Review Article

Paediatric cardiac catheterization: innovations

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In recent years interventional procedures have been introduced to the field of paediatric cardiac catheterization. These procedures continue to develop in complexity and increasingly are being applied to patients with reduced cardiovascular reserve, as an alternative to cardiac surgery or when cardiac surgery with cardiopulmonary bypass is contraindicated. More frequently anaesthetists are being called upon to provide support in sedating, anaesthetizing or/and resuscitating these patients. The purpose of this review is to give a comprehensive update of the interventional procedures and to review the anaesthetic management techniques as they apply to the catheterization laboratory. We will discuss possible complications and management strategies from our own experience and the experience of others. We have observed that as more complicated procedures are performed the anaesthetist plays a pivotal role in the management of the patient from arrival to departure from the cardiac catheterization laboratory, and in preventing mortality and major morbidity. Although the economic consequences of interventional cardiological techniques remain unclear, the field continues to expand and more complex procedures are continually being introduced.

Depuis peu, on réalise chez l'enfant des interventions par cathétérisme cardiaque. Ces interventions sont de plus en plus complexes et sont pratiquées sur des patients dont les réserves cardiaques sont diminuées, à la place de la chirurgie ou lorsque la circulation extracorporelle est contre-indiquée. On fait de plus en plus appel aux anesthésistes pour procurer à ces patients

la sédation, l'anesthésie et la réanimation. Ce survol a pour but de faire le point sur ces interventions et de passer en revue les techniques de gestion anesthésique telles qu'elles s'appliquent à un laboratoire de cathétérisme cardiaque. Nous examinerons les complications possibles et les stratégies de gestion à partir de notre expérience et de celle des autres. Nous avons observé que lorsqu'il s'agit de prévenir la morbidité et la mortalité, le rôle central de l'anesthésiste s'accroît proportionnellement à la complexité de l'intervention et depuis l'arrivée du patient en salle de cathétérisme jusqu'à son départ. Bien que les conséquences économiques de ce type d'intervention soient encore mal connues, ce champ d'activité est en pleine expansion et des techniques encore plus complexes font régulièrement leur apparition.

Key words

ANAESTHESIA: paediatric, cardiac;
HEART: catheterization, complications.

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Over the years the worldwide prevalence of congenital heart disease (CHD) and the relative frequency of the various congenital cardiac lesions has remained stable.^{1,2} Overall, approximately one percent of live births make up the population of patients with CHD. In North America, CHD accounts for almost all heart disease in children. The New England Infant Cardiac Program reports that, excluding premature infants with patent ductus arteriosus, three infants of every 1000 live births will need cardiac catheterization and/or surgery and/or will die from CHD in early infancy and many at some later time.²

The use of cardiac catheterization and angiography as a diagnostic tool for patients with CHD was first reported in 1947 by Bing *et al.* who described the haemodynamic data of 48 patients with cyanotic CHD.³ The idea that a catheter could be used for intracardiac therapy was introduced in 1964⁴ with the dilatation of acquired peripheral vascular stenosis by graduated rigid dilators. However, the balloon atrial septostomy developed by Dr. Rashkind and reported in 1966⁵ was the first paediatric and the first intracardiac transcatheter procedure to be used as a routine form of therapy. This procedure rapidly became the standard emergency therapy for the newborn with transposition of the great arteries.

Echocardiography as a diagnostic tool in evaluating congenital cardiac lesions has resulted in a decrease in the number of diagnostic cardiac catheterizations. However, the total number of catheterizations is steadily increasing because of the introduction of interventional cardiac catheterization procedures (Figure 1). Currently at The Hospital for Sick Children in Toronto and Children's Hospital in Boston 45–55% of all cardiac catheterizations are interventional. Patent ductus arteriosus is now closed routinely at cardiac catheterization.⁶ As experience with interventional cardiology increases, more complex procedures are performed on cardiovascularly unstable patients. Vascular stents are placed to reinforce the dilated vessels particularly in patients with peripheral pulmonary stenosis. Umbrella occlusion devices are used to close patent ductus arteriosi, atrial and ventricular septal de-

FIGURE 1 The increase in the total number of cardiac catheterizations is directly related to the increase in interventional procedures. (Courtesy Dr. M. Freed.)

FIGURE 2 The dramatic increase in electrophysiological ablation procedures from 1985 through 1992 is directly related to the advent of intracardiac catheter ablations (Courtesy Dr. E. Walsh.)

fects. It is possible that these devices may prove a satisfactory alternative to surgery in patients where surgical repair is difficult or contraindicated.

Patients with CHD, with or without surgery, may develop rhythm problems as their survival increases. This will dramatically increase the number of patients requiring electrophysiologic evaluation. While surgical treatment of arrhythmogenic foci or accessory tracts is possible, less invasive catheter ablation techniques may replace the surgical approach (Figure 2).⁷

The purpose of this review is to discuss the procedures currently performed in the catheterization laboratory, their potential complications, recommendations regarding their anaesthetic management and economic considerations for these new techniques.

Cardiac catheterization for haemodynamic assessment

Although many infants with CHD may be fully evaluated by noninvasive techniques such as echocardiography, select patients are taken to the catheterization laboratory for further haemodynamic assessment and/or anatomical definition. Femoral vessels, in both infants and children, are usually entered percutaneously employing the Seldinger technique.⁸ Alternatively, jugular and/or subclavian veins may be cannulated.

The "normal" mean right atrial (RA) pressure is approximately 3 mmHg. The left atrial (LA) tracing is very similar to the RA tracing in that it has the same three characteristic waves. The characteristics of the aortic trace will vary at different locations. The pulse pressure will widen as more distal measurements are obtained.⁹ In the patient with normal architecture of the pulmonary circulation, a pulmonary artery wedge pressure may be obtained to estimate left atrial pressure. This is widely utilized in the evaluation of patients who have pulmonary artery catheters in place.¹⁰ The left atrial pressure can be measured directly by advancing a needle across the atrial septum. This is referred to as a Brockenbrough procedure.¹¹ During catheterization, pressure gradients across valve and outflow chambers are also assessed.

Examples of haemodynamic abnormalities

The RA pressure may be increased by large left-to-right shunts at the atrial level, any ventricular to atrial communication as may occur in an endocardial cushion defect, abnormalities of the tricuspid valve that produce stenosis or regurgitation, and a decrease in right ventricular compliance. The LA pressure will be elevated due to volume overload or mitral valve disease, whether the valve is regurgitant or stenotic. A reduction in left ventricular compliance may also be reflected by an increased LA pressure. The left atrium is less compliant than the right atrium and, therefore, the left atrial pressure changes tend to be more dramatic than RA pressure changes.

By definition a nonrestrictive ventricular septal defect (VSD) will produce equalization of right ventricular (RV) and left ventricular (LV) pressures and the waveforms will be nearly identical.

Elevated pulmonary artery pressures may be caused by a number of aetiologies ranging from pulmonary vascular disease to anatomical pulmonic stenosis in the main or branch pulmonary arteries. High flow states as seen

in non-restrictive ventricular septal defects can also cause pulmonary hypertension.

In the evaluation of patients with pulmonary hypertension it is important to know if the pulmonary hypertension is reversible before undertaking corrective surgery. Oxygen is a potent vasodilator and pulmonary artery pressures are obtained first while breathing room air and then 100% oxygen. If the pulmonary artery pressure decreases in response to 100% oxygen, then the vessels are reactive. Nitric oxide, a direct-acting pulmonary vasodilator, may also be administered in known concentrations while serial pulmonary artery pressures and resistances are obtained. If the pulmonary artery pressure and resistance decrease, the pulmonary hypertension is in part reversible.¹² Such reversibility is important in the determination of the suitability of the patient to undergo many procedures.

