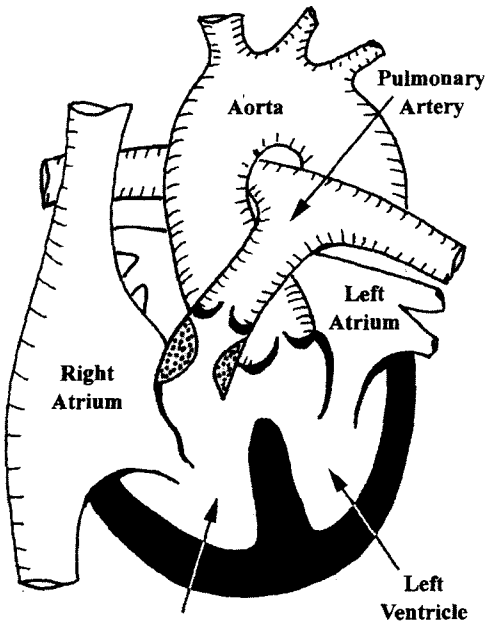

Right Ventricle Fig. 20.4 Schematic diagram of tetralogy of Fallot illustrating the infundibular stenosis (stippled area), ventricular septal defect, overriding aorta, and right ventricle hypertrophy.

Tetralogy of Fallot

Anatomy

Tetralogy of Fallot represents 10% of all congenital heart defects and is the most common form of cyanotic heart disease. A Danish scholar, Nicholas Steno, first described the defect in 1673, 200 years before Fallot. One hundred years later, numerous other reports were published from post-mortem cardiac examinations. The French physician, Arthur Fallot, was the first to make an accurate bedside diagnosis with later validation at post mortem. He later published a review of the French literature on the *maladie bleue* in 1888, his name then becoming popularly associated with the malformation[17] Tetralogy of Fallot comprises four anatomical abnormalities: (i) a large unrestrictive VSD; (ii) RVOT obstruction; (ill) overriding of the aorta above the RVOT; and (iv) RV hypertrophy (Fig. 20.4). In reality, there is a spectrum of abnormalities ranging from TOF with P A, where there is total obstruction to RV outflow, through TOF with PS (the "classic" tetralogy), to TOF with absent PV. Embryologically, TOF is believed to result from incomplete rotation and faulty partition of the conotruncus during septation.

The VSD is perimembranous, large (usually the same size as the aortic valve), and unrestrictive. It is also important to note that the cardiac conduction tissue lies in close proximity to the margins of the VSD and may be damaged during repair producing temporary or permanent heart block. Because of clockwise rotation, the aorta overrides the VSD and thus has a biventricular origin. Additionally, the aortic arch lies to the right in 25% of TOF patients and may be associated with mirror image branching of the head vessels. There may be an aberrant origin of the ipsilateral subclavian artery from the descending aorta in some patients, and rarely, there may be an isolated origin of the left subclavian artery from the pulmonary artery. These abnormalities have important implications when selecting the surgical approach for the placement of palliative shunts.

Coronary abnormalities occur in 5-12% of patients with TOF. Failure to detect these preoperatively can have serious consequences for a successful outcome because they may be damaged during 'Surgery. The most common abnormality consists of a left anterior descending artery that originates from the right coronary artery, and crosses the RVOT inferiorly. This arrangement makes it very susceptible to damage if the transannular incision is carried too far inferiorly across the RVOT. Indeed, an alternative surgical approach may be needed in relieving the subpulmonary obstruction, or a RV to MP A conduit may be required. Other coronary anomalies include a right coronary artery originating from the left coronary artery, and left coronary artery originating from the MP A. Precise definition of the coronary anatomy may be possible with echocardiography alone.[18] If there is uncertainty, then aortic root or selective coronary angiography may be used.

Other coexisting cardiac lesions include left SVC (LSVC), AV septal defect, PDA, ASD, and interrupted inferior vena cava. All of these require modifications to the surgical repair. It is particularly important to be aware of the presence of a LSVC because this usually drains into the coronary sinus in the RA. During surgery a venous drainage cannula is placed in the LSVC and this vessel is usually tied off at the end of the procedure. Therefore, a central venous line placed via the left internal jugular route will be rendered useless postoperatively.

