Anesthesia For the Patient With Congenital Heart Disease For Noncardiac Surgery

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Introduction

Congenital heart disease (CHD) is present in 9 per 1000 live births in the United States, making it the most common congenital defect requiring invasive treatment in the first year of life. With the current mortality for all congenital cardiac operations less than 5%, an increasing number of these patients survive, and it is currently estimated that there are between 650,000 and 1.3 million adults and children in the U.S. living with congenital heart disease. Of these, approximately 55% have simple lesions, 30% have moderately complex lesions, and 15% have complex lesions. The learning objectives of this lecture are: 1) Understand the pathophysiology of common congenital heart diseases and its impact on anesthetic management for non-cardiac surgery; 2) Understand the most common non-cardiac surgical procedures in patients with congenital heart disease; 3) Understand the updated SBE prophylaxis guidelines from the American Heart Association.

Basic Approach to Congenital Cardiac Lesions

One practical approach to assessing patients with CHD is to ask the following questions: 1. Is the patient cyanotic, with either obligatory right-to-left intracardiac shunting, or mixing lesions? And if cyanotic, does the patient have a functional single ventricle? If acyanotic is this a left-to-right shunting lesion, or an obstructive lesion? 2. Has the patient undergone corrective or palliative surgery, and if so, what is the resulting anatomy and residual defects? 3. What is the patient’s current status: well compensated with no limitations, moderately well compensated with few limitations, or poorly compensated with significant limitations? 4. What is the proposed procedure and what anticipated effects will the procedure and anesthetic management have on the patient’s pathophysiology? 5. Does this patient need infective endocarditis prophylaxis?

Pathophysiology of Common Cardiac Lesions

The common complex and moderately complex cardiac lesions include: Tetralogy of fallot (incidence 9-14% of CHD), transposition of the great arteries (10-11%), atrioventricular septal defects (4-10%), coarctation of the aorta (8-11%), hypoplastic left heart syndrome (4-8%), and ventricular septal defects (VSD)(10-20%). All of these lesions, with the exception of transposition of the great arteries, have a wide range of anatomical abnormality ranging from relatively mild, to severe, which must be assessed and which have great influence on the patient’s symptomatology and anesthetic considerations.

Tetralogy of Fallot

Tetralogy of fallot (TOF) consists of 1) large subaortic VSD, 2) right ventricular outflow tract obstruction/ pulmonary stenosis, 3) right ventricular hypertrophy, 4) right sided aortic arch (present in 25% of patients). In the unrepaired TOF patient, pathophysiology depends on the degree of right ventricular outflow tract (RVOT) obstruction. With significant RVOT obstruction, infundibular spasm constricts the RVOT, limiting blood flow into the pulmonary artery, forcing blood right to left across the VSD and resulting in significant arterial desaturation, or “Tet spells.” These spells are incited by catecholamine release due to pain, stress, light anesthesia, and emotional upset in the infant. Many of these patients receive oral propranolol to prevent these spells. Treatment involves intravascular volume administration to increase RV stroke volume, increasing sedation or anesthesia, avoidance of exogenous catecholamines, increasing FiO2, and increasing systemic vascular resistance to force blood left-to-right across the VSD and out the RVOT, increasing pulmonary blood flow and thus oxygenation. Surgical treatment of TOF with pulmonary atresia or significant pulmonary stenosis with spells involves either placing a systemic to pulmonary artery shunt in early infancy followed by a complete anatomic repair at 6-12 months, or complete repair.
in early infancy. Patients without cyanotic spells are normally repaired at 6-12 months. It is important to note that repaired TOF patients often have residual defects, usually involving varying degrees of pulmonary insufficiency, or residual RVOT obstruction. Assessment of these patients with periodic echocardiography or cardiac MRI is important, and planning the anesthetic should involve review of these data. Teenage or adult TOF patients may have RV dilation and are prone to ventricular arrhythmias.

