Exercise Testing in Patients With Arrhythmias

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Exercise testing has become an important method for evaluation of pediatric patients with known or suspected arrhythmias. It has proven useful in patients with exercise-induced symptoms, patients with congenital heart disease, and patients with pacemakers. Exercise has predictable effects on the normal electrocardiogram. Exercise can also bring out abnormalities in cardiac rhythm that may not be present at rest. The results of exercise testing can provide information that directs further therapy or evaluation. Exercise testing also helps to evaluate the efficacy of antiarrhythmic therapy in some patients.

Exercise testing has become a popular, noninvasive tool in the evaluation of cardiovascular disease. The first clinically popular testing used the Master’s 2-step test developed in 1929 (11). Exercise testing was first used by Wood and Wolferth in 1931 (4) to evaluate for coronary artery disease. It was abandoned, though, as it lacked sufficient sensitivity. In general, in the U.S. it has been replaced by graded exercise testing on a motor driven treadmill. In Europe and in many pediatric centers in the U.S., however, exercise testing is performed on a cycle ergometer (11, 22). Exercise testing in adults is most commonly used to elicit signs of myocardial ischemia manifested as changes in the electrocardiogram. In many pediatric exercise laboratories, though, patients with known or suspected arrhythmias constitute the most common group evaluated with exercise stress testing (22). Exercise testing can play a role in investigating symptoms brought on by exercise, such as syncope or palpitations, which may be caused by an arrhythmia (21).

Patients who are at increased risk for arrhythmias, such as some types of postoperative congenital heart disease, may have arrhythmias brought out by exercise. Patients with known or suspected arrhythmias may have arrhythmias induced by exercise, aiding in diagnosis. Other patients with a known arrhythmia can have a cessation or change in arrhythmia during testing. In some groups, exercise testing has proven useful in testing the efficacy of antiarrhythmic therapy. Pacemaker function can be evaluated by exercise testing, especially in patients with activity...
mode pacing. All of the above indications have been used in both clinical and research settings and will be discussed in this paper. Also, the normal exercise electrocardiogram and the use of exercise testing in arrhythmia evaluation at Texas Children’s Hospital will be reviewed.

**Normal Exercise Electrocardiography**

The first investigator of exercise electrocardiography was Einthoven, the inventor of the string galvanometer (the prototype of the electrocardiograph) (3). He found that the amplitude of both the P wave and QRS complex increased following exercise. Much has been learned about the normal exercise electrocardiogram since that time. Most of the collected data is from adult subjects, but the normal response in children appears to be similar. The heart rate increases as increasing work is performed until a maximum heart rate is reached (12). As the heart rate increases, the absolute P wave amplitude increases but the P wave axis does not change. This is thought to reflect an increase in right atrial pressure and volume. The lateral septal Q wave increases in amplitude, but at high heart rates the magnitude of the maximal depolarization decreases slightly. The magnitude of the deviation of the J point from the baseline increases and the ST segment shifts superiorly and rightward. This creates the J point depression with a rapidly upsloping ST segment associated with exercise (Figure 1) (15). Quickly following exercise, the T wave broadens and increases in amplitude. This returns to normal by 3 minutes into the recovery period.

![Figure 1](image.png)

**Figure 1** — Resting and exercise electrocardiogram from Lead V5 in a 14-year-old girl being evaluated for exercise-induced bronchospasm demonstrating physiologic J point depression.
Effects of Exercise on Cardiac Rhythm

Exercise appears to be safe in the majority of children, even in many children with congenital heart disease or arrhythmias that are not life threatening. Exercise does affect cardiac rhythm and electrical conduction through the heart. Many of these effects are mediated by changes in autonomic tone from the resting state (39). During exercise, the first change is a fall in parasympathetic tone promoting the initial increase in heart rate (3). This is followed by an increase in sympathetic activity. Cardiac sympathetic activity increases both as a result of increased circulating catecholamines during exercise and as a result of increased direct sympathetic stimulation of the myocardium. In the normal heart these changes in autonomic balance not only increase heart rate but also increase myocardial contractility and stroke volume. The rate of depolarization of the normal sinus node increases, atrioventricular conduction is enhanced (see Figure 2), and the

Figure 2 — This 13-year-old boy with atrial flutter was not recognized to be in atrial flutter when sent to the exercise laboratory. The top panel is the resting electrocardiogram from Lead II demonstrating atrial flutter with 2:1 atrioventricular conduction. The middle panel shows abrupt development of 1:1 atrioventricular conduction during exercise. The bottom panel shows the postexercise electrocardiogram with increased atrioventricular block and the typical “saw-tooth” appearance of atrial flutter.
ventricular repolarization rate is increased. The increase in myocardial work brings on an obligate increase in myocardial oxygen consumption (39). In the normal heart, this is well tolerated and is not a limiting factor to exercise and does not induce significant abnormalities in cardiac conduction or cause arrhythmias. In patients with coronary artery obstruction, coronary artery abnormalities, or myocardial hypertrophy, though, the increase in myocardial work and oxygen consumption may not be accompanied by an adequate increase in myocardial blood supply. The relative ischemia in any of these conditions during exercise may change the electrophysiologic properties of the heart.