Two important variants of TOF are P A with VSD and the absent PV syndrome. Pulmonary atresia with VSD is characterized by hypoplasia of the central and peripheral pulmonary arteries. The MP A may be absent or the branch PAs may be non-confluent or stenotic. Pulmonary blood supply is usually via MAPCAs. The surgical correction of this lesion is very different from that of classical TOF as described later in this chapter.J[9],20 The absent PV syndrome is characterized by combined PS and incompetence which *in utero* leads to increased pulsatile pulmonary blood flow *(PBF)* producing massive enlargement of the main and branch PAs. This produces the central characteristic feature of airway compression and tracheobronchomalacia. These babies typically present in the neonatal period with severe respiratory distress, cyanosis, and air trapping. Tracheal intubation with high levels of positive end-expiratory pressure *(PEEP)* may be useful in stenting the airways. Prone positioning may also be useful in relieving some of the obstruction. Infants with significant lung disease require urgent surgical intervention. However, symptoms commonly persist due to the underlying intrinsic airway abnormalities and such patients may need long-term ventilator support.

In the common form of TOF, the obstruction to the RVOT usually has dynamic and fixed components. The dynamic component consists of hypertrophied infundibulum and muscle bundle fibers in the RVOT. The hypertrophy occurs in response to the pressure load on the RV. Fixed obstruction

may be valvular, consisting of a thickened, hypoplastic, and often bicuspid PV. In 15-25% of patients the PV is atretic producing complete obstruction to the RVOT. These patients require an alternative of *PBF* such as a PDA, bronchial arteries or MAPCAs.

Beyond the PV, impedance to RV outflow occurs due to abnormalities in the pulmonary arterial tree. There is usually some degree of central pulmonary artery hypoplasia in all patients. There may also be localized narrowing of the MP A or BP As. Atresia or discontinuity of the MP A or BP As may occur, which will further complicate surgical correction, as restoration of continuity or augmentation of the pulmonary arteries will be required.

There is a weak association of familial inheritance of TOF. Indeed TOF is associated with major extracardiac malformations and may occur as part of a syndrome. Some examples are the V ACTERL (vertebral, vascular, anal, cardiac, tracheoesophageal, renal, and limb anomalies) association, DiGeorge syndrome, velocardiofacial syndrome, and CHARGE (coloboma, heart anomaly, choanal atresia, retardation, and genital and ear anomalies) association. Recent genetic studies have shown that TOF is associated with chromosome 22q11 deletion (catch 22 syndrome). This chromosomal abnormality is also responsible for DiGeorge syndrome, velocardiofacial syndrome, and conotruncal anomaly face syndrome. In one study of TOF patients, the prevalence of 22q11 deletion was 13%. This deletion is considered to be the most common genetic cause of TOF-associated syndromes.[21]

The clinical manifestation of TOF ranges from extreme cyanosis at one end of the spectrum, because of profound right-to-left shunting through the VSD, to normal saturation for patients who have minimal RVOT obstruction and who exhibit a net left-to-right shunt. The latter group is sometimes known as "pink tets" because of the absence of cyanosis. They may even show signs of CHF from pulmonary overcirculation. The presentation of symptoms with TOF is determined primarily by the degree of RVOT obstruction because the large non-restrictive VSD effectively equalizes the pressures in both ventricles. If the VSD is restrictive RV pressure may be suprasystemic. The RV undergoes hypertrophy in response to the high afterload and the wall thickness may become similar to the LV. Right ventricle hypertrophy is undesirable for several reasons. It can lead to diastolic dysfunction; surgical correction of the VSD and RVOT obstruction becomes more difficult through a thickened ventricle; and it becomes more difficult to protect the hypertrophied RV during aortic cross-clamping, which may contribute to postoperative RV dysfunction. In order to limit RV hypertrophy, surgical correction is undertaken in early infancy.