Most patients with CHD have some form of shunt communications, either intracardiac or extracardiac. The most common method of calculating shunt involves the measurement of oxygen saturations in various chambers/vessels of the heart. Spectrophotometers measure the absorbance of oxyhaemoglobin (wavelength = 600 nm) and deoxyhaemoglobin (wavelength = 506.5 nm). The respective concentrations are calculated and the portion of haemoglobin that is saturated is reported as a "percent" measurement. This newer method of measuring oxygen saturation directly is not affected by alkalosis, hypothermia or fetal haemoglobin.¹²

The Fick formula is used to determine pulmonary blood flow ($\dot{Q}_p$) and the systemic blood flow ($\dot{Q}_s$). the ratio of $\dot{Q}_p$ to $\dot{Q}_s$ determines if there is a shunt and the direction of the shunt. A $\dot{Q}_p/\dot{Q}_s$ of 1:1 is seen in an anatomically normal heart (no shunts present) in which the right-sided output equals the left-sided output. A $\dot{Q}_p/\dot{Q}_s > 2$ denotes a large left-to-right shunt and a $\dot{Q}_p/\dot{Q}_s < 1$ a right-to-left shunt.^{9,12,13} The values obtained for $\dot{Q}_p$ and $\dot{Q}_s$ can then be used to calculate pulmonary vascular and systemic vascular resistances using Ohm's Law.^{8,10,12}

The vast majority of diagnostic, non-interventional haemodynamic studies are performed while the patient is sedated. This is preferred as the sedated patient's haemodynamic values more closely approximate the awake values than those obtained with the child anaesthetized.

To obtain adequate conditions with sedation without producing airway compromise or hypoventilation, both of which will produce respiratory acidosis and artificially elevated pulmonary artery pressures and resistances, can be a challenge. As both FiO_2 and PCO_2 as well as positive-pressure ventilation influence right-sided pressures, general anaesthesia is avoided when possible in di-

agnostic haemodynamic catheterizations but may be necessary for the safe management of selected patients.

Cardiac catheterization for interventional procedures

Interventional cardiac catheterization procedures such as umbrella device insertion, embolization of vessels using thrombogenic coil devices, and balloon valvuloplasty have proved to be a considerable advance in the treatment of congenital heart defects. In some lesions, such as a PDA or secundum ASD, placement of an umbrella device is curative and avoids the need for surgery. Other interventions such as embolization of abnormal vessels using the thrombogenic coil devices and some VSD device closures are performed as part of a staged approach to management of more complex defects. Valvuloplasty will reduce ventricular or atrial outflow obstruction, although it is often a temporizing measure with definitive surgery required later.

Umbrella occlusion device insertion

Initially described for PDA closure by Rashkind in 1979,¹⁴ the device consisted of a six-arm single umbrella with "fish hooks" at the end of three of the arms. A large catheter size (15F) was needed to accommodate this device. Modifications lead to the development of a spring-loaded double umbrella, connected back to back with four arms on each umbrella covered by a fabric mesh (Figure 3).¹⁵ The double umbrella is designed to allow each umbrella to fold back against each other, using spring tension when opened to fix the device against a vessel wall or septum (Figure 4). This allowed the size of the delivery pod to be reduced to 11F. The fabric mesh becomes closed by clot and subsequently endothelializes.

The device, currently awaiting final FDA approval, has been used to close both atrial and ventricular septal defects successfully, as well as patent ductus arteriosi.^{16,17} Once the long sheath containing the delivery device and folded umbrella is positioned across the defect, the sheath is gently withdrawn allowing the distal umbrella arms to protrude and open fully. Once stable, the sheath is further withdrawn allowing the proximal arms to release and open. Before the device is finally released from the delivery sleeve, the arms are checked to insure correct position and stability to reduce the risk of device embolization, obstruction to outflow or entrapment of valve leaflets.

Passage of guide wires and large-bore sheath across valve leaflets and the atrial or ventricular septum may cause severe haemodynamic disturbances. This is less of a problem during PDA or ASD closure, but is a particular concern when VSD devices are placed. The size of the sheath limits the use of this device to patients

FIGURE 3 The double umbrella device demonstrating spring arms and fabric mesh.

FIGURE 4 The initial placement of the distal umbrella against the distal side of the septum (shown) is followed by withdrawal of the delivery catheter until the proximal umbrella is seated against the proximal side of the septum. The device is then released and the delivery catheter is withdrawn.

over 5 kg, and can lead to considerable blood loss and possible air embolism with frequent wire and catheter changes.

PATENT DUCTUS ARTERIOSUS

This was the first congenital defect to be corrected surgically and the first to be treated using a percutaneous technique. With refinement of the device and technique, transcatheter closure is a reliable and safe option for PDA closure for most patients outside the neonatal period (Fig-

FIGURE 5 The femoral approach is demonstrated for placement of the patent ductus arteriosus umbrella. In frame A, the umbrella's distal arms have been released into the aortic side of the patent ductus (arrow). In frame B the proximal arms have been released from the delivery pod which remains attached to the umbrella to retrieve it should it be malaligned. An aortic injection has been made to assess adequacy of device placement. In frame C the device has been released from the delivery pod. Aortic injection demonstrates no residual ductal flow.

ure 5). It is a relatively brief procedure, is associated with minimal haemodynamic disturbance and can be performed on an outpatient or short-stay basis.¹⁸

General anaesthesia is used infrequently as intermittent sedation will usually suffice. Patients need to be immobile when the device is being released from the sheath to ensure a stable position; extra sedation with a bolus dose of ketamine, 1 to 2 mg · kg⁻¹, may be required at this time.

ATRIAL SEPTAL DEFECTS

Umbrella devices have been used to close secundum atrial septal defects^{17,19-21} (Figure 6) and fenestrations within an atrial baffle following modified Fontan procedures.²² As with PDA occlusion, ASD closure is associated with few haemodynamic problems and can be performed as an outpatient or on a short stay basis.¹⁹ General anaesthesia is not routine unless transoesophageal echocardiography is necessary to assist in positioning the device, if associated cardiac defects are present or if there is a history of previous surgical repair, with limited haemodynamic reserve. Hypotension and rhythm disturbances are uncommon and transfusion is rarely required. Sedation along with local anaesthesia to the groin, is usually satisfactory, but patients must be still during device placement and occasionally a bolus of ketamine (1-2 mg · kg⁻¹) *iv* is necessary at this time.

Complications related to device placement include atrio-ventricular valve regurgitation resulting from the arms of the device impinging on valve leaflets, air embolism via the large delivery sheath and device embolization that may require surgical removal if transcatheter retrieval is not successful.

VENTRICULAR SEPTAL DEFECTS

In contrast to our experience with closure of PDA's or

FIGURE 6 The femoral approach is demonstrated for placement of the ASD umbrella.

ASD's, transcatheter VSD device closures are prolonged procedures, often associated with profound haemodynamic instability and blood loss with intensive care management frequently required following placement.²³ Close liaison between the anaesthetist and cardiologist is essential.