Abnormal conduction tissue or areas of myocardium with abnormal electrophysiologic properties (ectopic foci) may be affected as the heart attempts to adapt to the increased workload of exercise. An ectopic focus may be suppressed as the depolarization rate of the sinus node increases. Alternatively, the depolarization rate of an ectopic focus may accelerate and/or become the dominant pacemaker in the heart during exercise. Also, catecholamines which increase with exercise may increase delayed afterdepolarizations, which are thought to play a role in the onset of ventricular tachycardia (22, 37).

Chronic exercise has specific effects on cardiac rhythm that are different from acute exercise and also alters the effect of acute exercise on cardiac rhythm. Aerobic training lowers resting heart rate and heart rate at a specific, submaximal workload as compared to pretraining heart rate. In general, the heart rate at peak exercise is unchanged (although the work performed at peak exercise may be higher than pretraining work levels).

In some well-trained individuals, the resting heart rate may be very low (as low as 30 to 40 bpm). These individuals have an increased prevalence of escape rhythms, such as a slow rhythm from an atrial focus other than the sinus node, junctional rhythm, or ventricular rhythm. These rhythms are usually asymptomatic. The sinus bradycardia that brings on escape rhythms is probably due to changes in resting autonomic tone with a vagal predominance that develops during training. There is no direct change in the sinoatrial node tissue.

**Exercise Testing in Patients With Heart Disease**

A variety of studies have been published describing the use of exercise testing in the evaluation of patients with known or suspected arrhythmias and for provocation of arrhythmias in patients at risk for arrhythmias (e.g., postoperative congenital heart disease). The findings and recommendations of these studies will be discussed in the following sections.

**Exercise Testing in Patients With Arrhythmias**

**Bradycardia.** Evaluation of the heart rate and rhythm during exercise testing aids the pediatric cardiologist in evaluation of patients with known abnormalities of cardiac rhythm. Sinus node dysfunction is a common complication following certain surgical procedures, especially atrial baffle procedures in transposition of the great arteries. Sinus node dysfunction including sick sinus syndrome (or tachycardia-bradycardia syndrome) can also exist in patients with structurally normal hearts or unoperated congenital heart disease (4). Resting heart rate may be normal at a given point in time although Holter monitoring will usually demonstrate bradycardia at some point during the day.
Exercise testing in our laboratory was used in evaluating sinus node function in 29 patients with transposition of the great arteries after the Mustard operation (23). Treadmill testing employed a modified Bruce protocol. The resting heart rate was normal in 93% of the patients. During exercise, however, 83% had an abnormally low heart rate either during maximal exercise or during the recovery period. Sixteen of the patients with low heart rate during exercise also had an intracardiac electrophysiologic (EP) study. Sinus node dysfunction was confirmed in 9 patients. Exercise testing was more sensitive in detecting sinus node dysfunction than EP study. Ten of 29 patients were receiving chronic digoxin therapy during exercise testing. There was no difference in heart rate response between patients who were receiving digoxin and patients not receiving digoxin. There was no difference in incidence of sinus node dysfunction between patients with normal exercise duration and patients with subnormal exercise duration. In 12% of the tests there were sinus pauses during the recovery period. Abnormalities during exercise testing did not correlate with symptoms of bradycardia or with the presence of arrhythmias. Improved detection of sinus node dysfunction by exercise testing as compared to EP study may be related to differences in physiologic state between the two procedures. Varying degrees of sedation are usually necessary for EP study whereas exercise studies can be performed in a more normal state.

A prospective study, also done at Texas Children’s Hospital, failed to show any correlation between sinus node recovery time and sinoatrial conduction time as measured at invasive electrophysiologic study and findings during maximal treadmill exercise testing (5). Although clinical exercise testing in patients at risk for symptomatic bradycardia may demonstrate a low peak exercise heart rate, the significance of this finding is unclear. We currently rely on 24-hour ambulatory electrocardiographic (Holter) monitoring with a correlating diary of symptoms to assess the impact of bradycardia in an individual patient.

