

# **INTENSIVE CARE SOCIETY**

## **Guidelines for Adult Organ and Tissue Donation**

**Prepared on behalf of the Intensive Care Society by the Society's Working Group on Organ and Tissue Donation (November 2004)**

### **Chapter 5 - Clinical management of the potential heartbeating organ donor**

## 5. Clinical management of the potential heart beating organ donor

### 5.1 Pathophysiological Changes after Brain Stem Death

Widespread changes occur after brain stem death, which may jeopardise the function of potentially transplantable organs (Table 1).

Table 1. Approximate incidence of pathophysiological changes after brain stem death (%).

|                                        |    |
|----------------------------------------|----|
| hypotension                            | 80 |
| diabetes insipidus                     | 65 |
| disseminated intravascular coagulation | 30 |
| cardiac arrhythmias                    | 30 |
| pulmonary oedema                       | 20 |
| acidosis                               | 10 |

#### 5.1.1 Cardiovascular changes

There are usually two distinct phases, characterised by sympathetic overactivity and underactivity. The first phase is not seen in all patients.

- i) **Hyperdynamic phase:** Sympathetic overactivity causes a transient catecholamine surge, (particularly adrenaline and noradrenaline) which increases in heart rate, blood pressure, cardiac output and systemic vascular resistance. The catecholamine storm adversely affects the delicate balance between myocardial oxygen supply and demand.
- ii) **Cardiovascular collapse phase (Table 2):** Hypotension results from loss of sympathetic tone, profound vasodilatation and myocardial depression. Hypovolaemia secondary to diabetes insipidus (5.1.2) frequently contributes to hypotension in unsupported patients with brain stem death.

Table 2. Causes of cardiovascular collapse after brain stem death.

|                         |                                                 |
|-------------------------|-------------------------------------------------|
| Peripheral vasodilation |                                                 |
| Hypovolaemia            | diabetes insipidus                              |
|                         | osmotic diuretics (mannitol)                    |
|                         | hyperglycaemia                                  |
|                         | therapeutic fluid restriction                   |
| Myocardial depression   | depletion of high energy phosphate              |
|                         | mitochondrial inhibition                        |
|                         | possible reduction in T <sub>3</sub> production |
|                         | electrolyte disturbance                         |

Subendocardial myocardial ischaemia and ventricular dysfunction are common even in previously healthy hearts. Blood gas and electrolyte disturbances may also contribute to the ECG abnormalities which include ST segment and T wave changes, arrhythmias and conduction abnormalities. Most changes are usually temporary and reversible.

#### 5.1.2 Endocrine changes

Anterior and posterior pituitary failure causes significant reductions in the circulating levels of tri-iodothyronine (T<sub>3</sub>) and thyroxine (T<sub>4</sub>). The former may contribute to cardiovascular deterioration. Reduced production of anti-diuretic hormone (ADH) causes diabetes insipidus (DI) which occurs in up to 65% of organ donors. It is characterised by diuresis, hypovolaemia, plasma hyperosmolality and hypernatraemia. Reductions in cortisol production are unrelated to the degree of hypotension but may impair donor stress response.

Insulin secretion is reduced and contributes to the development of hyperglycaemia. This may be aggravated by the administration of large volumes of glucose containing fluids, if used to treat hypernatraemia and increased levels of catecholamines. Untreated hyperglycaemia leads to increased extracellular osmolality, metabolic acidosis, osmotic diuresis and cardiovascular instability.

#### *5.1.3 Pulmonary changes*

Pulmonary dysfunction is common in the organ donor and may be due to the development of pneumonia, aspiration of gastric contents, neurogenic pulmonary oedema or pulmonary trauma.

#### *5.1.4 Coagulopathy*

Haemostatic disorders may occur secondary to release of tissue thromboplastin by ischaemic or necrotic brain. Disseminated intravascular coagulation is common and its incidence increases with the duration of brain stem death.

#### *5.1.5 Hypothermia*

Hypothalamic failure after brain stem death results in impairment of temperature regulation. Heat production is reduced because of a fall in metabolic rate and loss of muscular activity. This is associated with an increase in heat loss because of peripheral vasodilatation. Active measures may be required to prevent hypothermia.

The clinical course of a ventilated but otherwise unsupported brain stem dead patient is short with asystolic cardiac arrest generally occurring within 72 hours. However cardiac and other body functions have been maintained for many days in fully supported brain dead patients.

### **5.2 Resuscitation and Maintenance of the Organ Donor**

Following the certification of death by brain stem testing and the lack of objection to donation, there is a change in the emphasis of care. Therapy previously aimed at preserving brain function is now directed at optimising transplantable organ function. There is no decrease in patient dependency because the need for therapeutic intervention and support of relatives continues. Adequate time must be allowed for confirmation of brain stem death but unnecessary delays should be avoided to minimise the risk of deterioration of the donor. High quality critical care including chest physiotherapy, aseptic precautions and antibiotics may be required. The patient has usually been rendered slightly hypovolaemic by brain protecting therapies. Although hypovolaemia should be corrected, it is important to avoid excessive volume replacement particularly in potential lung and heart donors. The primary goals are maintenance of adequate tissue perfusion and preservation of organ viability.