The indications for VSD device placement include closure of: (1) residual defect; (2) recurrent defect;²⁴ (3) preoperative closure of defects in patients with associated complex congenital heart defects who have muscular VSD's that may be difficult to locate on cardiopulmonary bypass and contribute to a significant shunt and volume load on the systemic ventricle,²⁵ and (4) closure of acquired defects such as post-myocardial infarction or trauma.²⁴

The technique of transcatheter VSD closure is shown schematically in Figure 7. Muscular VSD's are frequently multiple, may run obliquely through the septum and have more than one opening into the right ventricle due to

FIGURE 7 The VSD is initially crossed with a balloon catheter and wire from within the left ventricle either passed retrograde across the aortic valve (A) or antegrade across the mitral valve (not shown). The wire is passed into the right ventricle from where it is snared (B) and brought outside the body. For apical or mid-muscular VSDs the snare and wire are brought out from the internal jugular vein as the approach is straighter and the sheath is less likely to kink. The sheath, delivery pod and folded umbrella device are passed over the guide wire and across the VSD from within the right ventricle (C). For anterior and high defects the wire is snared and brought out the opposite femoral vein.

TABLE I

Diagnosis	Number	Mean age (mos)	Mean weight (kg)	Duration (min)	Transfusion ($\text{ml} \cdot \text{kg}^{-1}$)	CICU (admits)
Group 1	24	165 $\pm$ 99	36 $\pm$ 19.5	339 $\pm$ 96	10.8 $\pm$ 2.9	15
Group 2	23	26.5 $\pm$ 12.1	11.8 $\pm$ 3.2	310 $\pm$ 94	21.8 $\pm$ 11.1	9
Group 3	19	47 $\pm$ 37	14.8 $\pm$ 7.9	278 $\pm$ 66	20.4 $\pm$ 7.8	7
Group 4	4	538 $\pm$ 327	70 $\pm$ 2.5	245 $\pm$ 72	0.0 $\pm$ 0.0	4

Group 1, recurrent or residual VSD.

Group 2, primary muscular VSD.

Group 3, VSD closed preoperatively.

Group 4, acquired VSD after infarction or trauma.

septal muscle trabeculation. It is therefore easier to cross the VSD initially with a catheter and guide wire from within the left ventricle. However, the device is positioned from the right ventricle side of the septum to avoid the problems associated with the large bore delivery sheath across the aortic and mitral valves.

After positioning the sheath and delivery device, the umbrella is released slowly, allowing the distal arms to open on the LV side of the septum and the proximal arms on the RV side. Transoesophageal echocardiography is frequently used to determine the position of the device arms, evaluate residual shunting and atrio-ventricular valve function.

At Children's Hospital, Boston, we have reviewed the

charts and catheter data for transcatheter VSD device closure procedures over an 18-month period. Eighty-six devices were inserted in 70 patients. Seventeen patients required more than one device (Table I).²³

The median time for these procedures was 305 min (range 127 to 580 min). Transfusion was required during 38 procedures (54.5%) and the amount transfused was much greater in patients <10 kg.

During 28 procedures (40%), hypotension (>20% decrease in systolic blood pressure from baseline) occurred; 12 responded to volume replacement alone, whereas 16 required additional acute resuscitation with inotropes (13), vasopressors (6), transvenous pacing (3), or cardioversion (2) due to sustained hypotension. During four

procedures external cardiac massage was required. However, there were no deaths in this series.

Serious arrhythmias associated with hypotension and desaturation required treatment or catheter withdrawal during 21 procedures (30%). Ventricular arrhythmias were common with ventricular tachycardia (VT) occurring in six patients, requiring treatment with lidocaine (5) or cardioversion (1). Severe junctional bradycardia or complete heart block was induced on nine occasions, requiring treatment with transvenous pacing (3) and isoproterenol (1).

Factors contributing to haemodynamic instability include preexisting patient condition, blood loss, arrhythmias from catheter manipulation in the ventricles and across the septum, atrio-ventricular or aortic valve regurgitation from stenting of valve leaflets by the stiff-walled catheters, and device related factors such as malposition of the umbrella with arms impinging on valve leaflets or dislodgment from the ventricular septum.

Fifty percent of the patients were admitted directly to the cardiac intensive care unit (CICU) because of haemodynamic instability, procedure duration or concern over device position; 24 of these patients required mechanical ventilation.

Initially, patients undergoing these procedures were managed with sedation but, as our experience with these procedures has increased, general anaesthesia has replaced sedation because of: (1) associated haemodynamic instability; (2) TEE being required to assist device location; and (3) insertion of the introducer sheath via the internal jugular vein for apical VSD closure necessitating intubation and control of the airway.

Coil embolization

Systemic arterial-to-pulmonary artery collaterals vary in size, but may result in a considerable left-to-right shunt. Certain groups of patients, such as those with severe pulmonary atresia, may have large collaterals supplying lung segments or lobes; these vessels are suitable for unifocalization. Before surgery, thrombogenic coils can be embolized into smaller vessels that are not suitable for unifocalization (Figure 8) to prevent excessive pulmonary flow to lung segments and problems with aortic run-off from the aorta to the pulmonary circulation during cardiopulmonary bypass. The coils are inserted through catheters in a manner similar to umbrella devices and are coated with a thrombogenic material and, once introduced into the blood stream, rapidly induce clot formation occluding the vessel. When coils are placed in a large number of collaterals, particularly if the collaterals supply a large amount of lung, the decrease in left-to-right shunt may result in arterial haemoglobin desaturation.

FIGURE 8 Embolic coils are available in a variety of shapes and sizes which must be carefully chosen for the vessel being occluded. They are coated with thrombogenic material to induce rapid clot formation.

Venous collaterals may develop following single ventricle repairs and contribute to desaturation from an effective right-to-left shunt. Coiling of these vessels cause little haemodynamic change. General anaesthesia is rarely necessary for embolization, but may be considered if the procedure is likely to be prolonged due to multiple embolizations, or if the limits of sedation have been reached. Haemodynamic disturbances are rare.

Balloon dilatation

PERCUTANEOUS BALLOON VALVOTOMY

Percutaneous balloon valvotomy has been applied to patients to relieve congenital and acquired stenosis of aortic,²⁶⁻³¹ pulmonary^{26,32,33} and mitral valves.^{26,34,35} Congenital stenosis results from abnormal cusp development with leaflet thickening and commissural fusion while acquired stenoses are usually the result of leaflet calcification and fusion. Balloon valvotomy is usually palliative, improving antegrade flow and ventricular performance in the short to intermediate term, thereby avoiding the need for surgical intervention and CPB in infancy. For some patients balloon valvotomy may be curative requiring no further intervention, e.g., isolated congenital pulmonary valve stenosis.²⁶

On most occasions haemodynamic catheterization is performed prior to balloon valvotomy. Most often these procedures are performed with the patient sedated, breathing spontaneously and the catheter insertion site anaesthetized with local anaesthetics. This allows for more accurate assessment of transvalvar gradient and valve regurgitation. Once data are obtained, tracheal intubation and controlled pulmonary ventilation may be imple-

mented because of the potential haemodynamic disturbances associated with valve dilatation, particularly with aortic and mitral valves.

Balloon size and inflation pressure vary. The balloons are composed of latex and are inflated using a radio-opaque dye. The balloons are inflated to the prescribed pressure, noting the presence of a central "waist," which disappears at maximal balloon pressure. The balloon is rapidly deflated giving an inflation/deflation cycle of less than ten seconds: this may be repeated. Balloon rupture is not uncommon during dilatation of resistant stenosis but rarely produces intimal damage or embolic phenomena.

