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Care of the multiple organ donor

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Abstract. Successful organ transplantation offers patients with end stage organ failure the chance of a normal life. The recognition of brain death allowed the use of beating heart donors and this has enabled multiple organ procurement from a single donor. Suitable patients with severe brain injury resulting in brain death, who may be potential organ donors, are to be found on both neurosurgical and general intensive care units. The pathophysiological results of brain death are similar, irrespective of the underlying cause. Severe brain injury may result in the loss of temperature regulation, and the development of diabetes insipidus and cardiovascular instability. The management of brain injury before death often results in abnormalities of fluid balance, due to fluid restriction and diuretic therapy. Other problems such as acute endocrine failure and the impact of their correction on ultimate organ function remains to be elucidated. Good donor maintenance in the intensive care unit and operating theatre is essential if optimal function of the transplanted organ is to occur.

Key words: Organ donor – Critical care

The first successful transplantation of organs in humans took place in 1954 with the transfer of a kidney from one identical twin to another [1]. This followed the first reported human renal transplant by Voronoy in 1936 and later attempts in the early 1950s. Shortly thereafter kidneys were successfully transplanted from non-identical siblings using ionising radiation to provide immunosuppression. Since these early beginnings many different organs have been successfully transplanted. The kidney is able to withstand periods of ischaemia at body temperature for up to one hour (warm ischaemic time) and still function satisfactorily after transplantation. This allows the removal of kidneys after the circulation has ceased. The heart, liver and lungs will tolerate only short periods of warm ischaemia before irreversible cellular damage occurs. These organs must be isolated whilst the donor circulation is intact and cooled with cold perfusion fluids whilst in situ. The concept of brain stem death was coincidently becoming accepted at the same time as these needs were recognised and this has allowed the use of beating heart organ donors.

Brain death was first described by Mollaret and Goulon in France during 1959 [2] and was formally accepted in the United Kingdom (UK) during 1976 [3]. The initial aim was to to prevent the unnecessary prolongation of therapy when a hopeless prognosis exists. Since then large studies have repeatedly shown the validity of the diagnosis of brain death [4].

The success of transplantation has increased the demand for organs which has resulted in the removal of multiple organs from one donor. Studies after multiple organ procurement have shown no individual difference in organ function when compared with single organ donation [5]. It has been estimated that there are approximately 4000 potential donors per year in the UK but only 15–20% of these actually donate organs [6, 7]. These figures are derived from epidemiological studies of the incidence of subarachnoid haemorrhage and severe head injuries. They do not take into account regional variations in medical practice or pathology. In addition changes in patient admissions relating to seat belt and drunk driving legislation may have reduced the incidence of major head injury. Assertions, based upon these figures, that many potential organ donors are missed each year [8] may not be true. Studies in this centre have demonstrated that few suitable donors are lost (unpublished observations). The exact numbers of potential organ donors in the UK is

still unknown and it is hoped that a national study in 1989 will provide this information.

It is vital, for optimal organ function after transplantation, that the donor organs are kept in good condition [9] with particular emphasis placed on the maintenance of organ perfusion. Furthermore, if during life, the patient wanted to donate organs then the medical team have an obligation to ensure that organs are in the best possible state for the recipient.

The diagnosis of brain death

In the UK the donor must have satisfied the brain stem death criteria as defined by the Medical Royal Colleges in 1976 [3]. Other countries in the western world have set similar criteria. In the UK these criteria are based on clinical findings [10–12] and do not include electroencephalograph (EEG) examination and cerebral blood flow measurements that are mandatory elsewhere [13, 14]. The causes of death in beating heart donors during one year (1987), as reported to the United Kingdom Transplant Service, are shown in Table 1.

