The retrieval of thoracic organs: donor assessment and management

Andrew MacLean and John Dunning
Cardiothoracic Surgical Unit, Papworth Hospital, Cambridge, UK

The optimal management of the multi-organ donor is critical to the successful outcome of transplantation. It is a complex challenge demanding careful attention to detail, and requiring a shift in emphasis, since the pathophysiological processes have far reaching effects which many clinicians do not see on a day-to-day basis. The optimal management of haemodynamic and respiratory status is essential in order to maximise the yield of suitable thoracic donor organs, yet this process will also improve the condition of other organs at the time of procurement and thus enable prompt recovery of function following hepatic and renal transplantation. The process commences when a potential donor is identified, and is only complete after successful transplantation of all possible organs. In order to achieve this end, a dedicated, multi-disciplinary team is necessary, consisting not only of medical staff, but also support workers who organise logistics, and who play their own part in vital areas, such as transport of the donor team and organs. The co-ordinator’s role is pivotal in bringing together, in harmony, teams from different centres. It is important to remember that the effort of every person involved in the management and procurement of donor organs is primarily directed towards maximisation of the donor pool, and that our main responsibility is to the recipients on our waiting lists.

When thoracic organ transplantation was introduced in the late 1960s, outcomes were initially poor and clinical potential far from clear. However, with the introduction of cyclosporin to clinical practice, the success of cardiac transplantation was changed almost overnight, and cardiopulmonary transplantation is now an extremely effective therapy for a large number of patients with heart and lung failure. One year survival of around 80% and 5 year survival of more than 60% may now be expected for recipients of heart transplants. The major problem facing thoracic transplantation is not the success of the therapy but the availability of donor organs. The number of transplants carried out worldwide has reached a plateau determined solely by donor availability. Approximately 400 heart transplants are performed every year in the UK but there are around 6000 potential recipients. Waiting lists for thoracic transplantation in the UK are kept at current levels by application of rigorous selection criteria. The shortage of donor organs is, therefore, the key crisis currently affecting thoracic transplantation.
The overall availability of solid organ donors is decreasing with better road safety and consequently fewer deaths from road traffic accidents, fewer major industrial injuries, improved emergency trauma care and improvements in the prevention and management of spontaneous intracranial haemorrhage. All of these factors conspire to make the multiple organ donor an increasingly scarce commodity. This article will describe how brain stem dead multiple organ donors can best be utilised so as to maximise organ yield. Developments in donor selection criteria will be reviewed as will the general management and support of the brain stem dead patient.

For the purposes of this article, we will refer to legal definitions and organisational issues as they apply to our own centre in the UK and, unless stated otherwise, we will refer only to our own centre’s procedures and criteria.

**Donor selection criteria**

The risk-benefit assessment for thoracic transplantation is a stark one. The life expectancy without transplant for patients in end stage heart or lung failure is low, with a poor quality of life in the interim. Patients with relatively stable, slowly progressive conditions, such as Eisenmenger’s syndrome, often have relatively good life expectancy but appalling symptoms with a poor quality of life. Transplantation is indicated primarily for symptom relief. It is against this background of imminent death and intolerable symptoms that risks of poor outcome after the use of suboptimal donor organs must be weighed. This is the impetus for expanding the criteria for donor selection. Any increase in postoperative mortality or morbidity incurred from the use of such organs must be assessed against the outcome for untransplanted recipients on an intention-to-treat basis. Additionally, cardiopulmonary transplant recipients require immediate graft function in order to support life — a position which is not mirrored in renal transplantation, where delayed graft function may be overcome with continued dialysis support.

