions.

1.1. About this document

This Guideline provides information for managing the physiological effects of neurological death in an organ and tissue donor, and aims to standardise the clinical therapies delivered to the donor from the determination of neurological death to retrieval surgery in order to:

- fulfill the wishes of the deceased, their family and loved ones
- ensure that organs retrieved have been optimised for transplantation
- maximise the number of potentially transplantable organs

- facilitate a controlled approach to retrieval surgery with a physiologically stable organ and tissue donor.

This Guideline must be read in conjunction with:

NSW Health Policy Directives, Guidelines, Manuals and State Forms	
PD2022_035	*Organ and Tissue Donation, Use and Retention*
State Form SMR010517	*Neurological Determination of Death* (also called brain death)

State Forms can be ordered via the NSW Health intranet webpage *Ordering and Printing Forms*.

The Organ and Tissue Authority's *Best Practice Guideline for Offering Organ and Tissue Donation in Australia* also provides guidance in supporting families in the decision-making process for organ and tissue donation.

1.2. Legal and legislative framework

This Guideline is to be used for individuals who have been determined to have died by neurological criteria using the definition outlined in the *Human Tissue Act 1983* (NSW) (the Act).

As set out in section 33 of the Act, for the purposes of the law in NSW, a person has died when the following has occurred:

a) irreversible cessation of all function of the person's brain, or

b) irreversible cessation of circulation of blood in the person's body.

2. CARDIOVASCULAR SYSTEM

2.1. Physiology

Progressive brain stem ischaemia during herniation triggers an intense but transient sympathetic activation and markedly increased systemic vascular resistance. This leads to arterial and venous hypertension, decreased left ventricular output with risk of end organ hypoperfusion, pulmonary congestion and increased hydrostatic pulmonary capillary pressure, which may manifest as overt pulmonary oedema.

After cerebral herniation and brain stem ischaemia, arterial hypotension and venous pooling (relative hypovolaemia) occur and will result in decreased organ perfusion unless treated.

2.2. Hypertensive response

Hypertension rarely needs active management due to its transient nature but, if severe and prolonged, may require short-acting antihypertensives. Most commonly used drugs are:

- Esmolol (infusion 50-300 micrograms/kg/minute)
- Sodium Nitroprusside (0.5-4 micrograms/kg/min)
- Clevidipine (starting dose 1-2mg/hr, maintenance dose 4-6mg/hr) – not common in

children.

2.3. Hypotensive response

Hypotension is multifactorial, including decreased mean systemic filling pressure, low vascular resistance and decreased cardiac output.

Intravascular hypovolaemia is common due to the previous use of osmotic diuretics, onset of central diabetes insipidus (CDI) and hyperglycaemia induced diuresis.

Vasodilation is the result of a lack of sympathetic outflow, inflammatory activation, and possible vasodilatory effects of previous deep sedation for increased intracranial pressure (ICP) control. Reduced cardiac contractility is often due to catecholamine cardiotoxicity during the endogenous sympathetic surge.

Management

The management of hypotension is dependent on the clinical examination of the individual, the preceding clinical management and progress. Three management strategies are commonly adopted, and treatment is escalated dependent on the clinical response. These strategies are:

- volume expansion
- vasopressors
- inotropes.

The management of a patient with haemodynamic instability is significantly facilitated by the insertion of a central venous line (CVL) and arterial line.

2.3.1. Volume expansion

Choice of intravascular fluid

Crystalloids with balanced salt content are to be used to avoid hypernatraemia (concurrent CDI) and hyperchloraemic acidosis (increases renal vascular resistance, confounds base excess when used as resuscitation target).

There is no support for the use of artificial colloids.

Albumin solutions (20%, 4%) may be considered to reduce the amount of volume given, although this is usually only moderately effective; the high sodium content of 4% albumin-based solutions needs to be considered and compared with the donor plasma sodium.

The most commonly used fluids are Hartmann's solution, Plasmalyte, 0.9% NaCl, 0.18% NaCl (in the setting of hypernatraemia) and 4% or 20% albumin.

Monitoring

Serial clinical examination assessing the response to fluid bolus is a simple and effective way of determining appropriate intravascular volume. Central venous pressure is notoriously unreliable to assess the intravascular volume state, but changes during rapid infusions (<15

min) may indicate the degree of volume responsiveness. Pulse pressure variation is another method that has been used to determine optimal fluid status.