Aortic valve

Percutaneous balloon dilatation is used to treat critical aortic stenosis in neonates and in older children with progressive congenital or acquired aortic stenosis (AS). Valvotomy usually provides short to intermediate term palliation, with up to 70% of patients requiring re-dilatation after eight years (Dr. P. Moore, Children's Hospital Boston, personal communication). Studies in older children have demonstrated a successful reduction in peak systolic gradient of between 45 and 65%, with persistence of this initial fall up to 12 months post-dilatation.^{28,29} The valvotomy balloon should be 90–100% the diameter of the aortic annulus to reduce the risk of regurgitation.²⁶ The cardiac output immediately decreases during inflation causing hypotension, bradycardia and a transient left bundle branch block. On most occasions blood pressure and heart rate recover spontaneously provided preload has been maintained.

Neonates with critical AS present soon after birth with cardiac failure, low cardiac output, severe acidosis and collapse. Concentric hypertrophy may be extreme with a small LV cavity and subendocardial fibrosis. These neonates have a ductus-dependent circulation requiring PGE₁, inotropic support and mechanical ventilation. Balloon dilatation is performed once the patient is resuscitated and stable, but still carries severe risk. Preload must be maintained, excessive tachycardia avoided and any decrease in afterload avoided. They require close monitoring in the catheterization laboratory, and general anaesthesia with muscle relaxation and ventilation. To reduce the stress response and work imposed on the ventricle, synthetic opioids such as fentanyl and sufentanil are given. Potential complications during dilatation include sudden death from acute coronary ischaemia, arrhythmias during dilatation, including asystole, and severe aortic regurgitation following dilatation which may require surgical treatment.^{26,27,30} Infants presenting with failure to thrive, tachypnoea and cardiac failure from severe AS undergo semi-elective balloon dilatation. These

patients are usually more haemodynamically stable than are the neonates and less likely to have complications during dilatation. However, the risk of severe regurgitation remains. These patients are often initially managed with sedation with provision for immediate intervention if there are haemodynamic complications.

Pulmonary valve

Historically, the pulmonary valve was the first valve to be balloon-dilated and experience with this technique is now extensive. It is an effective method of relieving RV outflow obstruction and the treatment of choice for pure pulmonary valve stenosis.³²

Neonates with critical pulmonary valve stenosis are severely cyanosed at birth, with a ductus-dependent pulmonary blood flow requiring PGE₁ infusion and often mechanical ventilation. While dilatation is frequently successful in these patients, systemic saturations may remain low initially post-dilatation requiring continued prostaglandin infusion and ventilation until the RV pressure and pulmonary vascular resistance fall. The antegrade flow across the valve prior to dilatation is often small such that cardiac output is little affected when the balloon is inflated for short periods, occluding the RV outflow.³³

Neonates and infants with less critical stenosis, who have some antegrade flow across the pulmonary valve, undergo balloon dilatation to improve forward flow and relieve RV hypertension. When the balloon is inflated the cardiac output decreases dramatically and hypotension and bradycardia may result as pulmonary blood flow ceases. The end-tidal PCO₂ decreases and arterial haemoglobin desaturation may occur. The circulation usually recovers spontaneously. Older patients with pulmonary valve stenosis may have significant RV hypertension and hypertrophy, but also tolerate balloon valvotomy well. Most of these cases can therefore be performed under sedation alone.

Mitral valve

Mitral valve stenosis in children in the United States is rare, and when it occurs is usually congenital in aetiology. Mitral valve dilatation has been applied to patients with congenital mitral stenosis as well as patients with rheumatic mitral disease. Mitral valve dilatation has not been established as effective in congenitally deformed mitral valves but is established in mitral stenosis of rheumatic origin.^{34,35} Mitral valve dilatation increases mitral valve area and results in haemodynamic improvement in most patients. It results in a reduced transvalvar gradient, increased cardiac output, reduced mean LA pressure and pulmonary artery pressure, although LVEDP frequently rises temporarily after dilatation for reasons that are unclear.

A trans-atrial approach to the mitral valve is used, so that patients who have an intact atrial septum require trans-septal needle puncture. Balloon inflation may be associated with severe haemodynamic disturbances and general endotracheal anaesthesia is necessary for most cases. Mitral regurgitation is a major complication and results in early pulmonary oedema and low cardiac output due to the poorly compliant left atrium. If there is any concern, patients are best monitored in the CICU following dilatation and weaned from ventilation when haemodynamically stable. In most cases tracheal extubation can be performed within 24 hr. If severe mitral regurgitation persists, surgical repair may be necessary.

PERIPHERAL PULMONARY ARTERY STENOSIS

Peripheral or branch pulmonary artery stenosis may be a congenital or acquired lesion. The incidence of congenital pulmonary artery stenosis is approximately 2 in 10,000 births. Acquired lesions are more common and occur following palliative or corrective cardiac surgery particularly when pulmonary artery manipulations are performed. Patients present with single or multiple areas of stenosis and may also have areas of diffuse hypoplasia. Percutaneous balloon angioplasty or balloon dilatation of stenotic pulmonary arteries has been described as a treatment for this lesion.³⁶⁻³⁸ Percutaneous balloon angioplasty was first reported in humans by Lock,³⁶ and later by Ring.³⁷ Pulmonary arteries are dilated in an attempt to increase the size of the pulmonary vasculature, and to decrease the right ventricular pressure. Indications for balloon dilatation include those patients who have near-systemic, systemic, or supra-systemic right ventricular pressures, hypertension in unaffected portions of the vascular bed, a decrease in blood flow to a portion of the pulmonary vascular bed, or symptoms.³⁸⁻⁴¹ Although dilatation of one pulmonary artery branch at a time has been suggested, as the artery dilated first may dissect,³⁷ balloon angioplasty of bilateral pulmonary artery stenosis as a single stage procedure has been successful.^{42,43}

Prior to balloon angioplasty the pulmonary artery anatomy is angiographically defined and the angioplasty balloon-tipped catheter is then placed into the pulmonary artery. When the balloon is positioned optimally, it is inflated until a waist produced by the stenosis on the balloon begins to disappear, or until a maximum inflation pressure for that balloon diameter is achieved. Following the angioplasty, the balloon is removed and repeat angiography is performed to determine if a pressure gradient is present.

Complications of this procedure have been described and include transient pulmonary oedema distal to the dilated segment, tears in the pulmonary artery intima and media, aneurysm formation, pulmonary artery per-

FIGURE 9 Cardiac angiography demonstrates air bubbles and blood in the right mainstem bronchus following rupture of the pulmonary artery.

foration, pulmonary artery rupture (Figure 9) and pulmonary artery rupture with death.^{37,44,45} Other complications include transient haemoptysis, ventilatory failure, and iliac vein occlusion. Percutaneous balloon angioplasty is successful in 60% of patients, but has a mortality that approaches 1%, and has a risk of aneurysm formation of 3%.⁴⁶

General anaesthesia is rarely needed for these cases. Appropriate sedation can result in few respiratory or haemodynamic complications during cardiac and pulmonary catheter manipulation. The goal of anaesthetic management is to provide support treatment. This may include oxygenation, the control of ventilation, administration of additional anaesthesia and muscle relaxation, volume replacement, haemodynamic support and inotropic agents as necessary.

AORTIC COARCTATION DILATATION

Native uncomplicated aortic coarctation is not universally treated with balloon dilatation angioplasty. This lesion is often treated surgically because the rate of recurrent aortic coarctation after angioplasty may be higher than that seen following surgical repair. Although minimal residual gradients and a decreased incidence of complications have been reported,^{47,48} aneurysm formation at the dilated area postangioplasty, and potential other long-term effects, need to be considered.⁴⁹ Other complications of balloon angioplasty of a native aortic coarctation may include aortic rupture, vascular trauma, and thrombosis. Recurrent aortic coarctations may be dilated with balloon angioplasty.