A set of relative and absolute criteria for rejection of organs is used at our centre. The significance of each factor needs to be clearly understood, so that a balanced and integrated assessment of cumulative risk can be made when matching donors to recipients. Such a policy is inherently flexible and excludes a rigid algorithmic approach. The absolute contra-indications to organ donation (Table 1) are essentially those of potential disease transmission from donor to recipient. Other factors which are commonly misconstrued as
Table 1 Absolute contra-indications to heart and lung donation

- Blood borne sepsis
- Extra CNS malignancy
- Positive serology for hepatitis B or C
- HIV
- Direct myocardial toxicity (e.g. carbon monoxide, tricyclic antidepressants)
- Direct pulmonary toxicity (e.g. drowning, chemical injury, proven aspiration)

Absolute contra-indications to transplantation are listed in Table 2. Consideration of the likelihood of recurrence of tumours or sepsis in the recipient is fraught and a considered risk analysis often impossible. Sound evidence in support of such diagnoses as cancer or septicaemia should be sought in available case records and a careful history taken from the donor’s relatives. Frequently, however, thorough clinical examination of the donor and surgical assessment of the internal organs can help rule out such diagnoses. Infrequently, a problem arises with presumptive, radiological diagnosis of primary central nervous system (CNS) malignancy as the cause of brain death in a multiple organ donor who has a past history of non-CNS malignancy thought to have been cured. Often there is no tissue diagnosis available on the intracranial mass and, in such difficult cases, discussion with the specialist making the diagnosis, usually a neurosurgeon or neuroradiologist, as to the likelihood of inaccuracy may be helpful.

Given that the demand for thoracic organs far exceeds supply, the first principle of organ procurement must be to maximise organ yield. Rejection of donor hearts and lungs at a distance on the basis of reported physiological criteria alone is fraught with difficulties. Poor haemodynamic indices cannot be automatically attributed to irreversible organ injury. Donors are initially managed by teams inexperienced in donor care. There is good evidence that a significant proportion of donors with initially poor haemodynamic and pulmonary function can ultimately produce excellent thoracic organs. The reduced systemic vascular resistance which occurs in brain dead multiple organ donors leads to low arterial pressures that are understandably, but inappropriately, treated.

Table 2 Relative contra-indications to heart and lung donation

- Donor age
- Smoking history
- High inotrope requirement
- ECG changes of ischaemia
- Dysrhythmia
- Metabolic acidosis
- Pre-existing lung pathology
- Pre-existing cardiac pathology
with aggressive volume replacement to little avail. This does not necessarily reflect poor cardiac pump function and cardiac indices as high as 4.5 l/min/m² can be recorded in this group. Targeted haemodynamic and pulmonary assessment and intervention will frequently salvage such donors. Therefore, a low threshold for clinical review should be adopted by donor teams. Such a policy has significant resource implications. It will lead to more donor runs with a disproportionate increase in ‘empty handed’ runs. Concomitantly, there will be an increase in the total number of stressful ‘false alarms’ for recipients. However, the increase in total organ yield which such a policy provides compensates for these disadvantages.

Older donors have higher prevalences of acquired heart and lung disease, but age alone is a poor correlate of the presence of these conditions. There is clear evidence, however, that even in the absence of overt coronary artery disease there is an accelerated rate of allograft coronary occlusive disease (COD) in transplanted hearts from older donors. In International Registry figures, increasing age of donor is amongst the most significant risk factors for 1 and 3 year mortality following heart transplant. A donor age of greater than 55 years gives an odds ratio for death at 1 year post transplant of 1.75 compared to mean \( P = 0.006, \ CI \ 1.17-2.60 \)^1. This ranks among the most numerically significant risk factors predicting poor outcome in heart, and also lung, transplant. In addition, biological age is of significance in short term outcome, with most series showing marginally higher early, in-hospital mortality for older donor hearts. The exact nature of this phenomenon is unclear and multifactorial. Impaired preservation of right ventricular function and diminished ability of older ventricles to adapt to increased pulmonary vascular resistance (PVR) are implicated. However, many have shown that hearts from those donors over 55 years can be transplanted successfully and the traditional cut off figure of 40 years is certainly too conservative\(^6\). The assessment of a donor on the basis of age, in isolation from other factors, is of little value. We regularly use donors over 50 years of age and occasionally over 55 years and have no fundamental objection to even older donors in an appropriate clinical setting. There is increased acceptance of older patients for transplantation and some centres openly advocate the use of older organs for such recipients\(^7\). Additionally, the use of organs from older donors is now commonplace in both renal and hepatic transplant programmes.