Repeat bedside echocardiographic studies can also be used but must be done by an appropriately trained operator.

Haemodilution might make blood transfusion necessary to maintain haemoglobin above 70g/L for a cardiovascularly stable donor and above 90g/L for an unstable donor.

Goals of fluid therapy

- Urine output 0.5 ml/kg/hr (note – polyuria due to central diabetes insipidus or diuretics may be confounders; in children target of 1-3ml/kg/hr).
- Central venous oxygen saturation (ScvO2) >70% (note – low basal metabolism due to neurological death may be a confounder).
- Cardiac index >2.2 L/min/m^2 (note – high cardiac output state due to vasodilatory shock may be a confounder).

Complications

- Fluid overload worsens lung transplantation outcomes whereas a more restrictive fluid regimen does not appear to worsen renal transplantation outcomes.
- Fluid balance needs to be carefully assessed to avoid excessive fluid loading, especially when the lungs are considered for transplantation.

2.3.2. Vasopressors

Vasopressors are frequently needed to support mean arterial pressure once hypovolaemia has been excluded/ corrected.

Goals and monitoring

- Mean arterial pressure (MAP) >65 mmHg, or systolic blood pressure (SBP) >100 mmHg (refer to Section 6 for paediatric reference range)
- Cardiac output monitoring, such as echocardiographic studies, is recommended in patients with high vasopressor requirements to rule out myocardial dysfunction.

Choice of agent

Noradrenaline is the preferred vasopressor to improve/ support organ perfusion pressure in the absence of hypovolaemia.

- Tachyphylaxis from prolonged previous use of noradrenaline to support cerebral perfusion pressure and a systemic inflammatory response may lead to high doses of noradrenaline required to attain a mean arterial pressure >70 mmHg and a systolic blood pressure >100 mmHg.
- In the setting of escalating noradrenaline requirements consider vasopressin.

Vasopressin infusion is useful to reduce the noradrenaline requirements and helpful in the setting of CDI.

- Dose of vasopressin typically up to 0.5-2.4 units/hour [10-50 milliunits/kg/hour] – note when used as a vasopressor the dose is much higher than that for CDI (see Section 4.2 for CDI dose)
- The catecholamine sparing effect of vasopressin may improve cardiac function post transplantation.

Refer to Section 6 for paediatric reference range.

2.3.3. Inotropes

Inotropic agents, such as dobutamine and/or adrenaline, might be used in case of refractory or worsening cardiac failure that does not respond to vasopressors to maintain coronary perfusion pressure.

Dopamine is very rarely used in Australian clinical practice although some theoretical benefits (modulation of immune and ischaemia-reperfusion responses) are still the subject of experimental investigations.

Monitoring

The combination of vasopressors and inotropes make careful functional examination of the heart necessary and monitoring of cardiac output in adult potential organ donors is highly recommended.

Dosage

- Dobutamine – titrate for cardiac index >2.5
- Adrenaline – titrate for cardiac index >2.5

2.4. Arrhythmias

In the peri-herniation period, arrhythmias are usually related to autonomic instability and often refractory to treatment. Tachyarrhythmias developing post-herniation should prompt the clinical team to review the patient's volume and electrolyte status (K, Mg).

Ventricular and supraventricular tachyarrhythmias – amiodarone is the drug of choice.

Dosage

- Amiodarone – bolus 200-300 mg for adults, followed by infusion (max dose of 1200 mg/24h)
- Amiodarone – bolus 25 microgram/kg/minute over 4 hours for paediatric dose, followed by infusion (5-15 microgram/kg/minute)

Bradyarrhythmias – direct acting β-sympathomimetics (for example adrenaline or isoprenaline) should be used as atropine is ineffective due to the vagal breakdown following herniation.

Dosage

- Adrenaline, isoprenaline – titrate to desired heart rate (60-120 bpm).

3. RESPIRATORY SYSTEM

Many lung donors fail to meet eligibility criteria due to potentially preventable causes such as:

- atelectasis
- pulmonary oedema – neurogenic and fluid overload
- pneumonia (hypostatic pneumonia, ventilator-associated pneumonia acquired during deep sedation for intracranial hypertension management, aspiration pneumonia, pulmonary inflammatory response triggered by cerebral herniation).