Exercise testing has been an important part of the evaluation of patients with congenital complete atrioventricular block (CCAVB). It allows evaluation of peak heart rate and the ability to exercise at a normal level. The presence of decreased exercise performance can aid in the decision to place a permanent pacemaker. It is also useful for the provocation of ventricular arrhythmias. Ventricular tachycardia may be associated with syncope or sudden death in some patients with CCAVB. Thoren studied 15 patients with CCAVB by cycle ergometry (36). Fourteen of the patients had a linear increase in ventricular rate with incremental exercise. Mean maximal ventricular rate was 110 bpm and mean maximal atrial rate was 185 bpm. Peak oxygen consumption was measured in 10 patients and the mean value was 36 ml/min kg. This value was in the normal range for their laboratory. Two of the patients were above the normal range and two were below it. Some of the patients had ventricular extrasystoles.

Winkler evaluated the significance of exercise-induced ventricular ectopy in patients with CCAVB (38). Fourteen patients with CCAVB were studied on a treadmill with a modified Bruce protocol. One patient had uniform PVCs at rest. Ten patients had ventricular arrhythmia during exercise: Two patients had uniform PVCs, five had multiform PVCs, two had ventricular couplets, and one had ventricular tachycardia. The patients with PVCs at rest had multiform PVCs during exercise. Only patients over age 10 had ventricular ectopy. Also, patients with a prolonged QRS duration were more likely to have ventricular ectopy more
severe than uniform PVCs. Only one patient had decreased exercise tolerance. One patient subsequently received an electronic pacemaker. None of the patients died during follow-up. The findings in this study did not demonstrate a relationship between ventricular ectopy during exercise and symptoms.

Karpawich et al. retrospectively evaluated a group of 24 children with CCAVB who had an EP study (27). Eight of the children had a history of syncope (Stokes-Adams attacks). Only the presence of a persistent resting heart rate less than 50 bpm correlated with a history of syncope. Identification of the site of block by EP study was not helpful. Eleven of the children had an exercise test. The degree of increase in heart rate during treadmill exercise did not correlate with a history of syncope. Three children had ventricular ectopy during exercise, but only one of those patients had a history of syncope. Two of the patients without exercise-induced ventricular ectopy had a history of syncope. This patient population was relatively young, with a median age of 13 years. In older patients with more ventricular ectopy during exercise, there may be a correlation to syncope and Stokes-Adams attacks.

The response of the ventricular rate to exercise can predict the site of atrioventricular block. Pinsky et al. also reported on a group of patients with CCAVB from our institution (30). He reported on exercise testing in 12 patients. Each patient had an increase in ventricular rate during exercise although the degree of increase was variable (Figure 3). Patients with AV block above the His bundle showed a greater increase in heart rate.

Figure 3 — Resting and exercise electrocardiograms from a 7-year-old boy with congenital complete atrioventricular block. There is an increase in ventricular rate with exercise but no atrioventricular conduction.
Winkler et al. also reported on six patients with L-transposition of the great arteries, one patient with mitral regurgitation, and four patients following surgery for congenital heart disease with complete atrioventricular block (CAVB) (38). They found the incidence of ventricular ectopy to be similar to that of patients with congenital complete AV block. Patients with exercise-induced ventricular ectopy and complete AV block in this group, however, appeared to be at increased risk of sudden death. Six of the patients with CAVB had diminished exercise tolerance on the treadmill. Diminished exercise tolerance in a patient with complete atrioventricular block is an indication for an electronic pacemaker. Five patients with abnormal exercise tolerance received a pacemaker.

Exercise testing may demonstrate abnormalities in atrioventricular conduction in patients with exercise-induced symptoms. Alboliras et al. reported two patients with univentricular heart and exercise-related symptoms (1). The first patient had a modified Fontan procedure and initially did well. Two years after surgery she began having nausea and vomiting during exercise. Cycle ergometry was performed. At rest she was in normal sinus rhythm. During exercise she initially developed second-degree atrioventricular block (Mobitz Type I, 3:2 atrioventricular conduction), and then subsequently high-grade atrioventricular block with an atrial rate of 150 bpm and an irregular ventricular rate of 60 bpm. A dual-chamber pacemaker was placed and exercise testing was repeated. There was normal pacemaker function and no exercise-induced nausea. The second patient was a 12-year-old with univentricular heart. An aortopulmonary anastomosis had previously been performed. This patient also had recurrent nausea with exertion. He had first-degree atrioventricular block on supine resting ECG which progressed to second-degree atrioventricular block on standing. During cycle ergometry, the degree of second-degree AV block increased with an atrial rate of 146 bpm and a ventricular rate of 73 bpm. A modified Fontan procedure was performed and a dual chamber pacemaker was placed. He is asymptomatic.