The arterial angioplasty of coarctations and recurrent

FIGURE 10 A stenotic non-balloon dilatable right pulmonary artery (left frame) is seen to be markedly larger after stent placement (right frame).

coarctations is performed under local anaesthetic while the patient receives sedation. General anaesthesia is rarely required.

PATENT DUCTUS ARTERIOSUS DILATATION

Although vasoactive substances, decreasing concentrations of prostaglandins, and an increase in arterial oxygen tension are considered responsible for the closing of the ductus arteriosus at birth, the precise mechanism is not clearly understood. Maintaining a patent ductus arteriosus to supply systemic or pulmonary blood flow before palliative or definitive surgical repair may be crucial. Balloon dilatation of a closed ductus arteriosus in patients with CHD has provided immediate and sustained wide patency in infants who did not respond adequately to prostaglandin E_1 .⁵⁰ However, this therapy is not widely practiced due to the potential for rupture or paradoxical closure of the ductus.

Vascular stent placement

The placement of vascular stents into pulmonary arteries, aorta, conduits, and baffles is performed in an attempt to maintain the long-term patency of the previously stenotic or obstructed areas (Figures 10 and 11). The approach is similar to that described for balloon dilatation of these lesions and is usually performed under sedation. The potential for migration of the stent into the left heart and outflow tract exists.

Valved conduits have been used for right ventricle to pulmonary artery connection. However, progressive fibrointimal stenosis of the valved conduit may occur leading to an increase in the mean transconduit pressure gradient. Stenosis of these conduits is managed with balloon angioplasty and stent placement. Local anaesthesia to the involved femoral region or catheter insertion site combined with sedation is well tolerated. Following inflation of a balloon-tipped catheter a tubular slotted steel stent is introduced and positioned in the valve conduit

FIGURE 11 A postmortem picture of a vascular stent demonstrates how the mesh becomes endothelialized with time and how the mesh preserves collateral vessels.

TABLE II Arrhythmias amenable to radiofrequency ablation

<i>Accessory atrioventricular pathways</i>
Wolff-Parkinson-White syndrome
"Concealed" accessory pathways
Permanent junctional reciprocating tachycardia
<i>AV node reentry tachycardia</i>
Fast pathways
Slow pathways
<i>"Automatic" tachycardias</i>
Ectopic atrial tachycardias
Junctional ectopic tachycardia
Ectopic ventricular tachycardia

at the area of stenosis reducing the mean transconduit pressure gradient.

Transcatheter ablation

Many cardiac dysrhythmias can now be treated with transcatheter ablation in the cardiac catheterization laboratory. Most of these are tachyarrhythmias caused by accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) or AV node reentry while some are "automatic" tachycardias arising from abnormal foci in the atria or ventricles (Table II).

Until about 20 yr ago the only treatment for recurrent tachyarrhythmias was medical. Many antiarrhythmic agents fail to control the arrhythmias reliably or have significant side effects (Table III). The cardiotoxicity and dysrhythmic action of these agents may increase morbidity and mortality.^{51,52} Also, some patients, especially adolescents, can or will not comply with medical regimen.

During the decade from 1980 to 1990 surgical treatment of tachyarrhythmias was introduced. However, in many instances this treatment necessitated the use of cardiopulmonary bypass and the risk of major surgery. The

TABLE III Commonly used anti-arrhythmic agents

<i>Drug</i>	<i>Indication</i>	<i>Side effect</i>
Amiodarone	Ventricular tachycardia	Pulmonary fibrosis
	Supraventricular tachycardia	Thyroid dysfunction
	Paroxysmal atrial fibrillation	Hepatic dysfunction
Sotalol	Ventricular tachycardia	Bronchospasm
	Supraventricular tachycardia	Prolonged QT
Procainamide	Ventricular tachycardia	Lupus syndrome
	Supraventricular tachycardia	
Quinidine	Ventricular tachycardia	Nausea, diarrhoea
	Supraventricular tachycardia	Prolonged QT

success rate was not complete and the convalescent period prolonged.⁵³

In 1982 transcatheter ablation was described.⁵⁴ Initially, direct current was used; this created a high burst of energy causing an "explosion" with gas formation inside the heart and a risk of more extensive damage than intended. The use of direct current has now been replaced with radiofrequency energy. Radiofrequency energy is a low-power high frequency alternating current which causes controlled injury by creating heat at the tip of the catheter. In contrast to direct current, which can cause intense retrosternal pain, radiofrequency ablation is virtually painless.

Transcatheter ablation was first performed in adults, but during the last few years several reports of radiofrequency ablation of different arrhythmias in children have been published.^{7,55-57} Initially, catheter ablation of tachyarrhythmias in the paediatric population was only performed in children with structurally normal hearts. However, over the last few years catheter ablation has been performed with increasing frequency in children with congenital heart defects who present with arrhythmias following cardiac surgical repair (for instance in children who have undergone an atrial switch operation for transposition of the great arteries (TGA) during early childhood (Mustard or Senning operation)) or as a preparation for corrective surgery in children in whom post-operative arrhythmias will be poorly tolerated (as for example in children with tricuspid atresia having a right atrial to pulmonary artery connection (Fontan procedure)).^{58,59}

A recent multicentre study from the Pediatric Electrophysiology Society demonstrates radiofrequency catheter ablation to have a good success rate and a low complication rate in a paediatric population.⁶⁰ In that study approximately 15% of the children had CHD. The indications for ablation are presented in Table IV.

TABLE IV Indications for ablation

Life-threatening arrhythmia, syncope, seizures, cardiac arrest
Medically refractory arrhythmia
Adverse drug effects
Tachycardia-induced ventricular dysfunction
Impending cardiac surgery for congenital malformation (e.g., Fontan)
Patient's choice

Most cardiology reviews of transcatheter ablation procedure do not address the question of anaesthetic management except to point out that radiofrequency ablation does not require general anaesthesia as it is pain-free, in contrast to direct current ablation. It is, however, not always reasonable to expect small children and impatient adolescents to lie completely still for many hours, consequently in most paediatric series, general anaesthesia is described or implied. A recent study shows that adults also prefer general anaesthesia.⁶¹

While transcatheter ablation seems preferable with fewer side effects to surgical ablation or long-term medical treatment, it has been a question of which is most cost-effective. A recent study favours transcatheter ablation.⁶²

ANAESTHETIC AGENTS AND THE CARDIAC CONDUCTION SYSTEM

There is an extensive literature about anaesthetic agents and their cardiovascular effects but little about the agents' effect on the normal conduction system. The majority of the studies has been performed using surface ECG rather than direct electrophysiological measurements. Most studies have been limited to arrhythmic properties and the effects of anaesthetic agents in combination with endogenous and exogenous catecholamines.^{63,64} Furthermore, all these studies were performed in adults and it is unclear whether these studies can be directly applied to children.

A study of fentanyl and droperidol effects on the refractoriness of accessory pathways in patients with Wolff-Parkinson-White Syndrome was performed using intraoperative electrophysiological evaluations.⁶⁵ Fentanyl, 50 $\mu\text{g} \cdot \text{kg}^{-1}$, had no effect on the refractory period of the accessory pathways while droperidol (200–600 $\mu\text{g} \cdot \text{kg}^{-1}$) increased the refractory period. The authors concluded that, in clinical practice (though not for ablation procedures), droperidol may prevent arrhythmias in patients with Wolff-Parkinson-White Syndrome.