With a non-restrictive VSD and equalization of RV and LV pressure, the main determinants of the degree of shunting are the relative resistances of the systemic and pulmonary circuits. Both of these change depending on various factors and, therefore, the degree of cyanosis will vary. Right ventricle outflow tract obstruction usually has a dynamic and fixed component; prodUcing different propensities for cyanosis. An acute form of this RVOT obstruction occurs during a hypercyanotic or "tet spell" producing severe cyanosis and hypoxemia, which can lead to syncope or a stroke. The obstruction is thought to result from infundibular spasm leading to an acute decrease in *PBF* and shunting of desaturated blood into the systemic circulation. T~~e spells can occur spontaneously, but are usually in response to crying, defecation, agitation, injury or fright which increases sympathetic tone leading to increased contractility producing infundibular spasm. As well as acute increases in RVOT obstruction, "tet spells" may be precipitated by an acute fall in systemic vascular resistance *(SVR).* This may happen during induction of anesthesia (especially if the patient is hypovolemic), and it is very important that the anesthesiologist is well prepared in advance to treat a spell. The goal of treatment is to use maneuvers (described later in this chapter) which reverse the direction of right-to-left shunting. Other factors which can worsen cyanosis are anemia, acidosis, infection, stress and posture. "Tet spells" in the awake patient are usually accompanied by hyperventilation secondary to the metabolic acidosis and the hypoxemia. Children classically adopt a squatting posture during a spell to alleviate discomfort. Squatting increases intra-abdominal pressure leading to increased venous return and RV preload, which helps to "enlarge" the RVOT. This position also increases *SVR,* which favors left-to-right shunting.

The presenting features of TOF are variable depending on the degree of RVOT obstruction. Prenatal diagnosis is possible with ultrasonography. In the neonate, cyanosis and the presence of a murmur will lead to further diagnostic evaluation. In newborns with critical PS and ductal-dependent *PBF,* the clinical presentation may be delayed until ductal closure. Very rarely, the baby will be in CHF from pulmonary overcirculation if there is only mild RVOT obstruction-the socalled "pink tet." Sudden severe desaturation will occur during a hypercyanotic spell characterized by infundibular spasm and severe reduction in *PBF.*

Physical findings are not specific for TOF. The degree of cyanosis will vary, and pulse oximetry will demonstrate low hemoglobin saturation. Clubbing is a relatively late finding. Cardiac auscultation reveals a crescendo-decrescendo systolic murmur best heard at the upper left sternal border. The intensity of the murmur will be diminished during a hyper-cyanotic spell. Chest radiograph shows a characteristic "boot

shaped" heart, which is a reflection of RV hypertrophy and a concave upper left heart border from a small or absent MP A. The lung fields are oligemic from diminished blood flow. The ECG usually shows RV hypertrophy and right axis deviation.

Morbidity and mortality

Survival beyond the fourth decade is very rare in untreated patients. Without surgery, 25-35% of children with TOF die in the first year of life, 40-50% by year 3, 70-76% by year 10, 90% by year 21, and 95% by year 40. The outcome is even worse for the subset of patients with P A. Without surgery, most of these children do not survive beyond infancy. Even those children who are completely palliated show delayed growth and development compared with their normal counterparts. This is usually due to the associated non-cardiac conditions. The clinical course of patients with TOF is determined primarily by the degree of PS. Mortality in untreated patients is usually a result of hypoxemia or its hematologic consequences, or the result of problems such as endocarditis or brain abscess. With complete repair in early infancy or childhood, over 85% of patients are expected to survive to adulthood.

In the present era, most of the diagnostic information needed for surgical decision-making can be obtained from echocardiography, with cardiac catheterization only necessary in selected cases. As well as delineating the anatomy of the heart chambers, PV, MPA, BPAs, and aortic arch, Doppler studies can demonstrate the severity of RVOT obstruction and the location, type, and number of VSDs. In addition, it aids in localizing alternative sources of *PBF.* Coronary anatomy can also be described in many cases. However, echocardiography cannot reliably image pulmonary artery anatomy beyond the proximal branches. If echocardiography is insufficient in providing all the information needed for determining the surgical plan, then cardiac catheterization and angiography are performed. If only a palliative procedure is planned, echocardiography may be adequate. However, for planned complete surgical correction, and for patients who have had a palliative procedure (where there may be distortion of the vascular anatomy), cardiac catheterization may be indicated. Cardiac catheterization is not a benign procedure in the tetralogy patient, especially for those at high risk for hypercyanotic spells which may be precipitated by manipulation of the catheter across the RVOT. Such patients should be managed by experienced personnel with appropriate emergency drugs and equipment immediately available for the management of "tet spells" in the catheterization laboratory.