**D-Transposition of the Great Arteries**

In this lesion the great arteries fail to rotate and the aorta and coronary arteries arise from the right ventricle, and the pulmonary artery from the left ventricle. D- refers to dextro- meaning the aorta is rightward and anterior to the pulmonary artery. About 85% of these patients have an intact ventricular septum and 15% have a VSD. Since the systemic venous blood is recirculated to the RV and the aorta and is not oxygenated by this pathway, adequate arterial saturation depends on mixing between right and left sides of the heart, either at the VSD level, atrial septal defect level, or patent ductus arteriosus. For this reason, neonates with significant desaturation undergo a balloon atrial septostomy, which creates a much larger communication at the atrial level and normally increases oxygen saturation to the 80-90% range. Standard surgical treatment since the mid-1980’s has been the arterial switch operation, which includes translocation of the coronary arteries and leaves the normal physiology. The vast majority of infants do very well and have little if any residual defect, and thus can be treated as a well-compensated, corrected CHD patient. Residual problems might include coronary artery ischemia due to anatomic problems with the re-implanted coronary arteries, or regurgitation or stenosis in the neo-arte root. Older patients with D-TGA repaired before the mid-1980’s most often had the Mustard or Senning operations, which both re-routed the blood flow at the atrial level, leaving the aorta arising from the RV and pulmonary artery from the LV. These patients are now adults, and most have significant problems including systemic ventricle failure, as the RV is inadequate as a systemic pump; and frequent significant atrial arrhythmias from the atrial suture lines. These patients require thorough preoperative evaluation by a cardiologist addressing ventricular function and arrhythmias.

**Atrioventricular Septal Defects**

Atrioventricular septal defects (AVSD) are classified as partial, intermediate or transitional, and complete. AVSD are also known as atrioventricular canals (AVC), and derive from a deficiency or absence of the endocardial cushion during cardiac development. Partial AVC consist of a primum atrial septal defect, no VSD, and separate atriventricular valves with a cleft mitral valve. These defects have pathophysiology similar to that of a large atrial septal defect (ASD), and thus are rarely symptomatic in infancy, and are usually repaired at age 2-4 years. Residual mitral stenosis or regurgitation may be encountered. Intermediate or transitional AVC has a primum ASD, common single atrioventricular valve, often with regurgitation, and a small or absent VSD component. These patients are also usually not symptomatic in infancy and are repaired at 2-4 years of age, and also may have residual mitral valve disease. Complete AVC have both a large primum ASD, and VSD component and common atrioventricular valve. These patients have very large left-to-right shunts in infancy, and develop congestive heart failure that necessitates repair before 6 months of age. Residual mitral or tricuspid valve regurgitation may be problematic for these patients. The majority of CAVC patients also have Trisomy 21, and these patients develop severe pulmonary hypertension earlier than patients with CAVC and normal chromosomes. If these patients are not repaired in the first several years of life, the high pressure and flow in their pulmonary arteries may result in significant muscular development in smaller and more distal pulmonary arteries, and then produce severe pulmonary hypertension, that is equal to or even higher than systemic pressures. This results in right-to-left shunting, increasing cyanosis, and eventually results in Eisenmenger’s Syndrome, which denotes irreversible, fixed pulmonary hypertension. These patients are at high risk for anesthesia for any procedure. With early repair of CAVC that is offered to all patients, this syndrome is seen much less frequently over the past two decades.
Coarctation of the Aorta

Coarctation is a narrowing of the aorta most commonly in the juxtaductal area, or opposite the insertion of the ligamentum arteriosum near the left subclavian artery. As with nearly all congenital defects, there is a wide range of anatomy and patients may present as neonates in cardiovascular collapse when their patent ductus arteriosus closes if they have a very tight coarctation, or may not present until later in childhood with milder forms of coarctation. Later presentation is usually with hypertension in the right upper extremity; patients may also have a soft systolic murmur, or continuous murmur over the back due to development of extensive arterial collaterals which form to increase blood flow to areas below the coarctation. Repair is normally done as soon as the coarctation is diagnosed, usually via left thoracotomy by direct end-to-end anastomosis after resection of the coarctation segment. Residual coarctation may occur and is usually treated with balloon angioplasty and stenting in the cardiac catheterization laboratory. Patients may have residual hypertension, and adults with late treatment of coarctation often have severe coarctation, early coronary artery disease, and are prone to cerebral vascular accidents. Most repaired patients, however, have essentially normal cardiac function when presenting for non-cardiac procedures.