Another study examined the effects of alfentanil-midazolam anaesthesia during surgical interruption of accessory pathways in eight patients with the Wolff-Parkinson-White syndrome. After sternotomy using epicardial electrodes, a baseline electrophysiological study was performed and compared with the preoperative elec-

trophysiological values. Alfentanil-midazolam infusion had no effect on either the normal conduction system or the accessory pathways.⁶⁶

In a similar study during surgical ablation of accessory pathways in patients with Wolff-Parkinson-White syndrome the effects of volatile anaesthetic agents and Sufentanil were studied. It was found that sufentanil ($20 \mu\text{g} \cdot \text{kg}^{-1}$) combined with lorazepam ($0.06 \text{ mg} \cdot \text{kg}^{-1}$) had no clinically important effect on the accessory pathway. Of the volatile agents enflurane most, then isoflurane and least halothane increased the refractoriness within the accessory and normal atrioventricular pathways. The authors warn that these volatile agents may be contraindicated as anaesthetic agents during ablation procedures, while they may be agents of choice for incidental surgery in patients with pre-excitation syndrome.⁶⁷

In a recent study, we compared the effects of isoflurane and propofol with baseline anaesthesia (alfentanil- N_2O -pancuronium) in 20 children with supraventricular tachycardia. The study was performed during transcatheter ablation procedures. Neither isoflurane nor propofol changed any of the electrophysiological measurements when compared with baseline anaesthesia.⁶⁸

In a review of cardiac electrophysiology and conduction pathway ablation Renwick *et al.* concluded that balanced general anaesthesia or monitored anaesthesia care with benzodiazepines, propofol and opioids are acceptable.⁶⁹

CURRENT PRACTICE

As mentioned previously, intravenous sedation is suitable for adults undergoing catheter ablation unless direct current is used or cardioversion becomes necessary. In a paediatric practice it is unreasonable to expect the children to lie still for many hours so general anaesthesia is the rule. In some cases where the abnormal focus is close to the AV node it is imperative to prevent the child from moving at the time radiofrequency energy is applied, as movement might displace the catheter and cause permanent a-v dissociation. While the entire procedure is time consuming including a brief haemodynamic catheterization and up to hours of mapping the conduction system, the actual ablation only takes seconds (Figure 12).

The majority of paediatric patients presenting for radiofrequency ablation will have structurally normal hearts, and normal ventricular function. Some, patients who have had multiple episodes of tachycardia and who have been treated with different antiarrhythmic drugs, may have decreased left ventricular function. Also, an increasing number of children with complex congenital defects are now coming for either pre- or postoperative ablation of atrial arrhythmias and those patients often

FIGURE 12 ECG showing Wolff-Parkinson-White syndrome with conversion to normal sinus rhythm after 3.5 sec radiofrequency current.

have severe pathophysiology with intra- and extra-cardiac shunts and ventricular dysfunction. Anaesthetic management of these patients depends on their cardiac pathophysiology. Post-procedure management may include overnight stay in the cardiac ICU and even ventilatory support overnight. This is in contrast to the patients with structurally normal hearts, whose tracheas are extubated in the catheterization laboratory. After recovery in a post-anaesthesia care unit they are returned to the cardiac ward after a few hours.

Nausea and vomiting following the procedure is common. We administer either droperidol ($75 \mu\text{g} \cdot \text{kg}^{-1}$) or ondansetron ($150 \mu\text{g} \cdot \text{kg}^{-1}$) towards the end of the procedure to attenuate this problem.

Proper patient positioning is very important due to the length of these procedures. During our first two years of performing radiofrequency ablations the patient's arms were positioned over the head. Three patients experienced temporary brachial plexus injury. Since that time we have positioned the patient's arms at their sides with pressure points padded and there has been no further occurrence of nerve injury.

Until further studies reveal more about anaesthetic agents' effects on conduction through abnormal pathways, general anaesthesia using either an opioid-relaxant technique or isoflurane-relaxant are acceptable for radiofrequency ablation of abnormal pathways in the paediatric population as is monitored anaesthesia sedation with benzodiazepines and opioids in adults and mature adolescents.

Cardioversion

Nonpharmacological techniques for management of acute arrhythmias have gained in popularity. Interest in the use of electrical techniques to convert tachyarrhythmias

to a normal rate and rhythm has increased for four reasons: (1) electrical therapy is highly effective, (2) the response to its effect is immediate, (3) side effects are few, and (4) it can be applied repetitively although eventually cumulative effects become additive producing toxicity.

The transthoracic application of high-electrical-energy currents can effectively convert either supraventricular or ventricular tachyarrhythmias to sinus rhythm in paediatric patients. Cardioversion differs from defibrillation in that the electrical discharge is synchronized with the R wave to avoid inducing ventricular fibrillation by accidental discharge during the period of ventricular vulnerability. During ventricular fibrillation, R-wave synchronization is impossible because ventricular depolarization is continuous.

Restoration of sinus rhythm by cardioversion is most successful for reentrant arrhythmias. This technique is ineffective for terminating automatic tachycardias and is contraindicated in the treatment of arrhythmias due to digitalis toxicity. The arrhythmias most responsive to electrical cardioversion are: (1) supraventricular tachycardia, (2) atrial flutter, (3) atrial fibrillation, and (4) ventricular tachycardia.

COMPLICATIONS OF CARDIOVERSION

Arrhythmias

A variety of arrhythmias and conduction disturbances have been described following cardioversion (Table V). The occurrence and frequency of arrhythmias and conduction abnormalities depend on multiple factors: presence and type of pre-cardioversion arrhythmias, cardiac disease, metabolic derangements, digitalis toxicity, and shock strength.⁷⁰⁻⁷⁵

Morphological and function damage

A number of studies have demonstrated that electrical countershock can produce morphological and functional derangements.⁷⁶⁻⁷⁹ The extent of macroscopic and microscopic evidence of necrosis, ECG changes and serum enzymes changes (CK-BB), is related to the intensity of the electrical shock and inversely to the time between shocks.⁷⁹⁻⁸¹ However, when properly performed using moderate energy doses (beginning at $0.5 \text{ joules} \cdot \text{kg}^{-1}$) histological injury is rare. In addition to morphological damage, left ventricular systolic and diastolic functional impairment have been demonstrated in animal studies.⁸²

Embolism

Systemic and pulmonary emboli have been demonstrated in 1 to 2% of adult patients but is extremely rare in the paediatric population.⁷⁰ Although anticoagulation before conversion of atrial arrhythmias has proved very suc-

TABLE V Arrhythmias after cardioversion

<i>Tachyarrhythmias</i>
Atrial fibrillation
Supraventricular tachycardia
Ventricular tachycardia
Ventricular fibrillation
<i>Bradycardia and atrioventricular conduction abnormalities</i>
Sinus bradycardia
Atrioventricular block
Right bundle branch block
Left bundle branch block

cessful in reducing the risk of emboli in the adult population the risk-benefit ratio in the paediatric population is quite low.⁷³

Patients with pacemakers

Cardioversion of the patient with a pacemaker is becoming more common in the paediatric population and, although not without risk, can be performed safely with special precautions. The electrode paddles should be placed at a distance from the generator. Programming the sensing circuit to low sensitivity is unnecessary and will not protect the generator. Immediately after the cardioversion the sensing and pacing circuits of the pacemaker need to be reassessed and the programming mode reviewed as reprogramming to an alternative pacing mode may occur during direct-current countershock.

