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Chapter 5
The Pediatric Heart Donor: Evaluation of Age, Size, Cause of Death, Donor Heart Function, and Vitality

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The shortage of organ donations is one of the main limitations to the application of pediatric heart transplantation to end-stage pediatric heart disease [1,2]. In the United States, pediatric donors have continued to make up a declining percentage (14%) of all organ donors during the past decade [3]. Because of the shortage of pediatric donors, especially in the younger age groups, many of the efforts to increase graft availability have focused on increasing public awareness of organ donation. There also, however, are opportunities to increase the recovery of acceptable cardiac allografts by focusing on donor management and organ selection. Recent guidelines, developed at the 2001 conference on Maximizing Use of Organs Recovered From the Cadaver Donor, include more aggressive strategies for maximizing the use of available cardiac donors [4]. Improved management of potential donors based on a better understanding of the complex pathophysiology of brain death should result in improved quality of donor organs. Finally, developing a better understanding of the factors that make a donor heart acceptable (or not) should increase the recovery of cardiac allografts from potential donors.

ABO COMPATIBILITY

The reference standard in cardiac transplantation has been to match recipients with donors who have a compatible ABO blood type. This standard has been challenged in the last decade by the success of ABO-incompatible heart transplantations in infants [5–7]. Serum titers of anti-A and anti-B antibodies usually are low below the age of 12 months, and studies have shown that infant recipients of ABO-incompatible donor hearts fail to produce antibodies against the donor blood group. This approach, which is detailed in Chapter 4, may improve the use of available infant donor hearts significantly and may reduce the higher mortality among infants on the waiting list.

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SIZE MATCH

In adults, donor–recipient size matching generally allows about a 30% mismatch in weights, with successful transplantation occurring with both undersize and oversize hearts. In children, the problem of donor shortage is compounded by the need to divide the already small pediatric donor pool into multiple smaller pools based on size. Thus a strategy of using oversized organs in pediatric recipients has been advocated by some centers, which report good success. In most patients who have dilated cardiomyopathy, a larger allograft can be accommodated easily, although this may not be the case for patients with hypertrophic or restrictive cardiomyopathy. In addition, there are special situations, such as in patients who have elevated pulmonary vascular resistance, in which planned use of an oversized heart may reduce the risk of right-ventricular failure after transplantation.

In a review of 69 pediatric heart transplant recipients from Loma Linda, Fullerton and colleagues [8] examined the effect of heart donor and recipient size mismatches. Donor-to-recipient weight ratios ranged from 0.48 to 3.09. There was a 75% incidence of transient lobar or complete lung collapse in recipients who had high donor-to-recipient weight ratios, but there was no increase in the number of days of artificial ventilation, nor did size mismatch have any effect on left-ventricular function. In a follow-up study from the same center, Razzouk and colleagues [9] examined 291 pediatric patients who underwent transplantation for congenital heart disease and found that posttransplantation morbidity and survival were not affected adversely by the use of oversized allografts, defined as a donor–recipient weight ratio greater than 2.5. Use of oversized donor organs did result in a greater incidence of delayed chest closure in the early postoperative period (28% for those with a ratio > 2.5, versus 8% for those with a ratio < 2.5). Others have shown that the use of oversized donor hearts transiently influences left-ventricular remodeling after heart transplantation. In a study of 20 pediatric recipients, Kertesz and colleagues [10] found that left-ventricular mass index was increased 2 weeks after transplantation in patients who had a donor–recipient weight index greater than 1.2. Left-ventricular mass gradually regressed, however, until it reached normal values by 12 months after transplantation. Recent analysis of the International Society for Heart and Lung Transplantation (ISHLT) database (Fig. 1) [11] suggests that a donor–recipient weight ratio of less than 0.5 and more than 2.5 is associated with a significantly increased risk of 1-year mortality (relative risk, 1.5; $P = .004$) in pediatric heart transplant recipients.