### **5.3 Monitoring the Organ Donor**

Optimal haemodynamic management requires invasive monitoring. Because of the order in which the great vessels are ligated during the donor operation, any newly placed arterial cannula should be inserted into the left radial or brachial artery. Equally a new central venous or pulmonary artery catheter (PAC) should be inserted into the right internal jugular or subclavian veins.

Intravenous fluid administration must be carefully monitored as organs, particularly the lungs, which may have been damaged during the period of sympathetic hyperactivity, are susceptible to volume overload and capillary leakage.

Echocardiography is useful to exclude major structural abnormalities of the heart and to measure left ventricular ejection fraction. Some transplant units will only request insertion of a PAC in those patients estimated to have a left ventricular ejection fraction below 45%<sup>1</sup>.

Transthoracic echocardiography (TTE) may be technically difficult and better images may be obtained with transoesophageal echocardiography (TOE). Transient regional wall motion abnormalities are common and systolic inward motion and thickening may improve with haemodynamic optimisation. Assessment of right ventricular size and function is important and frequently challenging.

## **5.4 Supporting the Organ Donor**

### **5.4.1 Cardiovascular support**

The goals of haemodynamic management are to optimise cardiac output maintaining normal preload and afterload. Where possible, the use of high dose  $\beta$  adrenoreceptor agonists, other inotropes and vasopressors, which increase myocardial oxygen demand and deplete myocardial high energy phosphates should be avoided.

The following haemodynamic goals are generally appropriate in potential adult heart donors (Table 3).

Table 3. Appropriate haemodynamic goals for potential adult heart donors.

|                        |                                                                                         |
|------------------------|-----------------------------------------------------------------------------------------|
| Mean Arterial Pressure | 60-80 mmHg                                                                              |
| Preload                | Central venous pressure ~ 4-10 mmHg<br>Pulmonary artery occlusion pressure ~ 10-15 mmHg |
| Heart Rate             | 60 – 100 beats.min <sup>-1</sup>                                                        |
| Rhythm                 | Sinus rhythm is desirable                                                               |
| Cardiac Output         | Cardiac Index > 2.1 l.min <sup>-1</sup> .m <sup>-2</sup>                                |

Patients who do not achieve these goals may still be considered for donation of other organs. These goals can usually be achieved by the standard critical care therapies (Table 4).

Table 4. Principles of critical care management for adult heart donation

| Clinical status                              | Haemodynamic management                                                                             |
|----------------------------------------------|-----------------------------------------------------------------------------------------------------|
| ↓ Mean Arterial Pressure<br>↑ Cardiac Output | i) Preload optimisation and<br>ii) vasopressor to ↑ afterload                                       |
| ↓ Mean Arterial Pressure<br>↓ Cardiac Output | i) Preload optimisation,<br>ii) Vasopressor to ↑ afterload and<br>iii) Inotrope to ↑ contractility  |
| ↑ Mean Arterial Pressure<br>↓ Cardiac Output | i) Preload optimisation and<br>ii) Vasodilator to ↓ afterload ±<br>iii) Inotrope to ↑ contractility |

The choice of inotropic support varies between transplant units and may be guided by data from pulmonary artery catheterisation but:

- High dose adrenaline may result in detrimental vasoconstriction in donor organs.
- The vasodilator effects of dobutamine may lead to undesirable hypotension and tachycardia.
- Vasopressin is less likely to cause metabolic acidosis or pulmonary hypertension and may be a more appropriate than noradrenaline for the cardiovascular collapse phase.

#### 5.4.2 Endocrine support

Hormone replacement therapy may reduce inotrope requirements and should be considered in all organ donors<sup>2</sup>. The therapies used to correct the common endocrine disturbances are shown in Table 5.

Table 5. The common endocrine problems seen in HBDs.

| Clinical problem      | Management                                                                                                                                                                                                                                                                                                                                                                       |
|-----------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Diabetes<br>Insipidus | Maintain $\text{Na}^+ = 155 \text{ mmol.l}^{-1}$ with 5% dextrose <sup>3</sup><br>Maintain urine output about $1 - 2 \text{ ml.kg}^{-1}.\text{h}^{-1}$ with vasopressin ( <i>pitressin</i> ) 1 U bolus and $0.5-4.0 \text{ U.h}^{-1}$ infusion <sup>1</sup> . If vasopressin fails to control diuresis, intermittent desmopressin ( <i>DDAVP</i> ) may occasionally be required. |
| Hyperglycaemia        | Insulin infusion to maintain plasma glucose $4-9 \text{ mmol.l}^{-1}$<br>Maintain $\text{K} > 4.0 \text{ mmol.l}^{-1}$                                                                                                                                                                                                                                                           |
| Hypothyroidism        | Tri-iodothyronine ( $\text{T}_3$ ) 4 $\mu\text{g}$ bolus then infusion at $3 \mu\text{g.h}^{-1}$                                                                                                                                                                                                                                                                                 |

Debate continues over the value of  $\text{T}_3$  replacement. Practical difficulties identifying the subgroup of patients with decreased free  $\text{T}_3$  have led to most transplant units empirically commencing  $\text{T}_3$  infusions in all potential organ donors. High dose methylprednisolone  $15 \text{ mg.kg}^{-1}$  is commonly given as part of the hormone package to diminish the inflammatory response.