Hypoplastic Left Heart Syndrome

This syndrome (HLHS) has varying degrees of underdevelopment of the left ventricle, and has either significant stenosis, or atresia of both the aortic and mitral valves, resulting in very limited or no flow through the left ventricle and aortic valve. At birth systemic blood flow is dependent on a patent ductus arteriosus (maintained by prostaglandin E1 infusion) to supply blood flow to the lower body, and to augment blood flow to the brain and coronary arteries in a retrograde manner. Since the mid 1980’s these patients have survived in increasing numbers, and now there are teenagers and even young adults with HLHS presenting for anesthesia for non-cardiac procedures. The initial surgical palliation involves reconstruction of the aorta by using the native pulmonary valve, native aorta, and augmenting the repair with a cryopreserved homograft patch to create a neo-aorta to provide systemic blood flow, which arises from the single right ventricle. An atrial septectomy must be performed to allow unimpeded flow of blood from left atrium to right atrium to bypass the hypoplastic left heart. And, since the pulmonary valve has been used to construct the neo-aorta, a new source of pulmonary blood flow is provided, either a small 3-4 mm systemic to pulmonary artery shunt, or a larger right ventricle to pulmonary artery shunt. In either case, the single systemic RV in parallel with the pulmonary circulation, bridged by a shunt, is inherently unstable, and hemodynamic stability depends on balancing pulmonary to systemic flow ratio to approximately 1:1. Excessive pulmonary vasodilation with high FiO2, or low PaCO2 will steal flow from the systemic and coronary circulations, often resulting in myocardial ischemia and a vicious cycle ending in cardiac arrest. These patients are most precarious after the neonatal Stage I Norwood palliation, and unfortunately often need non-cardiac procedures such as fundoplication and gastrostomy tube, during this period. These patients must be managed carefully during non-cardiac procedures, and should be cared for in an intensive care setting after most procedures.

The second stage of the palliation sequence for HLHS is a bidirectional cavopulmonary connection done at 2-6 months of age, where the systemic-pulmonary, or RV-pulmonary shunt is replaced by direct anastomosis of the superior vena cava to the right pulmonary artery. This greatly stabilizes the circulation by unloading the systemic single ventricle by diverting SVC flow directly to the lungs, resulting in much improved myocardial function. Although the balance of pulmonary to systemic flow is still a consideration at this stage, and hyperventilation will result in hypocarbia, decreased cerebral blood flow and thus decreased SVC and PA flow which results in arterial desaturation, it is after the bidirectional cavopulmonary connection that elective non-cardiac procedures should be performed. These may include procedures such as cleft lip and palate repair, craniosynostosis repair, or other common procedures.
The third stage of HLHS palliation is the Fontan operation, whereby the inferior vena cava blood is routed to the pulmonary arteries, bypassing the right ventricle to form a total cavopulmonary connection, either intracardiac (lateral tunnel Fontan), or by an extracardiac conduit. This is normally performed at 18 months-4 years of age and results in a patient with a completely unloaded systemic single right ventricle, and no pumping chamber for the right side of the heart. Instead, blood flow into the thorax and out through the pulmonary arteries depends on the small transpulmonary pressure gradient from the vena cava into the pulmonary arteries, across the lungs, and into the pulmonary veins. This flow is augmented by normal breathing and its negative thoracic pressure augmenting the transpulmonary pressure gradient. After the Fontan operation, signs and symptoms of right sided cardiac failure may occur, such as pleural effusions, ascites, and lower extremity edema. Although after the Fontan the patient may no longer be cyanotic, because of the high right sided pressures a 3-5 mm fenestration is often created in the Fontan tunnel, to lower right sided pressures. Systemic cardiac output is augmented at the expense of a small amount of right to left shunting, producing patients with varying degrees of desaturation. It is important to determine if the patient has a fenestration when presenting for an anesthetic. After the Fontan operation, patients may be intolerant of excessive positive pressure ventilation as commonly performed during general endotracheal anesthesia, because it reduces the transpulmonary pressure gradient and thus flow into the Fontan circuit, resulting in low cardiac output. In addition, hypovolemia from prolonged fasting, third space loss, or blood loss is not well tolerated. These patients merit careful evaluation and planning for anesthesia, and should be monitored postoperatively in an intensive care setting after major interventions.

