Palliative or corrective heart surgery is currently available for most forms of congenital heart defects. Postoperatively, various electrocardiographic patterns are present. There is a significant incidence of postoperative arrhythmias, many of which are sudden, unexpected, and life-threatening. Improved operative survival, as well as an increased awareness of postoperative arrhythmias, has led to an apparent increase in these arrhythmias. New monitoring techniques and electrophysiologic studies have helped to identify the extent of significant postoperative arrhythmias in patients with congenital heart defects.

The risk of developing postoperative arrhythmias is present in all patients who undergo heart surgery. In certain defects, the propensity to develop arrhythmias is greater, and specific types of arrhythmias appear to be predominant. Children who undergo surgical correction of tetralogy of Fallot or transposition of the great arteries have an especially high incidence of postoperative arrhythmias. Repair of atrial septal defects, ventricular septal defects, and atrioventricular canal defects also result in a relatively high incidence. In addition, as surgical survival of patients with complex congenital heart defects improves, the incidence of postoperative arrhythmias will most likely increase.

**Tetralogy of Fallot**

Considerable controversy has existed over the significance of electrocardiographic changes present following repair of tetralogy of Fallot over the past decade. The typical preoperative electrocardiographic manifestations of tetralogy of Fallot are right atrial hypertrophy and right ventricular hypertrophy. Postoperatively, the mean QRS vector axis usually shifts leftward. Right bundle branch block is the most common electrocardiographic abnormality with a reported incidence of 59–100% [1, 2]. Right bundle branch block with left anterior hemiblock is also seen in these patients with a reported incidence of 7–25% [3–7]. Permanent complete AV block currently occurs in 1% of patients [4, 5, 8].

A relatively high incidence of ventricular arrhythmias including premature ventricular depolarizations and ventricular tachycardia has been noted postoperatively in patients with tetralogy of Fallot [9–11]. The incidence of sudden death has varied from 2–3% [4, 8, 10]. Recent studies have indicated an association between the previous presence of ventricular arrhythmias and sudden death [10, 12].

Several investigators have discussed the etiology of right bundle branch block and attributed the right bundle branch block to injury of the proximal right bundle branch produced by repair of the ventricular septal defect [3–13]. Other investigators utilizing intraoperative electrographic recordings have shown that the right bundle branch block may result from the ventriculotomy with disruption of distal branches of
the right bundle [1, 14]. Utilizing intraoperative epicardial and endocardial recording techniques in patients having transatrial repair of tetralogy of Fallot, Horowitz and co-workers [15] found that the right bundle branch block pattern may be explained by delay or block in the proximal right bundle branch (in the moderator band) or peripheral right ventricular Purkinje fibers. The pattern of right ventricular activation during right bundle branch block which followed repair of tetralogy of Fallot was studied in 20 patients, and the right ventricular specialized conduction system was mapped to identify the site of block in postoperative right bundle branch block. In 8 patients, right bundle branch conduction was interrupted proximally in the area of the ventricular septal defect. In 5 patients, right bundle branch conduction was interrupted in the area of the moderator band. In the remaining 7 patients, right bundle branch conduction was interrupted in the area of the terminal fascicular network. The surface electrocardiogram was not useful in determining the site of the right bundle branch block. At least three distinct types of postoperative right bundle branch block exist and can be identified by differences in right ventricular activation. The patterns of right ventricular activation during these three types of conduction delays are shown in figure 10–1.

In our institution, 38 patients have undergone complete electrophysiologic studies after correction of tetralogy of Fallot. All patients except one had right bundle branch block. Catheter endocardial mapping of right ventricular activation was performed to identify the site of block in the right ventricular conduction system. Activation of the right ventricular apex within the first third of the QRS was considered indicative of distal right bundle branch block, whereas later activation was considered to indicate proximal right bundle branch block. This method is similar to that of Sung [16] who defined distal right bundle branch block when the interval between the onset of the QRS and the activation of the right ventricular apex was