DONOR AGE

In children, advanced donor age is an issue mainly for adolescent candidates who can match by size with older adult donors. In these adolescent recipients, older donor age has been shown to be a significant risk factor for 1-year mortality after heart transplantation [11,12]. In an analysis of 79 pediatric heart transplants at Stanford [12], children who received a heart from a donor over age 40 years had a 1-year survival of 20%, compared with 78% among

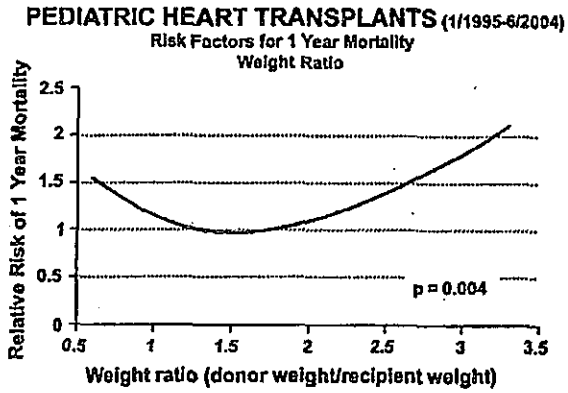

Fig. 1. Relative risk for 1-year mortality after heart transplantation for variations in donor/recipient weight ratios in pediatric heart transplant recipients in the ISHLT database.

those who received transplants from donors aged less than 40 years. The major cause of death in recipients of transplants from older donors was graft failure during episodes of acute rejection. Multivariable analysis of risk of death was significantly attributable only to the age of the donor. These authors also reviewed 267 adolescent recipients in the United Network for Organ Sharing (UNOS) database. One- and 2-year survival was 58% and 44%, respectively, for recipients of transplants from donors older than 40 years ($n = 12$), versus 85% and 79% for recipients of transplants from younger donors ($n = 255$). Based on the results of this study and a confirmatory review of data from the ISHLT [11], UNOS rules were changed so that adolescent recipients currently receive priority over adults when an adolescent donor heart is available [13].

DONOR GENDER

Although studies in adults have shown differences in posttransplantation outcome based on donor gender and donor–recipient gender match, donor gender did not influence survival in younger male recipients (those under 45 years of age) or female recipients of hearts from either gender [14]. In contrast to adults, donor gender seems not to be a risk factor in children, confirming the age-specific risk found by Al-Khaldi and colleagues [14]. In 152 pediatric transplants reviewed by Leman and colleagues [15], gender matching was not a predictor of posttransplant survival. This finding was confirmed by a review of more than 6000 pediatric heart transplants in the 2006 pediatric report of ISHLT [11].

DONOR-HEART ISCHEMIC TIME

Based on studies in the early days of heart transplantation, the standard acceptable donor ischemic time (DIT) traditionally has been in the range of 4 hours.

In pediatric transplantation, the difficulty of obtaining pediatric donors has led many centers to consider the use of donor organs with longer ischemic times [16–19]. In a study from Columbia, survival at 1, 5, and 10 years was not affected by DIT exceeding 4 hours [20]. Kawauchi and colleagues [17] studied 93 pediatric heart transplantations at Loma Linda with DIT ranging from 51 minutes to as long as 8 hours 17 minutes. Although there were differences in diastolic function at 1 week after transplantation, these differences had resolved by the second week. There was no correlation between DIT and primary graft failure, duration of inotropic support after transplantation, and long-term cardiac function. Outcome did not differ between 14 patients who had DIT greater than 8 hours and 14 patients who had short ischemic time (< 90 minutes) [21]. The 2006 pediatric report of the ISHLT, consisting of more than 6000 patients, also confirms that DIT was not associated with an increase in 1-year mortality [11], at least in the range of ischemic times observed. In contrast to these studies, Huang and colleagues [22] did find a relationship between donor ischemic time and primary graft failure in a study of 165 pediatric transplant recipients from Washington University in St. Louis. Ninety percent of patients who had primary graft failure survived with aggressive management, so DIT had no overall effect on mortality. With current methods of donor heart preservation, current practices reflect the general opinion that ischemic time up to 6 hours, and possibly 7 hours or more is probably safe, depending in part on additional factors such as pulmonary vascular resistance, complexity of reconstruction at implantation (warm ischemic time), and the degree of destabilizing bleeding in complex reoperations.