Thoracic organs are matched for size primarily on the basis of height. A 10% difference between donor and recipient height is broadly acceptable. Preferably, this will be smaller when the donor is female and recipient male. Similarly, a high transpulmonary pressure gradient (TPG) in the recipient cautions against use of a smaller heart. For lung volume we use a nomogram based on a standardised formula for estimating
Table 3  Formulae used to calculate total lung capacity (TLC) for males and females

<table>
<thead>
<tr>
<th></th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male TLC</td>
<td>Height (m) x 7.8 - 7.3</td>
</tr>
<tr>
<td>Female TLC</td>
<td>(Height (m) x 7.46) - (Age x 0.013) - 6.42</td>
</tr>
</tbody>
</table>

donor total lung capacity (TLC) on the basis of height and sex (Table 3). The formulae used are based on large population studies and accurately predict healthy lung volume. Recipients are listed with both measured and estimated TLC. The effect on lung and chest wall volume of lung pathology is, therefore, apparent. Again, a potential discrepancy of 10% is accepted with matching against recipient measured TLC.

Patho-physiological changes in brain stem death and management of the donor

Brain stem death leads to irreversible organ damage by a variety of mechanisms. It is a hostile environment for all organs but, in particular, the lungs and heart which have the lowest yield from multiple organ donors. A vicious circle of organ injury is initiated by disruption of normal neural and hormonal homeostasis, consequent ubiquitous cellular dysfunction and multiple organ dysfunction. This loss of homeostasis will ultimately cause irreversible cellular injury. There is defective aerobic metabolism with a reduction of intracellular high energy phosphates, reduced levels of tissue and circulating glycogen and poor mitochondrial function. This is manifest by intracellular and extracellular acidosis, disabled cell membrane function and water and electrolyte imbalance.

In addition to fundamental cellular dysfunction, there are secondary ischaemic insults to the heart which arise from hypoxia and hypotension. Catecholamine surges lead to periods of loss of local vasomotor tone and microvascular shunting interspersed by episodes of profound coronary vasoconstriction, both of which diminish myocardial perfusion significantly. Histopathologically, the cellular injury is manifest by cardiomyocytolysis, loss of myocyte integrity and finally coagulative necrosis. At an ultrastructural level, electron microscopy reveals contraction bands, sarcomere hypercontraction and mitochondrial disruption. Clinically, these changes are manifest by haemodynamic instability, ECG changes of ischaemia, including ST depression, and T-wave inversion. The only serious negative prognostic indicator for organ donation is the development of Q-waves. Bradycardia and J-waves due to hypothermia are common and can safely be ignored.
Dysrhythmias in this setting are also common. Atrial and even ventricular dysrhythmias, in themselves, are not a contra-indication to cardiac harvest. They are frequently associated with correctable acid-base and electrolyte abnormalities. An episode of ventricular fibrillation which has been rapidly and successfully treated, in which the underlying cause addressed and good pump function maintained is not a contra-indication to cardiac harvest. However, repeated episodes of dysrhythmia which are not corrected may result in diminished cardiac output, and raised venous pressures which may injure not only the thoracic organs, but may also irretrievably damage the liver.

Regional ventricular wall motion abnormalities are well documented in ultrasonographic studies of the donor heart. Some paradoxical septal motion is common and considered insignificant. Although formal ultrasonographic assessment of the heart is strongly advocated by some centres, its benefits are to a great extent obviated by early sternotomy and visual assessment of ventricular function by the harvesting surgeon. The most common and significant wall motion abnormality encountered is right ventricular dysfunction. The requirement for good right ventricular function in the recipient is high and some contra-indicative weight should be given to a poorly contracting right ventricle which does not respond to accurate haemodynamic management.