Tests for the criteria in the UK must be performed by 2 consultants or a consultant and senior registrar, who must be clinically independent and unconnected with the transplant team. The diagnosis must be certain, the patient having suffered severe and irreversible brain damage, the aetiology of which must be known and be totally dependent upon artificial ventilation. If any doubt exists then the diagnosis of brain death must not be made. Hypothermia, metabolic and endocrine abnormalities should be excluded. Prolonged drug action including alcohol should always be considered and can only be excluded by the passage of time. Measurement of plasma concentrations of sedative and analgesic drugs has been shown to correlate poorly with central effects [15] and cannot be relied upon. The tests used to confirm brain death are summarised in Table 2 and must be repeated by the same two doctors. The exact timing of the second set will vary according to the clinical condition of the patient and may be up to 24 h from the first set. Inevitably some patients will become asystolic whilst the diagnosis is awaited but may be suitable for cadaveric organ donation if consent has been granted.

Table 1. The causes of death in beating heart organ donors during 1987 (Figures supplied by the United Kingdom Transplant Service)

Subarachnoid haemorrhage	215
Cerebrovascular accident	14
Intracerebral haemorrhage	120
Head injury (road traffic accident)	188
Head injury (other causes)	94
Road traffic accident (associated injuries)	59
Cardiac arrest	18
Brain tumour	13
Meningitis	10
Asthma	11
Asphyxia	11
Respiratory arrest (other causes)	3
Anoxia/hypoxia	17
Overdose	6
Others	36
Total	815

Table 2. Clinical criteria for diagnosing brain death in the United Kingdom

Known cause of irreversible and severe brain injury
Absence of hypothermia, electrolyte and endocrine abnormalities
No residual sedative drug effects
No pupillary response to light
Absent corneal reflex
Absent caloric responses
No motor response within distribution of cranial nerves
No gag or bronchial reflex
Apnoea in the presence of adequate $PaCO_2$ (6.65 kPa/50 mmHg)

Prolonged elimination of drugs such as the benzodiazepines and opiates in critically ill patients is increasingly recognised [16–19]. The pharmacokinetic and pharmacodynamic effects of hypotension, hypothermia and endocrine failure on sedative and analgesic drugs has not been studied in the brain dead patient, but delayed drug elimination must be considered. In addition 6–10% of the population have been demonstrated to have pharmacogenetic abnormalities leading to delayed drug metabolism [20, 21]. The use of the specific opiate and benzodiazepine antagonists naloxone and flumazenil has been proposed as a test to exclude drug accumulation.

Improvement in conscious level, as shown by the return of reflexes or purposeful movements within the cranial distribution, after the administration of these antagonists disproves brain death. Conversely, with the lack of current clinical information, no improvement cannot guarantee lack of residual sedative effects. If there is doubt about the presence of sedative drugs then the diagnosis of brain death cannot be made.

Adverse effects of the two antagonists on haemodynamics and intracranial pressure make their use in the brain injured patient dangerous [22, 23]. If they are to be used then this should be postponed until all other tests have demonstrated brain death. A peripheral nerve stimulator should be used if the patient has renal failure and muscle relaxants have been used to exclude prolonged myoneural blockade.

Ventral pontine infarction ("the locked in syndrome"), idiopathic polyneuritis (Guillain-Barre syndrome), and brain stem encephalitis have been listed as conditions mimicking brain death [24]. The "locked in syndrome" is characterised by retained consciousness,

spontaneous respiration and vertical eye movements [25]. Polyneuritis has a characteristic history and presentation, consciousness is not lost although all muscle groups may be paralysed. Brain stem encephalitis is characterised by rousable stupor and retained purposeful limb movements [26]. All these conditions differ from the common causes of brain death (traumatic head injury, intracranial haemorrhage and cerebral hypoxia) in that they lack a defined severe cerebral injury as stipulated in the critera. Furthermore, if a careful clinical examination is performed patients with these conditions will not fulfill the criteria either.

Time of death in the UK is legally defined as the time when the second set of tests has confirmed brain death. Details of testing are best documented on a single form, variations of which are available in many hospitals.

Consent to organ donation

Consent may have been granted before death occurred and this will usually have been recorded on a donor card. If the patient carried a donor card there is no legal requirement to discuss consent with the relatives but it is usual and recommended practice to do so. Alternatively permission may be obtained from relatives. This is best discussed with them after the first set of tests has been performed. Waiting until after the second set of tests has been completed can result in unnecessary distress for the relatives and delay in obtaining the organs. When brain death and organ donation are being discussed with relatives the concept of the beating heart donor should be clearly explained. This will avoid the possibility of future distress should the media hightlight the issue at a later date.