CAUSE OF DONOR BRAIN DEATH

Causes of donor death in the pediatric population are quite different from those in adults [1,23,24]. Domestic trauma and asphyxia are the most common causes of death in pediatric donors, whereas road traffic trauma and cerebrovascular accidents are more frequent in adults. Data from the Pediatric Heart Transplant Study indicate that closed head trauma as a cause of donor death produces a greater likelihood of stable graft function in infant heart transplant recipients [25]. Few cases of the use of hearts from anencephalic donors have been reported [23,26,27]; the limited clinical experience indicates that these organs may have good function despite some morphometric differences from normal newborns [11]. In anencephalic neonates, however, the diagnosis of death is often impossible to achieve with current methodology for determining brain death [28,29].

Analyses of sudden infant death syndrome and anoxia as causes of infant death have yielded conflicting outcomes following infant heart transplantation [22,23]. It seems likely that the severity and reversibility of the anoxic cardiac insult (as reflected, for example, by higher troponin I levels) [30] are the major determinants of subsequent graft function.

Brain death is associated with many complex physiologic derangements, including hemodynamic, neuroendocrine, and metabolic alterations. In one review, 97% of donors required vasopressor support, 55% developed a coagulopathy, 46% diabetes insipidus, 30% cardiac ischemia, 25% lactic acidosis, 20% renal failure, and 13% acute respiratory distress syndrome [31]. Whether brain death caused by gunshot injury represents a risk factor remains controversial. Gunshot injuries often result in increased neuroendocrine stress and resultant myocardial dysfunction associated with a rapid increase in intracranial pressure. Karamlou and colleagues [32] speculated that the observed higher incidence of rejection and infection following heart transplantations from donors with fatal gunshot head injuries resulted from a combination of endothelial damage and up-regulation of proinflammatory pathways in the donor secondary to the rapid increase of intracranial pressure. In contrast, Hokl and colleagues [33] found troponin T levels and requirement for inotropic support were lower in donors who had died from cranial trauma than in those who had spontaneous intracranial hemorrhage.

In pediatric recipients, Odim and colleagues [34] examined the duration of donor brain injury as a potential risk factor for death after transplantation. Recipients of allografts with long periods from brain injury to declaration of brain death or from death to organ removal had significantly improved freedom from rejection but no difference in overall survival.

Finally, controversy exists regarding transmission of central nervous system malignancy from donor to recipient. Hornik and colleagues [35] examined the outcome of 32 heart transplantations performed with organs from donors who had primary brain malignancies. Unlike reports in the renal and hepatic transplantation literature, there were no cases of donor-transmitted malignancy in these heart recipients.

DONOR INFECTIONS

Hepatitis B

Donor hepatitis serologic testing includes hepatitis B surface antigen (HBsAg), hepatitis B core antibody (anti-HBc IgM and anti-HBc IgG), and hepatitis B surface antibody (anti-HBsAb). Individuals who have recovered from natural hepatitis B infection will be anti-HBc IgG positive, and 95% will also be anti-HBsAb positive. In contrast, those who have received the hepatitis vaccine will be only anti-HBsAb positive, and core antibody will not be present. The issue of hepatitis B donor hearts can be separated into those donors who are HBsAg positive and those donors who are anti-HBsAb positive but HBsAg negative (see Mawhorter and Avery for an excellent review of this issue) [36]. Wang and colleagues [37] examined the outcomes of 32 donor organs that were HBsAg positive, indicating the presence of active infection. When HbsAg-positive organs were placed into HbsAg-positive recipients, all recipients (n = 4) had hepatitis flare-ups after transplantation, requiring treatment. When HbsAg-positive organs were placed into anti-HBsAb–positive recipients, however, none (n = 26) developed hepatitis. These authors concluded that organs from donors who are positive for HBsAg may be used for critically

ill status 1 recipients who are anti-HBs positive. Vaccine-acquired immunity reduces the risk, but natural immunity may be even better [36].