ANAESTHETIC MANAGEMENT OF CARDIOVERSION

For elective cardioversion, patients are appropriately fasted. Cardioversion is performed in a setting with appropriate monitoring and resuscitation equipment. Routine monitoring includes blood pressure, ECG, precordial stethoscope, temperature and oxygen saturation. Patients with full stomachs who cannot be appropriately fasted will require tracheal intubation. An intravenous line is inserted for drug administration. Prior to sedation or induction of anaesthesia the patient is connected to an ECG monitor capable of R-wave synchronization. The technique of sedation/anaesthesia can be quite variable and consists of ketamine, diazepam, midazolam, propofol, and/or thiopentone. After ensuring the patient is satisfactorily sedated/anaesthetized the first countershock is applied with R-wave synchronization beginning at a dose of $0.5 \text{ joules} \cdot \text{kg}^{-1}$. If successful, the procedure is terminated and the patient is monitored for two hours in the recovery area and then overnight in his/her room. If unsuccessful, a second countershock is applied at double the energy level used for the first shock. Each shock should be separated by two to three minutes as the risk

of producing myocardial injury will be lower, and delayed reversion of the arrhythmia can occur up to two minutes after cardioversion.

Transoesophageal echocardiography

Transoesophageal echocardiography (TEE) can be utilized both as a diagnostic and monitoring tool in the catheterization laboratory. Transoesophageal echocardiography provides real-time imaging and has become an additional modality for monitoring interventional catheterization procedures.⁸³⁻⁸⁵ It can provide useful information for catheter placement,⁸³ valvular function and integrity, pre- and post-dilatation gradients and on-line assessment of ventricular contractility and filling volume. Transoesophageal echocardiography has also proved useful in monitoring blade and balloon atrial septostomy and positioning of double-umbrella occluding devices across various heart defects.⁸⁶ Advantages of TEE monitoring include decreased x-ray exposure and lesser amount of contrast material utilized. It can also provide imaging planes not available by radiology and it will not endanger the sterility of the field nor will it interfere with fluoroscopy.

The diagnostic uses of TEE are well described and TEE evaluation can be performed prior to catheterization when information obtained from transthoracic echocardiography is insufficient.

General anaesthesia and heavy sedation have both been advocated for TEE examination in children. Recently, paediatric biplane probes have been introduced and used in infants as small as 2.4 kg.⁸⁷ General endotracheal anaesthesia is well suited for smaller children as the airway is better protected against aspiration and possible compression from the TEE probe. Accidental extubation can occur especially at the time of introduction and withdrawal of the TEE probe. For this reason, we perform nasotracheal intubation to obtain greater stability of the endotracheal tube position. The patient's medical condition will dictate the choice of anaesthetic technique more adequately than the proposed procedure. For example, although propofol anaesthesia is an appealing choice for patients undergoing catheterization procedures on an outpatient basis,⁸⁸ the reduction in systemic blood pressure might not be well tolerated by patients with uni-ventricular heart physiology.

The risk of bacteraemia associated with TEE is extremely low and the American Heart Association position paper, concludes that endocarditis prophylaxis is not required for this procedure although the use of nasotracheal intubation is an indication.^{89,90}

Transoesophageal echocardiography is an invasive monitoring and diagnostic procedure and has been associated with some complications. The most expansive

study of TEE safety has been done by Daniel *et al.* who reviewed the experience of 15 European centres.⁹¹ The overall complication rate was 0.18% and death occurred in less than one in 10,000 patients. Complications included bronchospasm, hypoxia, arrhythmias, pharyngeal and oesophageal bleeding and vomiting. Bronchial and vascular compression with ensuing desaturation and hypotension has been described,^{92,93} and this phenomenon is not a rare occurrence in smaller infants and children, especially those weighing <10 kg. We have observed hypercyanotic episodes after probe insertion in patients with Tetralogy of Fallot, although a cause and effect relationship is difficult to prove. Other potential causes of desaturation include tracheal or bronchial compression, bronchospasm and/or light anaesthesia.

Anaesthetic management

Location

Cardiac catheterization laboratories are usually located in sites remote from the operating room. The facilities, having been primarily designed in the past when anaesthesia was infrequently utilized, are rarely configured to accommodate anaesthetic personnel. The result is that the anaesthetist may be required to work in cramped quarters and/or monitor the patient from a location where access to both patient and airway is awkward. Fortunately, as new facilities are developed and old facilities are upgraded, appropriate accommodation for anaesthesia support is being included.

Anaesthetic considerations

The anaesthetist who provides anaesthetic support for the catheterization laboratory is presented with a dilemma: should the patients be sedated, anaesthetized using a technique which enables early postprocedural extubation or does the patient and/or procedure oblige postoperative ventilation with intensive care support?

Routine patient monitoring includes ECG, blood pressure, arterial oxygen saturation and temperature continuously throughout the catheterization. Respiratory rate is measured using end-tidal carbon dioxide monitoring through nasal prongs in spontaneously breathing patients. Secure intravenous access is established to provide additional sedation or resuscitation. Once the cardiologist has arterial and venous catheters in place beat-to-beat monitoring of the arterial pressure, measurements of filling pressure and arterial blood gas analysis are readily available. To reduce the risk of infection, urinary catheters are not used routinely, but catheterization at the end of a procedure may be advisable especially after a long procedure or if large fluid volume replacement has been given.

A problem which has become very apparent with the introduction of general anaesthesia to the catheterization laboratory is the frequent occurrence of pressure damage and brachial plexus injury when patients have their arms positioned above their heads to make room for the lateral cameras. These are complications which cardiologists have been slow to appreciate as they did not occur commonly in awake or lightly sedated patients who could move and complain about the discomfort.

Antibiotic coverage is given for interventional procedures involving device or coil placement as cephalexin ($30 \text{ mg} \cdot \text{kg}^{-1}$), repeated after six hours. Antibiotic coverage is not recommended by the American Heart Association for haemodynamic catheterization.^{90,94}

Heparinization schedules vary depending on the procedure and the results. All patients receive a bolus of heparin ($100 \text{ U} \cdot \text{kg}^{-1}$) at the time of insertion of the arterial and venous catheters. The activated clotting time (ACT) is determined every 30 min and additional heparin administered as necessary to maintain the $\text{ACT} > 200 \text{ sec}$. Heparin is not reversed at the end of the procedure but allowed to wear off naturally to minimize the risk of thrombosis. Certain patients receive an infusion of heparin ($20 \text{ U} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) for 12 to 24 hr after the procedure. These include those who have had shunts manipulated by catheters, have had stents placed, have evidence of disruption of the pulmonary artery intima as in patients undergoing dilatation of the pulmonary artery, have undergone an ablation, or those in whom there is concern regarding the patency of the arterial cannulation site.^{95,96}