If there are no relatives, the Hospital Administrator (as the legal possessor of the body) may grant permission to donate organs. Where the nature of the patient's death requires statutory reporting, the consent of the Coroner, Procurator Fiscal or Medical Examiner may need to be obtained. This should also be sought after the first set of tests to avoid later delays.

Both good and bad publicity have surrounded the practice of organ donation leading to fluctuations in supply but there is still a large deficit of donor organs. Many people wish their organs to be donated after death but may not communicate this to their next of kin. In The UK an "opting in" system is practiced in the form of widely available donor cards. These were at one time issued as part of the driving license. This latter practice has been withdrawn to comply with European Economic Community (EEC) regulations. Donor cards have not been successful due to low acceptance rates by the public or the cards not being carried or lost at the time of accident or hospital admission. "Opting out" procedures have been implemented in some countries where the individual have to register on a central computer that they do not wish to donate organs.

"Required request" has been introduced as federal law in many states in the USA [27, 28] but there is no scientific evidence that the supply of donors has increased as a result. This legislation requires that the physician looking after a potential organ donor discusses the possibility of organ donation with the relatives. A compromise has been suggested in the form of "required discussion" where physicians must discuss potential donors with the local transplant team who could then approach the relatives [6].

At present in the UK it is usually a senior member of the medical team looking after the patient who asks for consent. They should have previously met the relatives and discussed the poor prognosis. The majority of relatives will gain some comfort out of the act of donating organs and this provides some relief from an otherwise tragic situation [29], some may even feel aggrieved if not approached about donation. A voluntary group "BODY" (British Organ Donor Society[1]) has recently been set up in the UK to offer help to relatives in these situations. Transplantation programmes are more successful in children than adults but are hampered by the lack of donors in children under five years. Children may cause particularly strong emotions both in favour and against organ donation.

Organ retrieval

The UK Transplant Service (UKTS) in Bristol maintains a computerised record of all patients in need of organ transplantation and acts as a coordinating centre for the use of organs. Local organisation of the surgical organ retrieval is performed by regional transplant coordinators. The different organs retrieved from a single donor may be used in several different centres around the UK. Organs are also exchanged throughout the EEC. Within Europe there are a number of different coordinating organisations including Eurotransplant (the Benelux countries, FRG and Austria), France-Transplant, Scandia Transplant, Swiss Transplant, North Italy Transplant, Barcelona Transplant and Luso Transplant (Portugal). The activities of these organisations have been reviewed in depth [30].

Unavoidable delays at the transplantation centre may be seen as procrastination by the donor hospital and may lead to frustration. These delays may be caused by the difficulties in organising several retrieval teams simultaneously and the frequent shortage of

[1] "BODY", Balsham, Cambridge CB1 6DL, England, Tel.: (0223) 893636

ICU bedspace for the recipient. Early referral of potential donors may reduce later delays to a minimum.

Emotional differences may exist between the medical personnel looking after the potential donor who feel their therapeutic efforts have failed and the members of the transplant team who are encouraged by the opportunity to help another patient. These differences have, on occasions, led to misunderstandings and both groups need to appreciate the other's feelings if these problems are to be prevented in the future.

Criteria for organ donation

A few absolute contraindications apply to all potential donor organs including Hepatitis B infectivity, the demonstration of antibodies to human immune deficiency virus (HIV), other known viral infections, a history of intravenous drug abuse, malignancy (apart from primary central nervous system (CNS) tumours) and concurrent bacterial sepsis (Tables 3 and 4). The potential donor who is known or suspected of being an active promiscuous homosexual should be carefully considered. It is possible to be infective with HIV despite negative serology (see below). The decision to use organs in these situations can only be resolved by individual discussion of each case with the surgeon reponsible for the recipient operation. Age, diabetes mellitus and the presence of other disease processes are relative contraindications. Some organs which do not fulfil the usual criteria may be used if there is a desperate need, such as in a patient with fulminant hepatic failure.