Because hepatitis is a chronic infection, donors who are anti-HBc positive are still capable of transmitting hepatitis to heart transplant recipients. Pinney and colleagues [38] found that transplantation of 33 hearts from anti-HBc–positive donors was associated with a small risk of transmission of virus but with equivalent posttransplantation survival. De Feo and colleagues [39] reviewed 25 heart transplantations and found no evidence of viral transmission. Haji and colleagues [40] examined the relationship between hepatitis B seropositivity and the development of graft coronary artery disease. When either donor or recipient was seropositive for anti-HBc, the incidence of graft coronary disease at 1 year was increased (46%, versus 24% in the seronegative group). In a review for the UNOS database, Babcock and colleagues found that anti-HBc IgG–positive/anti-HBc IgM–negative/HBsAg-negative donors were generally safe for heart transplantation with a low risk of disease transmission (W.D. Babcock, personal communication, 2006). Recipients receiving hearts from hepatitis-positive donors should be informed of the donor serology and counseled regarding the risks and benefits of receiving that organ. Finally, hearts from donors who received the hepatitis B vaccine (anti-HBs positive/anti-HBc negative) cannot transmit active infection, because the vaccine uses recombinant surface antigen only.

Hepatitis C

Hepatitis C serology consists of anti-HCV IgM, anti-HCV IgG, and hepatitis C virus-RNA polymerase chain reaction (PCR). There is a high rate of seroconversion (ranging from 25%–82%) in recipients of organs from hepatitis C–positive donors, so these organs usually are turned down outright. In extraordinary cases, an organ from a hepatitis C antibody–positive/PCR-negative donor could be used for a critically ill recipient [41].

West Nile virus

Although there are reported cases of transmission of West Nile virus from an infected donor to solid-organ recipients [42–46], routine donor screening for this virus is currently not performed. A known infection would be a contraindication to organ donation. Still, a high index of suspicion for West Nile virus should be maintained in any recipient who develops encephalitis after transplantation or blood transfusion.

Toxoplasmosis

If the donor is toxoplasmosis IgG positive but IgM negative, transplantation is acceptable. If the recipient of that organ is toxoplasmosis IgG negative, the patient should start either trimethoprim/sulfamethoxazole or pyrimethamine prophylaxis. If the donor is toxoplasmosis IgM positive, serum should be sent to a national reference laboratory to determine whether the donor is infected.

HIV

Routine donor testing for HIV is performed by ELISA. A donor whose HIV ELISA is positive is retested using Western immunoblotting. If the Western blot is positive, organ donation is contraindicated.

DONOR SUBSTANCE ABUSE

Alcohol or intravenous cocaine abuse in the donor may impair ventricular function and reduce survival after transplantation. In adults, the incidence of substance abuse in donors is substantial. Freimark and colleagues [47] found a history of chronic alcohol abuse in 17% of adult heart donors and a history of nonintravenous cocaine abuse in 16% [48]. Alcohol abuse was an independent risk factor for death after transplantation. One-year survival was 61%, versus 95% in recipients of organs from donors without alcohol abuse, mostly because of the development of severe ventricular dysfunction during episodes of rejection [47]. In contrast to intravenous cocaine abuse, which is a contraindication to organ donation, a history of nonintravenous cocaine abuse in the donor was not associated with increased risk after transplantation [48].