#### 5.4.3 Respiratory support

If the lungs are to be transplanted, the  $\text{FiO}_2$  should be kept at or below 0.4 to minimise the risks of oxygen toxicity. A modest level of positive end expiratory pressure (PEEP) (i.e.  $<5 \text{ cmH}_2\text{O}$ ) will prevent alveolar collapse. Strict asepsis should be continued during physiotherapy and tracheal toilet. Physiotherapy should include hourly gentle inflation of the lungs and two hourly side-to-side turning. The transplant team may request an up to date chest X-ray.

Suitable goals for respiratory support are:

- maintenance of normocapnia ( $\text{PaCO}_2 \sim 5.0\text{-}5.5 \text{ kPa}$ ).
- ventilation with the lowest  $\text{FiO}_2$  to maintain  $\text{PaO}_2$  of  $>10.0 \text{ kPa}$ .
- PEEP  $> 5 \text{ cmH}_2\text{O}$  may reduce cardiac output and is rarely required.
- high inspiratory pressures should be avoided.

The mode of ventilation needs to be carefully selected:

- consider pressure control ventilation.
- modes that allow patient triggered ventilation are not appropriate.
- Very sensitive ventilatory triggers may allow cardiac cycle induced pressure changes to trigger the ventilator. This may cause diagnostic confusion by giving the appearance of a spontaneous breath.

#### *5.4.4 Renal support*

Hypotension is associated with acute tubular necrosis and failure of transplanted kidneys. Although low dose dopamine is now unfashionable in the general critical care setting, there is some evidence that donor pre-conditioning with dopamine improves initial graft function after kidney transplantation<sup>4</sup>.

#### *5.4.5 Haematological support*

The haemoglobin concentration should be maintained over  $9 \text{ gm.dl}^{-1}$ . Deranged coagulation should be treated with fresh frozen plasma and platelets. Antifibrinolytics such as epsilon aminocaproic acid may cause microvascular thrombi in donor organs and should be avoided.

#### *5.4.6 Temperature support*

Hypothermia should be anticipated and heat loss prevented by using:

- warmed intravenous fluids
- warming blankets
- heated and humidified inspired gases
- increased ambient temperature

### **5.5 Nursing and Psychological Care**

Nursing staff working in critical care areas play a key role in caring for potential organ donors and their families. The complex care required by these patients and families has been described as emotionally demanding and stressful<sup>5-7</sup>. The donor's family, friends and carers will require considerable psychological and pastoral support<sup>8</sup>. The circumstances of the donor's death may engender feelings of remorse guilt or anger. Nurses may have difficulty caring for a patient in whom death has been declared when, previously, care was directed at saving life. Explaining futility of care to families and friends is difficult. The attitudes and actions of nursing staff will significantly affect the family<sup>5-7</sup>. Therefore it is necessary for critical care units to ensure that nurses caring for these patients have a sound knowledge of:

- Organ/tissue donation, transplantation and ethical issues.
- The diagnosis of death by brain stem testing and its certification.
- The continued clinical management of the potential organ donor.
- Communication skills.
- The ability to support families during a stressful time.
- Awareness of religious and cultural issues with regard to organ/tissue donation.
- The role of donor transplant co-ordinators.

These educational elements should be included in local orientation and on going competency training in critical care. Designating an experienced 'Link Nurse' in the critical care unit, to liaise with the donor liaison nurse and donor transplant co-ordinator will assist in the delivery of such education and updates of any practice changes<sup>9-11</sup>. Donor transplant co-ordinators and donor liaison nurses can also support staff in debriefing and reviewing any aspects of individual donations<sup>6,10</sup>.

Important aspects of medical and nursing care that must be continued are described above; these will require careful explanation to families. As well as continuing to care for the patient, nursing staff will also help families understand the necessity for:

- Continued invasive monitoring.
- Adherence to infection control procedures.
- Mouth and eye care.
- Hygiene needs as required as individual to the patient.
- Regular patient repositioning.

Relatives of potential organ donors report that they derive great comfort in seeing staff maintain care and respect for their dead relative as if they were alive<sup>8,11,12</sup>.

Effective documentation and communication between medical and nursing staff is vital to ensure the families of organ donors are supported and kept well informed of the progress of events.

Staffing levels and skill mix may need to be reviewed to ensure experienced critical care nurses are available to care for the potential organ donor and their family. All aspects of care will require the full attention of a trained nurse even if the patient's physical needs are minimal, as more nursing time will be required for the family's ongoing support, explanations and discussions.

Further details about managing organ donation will be described in the UK Transplant Donor Management Guidelines which will be available in early 2005, and will be placed on the website [www.uktransplant.org.uk](http://www.uktransplant.org.uk).

## References

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