Kidney donors should have a urine output greater than 0.5 ml/kg/h and normal plasma urea and creatinine concentrations. However, kidneys have been sucessfully transplanted after a period of anuria or oliguria in the donor [31].

The potential heart donor must be carefully assessed for ischaemic and other cardiac disease. The history, clinical examination, chest X-ray and ECG examination must all be within normal limits. No period of prolonged cardiac arrest should have occurred and the heart should not require significant inotropic support. Most heart transplant units will not accept donors over the age of 50 years because of the high incidence of unrecognised ischaemic heart disease.

Lung donors require special attention. Pulmonary gas exchange must be good and a fractional inspired oxygen concentration of less than 30% inspired oxygen, to achieve normal arterial oxygen tensions, is required. The trachea should ideally have been intubated for less than 24 h because of the rapid bacterial colonisation of the airways in artificially ventilated patients. The match of donor lung size to that of the recipient pleural cavity is important and guidelines exist for measuring the lung size from the chest X-ray. These are available from transplant coordinators.

Table 3. General criteria for organ donation

<70 years	
Free from transmissable disease:	bacterial
	fungal
	protozoal
	viral infections
Hepatitis B antigen negative	
HIV antibody negative	
No widespread atherosclerosis	
No trauma, infection or chronic disease in organ to be transplanted	
Free of malignant disease except primary CNS	

Table 4. Specific criteria for individual donor organs

Corneas	<90 years
	No past history of intraocular disease or surgery
	May be removed up to 12 h after cessation of the circulation
Kidneys	<70 years
	No history of renal disease
	Adequate renal perfusion
	Adequate urine output[a]
Liver	<55 years
	Satisfactory donor height, weight, abdominal girth
	Liver function tests normal
	No alcohol abuse
Heart	<50 years
	Satisfactory donor height, weight, chest circumference
	Chest X-ray and ECG normal
	No long period of cardiac arrest
	High dose inotropic support not required
Heart/lung	As for heart plus:
	No pulmonary trauma or infection
	Artificial ventilation less than 24 h if possible
	Good gas exchange required $F_IO_2 < 30\%$
	Satisfactory thoracic measurements for recipient
	Non-smoker
Heart valves	<60 years
	No history of valve disease
	May be removed up to 12 h after cessation of the circulation
Pancreas	<50 years
	Normal plasma amylase
	No family history of diabetes mellitus

[a] In certain instances kidneys can be removed from donors who are anuric/oliguric

Donor transmitted disease

Transmitted bacterial infection should be avoidable by careful screening of donors for clinical and laboratory signs of sepsis and the use of routine prophylactic broad spectrum antibiotics during organ procurement. Viral and protozoal infections are more of a problem due to their silent carriage in donor organs, and once recognised, the lack of effective drugs for their treat-

ment. Screening for hepatitis and HIV is routinely performed in potential donors. However, using currently available assays, seroconversion occurs several weeks after primary infection. Antibody titres against HIV may be diluted by massive blood transfusion and blood products may themselves transmit infection to the donor. Testing the blood samples originally sent to the transfusion laboratory for crossmatching when the patient was first admitted may be helpful in such cases. Thus tests failing to demonstrate antibodies to HIV in donors cannot exclude HIV infection. Both HIV [32] and Hepatitis B [33] have been transmitted via transplanted organs and are likely to run a fulminant course in the immunosuppressed patient.

Cytomegalovirus and toxoplasmosis gondii both exist in latent forms in the normal population and may be reactivated in the immunosuppressed patient, particularly in the presence of other infections [34–36]. Both organisms may be transmitted to seronegative recipients via infected donor organs or blood transfusions and subsequently cause a primary infection. In the immunosuppressed patient both organisms may give rise to life threatening infections. Heart and heart/lung recipients appear particularly at risk from these organisms; some transplant units routinely screen all donor organs for them and then avoid giving seronegative recipients seropositive organs or blood [37]. Pyrimethamine may be given prophylactically to prevent toxoplasmosis gondii infection in mismatched donors [38].