DONOR RESUSCITATION TIME, HEART FUNCTION, AND CATECHOLAMINE REQUIREMENT

Cardiac function often is depressed after brain death, related to a combination of brain injury–induced neurohumoral storm and exposure to exogenously administered catecholamines. Cardiac function in the donor heart usually is assessed by echocardiography, although dramatically altered loading conditions may complicate this assessment. Additional studies that may be useful include 12-lead ECG and serum enzyme markers (creatine phosphokinase-MB fraction and troponin). Tissue Doppler imaging may become a valuable tool for assessing load-independent donor cardiac function [49], although there currently are no controlled studies of its sensitivity and specificity. In addition, for adult male donors over age 40 years and for female donors over age 45 years, coronary angiography usually is performed in the donor hospital, if feasible.

The use of high-dose inotropic support in the donor is not an absolute contraindication for heart donation but must be placed in context of other factors such as length of donor cardiopulmonary resuscitation, ejection fraction, age of donor, presence of elevated pulmonary vascular resistance, and complexity of the recipient operation. Use of inotropic support in adult donors has been associated with an increase in 1-year mortality, independent of any effect on ejection fraction [50]. Using load-independent indices of right-ventricular function, Stoica and colleagues [51] compared the function of hearts from recipients who were receiving low- or high-dose norepinephrine with those who were not receiving norepinephrine. All hearts showed subclinical decreases in indices of right-ventricular contractility, and those indices were lower in patients receiving norepinephrine. Recipient survival at 1 year was lower in patients who had depressed right-ventricular function.

Donor resuscitation time is another potential risk factor, although studies in children have not shown an influence on posttransplantation survival. In a review of 165 pediatric patients, Huang and colleagues [22] found that increased donor cardiopulmonary resuscitation time predicted a slightly but not significantly increased risk of primary graft failure (odds ratio, 1.1; $P = .1$), although there was no increase in mortality. In addition, there was no relation between greater donor inotropic requirement and the occurrence of primary graft failure. De Begona and colleagues [52] compared 72 pediatric recipients who received donor hearts not subjected to cardiopulmonary resuscitation with 68 patients whose donors had cardiopulmonary resuscitation for a mean of 18.8 ± 14.6 minutes, the longest time being 60 minutes. Early cardiac function, the number of days on assisted ventilation, and the amount of inotropic support were not different in the two groups. Systolic and diastolic left-ventricular function also were not different at 2 years.

DONOR HEART DISEASE

Simple congenital heart lesions in the donor heart, such as atrial septal defect, have been routinely repaired at the time of transplantation or afterwards using a transcatheter approach [53]. This concept has been expanded recently to include repair of ventricular septal defects [54] and mitral valve abnormalities [55,56]. More significant congenital heart lesions generally are considered a contraindication to organ donation.

Pre-existing coronary artery lesions sometimes are found at the time of donor angiography (in the case of an older donor) or at the time of first posttransplantation angiography. Li and colleagues [57] examined the influence of pre-existing donor coronary atherosclerosis on the development of adult-recipient graft coronary artery disease. Although the specific sites with donor lesions did not show an increase in intimal area by intravenous ultrasound when compared with nonlesion sites, the overall incidence of graft coronary artery disease was substantially higher (25% versus 4%) in grafts with pre-existing coronary lesions than in those with normal coronaries. Despite this finding, 3-year mortality was not different.

USE OF NONBEATING HEARTS

Some have argued that organ availability could be increased if organs could be used from donors who had nonbeating hearts (grafts removed after asystolic cardiac arrest). In a review of the records of local organ procurement organization in Philadelphia, Singhal and colleagues [58] screened for potential donors in which the time between withdrawal of care to cross-clamp of the aorta could have been under 30 minutes. Of 119 potential donors with nonbeating hearts, 82 met this time frame criterion, and 20 met all other criteria for potential donation. Thus, 12% to 18% of potential donors with nonbeating hearts would be acceptable heart donors, representing a 4% to 6% increase in the total number of transplantations that could have been performed in that region during the 3-year time frame of the study. The utility of organs from donors with nonbeating hearts in pediatrics has yet to be examined.

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