3.1. Ventilator management

The focus of care must change from "cerebroprotective" to "lung protective" with the main goal of optimal dynamic compliance in the donor lungs[15]. This may include cessation of spinal precautions to optimise patient positioning.

The main target of mechanical ventilation is to minimise lung trauma by using low tidal volumes, low mean airway pressure and optimal positive end-expiratory pressure using recruitment manoeuvres.

- Tidal volumes of 6-8 ml/kg
- Positive end-expiratory pressure 5-10 cm H_2O to maintain lung recruitment
- Preferably with a mean airway pressure <30 cm H_2O on volume-controlled mode
- Respiratory rate adjusted to obtain normocapnia, aiming at a physiological pH range
- Adjust fraction of inspired oxygen (FiO_2) to the minimal fraction necessary to obtain partial pressure of oxygen (PaO_2) 80-100 mmHg
- Recruitment manoeuvres can be performed after endotracheal suctioning that is frequently needed to maintain patent airways
- Repeated arterial blood gas analyses are necessary to optimise ventilation and assess lung function
- Head of bed elevation >30 degrees
- Continue/ commence standard Intensive Care Unit (ICU) chest physiotherapy.

A bronchoscopy could be considered early in the management of the donor. This must be done by a skilled operator who can navigate the airway anatomy while minimising mucosal trauma. The aim of the bronchoscopy is to evaluate signs of bronchitis, aspiration, to obtain a bronchoalveolar lavage sample for culture (if there is a clinical suspicion of infection) and to clear stagnant secretions resulting in atelectasis.

4. METABOLIC AND ENDOCRINE SYSTEMS

The development of progressive brain ischaemia may be associated with variable endocrine changes, including decreased levels of antidiuretic hormone, cortisol, insulin, and T_3.[16] These changes are not uniform and experimental and clinical studies have provided conflicting results. Hence there is a lack of evidence to guide replacement therapies.

4.1. Anterior pituitary

Anterior pituitary function is often preserved in donors who have died by neurological criteria. Despite this, hormone replacement therapy, notably the administration of the thyroid hormone triiodothyronine (T_3) also has positive inotropic effects. However, there is no data from prospective, controlled randomised trials to support the use of hormone replacement therapy.

Recommendations for its use in marginal heart donors is based on retrospective analyses and clinical experience/ case series.[17] In clinical practice, T_3 is usually considered together with vasopressin, methylprednisolone and insulin as part of a "hormone replacement therapy cocktail".

Echocardiography is used to exclude structural abnormalities of the heart, whereas any functional impairment (ejection fraction <45%, wall motion abnormality) may still be reversible and responsive to further supportive treatment, including hormone replacement therapy.

Glucocorticoid replacement

Use of glucocorticoids has more support as an anti-inflammatory therapy than hormone replacement therapy. As such, a supraphysiological dose of methylprednisolone has frequently been used to suppress the inflammatory response associated with cerebral herniation, particularly in heart/ lung donors.

Administration of steroids can be done in isolation and does not necessarily form part of hormone replacement therapy. A trial of hydrocortisone to facilitate weaning of vasopressors (noradrenaline) was terminated early since clinical practice had changed to routinely include steroids.[18]

Dosage

- Methylprednisolone 15 mg/kg intravenous bolus once only

Thyroid hormone replacement (T_3)

This remains contentious in the absence of robust clinical evidence, yet there is no evidence to suggest harm from this practice.

Several case reports have documented reversal of even severe systolic cardiac dysfunction following T_3 administration, and T_3 should be considered in the haemodynamically unstable heart donor and discussions with the surgical transplant team are encouraged.

Dosage

- T_3 infusion 4 mcg/hr (0.1-0.15 microgram/kg/hour)

4.2. Central Diabetes Insipidus (CDI)

Polyuria is commonly defined as a urine output >3 L/day but this definition does not apply to the acute setting. A common practice point is to use a urine output >3 ml/kg/h for two consecutive hours. Central Diabetes Insipidus (CDI) may be confirmed by a combination of high plasma osmolality (>300 mOsm/kg) and a low urine osmolality, usually <100 mOsm/kg.