Donor maintenance

After the criteria for brain stem death have been satisfied and consent obtained, patient care becomes donor maintenance and there is a change in emphasis from cerebral to donor organ protection. Failure to ensure that the organs are in optimal condition on removal may result in graft failure or malfunction in the recipient. Special problems arise when managing these donors, due to the nature of the severe intracerebral damage and resultant disturbances of cardiovascular function, temperature regulation and diabetes insipidus.

The cardiovascular system

Hypotension is a common finding in the brain dead patient. The vasomotor centre is damaged in common with the rest of the brain stem resulting in progressive vasodilatation. Dehydration from fluid restriction and diuretic administration is a recognised treatment for cerebral oedema and may result in hypovolemia. Myocardial function has also been shown to deteriorate in the brain dead baboon with increasing anaerobic metabolism [39]. Bradycardias are common in the presence of severe cerebral injury due to loss of sympathetic drive. The destruction of the nucleus ambiguus in the brainstem abolishes resting vagal tone, therefore atropine fails to reverse bradycardia in this situation and this has been used as a diagnostic test for brain stem death [40, 41]. Bradycardias continue to respond to sympathomimetic drugs which act directly on beta-adrenergic receptors in the heart such as isoprenaline.

The first step in the correction of hypotension is to expand the intravascular volume using the measurement of central venous pressure (CVP) as a guide to adequate replacement. Urine output and core to peripheral temperature gradients are additional useful guides in this situation. Blood losses should be replaced with whole blood or packed cells to maintain a haematocrit of 30%. This haematocrit maximises oxygen supply by optimising the balance between oxygen transport by the red blood cell mass and blood flow related to changes in viscosity [42]. The choice of other fluids for the correction of hypovolaemia is controversial, particularly when large volumes need to be given quickly to resuscitate a hypotensive donor. At this centre a modified gelatin solution is used but it remains to be determined whether crystalloid or colloid solutions are better in this situation. If a low blood pressure persists after correction of hypovolaemia the circulation may be supported by infusion of an inotropic agent. Dopamine is currently the most popular drug because it causes renovascular dilatation at doses up to 5 μg/kg/min. Higher doses of it and other inotropes lead to progressive renal and systemic vasoconstriction. Drugs with predominantly vasoconstrictor properties (eg. aramine, ephedrine, metaraminol) should only rarely be required if the above steps are followed. Their inappropriate use may cause splanchnic vascoconstriction reducing liver and kidney perfusion. With worsening brain damage it may well become impossible to maintain an adequate circulation [4]. The blood pressure should not be considered in isolation, as a maximally vasodilated circulation may provide good organ perfusion despite low measured blood pressures.

Fluid balance

Fluid restriction and diuretic therapy are routine practice in many units looking after acute neurological injuries. Diabetes insipidus, glycosuria (due to steroid therapy), and hyperthermia (before brain death) are also common and increase water losses. When assessing organ donors, fluid intake and losses should be calculated for the previous period of intensive care. Clinical assessment of skin turgor and mucous membrane hydration, together with measurement of

peripheral temperature, urine output and central venous pressure and the laboratory estimation of plasma urea and electrolytes and haematocrit guide fluid replacement.

The urinary losses in diabetes insipidus should be replaced by 5% dextrose with added potassium [43] or preferably with a solution based on the measured urinary losses of electrolytes. The inappropriate use of 0.9% sodium chloride or plasma expanders containing 0.9% sodium chloride over a period of days may lead to progressive hypernatraemia.

Temperature control

Extensive damage to the brain stem causes loss of the normal central control of body temperature, the body effectively becoming poikilothermic. Without control of the temperature by passive warming the donor temperature will fall to that of its environment. Hypothermia is harmful as it causes progressive vasoconstriction and cardiac instability as the core temperature falls. Monitoring of body core temperature should be carried out and steps taken to conserve heat. Intravenous blood and fluids should be warmed, inspired gases should be heated and humidified, the donor should be placed on a warming mattress and covered by reflective insulating blankets.