**Supraventricular Tachycardia.** There are few data on the effects of exercise or the results of exercise testing in children and adolescents who are known to have supraventricular tachycardia (SVT). Exercise testing probably has little utility in patients with SVT once the diagnosis is made. If there is a history of symptoms during exercise, though, exercise testing may help to establish the etiology. Supraventricular tachycardia in pediatric patients most often involves a reentry circuit. Less commonly an automatic, ectopic focus is the cause of supraventricular tachycardia. It is uncommon for patients with reentry SVT to have SVT induced by exercise testing. Only 23 children out of 2,761 developed SVT during exercise testing at Texas Children’s Hospital (10) (Figure 4). In eight of these children, though, there was a history of tachycardia that had not been previously recorded. The majority of the children had an accessory atrioventricular connection (e.g., bundle of Kent) associated with their tachycardia. There were no complications related to the development of tachycardia and none of the patients required emergent treatment for SVT.

Rozanski et al. reported the results of exercise testing in seven patients with known supraventricular arrhythmias (34). Two patients had sinus bradycardia, two had premature junctional complexes, and three had premature supraventricular complexes. Each patient had two exercise tests separated in time. All of the patients with premature complexes had suppression of ectopy with exercise, but one developed premature ventricular complexes during exercise. After suppression of premature supraventricular complexes in another patient, short episodes of
atrial ectopic tachycardia developed during exercise. No further information is available about either of these patients.

We reported the results of exercise testing in a group of children with Wolff-Parkinson-White syndrome (7). Seventeen patients were studied with a modified Bruce treadmill protocol and intracardiac electrophysiologic (EP) study. Treadmill times were normal. Two patients had premature ventricular complexes during exercise and two patients developed supraventricular tachycardia during exercise. The response of preexcitation (delta wave) was evaluated. One patient did not have preexcitation at rest or during exercise but a delta wave was apparent during the recovery period. In four patients the delta wave disappeared during exercise. In one patient the disappearance was abrupt and in three patients the disappearance was gradual. Six patients had partial normalization of the QRS complex but a small delta wave persisted throughout exercise. Six patients had persistent preexcitation. No patient with a normal QRS complex during the preexercise ECG developed preexcitation during exercise. During the electrophysiologic study, the anterograde effective refractory period of the accessory connection (ERP-AP) was measured.

These data were compared to the changes in the QRS complex during exercise. One of the patients with persistent preexcitation during exercise had a nodoventricular (Mahaim) fiber at EP study. In the other patients with persistent preexcitation, the ERP-AP ranged from 180 to 290 ms. In the group with partial normalization of the QRS, the ERP-AP ranged from 206 to 325 ms. In the children with disappearance of the delta wave during exercise, the ERP-AP ranged from 360 to 390 ms. In the patient without preexcitation at rest or during exercise, the ERP-AP was 522 ms. The response of the accessory pathway to exercise may be affected by changes in autonomic tone during exercise (increased sympathetic stimulation and parasympathetic withdrawal). Patients with a short ERP-AP have an increased risk of sudden death due to ventricular fibrillation following rapid atrioventricular conduction through the accessory pathway during atrial flutter or fibrillation (13). Patients with disappearance of preexcitation during exercise can be expected to have a long ERP-AP and may be at decreased risk of sudden death compared to patients with persistence of preexcitation. These patients may not need intracardiac EP study to determine their ERP-AP, although we continue to rely on intracardiac testing to determine definitely the ERP-AP.

**Ventricular Arrhythmias.** Ventricular arrhythmias are the most common
cardiac reason for pediatric exercise testing (22). Specifically, the most common indication is frequent PVCs in patients thought to have a normal heart. If the PVCs are suppressed by formal exercise testing, no further evaluation or treatment is necessary (Figure 5). Some physicians use informal exercise testing such as running in the hall to achieve suppression. This may suppress the PVCs but it is a submaximal test. In patients with PVCs that are associated with ventricular tachycardia, the PVCs may initially be suppressed with exercise, only to return as PVCs, complex PVCs, or ventricular tachycardia at higher levels of exercise. Following maximal testing, complex PVCs or ventricular tachycardia may develop in the early recovery period. This is less likely to occur during submaximal testing. Patients who have exercise-induced ventricular arrhythmias require further evaluation. Some of those patients will have cardiac abnormalities on more thorough evaluation. Also, exercise testing is useful in the evaluation of efficacy of anti-arrhythmic medication in patients with ventricular tachycardia.