Other forms of single ventricle such as tricuspid atresia have been treated with the Fontan operation since the 1970’s. These patients are often adults with the older Fontan configurations, such as a direct atrio pulmonary connection between the right atrial appendage and the pulmonary artery. These patients often have severe refractory atrial arrhythmias and signs of significant right heart failure. Before anesthesia these patients need careful evaluation, and many undergo Fontan conversion to a more hemodynamically favorable arrangement, or heart transplant.

Ventricular Septal Defects

VSDs are the most common single isolated defect, and are a component of many more complicated defects as well. In isolation, VSDs range from tiny asymptomatic muscular VSDs seen at birth that close spontaneously, to large or multiple “Swiss cheese” defects in the ventricular septum that produce severe symptoms and signs of congestive heart failure in early infancy. The degree of left to right shunting depends on the size of the defect, and the relative ratios of pulmonary to systemic pressure and resistance and the compliance of right and left ventricles. Neonates and infants with large unrepaired defects may have significant cardiomegaly and myocardial dysfunction, and in addition if intubated and hyperventilated with high FiO2 may deteriorate further from increased pulmonary blood flow, steal from the systemic blood flow, and further increases in end-diastolic volume. Patients with large defects who are unrepaired can progress to Eisenmenger Syndrome. Patients with smaller defects are much more hemodynamically stable, and patients with repaired VSDs and no residual defects most of ten have normal myocardial function and can be treated as corrected well compensated patients for non-cardiac procedures.

Common Simple Lesions

Atrial Septal Defect

This common lesion may be essentially asymptomatic and not diagnosed until adulthood in some patients. The three major types are: 1. primum ASD which is low in the atrial septum and usually associated with atroventricular canal and repaired in the first five years of life; 2. secundum ASD in the center of the atrial septum, often closed in the cardiac catheterization laboratory with ASD closure devices; 3. sinus venosus ASD, high in the atrial septum near the superior vena cava and most often associated with partial anomalous pulmonary venous return. The latter two types of ASD may be undiagnosed until adulthood; pulmonary vascular disease usually will not develop until the 4th
or 5th decade or later. These patients may come to medical attention because of transient ischemic attacks or strokes due to right to left movement of platelet fibrin thrombi during valsalva maneuver. If unrepaired, besides assessment for cardiac symptoms, meticulous attention needs to be paid to avoid introduction of air via intravenous infusion, or during the surgical procedure itself.

**Patent Ductus Arteriosus**

A small patent ductus arteriosus (PDA) is usually asymptomatic, and may not warrant treatment. Larger PDA produce significant left to right shunting, and are closed, often in the cardiac catheterization laboratory with PDA closure devices. Large PDA are often components for more complex cardiac lesions and are addressed during surgery. If isolated and not suitable for catheterization laboratory closure, they are closed via left thoracotomy, and the patient is treated as a well compensated simple cardiac patient for future anesthetics. If a PDA is large, and not closed in childhood, pulmonary vascular disease and even Eisenmenger Syndrome can develop, and these patients are approached with great caution.

**Preoperative Evaluation**

A thorough history and physical examination, focusing on cardiac signs and symptoms, previous surgical and catheterization procedures, and available data such as recent echocardiograms, is important for all patients with CHD presenting for non-cardiac procedures. Cardiac rhythm status is important to determine, especially in older patients with residual defects or in single ventricle patients. Common medications in the CHD population include ACE inhibitors for single ventricle patients, or those with significant CHF or mitral regurgitation; beta blockers for TOF patients or those with atrial arrhythmias such as paroxysmal supraventricular tachycardia; amiodarone for patients with significant atrial or ventricular arrhythmias; and diuretics for patients with CHF. Digoxin is rarely used in the modern era because of its lack of effectiveness in either the adult or pediatric population. Endothelin antagonists, phosphodiesterase-5 inhibitors, or prostaglandin analogs are used in pulmonary hypertensive patients. Many patients with CHD are taking aspirin or other antiplatelet therapies to decrease risk of thrombus formation in shunts or conduits. Finally, some patients are taking coumadin for mechanical cardiac valves, which will merit careful planning of perioperative anticoagulation regimens. Many patients, particularly adults with CHD will have implanted transvenous or epicardial pacemakers or automated defibrillators, and it is critical to understand the patient’s underlying cardiac rhythm, the reason for placement of the device, and the current modes and settings of the device.