Endocrine function is profoundly disrupted in brain death. Primary hypothalamic injury causes pituitary dysfunction and systemic endocrine disturbance ensues. Thyroid dysfunction is present. Instead of the normally predominant peripheral conversion of thyroxine (T4) to highly active tri-iodothyronine (T3), it is mainly the inactive metabolite of reversed tri-iodothyronine (rT3) that is produced with loss of normal thyroid tone. In addition, there is a reduction in the density of peripheral tri-iodothyronine receptors. To counter this sick euthyroid syndrome, we initiate an infusion of T3 early in donor management. Active management of this problem is by necessity presumptive. T3 has extra-nuclear effects on myocardicytes, and other cells. These effects are independent of secondary nuclear protein expression and are, therefore, almost immediate. The beneficial effects on cardiovascular function during the clinical use of tri-iodothyronine infusions in donors is apparent after about 15 min reflecting this effector mechanism.

Anti-diurectic hormone (ADH) practically disappears in brain stem death and this is manifest most obviously by the development of diabetes insipidus. As well as renal tubular effects, this phylogenetically diverse and primitive hormone acts on a variety of cells in synergy with other hormones. In particular, it acts with endogenous catecholamines to maintain vasomotor tone and endothelial cell integrity. ADH infusion in the organ donor will help maintain an adequate systemic vascular resistance and overcome peripheral vasodilation. It can frequently
replace noradrenaline in this role. It controls diabetes insipidus, abrogating its subsequent fluid and electrolyte management problems whilst simultaneously decreasing pulmonary and systemic capillary permeability.

Insulin and the hormones of glycaemic control are frequently disturbed. Therefore, careful monitoring of blood glucose is required, and appropriate management, with administration of glucose and insulin replacement therapy, should be initiated. In addition, loss of normal hypothalamic and pituitary function leads to a relative deficiency of corticosteroids. Early administration of high dose intravenous methyl prednisolone, or similar, is an essential feature of donor management and, in our protocol, a 1 g dose of methyl prednisolone is administered on arrival and prior to organ explant.

Vascular dynamics in brain death are profoundly disrupted. Brain stem injury causes sudden rises in circulating levels of endogenous catecholamines. These episodes known as 'autonomic storms' cause systemic and pulmonary vasoconstriction, rises in systemic vascular resistance and severe pulmonary and systemic hypertension. Vascular smooth muscle energy stores are depleted with consequent periods of profound systemic and pulmonary vasodilation and hypotension. This phenomenon is associated most strongly with brain stem 'coning'. It should not be regarded as an isolated event and recurs unpredictably. It is palliated by vigorous hormone replacement and treatment of hypotensive episodes in the donor should, therefore, be based on accurate haemodynamic diagnosis and pulmonary artery flotation catheter (PAFC) parameters. Lowered systemic vascular resistance, as alluded to above, is best managed by increasing the rate of ADH infusion, if necessary backed up by a low dose infusion of synergistic adrenalin or dopamine.