Interventional catheterization procedures are having an increasing role in the management of CHD. In some centers, balloon dilation of the RVOT is considered as a palliative alternative to a systemic to pulmonary artery shunt.[22] Correct technique avoids the disadvantages of surgical intervention, including distortion of pulmonary vascular anatomy, which can occur with shunting procedures. Balloon dilation and stent placement also has an important role in those patients who have undergone complete surgical correction, but are left with residual pulmonary artery or conduit stenosis.

All patients diagnosed with TOF require some form of surgical intervention. However, there is ongoing debate regarding the timing of repair, and whether this should be done as a staged technique (palliative shunt followed by complete repair) or primary total repair.[23],24 There is a trend now towards early total correction, whether the patient is asymptomatic or not, with some institutions performing surgery in the neonatal period with good results.[2s-27] Others prefer to adopt a slightly more conservative approach, opting for repair at 3-6 months of age or even later.[28],29 Other factors to consider are the institution's capability of providing perioperative critical care of neonates and small infants undergoing complicated cardiac surgery, or specific anatomical features that are contraindications to primary repair. Examples of unfavorable anatomy include the presence of coronary abnormalities such as the left anterior descending arising from the right coronary and crossing the RVOT, the presence of multiple VSDs, and inadequate pulmonary artery anatomy. In these cases it is reasonable to place a palliative shunt and allow the baby to grow, facilitating the eventual complete repair. The two-stage approach does have certain disadvantages.[3o] It subjects the baby to an additional surgical procedure with attendant risks and complications. These include injury to the recurrent laryngeal and phrenic nerves, inadequate or excessive *PBF* requiring subsequent shunt revision, potentially fatal shunt obstruction, and potential distortion of the pulmonary artery at the anastomotic site. If the shunt is placed centrally through a median sternotomy, subsequent surgery may be more hazardous due to increased risk of damage to the heart or major vascular structures during dissection. As far as timing of surgery, there are certain disadvantages to operating in the neonatal period. In addition to the risks of performing complicated surgery on tiny neonates and the effects of CPB (and possibly circulatory arrest) on immature organ systems, the surgical procedure is technically more challenging. Although most centers perform the repair using a transatrial-transpulmonary approach, smaller patients more commonly require a ventriculotomy to facilitate repair. Ventriculotomy may result in late RV dysfunction and dysrhythmias. Nevertheless, some centers strongly favor this approach.[25] Proponents of early repair point out the desirability of operating before significant RV hypertrophy has occurred. Right ventricle hypertrophy may require extensive myectomy, making VSD closure more difficult. Severe RV hypertrophy may also impair myocardial protection during the period of aortic cross-clamping. Early repair may also lead to more normal growth and development of

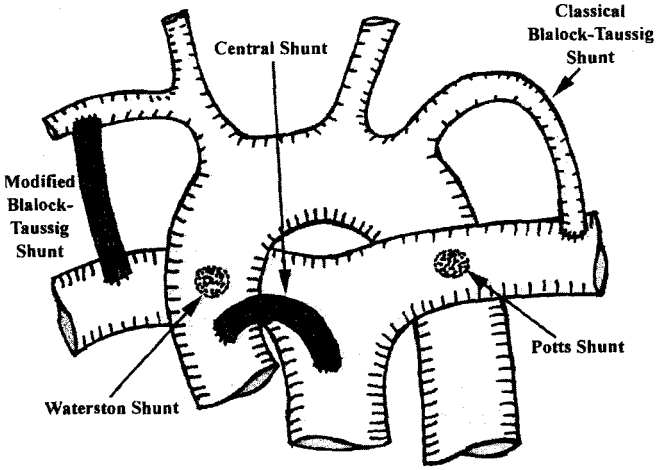

Fig. 20.5 Diagram showing the various types of shunts used to increase pulmonary blood flow. The modified Blalock-Taussig shunt and central shunts are the only shunts used in the modern era.

the pulmonary vasculature and avoids the long-term effects of cyanosis and hypoxemia. It is not possible to perform a complete repair on all patients with TOF and the debate over the optimal surgical approach will continue.