Jacobsen et al. reported 17 patients with PVCs and an anatomically normal heart (24). Five of the patients also had resting sinus bradycardia. The PVCs were uniform in all patients and disappeared in all patients during maximal treadmill testing. PVCs also reappeared in all patients during the recovery period. In one patient, ventricular tachycardia developed during the recovery period. The patients were followed for an average of 7.2 years. Eight of the patients still had PVCs on follow-up. None of the patients required treatment for ventricular

Figure 5 — Resting and exercise electrocardiograms from a 9-year-old boy with a normal heart. There are frequent premature ventricular contractions at rest and during early stages of exercise. The premature ventricular contractions are abolished with more intense exercise.
arrhythmia. One patient was subsequently diagnosed with mitral valve prolapse during the follow-up period. If PVCs were present at follow-up, a repeat exercise test was performed. The PVCs also disappeared in all eight patients during this test. The PVCs returned during the recovery period in only one patient. The patient with ventricular tachycardia during recovery after the first test continued to have asymptomatic ventricular tachycardia for 2 years following the initial evaluation. No treatment was required. Cardiac catheterization was performed and the heart was anatomically normal.

Rozanski et al. also performed maximal treadmill testing in 12 patients with ventricular arrhythmias (34). Exercise testing was repeated in each patient. All of the patients had an anatomically normal heart. Nine of the patients had isolated PVCs, one had PVCs and accelerated ventricular rhythm, one had ventricular tachycardia, and one had ventricular parasystole. Eight of the children with PVCs had suppression of PVCs with exercise. PVCs returned following exercise in all patients. Two of the patients had ventricular couplets during the recovery period. PVCs were not suppressed in one patient in spite of peak heart rates of 200 and 240 bpm. The patient with accelerated ventricular rhythm continued to have accelerated ventricular rhythm during exercise. In the other two patients the rhythm was unchanged during exercise. No follow-up was reported.

Fulton et al. studied 26 children with ventricular tachycardia (16). All of the patients had an anatomically normal heart. Eight of the children had symptomatic ventricular tachycardia. Sixteen patients had treadmill testing. Three patients had frequent uniform PVCs at rest and ventricular tachycardia (asymptomatic) during exercise. Six patients with ventricular tachycardia at rest had ventricular tachycardia throughout exercise. Seven patients with ventricular tachycardia at rest had suppression of ventricular tachycardia during exercise. None of the patients died during the follow-up period. Sixteen patients received antiarrhythmic drugs at some point and six patients were still receiving medication at the time of the report. Fulton et al. did not explain how exercise testing was used in evaluation of their patients.

Rocchini et al. reported on 38 patients with ventricular tachycardia (33). Exercise testing was performed in 21 patients. Twenty-one patients had underlying heart disease. Seventeen of the patients with structural heart disease and six of the patients with normal hearts had symptomatic ventricular tachycardia. Eleven of the symptomatic patients had exercise testing. Eight had an increase in the degree of ventricular arrhythmia. Two of the patients in this group had a previous exercise test with ventricular couplets. Five patients had symptoms during tachycardia and one patient required electrical cardioversion. Ten asymptomatic patients had exercise testing. All but one had a decrease in the degree of ventricular arrhythmia during exercise.

In this study, Rocchini et al. also used exercise testing to assess the efficacy of antiarrhythmic therapy. All 21 patients with underlying heart disease were treated. Three patients had ventricular tachycardia during exercise. In that group no patient had VT on Holter monitoring. Six patients had PVCs during exercise. In that group one patient had no PVCs on Holter monitoring. One patient had ventricular tachycardia on Holter monitoring and had no arrhythmia during exercise. Nine asymptomatic patients were treated with antiarrhythmic therapy. One patient had VT during exercise testing without VT on Holter monitoring. Three patients out of the entire group died from complications of underlying heart
disease but none died suddenly from arrhythmia. Another patient had VT on Holter monitoring and no arrhythmia during exercise. It appears that Holter monitoring and exercise testing are complimentary in detecting ventricular tachycardia. Alpert et al. has also demonstrated the utility of exercise testing in assessing the efficacy of antiarrhythmic therapy for VT (2).

Several authors have reported on the inducibility of ventricular tachycardia due to arrhythmogenic right ventricular dysplasia (ARVD) during exercise (22). Dungan et al. reported three patients out of a group of 26 patients with recurrent VT (14). Ten patients were initially thought to have a structurally normal heart but three of these patients were subsequently found to have ARVD. One was an infant and the other two were adolescents, both of whom developed ventricular tachycardia during exercise.