However, in the setting of the neurological determination of death it is highly advisable to start therapy with vasopressin analogues even before the results of the plasma and urine osmolality. CDI is commonly seen in neurological death as part of posterior pituitary dysfunction.

Complications of CDI

- Hypovolaemia → haemodynamic instability
- Hypernatraemia

Treatment

Responds well to either desmopressin or vasopressin.

	Desmopressin	Vasopressin
Source	Synthetic analogue	
Antidiuretic effect	Excellent	Excellent
Vasopressor effect	Minimal - 0.1% of Vasopressin	Potent
Dose	• 1 – 4 mcg intravenous bolus q4-8h, titrated to antidiuretic effect • Monitored by urine output and serum/urine osmolality	• Mainly used in children with a dose range: 0.5 - 3 milliunits/kg/hour • Higher doses are not usually warranted unless the patient is in vasodilatory shock • In hemodynamically unstable patients, vasopressin is considered the better agent as it is viewed as an ADH replacement therapy
Duration	~12 hours (range 8 – 20 h)	Minutes
Clinical use	• Haemodynamically stable patients • Intravenous bolus dose	• Haemodynamically unstable patients with hypotension related to vasodilatation • Infusion
Complications	• Hyponatraemia	• Hyponatraemia • Caution – potent coronary and splanchnic vasoconstrictive properties • Essential to correct hypovolaemia before commencement

Goals of management

- Maintenance of euvolaemia

- Normal serum sodium (aim 135-145mmol/L)
  - There is an association between hypernatraemia (155-170 mmol/L) and poor hepatic transplant outcomes.

4.3. Plasma glucose management

Maintaining normoglycaemia is part of standard intensive care unit care and infusion of short acting insulin is often needed due to decreased insulin secretion and increased insulin resistance following the neurological determination of death.

Maintain previous feeding or infusion sources of glucose. The anti-inflammatory properties of insulin do not seem clinically relevant, but hyperglycaemia adds to a pro-inflammatory state and might be particularly deleterious for kidney/ pancreas donors.

- Administer Actrapid titrated to BSL level 6-10 mmol/L.

5. GENERAL CARE

5.1. Temperature homeostasis

Hypothermia is common as part of acquired poikilothermia following the neurological determination of death and due to body exposure and infusion of fluids at room temperature. Active warming is frequently needed, and body exposure is to be kept to a minimum to maintain normothermia (aim >36°C). A study investigating the effects of controlled in-vivo hypothermia (32-34°C) in donors who have died by neurological criteria demonstrated a decrease in delayed graft function in renal recipients.[19]

Hyperthermia is usually of infectious aetiology and must be managed with antipyretics and antibiotics and, if extreme, with cooling blankets to return to normothermia.

5.2. Coagulation

Blood products (packed red blood cells, frozen fresh plasma and platelets) are used as per standard intensive care unit practice to correct pre-existing traumatic coagulopathy or to treat coagulopathy disseminated intravascular coagulation triggered by the release of tissue factor from necrotic brain tissue.

An international normalised ratio <1.5 and platelet count >50,000 should be maintained until retrieval surgery occurs. Packed red blood cell transfusions are used to ensure the haemoglobin is greater than 70 g/L in physiologically stable donors, and greater than 90 g/L in those donors that require significant inotropic support. Anticoagulants for deep vein thrombosis prophylaxis should continue in view of the high incidence of pulmonary embolism reported at lung retrieval surgery.

Heparin 5000 U subcutaneously twice-daily is common practice and reversible if needed.

5.3. Antibiotics

Clinical signs of infection and, more importantly, microbiology results should guide the use of antibiotics that are usually indicated by pre-mortem findings. Prophylactic use of antibiotics

following the neurological determination of death does not seem clinically warranted. Review by an infectious disease specialist is encouraged.