Neurogenic pulmonary oedema is a further frequent sequel to brain injury. Its origin is complex and multifactorial. Rises in systemic vascular resistance in association with left ventricular dysfunction result in increased left ventricular end diastolic pressure which may, on occasion, even exceed mean pulmonary artery pressure. In addition, a degree of functional mitral regurgitation can occur. The poor pulmonary haemodynamics are compounded by primary right ventricular dysfunction. High pulmonary capillary pressures result in fluid shifts across the alveolar–capillary membrane and subsequent oedema. In addition, there is primary pneumocyte endothelial dysfunction. This loss of alveolar integrity and capillary vascular injury causes a protein rich alveolar exudate and the tendency to oedema is aggravated by the associated systemic problems of loss of osmotic homeostasis and diabetes insipidus. In addition, it is compounded, on occasion, by the iatrogenic problems of massive crystalloid infusion and volume overload as an inappropriate
response to hypotension. The first manifestation of neurogenic lung injury is a decreasing arterial oxygen tension ($\text{PaO}_2$) in the face of a steady inspired oxygen fraction. Pulmonary dysfunction precedes radiological and clinical evidence of pulmonary oedema and $\text{PaO}_2$ is a sensitive early indicator, generally preceding drops in capillary oxygen saturation$^{24}$. Later, lung compliance will decrease, inflation pressures rise and the lungs will become heavy and ‘doughy’ on surgical inspection. Appearance of pulmonary oedema fluid in the endotracheal tube is a late and frequently irreversible development. The potential pitfall of attributing bloody endo-tracheal aspirant to pulmonary oedema is avoided by correlating this finding to other evidence and inspection of the airways by flexible bronchoscopy. Attention to an apparent need to increase the donors FiO$_2$ and frequent sampling of arterial blood gases may allow early intervention to prevent progressive pulmonary oedema.

To compound neurogenic lung injury, pulmonary complications associated with endotracheal intubation and positive pressure ventilation may occur. Aspiration at initial intubation or the initial traumatic event which generates an organ donor is not uncommon and, if significant, is a contra-indication to lung transplant. Sputum retention and basal atelectasis is inevitable in the multiple organ donor and contributes to poor gas exchange. Aggressive bronchial toilet with bronchoscopy and bronchial lavage for every potential donor will help clear mucous plugs, lower the bronchial infective load and get good samples of donor sputum for future culture. In addition, surgical inspection of the lungs can reveal dependent areas of atelectasis amenable to clearance by hand ventilation under direct observation. Many multi-organ donors reach the operating room with chronic underventilation of the lungs.

In donors dying of cranial trauma, concurrent blunt pulmonary injury is common. Direct inspection of the lungs will frequently reveal otherwise unrecognised contusion. In the absence of functional sequelae, this is rarely a problem but direct tears of the pleural surface from rib fractures will require careful assessment. The transplanted lung has a low compliance and, in the setting of positive pressure ventilation, such a tear, if large enough, can cause a significant air leak, which may be accompanied by infective complications in the immunosuppressed recipient.

Careful consideration should also be taken in the setting of multiple long bone fractures where fat embolus is common. This may not be manifest in donor gas exchange. However, our experience shows that in combination with pulmonary reperfusion injury, pulmonary fat embolus can be significant causing early organ failure. A low threshold for suspicion is necessary.
Aggressive pulmonary management will pay dividends in increased lung yield. Careful chest X-ray assessment, bronchoscopy with suction and fluid balance management guided by PAFC indices are essential. The appropriate use of diuretics and venesection, or volume repletion with blood or colloid are critical. Ventilation is carried out at a low rate, low inflation pressure (<7.5 cmH₂O) and as high a tidal volume as possible (up to 15 ml/kg). This is greatly facilitated by early sternotomy and wide sternal retraction. The inspired oxygen fraction should be kept as low as possible. Not only will this reduce the possibility of oxygen related injury to the lung during storage and transport, but often early, subtle changes in gas transfer will not be detected if the FiO₂ is high and hence an opportunity for early corrective measures will be missed.

The fundamental cellular dysfunction occurring in the multiple organ donor is manifest in other organ systems. Haematological dysfunction is common with thrombocytopenia and thrombopaeresis frequent. In addition, there is an almost invariable functional coagulopathy which may not be manifest in coagulation studies. It is apparent to the surgeons operating as a general ‘ooziness’. Losses during this prolonged procedure can be significant enough to require transfusion of clotting factors and platelets. Hypothermia as a result of poor vascular tone, peripheral vasodilation, loss of normal temperature homeostasis and a massive incision contributes to coagulopathy and is not actively treated because of its beneficial effects of lowering cellular metabolism as a contribution to organ preservation.