Echocardiography is the mainstay of diagnostic testing in the CHD population, and the latest echocardiography results should be reviewed. If there is any history of cardiac rhythm abnormality a recent ECG or Holter examination should be reviewed. Cardiac catheterization is most often performed for interventional procedures, but valuable diagnostic data is also available from this modality. Cardiac MRI has assumed an increasingly important role to assess anatomy, function, and progression of pathophysiology, and results should be reviewed.

A frequent question in the preanesthetic evaluation of the CHD patient for non-cardiac procedures is whether the patient should be evaluated by their cardiologist prior to the procedure. In general, if the patient has a simple or moderately complex lesion that has been completely corrected and is well compensated and so is followed by their cardiologist on an infrequent basis, a standard preanesthetic visit without a cardiology consultation is appropriate. A patient with a moderately complex lesion who is not well compensated, any cyanotic or single ventricle patient or patient with a complex lesion needs a recent cardiology evaluation that usually includes at least an echocardiogram, within no more than 3-6 months before the surgery. If the patient’s condition has changed significantly since the last evaluation, they should be seen again by the cardiologist.
In general, all cardiac medications should be continued through the perioperative period for most types of surgeries. Low dose ASA use is not a contraindication for most simple, superficial surgeries, but if the surgery is major, including intracranial, a discussion with the patient’s cardiologist and surgeon should take place and ASA is usually stopped 7-10 days preoperatively. Standard NPO orders apply to the CHD population including clear liquids up to 2 hours before anesthesia, breast milk up to 4 hours before, and infant formula, milk, or solids up to 6 hours before. It is critically important that patients with cyanotic lesions, shunt-dependent patients, and those with outflow tract obstruction are not left NPO for long periods of time. Hypovolemia can be a critical problem for these patients, especially with induction of anesthesia and institution of positive pressure ventilation. These patients should be scheduled early in the day, and if there are delays should be fed clear liquids until 2 hours before induction.

**Conduct of Anesthetic**

The setting for the non-cardiac procedure for the well compensated patient with a simple or moderately complex lesion can be in a normal well equipped and staffed community hospital setting. However, with less well compensated patients and complex and single ventricle patients, the procedures should be done in centers with expertise in CHD, and necessary backup support in case these patients deteriorate. In general, the more complex patients can have outpatient surgery, but admission to the hospital must be readily available. For complex patients having major surgery, postoperative care in an ICU is essential and must be planned in advance.

Standard monitors including ECG, non-invasive blood pressure, pulse oximetry, and capnography are essential for all procedures, including diagnostic imaging procedures, where end-tidal CO₂ can be monitored via a nasal cannula for sedation procedures. The decision to institute more invasive monitoring such as invasive arterial pressure, central venous pressure, continuous central venous oxygen saturation monitoring, or cerebral/somatic near-infrared spectroscopy for brain and tissue oxygenation, is anesthesiologist-dependent to a large extent and depends on the assessment of the potential for hemodynamic and respiratory instability due to the patient’s pathophysiology and the invasiveness of the planned procedure. Patients with significant baseline myocardial dysfunction, pulmonary hypertension, or cyanosis often benefit from preoperative intravenous access if possible.

Any anesthetic and sedation technique may be used in CHD patients, with careful attention to the particular pathophysiology of the patient and the desired hemodynamic goals. Premedication, often with oral midazolam, is well tolerated. Since halothane is no longer available, inhalation induction with sevoflurane is appropriate and well tolerated for many CHD patients. Propofol may be used for induction and maintenance in CHD patients, with careful attention to vaso- and vasodilatation produced by this agent. Ketamine, either IM or IV, is a very useful agent for many of these patients, preserving myocardial function and providing sedation and analgesia. Etomidate has little effect on myocardial contractility and hemodynamics, and is an excellent agent for the patient with impaired myocardial function. Dexmedetomidine is increasingly used for sedation in patients with CHD, and is usually well tolerated if the patient can withstand the bradycardia, and hypotension that sometimes results from the use of this agent. Any opioid can be used, and single shot caudal and nerve block techniques can also be used, even with low dose ASA use. Major neuraxial techniques, i.e. lumbar/thoracic epidural and spinal are best avoided with ASA use.