Surgical palliation

The proposal by Blalock and Taussig to anastomose the subclavian artery to the pulmonary artery in an end-to-side fashion to alleviate cyanosis resulted in the first successful palliation of TOF in 1944. This "classic" Blalock-Taussig (BT) shunt is very rarely used today. Potts and Waters ton later described direct aorta to pulmonary artery shunts. Although these shunts prOVided good palliation, their size was difficult to control and commonly resulted in too much *PBF.* They were also extremely difficult to take down during corrective surgery and thus were largely abandoned. A "modified" BT shunt (MBTS) is created by interposition of a graft of synthetic material, usually polytetrafluoroethane, between the subclavian artery or brachiocephalic artery and the ipsilateral pulmonary artery. This is the most common palliative procedure carried out in the current era (Fig. 20.5). The advantages of the MBTS are many: (i) it preserves blood flow to the arm; (ii) it can be used on either side, although most are done on the right side because the pulmonary anastomosis can be placed more centrally allowing easier control of the shunt during subsequent repair; and (iii) it avoids excessive *PBF* when appropriately sized. A central shunt, between the ascending aorta and the MP A, using graft material is used as an alternative to the MBTS when the vascular anatomy precludes placement of the latter.

Complete surgical repair

Lillehei carried out the first successful repair of TOF in 1954.

There are three major goals of the TOF repair: (i) maximal relief of RVOT obstruction; (ii) separation of the systemic and pulmonary circulation by closure of the VSD; and (iii) preservation of RV function in the short and long term. It is particularly important to delineate the coronary anatomy, determine the levels of obstruction of the RVOT, and the size and continuity of the pulmonary arteries. The details of the operative procedures are well described elsewhere.[31],32 After cardioplegic arrest the repair is done using a transatrialtranspulmonary approach. Right ventriculotomy is avoided if possible to preserve RV function.[33] The PV is examined through a longitudinal incision in the MP A, and if necessary, a commissurotomy is performed. The RVOT is exposed through the RA and TV and resection of the infundibular septum is carried out. Hegar dilators are passed through the TV into the MP A to calibrate the RVOT. If the size of the RVOT is judged to be inadequate, the MP A incision is extended downwards across the annulus and onto the RV free wall. The VSD is then closed through the RA. Any ASD is also closed although some surgeons prefer to leave an atrial communication to act as a "pop off' valve in case of severe RV dysfunction postoperatively. This will prodllce some hypoxemia via right-to-Ieft shunting. The adequacy of repair is assessed using several methods. The RV : LV pressure ratio can be measured, less than 0.75 being considered acceptable. It is important to emphasize, however, that pressure measurements in the early post-CPB period do not reflect measurements made at follow-up and may lead to unnecessary revisions of the repair.[34] Transesophageal echocardiography (TEE) is useful in demonstrating gradients across the RVOT, showing residual VSDs, and assessing ventricular function.[35],36 Blood gas measurements from the vena cava and the MP A are also useful in detecting residual shunts.

In patients who have a coronary artery which crosses the RVOT, a transatrial-transpulmonary repair is still feasible if the transannular incision is limited.[37] Many of these patients, however, require a valved RV-P A conduit to avoid damage to the coronary artery.

Management for surgical palliation

Many of these patients are critically ill due to severely reduced *PBF.* They may be mechanically ventilated and receiving an infusion of PCEl to maintain ductal patency. If intravenous access is available then anesthesia can be induced with a combination of ketamine and fentanyl and maintained with low concentrations of a volatile agent. It is important to maintain adequate *SVR* in order to limit right-to-left shunting through the VSD; in this regard, sevoflurane is a good choice as this agent has the least effect on *SVR.38* The myocardial depressant effect of volatile agents is also useful in limiting infundibular spasm. Low *SVR* is treated with phenylephrine or

norepinephrine; and preload is augmented with fluid boluses. It is important to avoid most inotropes, as these will worsen infundibular spasm by increasing heart rate and contractility. If there is no intravenous access, induction can be carried out rapidly and smoothly with sevoflurane. An alternative is to use intramuscular ketamine in unstable patients. Central venous access is obtained for the infusion of fluids and vasoactive agents. A radial arterial line is placed on the side opposite to that of the MBTS in order to get a true assessment of the blood pressure because after the shunt is opened there may be significant "steal" from the ipsilateral subclavian artery. A femoral arterial line can also be placed as long as care is taken to observe for evidence of distal lower extremity ischemia.