Endocrine failure

The incidence of posterior pituitary failure manifest by diabetes insipidus is high in brain death [44]. Autopsy findings in such cases have shown necrosis, infarction or oedema of the pituitary as a consequence of the initial injury [44]. Polyuria results from lack of antidiuretic hormone with excretion of large volumes of dilute urine and this needs to be replaced on an hourly basis if marked fluid depletion is not to occur. Fluid therapy is discussed elswhere in the text. A diuresis greater than 150 ml/h in an adult can be controlled using vasopressin or its synthetic analogues. Early use of vasopressin considerably simplifies the fluid management of these patients. Vasopressin may be effectively given as intramuscular injections or a low dose infusion of 1–2 units per hour [45]. The synthetic form dDAVP is more potent as an antidiuretic, has a longer duration of action and has less vasoconstrictor properties.

It would be surprising if anterior pituitary function was not damaged in a similar fashion to posterior pituitary function when brain death occurs and this has been confirmed experimentally in animals with brain stem injury [39]. Studies in humans have been less clear. Hall et al. [46] measured thyroid stimulating hormone, prolactin and cortisol in 5 patients with brain death and could demonstrate no abnormality except loss of the diurnal cortisol variation. Novitsky et al. [47] measured triidothyronine (T3), insulin and cortisol in 21 brain dead patients and found a decreased T3 and a low normal cortisol and insulin concentration. On the basis of this and their animal work they gave T3, cortisol and insulin to all their subsequent organ donors and appeared to have less cardiovascular and metabolic problems in them, compared to historical controls who did not receive hormone therapy. This study was both uncontrolled and retrospective with the attendant methodological problems but does indicate the need for further studies in this area.

Protection of organ function

In addition to the maintenance of adequate blood pressure, cardiac output and the avoidance of vasoconstriction specific protective treatments for individual organs are used. Renal protection is thought to be aided by low dose dopamine (2 μg/kg/min), mannitol infusions and frusemide (10 mg intravenous boluses). Most transplantation units give mannitol as a bolus of 20 g over 30 min immediately before kidney removal. Adequate flushing of kidneys before removal is important to wash out residual red cells. Handling of the renal vessels during donor nephrectomy may precipitate vascular spasm. This spasm has been reduced in experimental models by the administration of chlorpromazine, phenoxybenzamine, phentolamine, verapramil and prostaglandins [48]. There is a lack of comparative clinical studies for all these treatments and little information regarding other organs like the heart and liver.

There have been major advances in artificial organ preservation fluids for use immediately before organ removal and afterwards during storage. Many are still being investigated with different units using varied solutions. All are based on cold solutions, containing sugars as an impermeant and usually having a high potassium content. Together these reduce cellular swelling and metabolism. There has also been considerable interest in continuous hypothermic perfusion of organs in vitro. These topics have been reviewed in depth elsewhere [48, 49].

Ventilatory support

Continued artifical ventilation is necessary in the organ donor. The ventilator should be adjusted to give a $PaCO_2$ of 5.3–5.6 KPa and added oxygen given to maintain PaO_2 greater than 10 KPa. Oxygenation may be a problem if aspiration of stomach contents, acute lung injury, neurogenic pulmonary oedema or traumatic damage have occurred and arterial blood gases should be measured frequently. Positive end ex-

piratory pressure (PEEP) should not be used unless there are problems with oxygenation not responsive to increases in inspired oxygen concentration. PEEP increases mean intrathoracic pressure leading to decreases in thoracic venous return and a fall in cardiac output and renal blood flow. Other humoral mechanisms including release of antidiuretic hormone (ADH) and activation of the renin/angiotensin/aldosterone system have also been implicated [50]. PEEP should be avoided in the presence of haemodynamic instability and when not indicated specifically to improve PaO_2. Carbon dioxide production is low in the absence of cerebral blood flow, sympathetic drive and muscle tone [51, 52]. Low minute volumes or the addition of a dead space to the ventilator circuit may be necessary to maintain normocapnia.

Lung transplantation requires special consideration. Oxygen at unnecessarily high concentrations (greater than 60%) should be avoided due to the risk of pulmonary oxygen toxicity. Low pressures of PEEP (5 cm water) are routinely used in these cases to preserve lung volume by preventing alveolar collapse. Salt and water overload must be avoided. The lungs must not become infected and "aseptic techniques" of tracheal suctioning continued.