Cardiologists prefer the patients to be breathing room air, spontaneously, as this causes the least disturbance of the haemodynamic values, saturations and, consequently, the least effect on calculations of cardiac output, shunt fraction and vascular resistances. Therefore, the vast majority of diagnostic, non-interventional, haemodynamic studies are performed while the patient is sedated. At our institution, standard sedation for infants $< 10 \text{ kg}$ consists of chloral hydrate ($75\text{--}100 \text{ mg} \cdot \text{kg}^{-1}$) administered *po* or *pr*. Children between 10 and 20 kg receive "Demerol Compound" (meperidine 25 mg, promethazine 6.25 mg and chlorpromazine $6.25 \text{ mg} \cdot \text{ml}^{-1}$) *im* up to a maximum of $0.11 \text{ ml} \cdot \text{kg}^{-1}$ or 2.0 ml.⁹⁷ A 2% lidocaine solution (without epinephrine) is injected into the femoral area to provide analgesia for the percutaneous catheter insertion. Sedation is maintained by carefully titrating doses of morphine ($0.05 \text{ mg} \cdot \text{kg}^{-1}$) and midazolam ($0.05 \text{ mg} \cdot \text{kg}^{-1}$). If the infant/child is difficult to sedate or requires high doses of medications, ketamine ($0.2\text{--}0.5 \text{ mg} \cdot \text{kg}^{-1}$) by intermittent bolus or by continuous infusion ($1 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) can be used. Ketamine has minimal haemodynamic effects on the patients un-

dergoing cardiac catheterization.⁹⁸ Alternatively, in children with good myocardial function and not dependent upon systemic-to-pulmonary shunts or high preload, propofol by intermittent bolus or infusion may be used.⁹⁹ There is a small incidence of airway problems associated with such sedation techniques.^{19,100} If ventilation becomes compromised and the patient is still uncooperative, general anaesthesia with tracheal intubation or use of a laryngeal mask airway may be instituted. In non-interventional procedures general anaesthetic techniques are reserved for uncooperative patients, with or without developmental delay, when sedation techniques prove inadequate. Every effort is made to maintain spontaneous ventilation and minimize inspired oxygen concentration so that the haemodynamic data obtained by the cardiologist will be meaningful.

Certain interventional procedures can be performed safely using sedation techniques. Patent ductus arteriosus and atrial septal defect occlusion, coil embolization and balloon dilatation of peripheral pulmonary artery stenosis are examples of procedures that in many instances can be safely performed with the patient sedated. However, each patient needs to be carefully assessed in cooperation with the attending cardiologist. As an example some cooperative patients who can be adequately sedated for peripheral pulmonary artery stenosis dilatation are at risk to develop high pressure pulmonary oedema distal to the stenotic site after dilatation. Many of the at-risk patients can be identified prior to the procedure by the cardiologist. These patients receive general anaesthesia with tracheal intubation and positive-pressure ventilation prior to dilatation. Most will also require positive-pressure ventilation in the ICU postprocedure. Ventricular septal defect occlusion techniques are another procedure that requires the use of general anaesthesia techniques because of the severe haemodynamic disturbances described earlier and the possible need for resuscitative procedures.²³

Patients who are to undergo general anaesthesia from the outset of the procedure are frequently anxious on arrival in the preprocedural area. The previous application of EMLA cream may enable the establishment of an intravenous infusion and the use of *iv* sedation such as midazolam ($0.05\text{--}0.1 \text{ mg} \cdot \text{kg}^{-1}$). Alternatively we have found midazolam ($0.75\text{--}1.0 \text{ mg} \cdot \text{kg}^{-1}$) *po* or ketamine ($3\text{--}5 \text{ mg} \cdot \text{kg}^{-1}$) *im* to be useful to facilitate parental separation.

Most of these children tolerate induction of anaesthesia with thiopentone and maintenance with a combination of inhalation anaesthesia and opioid. We currently use isoflurane with small to moderate doses of fentanyl ($3\text{--}10 \mu\text{g} \cdot \text{kg}^{-1}$). In children with depressed myocardial function anaesthesia can be induced with ketamine ($1\text{--}2$

mg · kg⁻¹). If combined with fentanyl, pancuronium has a minimal effect on the heart rate and in many young children the relative tachycardia may be an advantage as the cardiac output is rate-dependent. In children with near terminally depressed myocardial function and depleted catecholamine stores, ketamine may be contraindicated, acting as a direct myocardial depressant.¹⁰¹⁻¹⁰⁴ In these patients careful titration with fentanyl and midazolam for induction or, alternatively, etomidate may be used.¹⁰⁵ It must be remembered that injection of etomidate is quite painful and may upset the child needlessly. Recently, the use of caudal blockade has been proposed for cardiac catheterization. Although initially attractive, this technique does not sedate the child who has to lie still for several hours; it does not enable the easy insertion of a subclavian or internal jugular vein catheter which many procedures now require; and it may produce haemodynamic alterations.

Most in-patients who come to the catheterization laboratory have a functioning indwelling intravenous catheter which facilitates induction. As the population of out-patients increases, the presence of an intravenous infusion is no longer the rule. Premedication with oral midazolam (0.5–1.0 mg · kg⁻¹) has proved very useful. In children where this fails and in whom local transdermal anaesthetics (EMLA® cream) fails, mask induction with halothane or intramuscular induction with ketamine are options.

Postprocedure management

After catheterization, patients who are not admitted to the ICU recover for a minimum of 30 min in a post-anaesthesia recovery facility where the patient can be monitored for both emergence from anaesthesia and possible adverse events related to the procedure. Haemodynamic status, arterial oxygenation status, respiration, level of consciousness, discomfort and presence of peripheral pulses are monitored. Parents are encouraged to be present during the recovery phase of the patient. Discomfort, particularly from the femoral cannulation site and need for pressure dressing, can be considerable and is treated with opioids; most commonly we use morphine (0.05 mg · kg⁻¹) repeated until the patient is comfortable. Nausea and vomiting can be a problem; the straining associated with nausea and vomiting can result in reopening of the femoral cannulation sites. Patients who experience nausea and vomiting will receive either droperidol (75 µg · kg⁻¹) or ondansetron (150 µg · kg⁻¹). The loss or decrease of a peripheral pulse is usually an indication for 12–24 hr of a low-dose heparin infusion, as described previously.

When the patients are adequately awake and are stable they are discharged to the cardiac ward where they un-

dergo 2:1 nursing coverage overnight. Monitoring on the ward is the same as in the post-procedural area. Patients are frequently discharged home the following day although same-day discharge has been reported.¹⁸

Economics

The rapidly escalating costs of health care have resulted in a greater emphasis on cost containment.¹⁰⁶ The consequence of this is to question the cost-efficiency of health care choices^{107,108} such as interventional cardiological procedures, over time-tested surgical procedures. New procedures are infrequently evaluated in randomized trials and the trials that are conducted rarely include cost analysis. The evaluation of costs and outcomes of concurrently available treatment modalities is problematic as the results of long-established and refined procedures are compared with those of an evolving alternative. A recent cost and outcome comparison analysis was performed on patients undergoing closure of PDA either by traditional surgical approach or by transcatheter occlusion.¹⁰⁷ The results of this analysis suggested that transcatheter closure was associated with increased cost compared with its surgical counterpart. Although the results of this study do not support the widespread use of the occluder procedure for the management of this common congenital lesion the results of this analysis are not universally accepted.¹⁰⁸ Outcomes and costs of interventional techniques may improve as the catheters and devices, procedures and patient-selection criteria are refined.^{6,109-112} Independent of outcome, costs associated with these new procedures may decline over time¹¹³ and should decrease with increasingly efficient provision of care.¹⁹

Conclusions

Over the past decade the introduction of interventional techniques has revolutionized the treatment of CHD and many procedures such as balloon pulmonary valvuloplasty, are highly recommended.¹¹⁴ Interventional techniques should not be viewed in isolation but considered as part of the overall approach to the patient with CHD. As has been described, interventional techniques may be used in conjunction with a surgical approach to the patient. Initial problems with morbidity in these new and innovative procedures is not surprising.¹⁰⁷ In 1952, Gross cited a failure rate of 20% in the early surgical attempts to ligate the ductus arteriosus¹¹⁵ which compares with a current surgical failure rate of 0.2%.¹⁰⁷ Such a period of development in all new procedures is common.¹⁰⁸ With the effects of time, competition and new technology, interventional cardiological procedures should become competitive with and possibly replace corresponding surgical procedures.

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