Most MBTS are performed via a thoracotomy. The median sternotomy approach is used when the surgeon feels that the patient will not tolerate lung retraction or side-clamping of the P A, when there is a possibility that CPB may be required, and for central shunt placement. Low dose heparin (l00 U /kg) is administered prior to shunt placement. Lung retraction can severely impair oxygenation and ventilation, and intermittent reinflation may be required. Similar decompensation can occur during partial clamping or obstruction of the PA during the construction of the anastomosis. Such decompensation is managed with fluids, vasopressors, and ventilation adjustments. However, attempting to normalize Pco_2 during single lung perfusion may overdistend the dependent lung, increasing *PVR* and impairing venous return. For central shunts, partial clamping of the ascending aorta is required, but it may be poorly tolerated in the presence of LV dysfunction. Inotropic support with dopamine is usually helpful. Once the shunt is open, oxygen saturation usually improves immediately. However, blood pressure may drop significantly, requiring volume infusion and vasopressors. If the diastolic pressure becomes very low, coronary flow will be reduced and ischemic changes may be seen on the ECG. Ventilation and inspired oxygen are adjusted to mimic spontaneous, non-anesthetized values for an accurate assessment of the shunt flow. An oxygen saturation of near 80% is optimal as this represents balanced pulmonary and systemic blood flow. A high saturation suggests pulmonary overcirculation and the shunt size may have to be reduced. Conversely, a low saturation suggests inadequate *PBF,* and a larger diameter shunt may be needed. In cases of persistent hypoxemia after apparently uneventful shunt placement, it is important to rule out the possibility of endobronchial intubation because failure to do so may lead to unnecessary shunt revision or even sternotomy. After chest closure, the patient is transferred to the cardiac ICU where mechanical ventilation is typically necessary for at least 12-24 hours. Increased *PBF* can cause unilateral pulmonary edema or pulmonary hemorrhage. Diastolic hypotension may cause myocardial ischemia, requiring close monitoring of intotropic medications and volume. Other complications include injury to the

phrenic and recurrent laryngeal nerves, Horner's syndrome, chylothorax, and shunt thrombosis. Patency of the shunt can be clinically confirmed by briefly disconnecting the patient from the ventilator and auscultating over the end of the endotracheal tube. The murmur is transmitted via the tracheal tube due to the proximity of the shunt to the bronchus. A low dose heparin infusion is started (8-10 U/kg/hour) to maintain shunt patency when the risk of post-surgical hemorrhage has diminished. After enteral intake has begun, the patient is prescribed aspirin until the time of corrective surgery. Platelet transfusions are generally avoided for patients undergoing shunt placement due to the risk of shunt thrombosis.

Management for complete repair

Procedures for complete repair necessitate additional considerations of the effects of CPB. The anesthetic induction does not differ from that described above. Generally, we utilize a total dose of fentanyl to 20-50 Ilg/kg and administer inhalational agents to supplement anesthesia. A recent study showed that a ketamine infusion provided more hemodynamic stability by preserving *SVR* in the pre-CPB period when compared with isoflurane.[39] The lower dose of fentanyl usually allows for extubation within 4-8 hours after surgery. Transesophageal echocardiography is used for almost all patients. However, it is important to monitor the effects of probe insertion on ventilation. The TEE probe may compress the trachea or mainstem bronchi, compromising ventilation, requiring removal. If TEE is not possible or unavailable, epicardial echocardiography can be performed post-CPB to assess repair. In addition to routine monitors, brain oxygen saturation trends can be followed with near infrared spectroscopy. Other monitoring alternatives include electroencephalography and transcranial Doppler. Neurologic monitoring is discussed elsewhere in this volume.