6. SYSTEM SPECIFIC MANAGEMENT

Cardiovascular management – for organ perfusion NOT cerebral perfusion Aim for CVP 6-10 mmHg, HR <120 bpm, MAP 60-80 mmHg For paediatric donors HR and MAP please note reference below		
Clinical Concern	**Possible Aetiology**	**Management Strategies**
Hypertension SBP >180 mmHg	Normally associated with herniation and is self-terminating	Short acting beta blocker (esmolol 50 - 300 microgram/kg/min) Vasodilator (sodium nitroprusside 0.5 - 4 microgram/kg/min)
Hypotension SBP <100 mmHg	Hypovolaemia Secondary to inadequate cardiac pre-load i.e. blood loss, polyuria (central diabetes insipidus), diuretics, hyperglycaemia or through therapeutic dehydration to ↓ICP. Secondary to low afterload. Vasoplegia caused by absent central vasomotor control and decreased vascular resistance or after re warming.	**Replace volume** Avoid hyperchloraemia and hypernatraemia and starch-based colloids **Blood transfusion** Aim for Hb >70 g/L STABLE donor Aim for Hb >90 g/L UNSTABLE donor **Vasopressors** Noradrenaline is the vasopressor administered most. If noradrenaline is >0.2 microgram/kg/minute then vasopressin (10-50 milliunits/kg/hour; max adult dose 2.4 unit/h) may allow reduction of noradrenaline
Arrhythmias	Supraventricular and ventricular → Tachycardia Bradycardia →	**Normalise physiology** Maintain normal serum electrolytes (optimise K^+, Mg^+ Ca^{2+}), optimise fluid status, normalise temperature. **Arrhythmia management** Standard arrhythmia management should be initiated (amiodarone, cardioversion). Consider adrenaline, isoprenaline or pacing. Normally **resistant** to atropine or glycopyrrolate.

Paediatric donors HR and MAP age range*#				
	Heart rate (beats per minute)	**Systolic Blood Pressure (MAP mmHg)**	**Mean Arterial Blood Pressure (MAP mmHg)**	**Diastolic Blood Pressure (MAP mmHg)**
Birth to 2 months	95 – 180	70 – 85	40 – 55	35 – 40
2 months to 1 year	100 – 170	75 – 90	45 – 60	35 – 45
1 year to 5 years	90 – 140	80 – 100	50 – 75	40 – 55
5 years to 10 years	80 – 120	90 – 105	60 – 80	50 – 65
more than 10 years	60 – 100	100 – 120	65 – 80	60 – 70

* Haque IU, Zaritsky AL. Analysis of the evidence for the lower limit of systolic and mean arterial pressure in children. Pediatr Crit Care Med. 2007 Mar;8(2):138-44. doi: 10.1097/01.PCC.0000257039.32593.DC. PMID: 17273118.

National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents; The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents. Pediatrics August 2004; 114 (Supplement_2): 555–576. 10.1542/peds.114.S2.555

Respiratory management – optimise tissue oxygen, prevent lung injury and infection Aim for pH 7.35-7.45, $PaCO_2$; 35-45 mmHg; PaO_2 80-100 mmHg; SaO_2 >95%		
Clinical Concern	**Possible Aetiology**	**Management Strategies – Adult and Paediatric**
Hypoxaemia	De-recruitment of lungs due to no cough or respiratory drive Orthostatic pneumonia and sputum retention Neurogenic pulmonary oedema Aspiration Acute lung injury Trauma	**Optimise mechanical ventilation** TV = 6-8mls/kg (ideal body weight) PEEP 5-10cm H_2O Plateau PIP <30 cm H_2O Normocapnia = $PaCO_2$ 35-45, normal pH FiO_2 = lowest possible to maintain PaO_2 80-100 mmHg and SaO_2 >95% **Lung recruitment** Early and continued physiotherapy (ideally 4-hourly), suctioning and re-positioning to promote alveolar recruitment. **Optimisation of fluid management** Aim for a negative fluid balance if cardiovascular stability allows this to occur. **General management** 2 to 4-hourly ABG as clinically indicated HOB elevation >30 degrees CXR if PaO_2 starts to decrease or oxygen requirements start to increase • Add broad spectrum antibiotics if clinically indicated.