Ryujin et al. performed exercise testing on 196 children with premature ventricular complexes on resting electrocardiogram (35). Nine patients also had Wolff-Parkinson-White syndrome, two had mitral valve prolapse, and one patient each had atrioventricular dissociation, first-degree atrioventricular block, and right bundle branch block. There was no other evidence of heart disease in any patient by physical examination, chest roentgenogram, or echocardiography. A graded exercise test was performed. The test was a modification of the Bruce treadmill protocol with 30 seconds at Stage 1, followed by 3 minutes each at Stages 3, 5, 7, and 9. The degree of ventricular arrhythmia increased in 29 patients. Eight patients had ventricular couplets, eight patients had three or four consecutive ventricular complexes, and 13 patients had ventricular tachycardia. These patients had further evaluation including electrocardiography during a two-step exercise test consisting of 3 minutes of exercise and 8 minutes of postexercise monitoring. They also had repeat treadmill testing and Holter monitoring. A total of 141 electrocardiograms, 77 two-step exercise tests, 77 treadmill tests, and 46 Holter monitor electrocardiograms were performed on the 29 patients.

A test was considered positive if it reproduced the findings of the initial treadmill test. In all, 3% of the ECGs, 15% of the two-step exercise tests, 26% of the Holter ECGs, and 51% of the treadmill tests were positive. In an individual patient, any testing mode may have shown normal results while another test showed ventricular arrhythmias, possibly even ventricular tachycardia. One of the patients with ventricular tachycardia on the initial exercise test died suddenly. She had bidirectional ventricular tachycardia and was treated with beta-blockers and disopyramide. Her QT interval was not commented upon. In this study, exercise testing was the most sensitive method of detecting serious ventricular arrhythmias in patients with PVCs and a normal heart.

We also evaluated ventricular tachycardia during exercise testing (9). There were 22 cases of VT in a total of 2,761 patients who had exercise testing for any reason over a 10-year period (Figure 6). Five patients had sustained VT (over 30 seconds). There were no complications during testing and no patient required acute treatment for tachycardia. Two patients had prolonged QT syndrome, four had arrhythmogenic right ventricular dysplasia, two had mitral valve prolapse, three had cardiomyopathy, and six had congenital heart disease. Only five patients had a normal heart. Ventricular tachycardia had not been documented prior to exercise testing in 11 patients. Seventeen patients had an intracardiac electrophysiologic study but only five had inducible ventricular arrhythmia. Only one patient was not treated with antiarrhythmic drugs. One patient required surgical
Figure 6 — An exercise electrocardiogram from a 17-year-old with a previous diagnosis of supraventricular tachycardia. During exercise, ventricular tachycardia developed. Note the atrioventricular dissociation at the onset of tachycardia which differentiates ventricular tachycardia from exercise-induced bundle branch block or supraventricular tachycardia with aberrancy. Invasive electrophysiology study confirmed the diagnosis of ventricular tachycardia. Serial exercise tests were performed to assess the response to antiarrhythmic therapy.

Ablation of a ventricular ectopic focus. Two of the patients died suddenly, both with congenital heart disease.

**Bundle Branch Block.** We examined the significance of exercise-induced bundle branch block in a pediatric exercise laboratory (8). Bundle branch block developed in six out of 2,761 patients evaluated by treadmill exercise testing (Figure 7). Five patients had congenital heart disease and one had a cardiomyopathy. Four patients had right bundle branch block and one had left bundle branch block. Three of the patients had aortic or subaortic stenosis. A total of 153 patients with aortic or subaortic stenosis were tested on the treadmill. Three of the patients had ST segment changes consistent with ischemia prior to onset of bundle branch block. The changes in ST segment and the development of bundle branch block could also be due to autonomic changes during exercise.

**Exercise Testing and Arrhythmias Following Surgery**

Arrhythmias are one of the most common long-term complications of surgery for congenital heart disease. They commonly occur after intracardiac repair of tetralogy of Fallot, atrial baffle procedures for transposition of the great arteries, the Fontan procedure, and repair of aortic stenosis. Ventricular arrhythmias are particularly common after surgery for aortic stenosis if there is residual valve stenosis or insufficiency. Ventricular arrhythmias are associated with sudden death in tetralogy of Fallot if the arrhythmia is not treated successfully. Atrial flutter and ventricular tachycardia following the Mustard or Senning operation are
accompanied by increased morbidity and mortality. Any arrhythmia may be induced during exercise testing, including all the arrhythmias just mentioned. Several studies have been done that address the postoperative status in this group of patients. We will discuss a few of the studies that specifically relate experience with arrhythmias and exercise testing in the postoperative setting.