Patients who do not have a palliative shunt may develop a "tet spell" during the pre-CPB period and without prompt and aggressive treatment severe hypoxemia may progress to cardiovascular collapse. Particularly vulnerable periods are during anesthetic induction before surgical stimulation, when reduced sympathetic tone causes a fall in the *SVR* leading to increased right-to-Ieft shunting. Manipulation of the great vessels by the surgeon may also result in sudden rightto-left shunting. The primary goal of management of a spell is to correct the hypoxemia by relieving the infundibular spasm and reversing the shunt. Some or all of the following maneuvers can be employed:

1 Administer oxygen. This does not relieve the spasm but helps reduce hypoxic pulmonary vasoconstriction.
2 Phenylephrine, 5-10 Ilg/kg and titrated to effect to increase *SVR.*
3 Volume infusion to support the blood pressure and increase right heart filling thereby reducing RVOT obstruction during systole.

4 Compress the abdomen to directly compress the aorta and place the child in a knee-chest position to increase the *SVR.*

5 Titrate esmolol, 50 Ilg/kg to effect. The negative inotropic effect and reduction in heart rate will help to reduce the infundibular spasm. Propranolol (0.1 mg/kg given slowly) also works but is slower in onset.

6 Increase the depth of anesthesia with a volatile agent to decrease contractility thereby reducing the infundibular spasm. Although halothane is traditionally used for this purpose, a recent echocardiographic study showed that

6'~~as less effect on the *SVR* index than halothane or iso urane at 1.5 minimum alveolar concentratjon[38] and therefore may be superior. Isoflurane is a poor choice because it is a potent vasodilator and also causes tachycardia, which increases contractility. Although morphine is frequently recommended for the treatment of "tet spells" in the awake patient, it produces excess vasodilation under anesthesia and is, therefore, not recommended.

7 If all these measures fail and the patient continues to deteriorate, the chest may have to be opened quickly, and the aorta may need to be compressed to reverse shunting. During the rewarming phase of CPB, preparations are

made for weaning from CPB. In general, three problems may be anticipated:

1 Right ventricle dysfunction, especially if the transannular incision was extended down the RV free wall. The mainstays of treatment are fluid loading to higher filling pressures, inotropic support, and reduction of RV afterload. Dopamine at 5Ilg/kg/minute is started when rewarming commences. ~cerin is also added at 2 Jl.g/kg/minute to decrease RV afterload. Other acceptable altema-

-' tives for inotropic s~port are dobutamine or milrinone. These also have beneficial effects on the pulmonary vasculature. Ventilation is adjusted to reduce *PVR* prior to weaning.

2 Arrhythmias and heart block. These are common after VSD repairs because of the close proximity of the conduction system. Epicardial pacing may be needed to accomplish weaning from CPB. In most instances heart block is a transient phenomenon due to the edema around the VSD patch. If it does not resolve after 7-10 days, permanent pacing may be required. Junctional ectopic tachycardia is seen occasionally, although the onset is usually 12-24 hours later. This is characterized by A V dissociation and rapid junctional rates as high as 200-230 beats/minute. Treatment consists of cooling the patient to 34-35°C, and drug therapy with amiodarone or procainamide. Atrial overdrive pacing can also be used to re-establish A V synchrony.

3 Post-CPB bleeding. Coagulopathy results from hemodilution of coagulation factors and the effects of CPB on platelet number and function, and may require transfusion of multiple component blood products. The use of antifibrinoly-

tics such as e-aminocaproic acid, or protease inhibitors such as aprotinin, may reduce post-CPB bleeding and minimize the use of blood products.

Once the chest is closed the patient is transferred to the ICU. Analgesia is provided with a continuous infusion of morphine at 20-40 Ilg/kg/hour, supplemented with intermittent boluses of midazolam for sedation. Hemodynamically stable patients with minimal bleeding are good candidates for early extubation, usually within 4 hours. After the patient is extubated, analgesia can be reliably provided with a combination of acetaminophen and a non-steroidal anti-inflammatory agent such as ibuprofen, with morphine as needed.