The donor operation

Reflex movements, particularly in the limbs, may occur following stimuli in the presence of brain death. The intact lower spinal cord retains certain spinal reflexes which may give rise to muscle spasms and twitches on stimulation. Superficial reflexes are more common than deep muscle reflexes [53]. No reflexes occur in the distribution of the cranial nerves. Anxieties may arise as to the validity of brain stem death criteria, unless these changes are anticipated and understood by the attending staff. True decerebrate or decorticate posturing implies intact pathways within the brain and is not seen in brain stem death. Tachycardia and hypertension may also occur after surgical incision. The neuronal pathways for these haemodynamic responses are not clear but may reflect a spinal vasoconstrictor response or a humoral mechanism such as adrenal medullary stimulation by a spinal reflex [54].

Tubocurarine is often recommended for abolishing muscle movement but usually causes a drop in blood pressure due to histamine release and ganglionic blockade. Pancuronium or vecuronium, more cardiostable muscle relaxants are a better choice, given the high incidence of hypotension in donors.

The question of the use of analgesic and anaesthetic agents is difficult. Their use depresses the possibly harmful tachycardia and hypertension related to surgical incision, otherwise their use is illogical in a brain stem dead donor. Nevertheless they are used by some experienced anaesthetists. The addition of volatile anaesthetic agents, in anaesthetic concentrations, to the inspired gases, overcomes some of the objections to organ donation expressed by some clinicians but others would regard this as an illogical position (D. Hill, personal communication). Most anaesthetists continue to use nitrous oxide as a carrier gas to avoid the administration of 100% oxygen from anaesthetic machines without a supply of compressed air.

Exact operative details differ between centres [55] and the procedure described is that used at this centre [56]. If all transplantable organs are to be removed the chest and abdomen are opened with a long midline incision from the jugular notch to the symphysis pubis. After a thorough inspection to exclude an unsuspected disease all organs are dissected out until attached only by their vascular pedicles. This dissection may take up to three hours, the most frequent delay being due to the presence of accessory vessels. The liver is dissected first followed by kidneys and pancreas.

When dissection is completed heparin is given intravenously to avoid coagulation around perfusion cannulae (15,000 units in an adult). Cannulae are then placed in the lower abdominal aorta, the inferior vena cava, and the portal vein in preparation for cold perfusion of the abdominal organs. The heart is perfused first with cold cardioplegic solution via a cannula in the aorta and fluid is vented by incising the superior pulmonary vein. Artificial ventilation is then discontinued. The liver is then perfused with ice-cold Ringer's lactate followed by 4.5% albumin solution. The kidneys are perfused with ice-cold Marshall's hypertonic citrate solution.

After cold perfusion the organs are removed and put in sterile bags and transported packed in ice to the recipient. Kidneys may be stored for up to 48 h (although organ survival after 72–96 h has been documented), livers up to 10 h, and heart and heart/lungs 4 h using these techniques. However, recent advances in preservation fluids may allow longer times in the future. Successful organ retrieval requires close cooperation between the different surgical teams from different centres. Efforts are being made in some areas to organise and train one surgical team to remove all organs rather than different teams each removing one organ.

Losses of fluids by bleeding and evaporation from an open abdomen and chest during the dissection phase are significant. Blood transfusion may be required during this dissection phase and blood should be crossmatched in anticipation.

Hypothermia may be a problem in the operating theatre when the donor has an open chest and abdo-

men. Open body cavities give rise to large heat losses from evaporation and radiation. Heat losses should be minimised by a warm theatre, warming all infused fluids, using heated humidifiers on ventilator circuits and using warming blankets beneath the donor.

Conclusions

The demand for organ donors is likely to increase each year and is likely to outstrip available supplies in the foreseeable future. Every effort should be made both to encourage organ donation and when offered, the donor organs should be kept in the best possible condition. This will mean allocation of medical and nursing time, plus resources similar to that given to other intensive care patients. Further studies need to be performed to identify all the consequences that result from brain death, in order that these may be corrected before organ removal.

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