**Tetralogy of Fallot.** The group of postoperative patients that has been most extensively studied is postoperative tetralogy of Fallot. There is an increased incidence of ventricular arrhythmias in postoperative tetralogy of Fallot. A subset of patients following surgery for tetralogy of Fallot is at risk for sudden death, and the sudden death appears to be related to the occurrence of ventricular tachycardia. James et al. reported four patients who had sudden cardiac arrest after intracardiac repair of tetralogy of Fallot (25). They postulated that ventricular tachycardia was the cause of death in all patients. One of the patients had a previous maximal exercise test that showed multiformal PVCs and short runs of VT during the recovery period. James et al. later reported an incidence of 9% uniform PVCs and 12% multiformal PVCs during exercise testing following surgery for tetralogy of Fallot (26). Garson et al. stressed the association of PVCs on resting ECG with elevated right ventricular systolic and end diastolic pressure as well as an increased incidence of sudden death (19). Half of the patients with sudden death died during exercise. Garson et al. recommended a treadmill exercise test for all patients following surgery for tetralogy of Fallot (Figure 8).
Garson et al. later reported on the incidence of ventricular arrhythmias during treadmill testing in a group of 104 patients following repair of tetralogy of Fallot (17). Fifteen patients had at least one PVC on resting ECG. Maximal exercise testing using a modified Bruce protocol was performed. Thirty-one patients had ventricular arrhythmia during exercise testing. Twenty-one patients had at least one PVC, four had multiform PVCs, four had at least one couplet, and two had VT. Five of the patients with resting ventricular arrhythmia had no arrhythmia during exercise testing. Twenty-one patients had ventricular arrhythmia during exercise and no ventricular arrhythmia on resting ECG. The patients with ventricular arrhythmia were older at the time of study and farther from time of operation. Cardiac catheterization was performed on 82 patients. All 15 patients with ventricular arrhythmia at rest and 22 of the patients with ventricular arrhythmia during exercise had cardiac catheterization. In all, 53% of patients with ventricular arrhythmia at rest and 50% of patients with ventricular arrhythmia during exercise had a right ventricular (RV) pressure over 60 mmHg, as compared to 27% without arrhythmia at rest and 16% without arrhythmia during exercise. There also was a positive correlation between an elevated RV end diastolic pressure and ventricular arrhythmia during exercise. All of the patients with couplets, or VT, and three of four patients with multiform PVCs had an RV systolic pressure over 60 mmHg. Garson et al. felt that all patients with ventricular arrhythmia after correction of tetralogy of Fallot should be treated with antiarrhythmic medication. They also demonstrated that exercise testing increased the ability to diagnose ventricular arrhythmia in postoperative tetralogy.

In 1985 Garson et al. again reported their experience with ventricular arrhythmias in a group of 104 patients following repair of tetralogy of Fallot. They found that 27% of patients had at least one PVC on resting ECG. Maximal exercise testing using a modified Bruce protocol was performed. Thirty-one patients had ventricular arrhythmia during exercise testing. Twenty-one patients had at least one PVC, four had multiform PVCs, four had at least one couplet, and two had VT. Five of the patients with resting ventricular arrhythmia had no arrhythmia during exercise testing. Twenty-one patients had ventricular arrhythmia during exercise and no ventricular arrhythmia on resting ECG. The patients with ventricular arrhythmia were older at the time of study and farther from time of operation. Cardiac catheterization was performed on 82 patients. All 15 patients with ventricular arrhythmia at rest and 22 of the patients with ventricular arrhythmia during exercise had cardiac catheterization. In all, 53% of patients with ventricular arrhythmia at rest and 50% of patients with ventricular arrhythmia during exercise had a right ventricular (RV) pressure over 60 mmHg, as compared to 27% without arrhythmia at rest and 16% without arrhythmia during exercise. There also was a positive correlation between an elevated RV end diastolic pressure and ventricular arrhythmia during exercise. All of the patients with couplets, or VT, and three of four patients with multiform PVCs had an RV systolic pressure over 60 mmHg. Garson et al. felt that all patients with ventricular arrhythmia after correction of tetralogy of Fallot should be treated with antiarrhythmic medication. They also demonstrated that exercise testing increased the ability to diagnose ventricular arrhythmia in postoperative tetralogy.
arrhythmias and sudden death in patients following surgery for tetralogy of Fallot in 488 patients (20). Exercise studies were not specifically addressed. Ventricular arrhythmia, usually only uniform PVCs, was present on resting ECG in 100% of patients who died suddenly, as compared to 12% who did not die suddenly. In patients with ventricular arrhythmia who were treated successfully with anti-arrhythmia medication, there was no sudden death. In light of this study, since all patients who died had PVCs on resting ECG, it is unclear the significance of the added sensitivity of diagnosing ventricular arrhythmia by exercise testing.