Endocrine and Metabolic Aim for normal range for all of the below: Temperature 36-37.5°C; Electrolytes (Ca, Mg, KPO_4, K, Na); Blood sugar 5-10 mmol/L; Urine output 0.5-1ml/kg/h		
Clinical Concern	**Possible Aetiology**	**Management Strategies – Adults and Paediatric**
Hypothermia May cause an increased risk of arrhythmias, coagulopathy and a delay in the neurological determination of death.	Hypothalamic/pituitary malfunction	Ensure temperature is maintained >36˚C Early use of warming blankets, fluid warmers for large fluid volumes and humidification devices.
Central diabetes insipidus	Lack of ADH hormone secretion from the posterior pituitary gland. Results in polyuria, hypernatraemia and hypovolaemia. Deficiency of ADH can lead to a systemic vasodilation induced by the loss of sympathetic activity. Common in donors who have died by neurological criteria (seen in >60%).	Start either vasopressin infusion or desmopressin if urine output >3 mls/kg for 2 consecutive hours, associated with a rising plasma sodium. Send paired urine and plasma electrolytes and osmolality to make a diagnosis but DO NOT delay commencement of treatment. **Vasopressin** infusion normal up to a maximum of 0.6 units/hour (paediatric dose range 0.5-3 milliunits/kg/hour) **Desmopressin** 2-4 micrograms every 2-6hr (not commonly used in children) **Fluid replacement**: use fluids that have a low sodium concentration as free water is lost and hypernatraemia develops. Use 5% dextrose for maintenance fluids where sodium levels are high (e.g. >155 mmol/L). For fluid resuscitation consider 0.45% saline or Hartman's solution.
Hypernatraemia (Na >155 mmol/L) Adverse effect on the outcome of Liver recipients	As a consequence of central DI or intracranial hypertension management.	Remove all sources of sodium in IV solutions Review if central DI contributory and follow central DI management as above.
Hyperglycaemia	May be pre-existing – IDDM NIDDM or as a consequence of high volumes of 5% dextrose solution.	Insulin as per the ICU policy should be administered to achieve plasma glucose levels 6 - 10 mmol/L
Hormonal Replacement Therapy	Loss of the hypothalamic/pituitary axis can impact on the haemodynamic stability in the donor who has died by neurological criteria.	T_3 Infusion 0.1 - 0.15 microgram/kg/hour Methylprednisolone 15 mg/kg Lack of agreement exists in the benefits of hormonal replacement therapy following neurological death. It is advised that hormonal replacement therapy be used in haemodynamically unstable patients (MAP<60 mmHg with CVP >12 mmHg and/or Noradrenaline infusion >0.2 mcg/kg/min), or in patients that have an evidence of cardiac dysfunction (LVEF <45% or major LV wall motion abnormality).

Infection Control and General Care
General measures of infection control apply. These include: 1. Hand hygiene – as per standard medical/ nursing care 2. Physiotherapy and bronchial toilet – improves elimination of secretions and improves chances of lung donation. 3. Eye care – care to ensure no corneal abrasions or ulcers to improve the chance of corneal donation. 4. Bowel care – continue nutrition, gastroparesis and bowel management as necessary More detailed information on infection control is available in the NSW Health Policy Directive *Infection Prevention and Control Policy* (PD2017_013).

7. MONITORING, INVESTIGATION AND GENERAL CARE

Parameter	Interval
Monitoring	
Cardiovascular	
ECG	Continuous
BP (invasive)	Continuous
Respiratory	
Saturations	Continuous
End tidal CO_2	Continuous
General	
Urine output (ml/hr)	Hourly
Temperature	Hourly
Investigation	
ECG	Baseline and following neurological determination of death (then PRN)
CXR	Baseline (then PRN)
Bloods:	
FBC	Daily
Coagulation	Daily
Liver function	Daily
Electrolytes	6-hourly
Urea & creatinine	6-hourly
ABG	Baseline (then as clinically needed)
Cultures:	
Blood	Once
Urine	Once
Sputum (from ETT)	Once

Special Investigation	
ECHO	Following neurological determination of death to review functionality and structure of heart for all heart donors
PA catheter and PICO	As per ICU protocol
Bronchoscopy	Selected lung donor
Coronary angiography	Selected heart donors - extended criteria category with risk factors for coronary artery disease. As clinically indicated following consultation with heart transplant team
Renal or hepatic imaging	As clinically indicated following consultation with transplant team
Medication chart	
Review all medications and cease the following:	Anticonvulsants Analgesics Sedatives Laxatives gastrointestinal motility agents Anti-nausea Antihypertensive Mannitol Diuretics
Nutrition Enteral nutrition should be continued	