Ideal donor management relies on the co-operation of teams from different centres and different disciplines. Their aims, namely the highest yield of best preserved organs possible, are identical. Active haemodynamic and endocrine management based on accurate monitoring throughout the donor procedure will make the donor inherently more stable. Avoidance of the tendency to ‘cut and run’ in the face of reversible episodes of instability will aid this common aim, and physiological stability is putatively beneficial to all harvested organs, not just those most sensitive to instability. In addition, it facilitates calm and unhurried surgical dissection and cannulation.

Planning and logistics

In the UK, the use of tissues and organs for transplantation is strictly controlled by law under the Human Organ Transplant Act 1989. Distribution of organs is co-ordinated by a special health authority, the central United Kingdom Transplant Support Services Authority. Since November 1993, a zoning system has been applied to procurement of
There are a total of 9 centres for thoracic transplantation, 8 adult cardiac zones and one centre carrying out only paediatric thoracic transplantation. Six of these centres undertake pulmonary transplantation. Each centre has first refusal of donors and responsibility for organ procurement within its own zone. Should a centre have no suitable recipient for zonal organs, it will procure the donor organs and then export to other centres. Occasionally, teams will travel outwith their zone, either to Ireland or mainland Europe, or more commonly in response to overstretched logistics in a neighbouring zone. This system has the advantage of increasing familiarity with standard working practices and procedure within local donor hospitals and between the different transplant teams within each zone. It encourages the development of universal, workable and familiar management protocols for organ procurement, alleviates logistical and travel costs and increases efficiency of organ procurement as a whole. Ischaemic and procedure times for local donors are decreased. The main disadvantages occur when transporting organs from one zone to another. Preservation and procurement techniques are different between centres. Different teams for procurement and implant can impose organisational stresses and demands good communication and trust between distant teams. In the UK, there is a well established system of local donor co-ordinators, usually from a nursing background, for each region of the country. Their role includes initial data acquisition and inspection of potential donors, counselling of relatives and gaining consent for organ donation. They co-ordinate timings and organisation at the donor hospital and act as theatre and intensive care liaison throughout the donor procedure.

Our donor team consists of four members: a cardiothoracic surgeon, an anaesthetist, a cardiopulmonary perfusionist and a theatre nurse. Tight organisation ensures a 1 h call out time and a dedicated vehicle is ready with the perfusionist as a trained driver. The provision of modern cellular radiocommunications is essential for co-ordination of the run. All operative and monitoring equipment is prepacked and our aim is to be as independent of local donor hospital staff and resources as possible. In so doing, the burden on local resources is decreased and good relationships with donor hospitals nurtured.

Haemodynamic indices such as blood pressure, heart rate and central venous pressure are of little immediate value in assessing a donor on initial referral. Clearly there is a subpopulation of these patients who have sustained irretrievable cellular and organ injury, but these are uncommon and distinguished only by a trial of resuscitation. For functional assessment and management of heart donors, we have adopted a standardised rationale for characterisation and correction of haemodynamic disturbance. It is based on haemodynamic monitoring by means of an arterial line and pulmonary artery flotation catheter.
Management of the multi-organ donor

There is constant surveillance of these indices by the team's anaesthetist and surgeon. Constant adjustments of calibration and intervention are necessary and, as in other aspects of cardiothoracic surgery, demands good communications between surgeon, perfusionist and anaesthetist. A nomographic model has been developed for assessment of haemodynamics based on the following premises. The circulation consists of two pumps and resistances in series, with the same flow through each. Flow, volume, pressure and any other haemodynamic variable, measured or derived, in the system are multidimensional and interdependent. Unidimensional, single factor analysis is, therefore, inadequate and multidimensional analysis is required. Therefore, it is necessary to make two assessments of the system, before and after an intervention, to fully elucidate the true import of haemodynamic indices. An intervention can reasonably be considered in terms of preload, afterload and volume but will never act on any one of these indices in isolation. Based on these premises, a physiological nomogram which plots developed pressure (P) against cardiac output (CO) is calculated for the systemic and pulmonary resistances as follows:

Right ventricle \((\Delta P) = MPAP - LAP\)
Left ventricle \((\Delta P) = AoP - RAP\)

where \(MPAP = \) mean pulmonary arterial pressure, \(RAP = \) right atrial pressure, \(AoP = \) mean aortic pressure, \(LAP = \) left atrial pressure assumed equal to pulmonary artery wedge pressure (all in mmHg).