Transposition of the Great Arteries. Until recently, the most common operation for transposition of the great arteries was an atrial baffle procedure (Mustard or Senning). Atrial arrhythmias occur frequently following this operation and the incidence of atrial arrhythmias increases as the postoperative period lengthens (29). The presence of atrial arrhythmias, especially atrial flutter, has been associated with an increased morbidity and mortality in this group of patients. Ventricular tachycardia has been recognized as a cause of sudden death in this group of patients. The incidence of sinus node dysfunction is also increased. Cardiac rhythm during exercise is frequently abnormal and exercise testing can be useful in eliciting arrhythmias following the Mustard or Senning procedure.

Exercise testing is a sensitive method for diagnosing sinus node dysfunction in patients following atrial baffle procedure. Reybrouck et al. demonstrated that half of a group of patients had a subnormal heart rate response to maximal exercise after atrial baffle procedure (32). The decreased chronotropic response to exercise is related to decreased exercise performance in this group of patients. Hesslein et al. also demonstrated the sensitivity of exercise testing for diagnosing sinus node dysfunction in patients following the Mustard operation (23). This study was discussed above in the section on exercise testing in patients with arrhythmias.

Mathews et al. performed maximal treadmill exercise testing on 21 patients after the Mustard operation (28). Fifteen patients were in sinus rhythm at rest. In this group, two patients had premature atrial complexes, two were in junctional rhythm, four had uniform PVCs, and one had multiformal PVCs during exercise testing. One of three patients with AV dissociation at rest had multiformal PVCs during exercise. A relationship between exercise-induced arrhythmias and sudden death in this group of patients has not been demonstrated, but large studies have not yet been performed.

Aortic Stenosis. The incidence of arrhythmias during exercise testing in aortic stenosis has not been evaluated. Sudden death is a known complication of aortic valve disease in pediatric cardiology and it appears to be related to the presence of arrhythmias (18). Ventricular arrhythmia, including VT, can occur during exercise testing. (9). Although the incidence of ventricular arrhythmia during exercise testing in aortic stenosis is unknown, it appears to be infrequent.

Other forms of congenital heart disease also appear to be associated with both supraventricular and ventricular arrhythmias (9, 10, 33). The significance of exercise-induced arrhythmias in this group of patients is unclear at this time. Careful studies in the future may delineate its relationship to prognosis and morbid events.
Exercise Testing and Pacemakers

Preston et al. evaluated the effect of exercise in a small group of early pacemaker patients and found that exercise consistently lowered the myocardial threshold by 11 to 37% (31). More recently, we used exercise testing to evaluate a group of pediatric patients with pacemakers (6) (Figure 9). All 24 patients had dual-chamber pacemakers. The indications for electronic pacing were sinus node dysfunction with symptomatic bradycardia (9), congenital complete AV block (7), refractory SVT requiring His bundle ablation (7), surgical complete AV block (3), and high-grade second-degree AV block (2). Maximal exercise testing was performed using a modified Bruce protocol. Ten patients reached the programmed

Figure 9 — Resting and exercise electrocardiograms from a 12-year-old boy who had an electronic atrial pacemaker placed for the treatment of severe sinus node dysfunction. A comparison of the tracings shows improved atrioventricular conduction with exercise.
maximal upper rate limit, and in six patients this was the maximal rate possible for the pacemaker. This suggests that the maximum programmable rates in some pacemakers may be inadequate for some pediatric patients who rely on a higher heart rate than do adults to support cardiac output during exercise. Ventricular sensing was normal in all patients. Two patients had atrial undersensing during exercise which resulted in a reduced ventricular rate. One patient had a lead fracture and the other had a malfunctioning pulse generator. Seven patients had myopotential inhibition during exercise which caused reversion to the noise rate and also limited the heart rate response to exercise.

Information from exercise testing can also be valuable when choosing a pacemaker system prior to implantation. In patients with sinus node dysfunction and a maximal heart rate during exercise that is significantly reduced from normal, a conventional pacemaker may not provide total relief from symptoms, especially symptoms during exercise. In this group of patients a pacemaker that is activity responsive may be beneficial, resulting in a more normal exercise heart rate and more normal exercise performance.

Summary

Exercise is a perturbation from the resting state. In some patients this perturbation leads to changes in the autonomic tone and/or the myocardium, which leads to changes in the cardiac rate or rhythm. These changes allow exercise testing and ambulatory electrocardiography during exercise to increase the diagnostic sensitivity for many arrhythmias. The significance of this increase in diagnostic sensitivity is unclear. Few studies specifically addressing this point are available. Presently, the unknown significance of exercise induction of arrhythmias also forces restriction from competitive athletics in many patients with arrhythmias in pediatrics. Future studies addressing exercise-induced arrhythmias and the stress of competition in patients with arrhythmias may help in the development of additional recommendations in the future.

References