8. REFERENCES

1. Beckman EJ. Management of the Pediatric Organ Donor. J Pediatr Pharmacol Ther. 2019 Jul-Aug;24(4):276-289. doi: 10.5863/1551-6776-24.4.276. PMID: 31337990; PMCID: PMC6633274.
2. Malinoski DJ, Daly MC, Patel MS, Oley-Graybill C, Foster CE 3rd, Salim A. Achieving donor management goals before deceased donor procurement is associated with more organs transplanted per donor. J Trauma. 2011; 71:990-5.
3. Franklin GA, Santos AP, Smith JW, Galbraith S, Harbrecht BG, Garrison RN. Optimization of donor management goals yields increased organ use. Am Surg. 2010; 76:587-94.
4. Dare AJ, Bartlett AS, Fraser JF. Critical care of the potential organ donor. Curr Neurol Neurosci Rep. 2012; 12:456-65.
5. McKeown DW, Bonser RS, Kellum JA. Management of the heartbeating brain-dead organ donor. Br J Anaesth. 2012; 108(S1):96-107.
6. Dictus C, Vienenkoetter B, Esmaeilzadeh M, Unterberg A, Ahmadi R. Critical care management of potential organ donors: our current standard. Clin Transplant. 2009;23(S21):2-9.
7. Bugge JF. Brain death and its implications for management of the potential organ donor. Acta Anaesthesiol Scand. 2009; 53:1239-50.

8. Mascia L, Mastromauro I, Viberti S, Vincenzi M, Zanello M. Management to optimize organ procurement in brain dead donors. Minerva Anestesiol. 2009; 75:125-33.
9. Kutsogiannis DJ, Pagliarello G, Doig C, Ross H, Shemie SD. Medical management to optimize donor organ potential: review of the literature. Can J Anaesth. 2006; 53:820- 30.
10. Wood, K.E., Becker, B.N., McCartney, J.G., D'Alessandro, A.M., Coursin, D.B. Care of the potential organ donor. N Engl J Med. 2004; 351:2730-39.
11. Shemie SD, Ross H, Pagliarello J, Baker AJ, Greig PD, Brand T, Cockfield S, Keshavjee S, Nickerson P, Rao V, Guest C, Young K, Doig C; Pediatric Recommendations Group. Organ donor management in Canada: recommendations of the forum on Medical Management to Optimize Donor Organ Potential. CMAJ. 2006; 174: S13-32.
12. Australian and New Zealand Intensive Care Society (ANZICS). The Statement on Death and Organ Donation (Edition 4.1). ANZICS.2021.
13. Kotloff, Robert M, Sandralee Blosser, Gerard J Fulda, Darren Malinoski, Vivek N Ahya, Luis Angel, Matthew C Byrnes, et al. Management of the Potential Organ Donor in the ICU. Critical Care Medicine 43, no. 6 (June 2015): 1291-1325.
14. Ware LB, Koyama T, Billheimer D, Landeck M, Johnson E, Brady S, Bernard GR, Matthay MA; California Transplant Donor Network. Advancing donor management research: design and implementation of a large, randomized, placebo-controlled trial. Ann Intensive Care. 2011; 1:20.
15. Mascia L, Pasero D, Slutsky AS, Arguis MJ, Berardino M, Grasso S, Munari M, Boifava S, Cornara G, Della Corte F, Vivaldi N, Malacarne P, Del Gaudio P, Livigni S, Zavala E, Filippini C, Martin EL, Donadio PP, Mastromauro I, Ranieri VM. Effect of a lung protective strategy for organ donors on eligibility and availability of lungs for transplantation: a randomized controlled trial. JAMA. 2010; 304:2620-7.
16. Ranasinghe AM, Bonser RS. Endocrine changes in brain death and transplantation. Best Pract Res Clin Endocrinol Metab. 2011; 25:799-812.
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. Crit Care Med. 2012; 40:1635-44.
18. Principal Investigator: Antman, AM. "Organ Donation and Hydrocortisone Treatment (HYDRO)". Organ Donation and Hydrocortisone Treatment - Full Text View - ClinicalTrials.gov, December 2011; NCT00675272; accessed in June2015.
19. Niemann, Claus U, John Feiner, Sharon Swain, Scott Bunting, Melissa Friedman, Megan Crutchfield, Kristine Broglio, Ryutaro Hirose, John P Roberts, and Darren Malinoski. Therapeutic Hypothermia in Deceased Organ Donors and Kidney-Graft Function. N Engl J Med. 2015; 373:405-414.