Lines of constancy, or isolines, are plotted for the target maximum and minimum values of each variable (Table 4) and each donor is assessed in terms of these isolines (Figs 1 & 2). The isolines for ventricular static power are referred to as power bands. In practice multiple points, before, during and after a given intervention, are plotted. In this way, the donor's cardiac function is characterised and interventions made as necessary. Figures 3 & 4 indicate the predicted effects of specific haemodynamic interventions.

**Table 4** Target maximum and minimum values for each haemodynamic variable

<table>
<thead>
<tr>
<th>Definition</th>
<th>Pulmonary circulation</th>
<th>Systemic circulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum vascular resistance</td>
<td>(\frac{\Delta P \times 79.9}{CO})</td>
<td>No minimum</td>
</tr>
<tr>
<td>Maximum vascular resistance</td>
<td>(\frac{\Delta P \times 79.9}{CO})</td>
<td>200 dynes.s cm(^{-3})</td>
</tr>
<tr>
<td>Minimum ventricular static power</td>
<td>((\Delta P - CO) \times 2.2167 = 3)</td>
<td>0.01 W</td>
</tr>
<tr>
<td>Maximum ventricular static power</td>
<td>((\Delta P - CO) \times 2.2167 = 3)</td>
<td>0.125 W</td>
</tr>
<tr>
<td>Minimum developed pressure</td>
<td>(\Delta P)</td>
<td>No minimum</td>
</tr>
</tbody>
</table>
In a functionally intact haemodynamic system with good myocardial and vascular function the following is predicted:

- adjustment of preload moves the status point across power bands at an angle and magnitude which is dependent on the functional integrity of the ventricular myocardium.
- variations in inotropes have similar effects dependent on receptor status and myocardial energy stores.
• variations in vascular impedance, in contrast, move the status points parallel to the power bands.

If dysfunction of vascular contractility or myocardial pump function occurs, then the progress of the status point will fail to comply with that predicted. In this way, specific intervention can be initiated. In practice,
the nomogramic approach is supplemented by other clinical data, in particular visual inspection of the beating heart. In addition, sudden unpredicted changes in haemodynamics, which are common during dissection and cannulation, make use of the nomogram difficult. It depends on meticulous instrument calibration and accurate measurement of the variables used together with experience in interpretation of the measured variables. The nomograms are rarely, if ever, actually plotted. However, as a model for the cardiothoracic donor team to base its overall haemodynamic management on, we find it excellent. The model's repeated, regular use provides a sound conceptual framework for the whole team and aids both consistency of management and discussion of donor cardiac function with the recipient team and senior staff.

References

4 Wheeldon D, Potter C, Oduro A, Wallwork J, Large S. Transforming the 'unacceptable' donor: outcomes from the adoption of a standardised donor management technique. J Heart Lung Transplant 1995; 14: 734-42
8 Wheeldon W, Cooper D, Novitzky D. Impairment of renal slice function following brain death, with reversibility of injury by hormonal therapy. Transplantation 1985, 41: 29-33
15 Galinaanes M, Hearse D. Brain death induced impairment of cardiac contractile performance can be reversed by explantation and may not preclude the use of hearts for transplantation. Circ Res 1992; 71. 1213-9


19 Clark R. Triiodothyronine: to be or not to be, that is the question. *Ann Thorac Surg* 1991; 51: 5

20 Davis P, Davis F. Acute cellular actions of thyroid hormone and myocardial function. *Ann Thorac Surg* 1993; 56 (Suppl): s16-s23