Airway management may range from sedation with spontaneous respiration, to mask or LMA general anesthesia, to endotracheal anesthesia, again being mindful of the effects of hyper- or hypocarbia, and positive pressure ventilation for the individual patient. With endotracheal anesthesia, the decision to extubate at the end of the procedure of course must take into account the severity of the underlying pathophysiology, and the magnitude of the surgical procedure. In single ventricle infants undergoing major abdominal procedures, for example, it is very often prudent to ventilate the patient in the early postoperative period.

Pacemakers and defibrillators must be interrogated before the procedure, and a discussion held with the patient’s cardiologist as to the underlying cardiac rhythm and reason for placing the device. In general, the pacemaker should
be converted to asynchronous mode just before the surgery, to avoid electrocautery interference inhibition of the pacemaker in demand mode. The defibrillator function must also be turned off, and external defibrillator capability immediately available. The device settings are restored as soon as possible after the completion of the procedure.

Common Non-Cardiac Procedures

Although CHD patients may undergo any surgical or diagnostic procedure, there are several common procedures that merit discussion. Young CHD patients often present for dental restorations and extractions under general anesthesia, due to the need for excellent dental hygiene to prevent infective endocarditis. A thorough preoperative evaluation is essential, and these procedures must be performed in the hospital setting with adequate expertise and backup in case of decompensation. Laparoscopic procedures such as fundoplication and gastrostomy tube, and cholecystectomy, are performed in CHD patients, but careful attention must be paid to the effects of CO₂ insufflation on PaCO₂, and increased intra-abdominal pressure. The combination of acute hypercarbia and decreased venous return is not well tolerated by single ventricle infants and patients with a Fontan circulation, and careful monitoring, or consideration for an open procedure are important. Scoliosis surgery is often done in patients with CHD, and must be very carefully planned, especially in patients with the Fontan circulation, who do not tolerate hypovolemia and hypotension. Careful monitoring, blood and volume replacement, and careful consideration of anesthetic technique in light of the need for spinal cord monitoring must be performed for these complicated patients. Craniofacial surgery, especially cleft lip and palate, and craniostenosis repair, are done in the CHD population, with careful consideration for prevention of air embolus, and monitoring and replacing blood loss. As noted above, in single ventricle infants, elective surgery is best performed after the cavopulmonary connection, which yields a much more stable circulation to withstand the stresses of major surgery. Diagnostic imaging procedures, especially MRI, are commonly performed in the CHD population, and either sedation or general anesthesia techniques can be used, again with careful attention to the hemodynamic and respiratory goals of each individual patient.

Infective Endocarditis Prophylaxis

The American Heart Association significantly changed its guidelines for prevention of infective endocarditis (IE) in 2007, resulting in a narrowing of the indications for administering IE prophylaxis. This has resulted in confusion for patients, parents, surgeons, and even cardiologists, and it is important to understand the new indications, which were based on an extensive review of the data and discussions among a large panel of experts. The patient must have BOTH a cardiac indication, and a surgical/procedural indication. The major cardiac indications are: 1. prosthetic cardiac valve or material; 2. previous IE; 3. congenital heart disease, but ONLY a) unrepaired cyanotic CHD including palliative shunts and conduits; b) completely repaired CHD with prosthetic material or device during the first 6 months after the procedure; c) repaired CHD with residual defects at or near the site of a prosthetic patch or device; 4. cardiac transplant recipients who develop valvulopathy. The surgical/procedural indications are 1. all dental procedures that involve manipulation or the gingival tissue or perforation of the oral mucosa; 2. respiratory tract procedures or procedures on infected skin, or musculoskeletal tissue. IE prophylaxis is not recommended for simple gastrointestinal or genitourinary procedures where the mucosa is not incised, i.e. simple endoscopy and cystoureteroscopy, but is recommended for surgery where mucosa is incised. For dental prophylaxis, a single dose of ampicillin 30-60 minutes before the procedure, or as soon as IV access is obtained, is the recommended regimen, with clindamycin, cefozolin, or ceftriaxone acceptable for penicillin allergic patients.
Summary

The large and growing population of patients who are living with CHD requires anesthesia care for the same types of non-cardiac surgeries and other procedures as the non-CHD population, and anesthesiologists will increasingly care for these patients in a variety of settings. Knowledge of the pathophysiology of the common CHD lesions, as well as careful preoperative assessment and preparation, and communication with the patient’s cardiologist and surgeon, are essential to provide optimal care in the best setting for these patients.

Bibliography


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