Errors in the Recognition and Treatment of Heart Disease

By R. Bruce Logue, M.D., and J. Willis Hurst, M.D.

The medical literature is replete with communications on heart disease. Despite this abundant literature, errors are made in recognizing and treating diseases of the heart. This report describes some of the more common errors encountered in practice and offers certain clues that may be helpful in preventing some of the mistakes.

Chest Pain

The diagnosis of angina pectoris is established by the presence of a characteristic history and does not depend upon the presence of electrocardiographic abnormalities. The pain is usually substernal and occurs characteristically during effort, emotional stress, exposure to cold or after a large meal. It is not well appreciated that it may occur on assuming the recumbent position. The duration is one to five minutes as a rule and relief may be prompt with rest or the administration of nitroglycerin. In inquiring regarding angina, it is not sufficient to ask about pain alone. Since pain may be denied, one should therefore ask about indigestion, tightness, squeezing, burning, heaviness or choking. Discomfort may occur in the arms, epigastrium, back or jaw without being present in the anterior chest. While precordial pain is rare in angina pectoris, it may occur. The sudden onset of angina for the first time or the change in its frequency or severity usually signifies coronary occlusion and rarely myocardial infarction. Judicious periods of rest when such changes occur may allow collateral circulation to develop and forestall infarction. In the elderly sedentary patient, discomfort may occur only after meals and one should be suspicious of "indigestion" accompanied by sweating or weakness. Many patients have prolonged pain without accompanying evidence of myocardial necrosis such as persistent electrocardiographic changes, fever, leukocytosis and an elevated sedimentation rate. These patients require sharp reduction in physical and mental activity and should be treated much like the patient with infarction, though with more freedom about the room and with shorter periods of rest. There is still a reluctance to make a clinical diagnosis of myocardial infarction without a drop in blood pressure or pulmonary rales, even with prolonged pain. It seems hardly necessary to emphasize that infarction often occurs without any changes on physical examination, and without leukocytosis, fever or elevation of the sedimentation rate.

The pain associated with emotional tension may be confused with coronary disease. While the precordial and inframammary location of the sharp, sticking or lancinating pain and accompanying hyperesthesia is well known, it is not widely known that precordial oppression or dull aching may occur. This is differentiated from coronary disease by the fact that it may last for hours or days, is unrelated to effort and unrelieved by nitroglycerin. It is frequently accompanied by the hyperventilation syndrome. There may be sighing respiration, weakness, giddiness, palpitation and heart consciousness. This pain is more often unrelated to heart disease, but at times is the disabling symptom.
due to anxiety accompanying organic heart disease.

We should not neglect to determine whether pain is aggravated by deep breathing, turning, coughing or swallowing, for this suggests pericarditis. The sudden onset of such pain accompanied by the early presence of a pericardial friction rub and fever may indicate acute benign pericarditis, a condition which is often misdiagnosed myocardial infarction.

Patients with pulmonary hypertension may experience pain indistinguishable from that of coronary disease. It is usually accompanied by dyspnea and occasionally by cyanosis. Nitroglycerin may give no relief, but intravenous aminophylline may be followed by dramatic improvement. Asthma, emphysema, mitral stenosis and interstitial defect are the more common causes of pulmonary hypertension. A history of repeated attacks of "coronary occlusion" in such a patient should arouse suspicion of pulmonary hypertensive pain.

The pain of gall bladder disease may closely simulate that of coronary disease. Intermittent, epigastric or lower substernal cramping pain unrelated to effort, particularly when associated with nausea, chilliness, or back pain suggests gall bladder disease.

Degeneration of a cervical disc may produce pain in the shoulder, arm and upper chest. The occurrence of prolonged numbness and paresthesias in the arms and hands without relation to exertion but with aggravation on lateral flexion or hyperextension of the head suggests the diagnosis. Tenderness on palpation over the course of the nerve trunks at their exit is common.

The pain of dissecting aneurysm may be mistaken for myocardial infarction. Clues of dissecting aneurysm which may be overlooked are tearing chest pain with persistence of hypertension, the development of aortic insufficiency, pulse or blood pressure differences between the arteries of the neck, upper extremities or lower extremities, pulsation of a sternoclavicular joint, or chest pain accompanied by transient paralysis of the legs.

One should resist the temptation to ascribe substernal discomfort to the presence of an hiatal hernia so commonly demonstrated by x-ray examination since these are rarely responsible for pain. Furthermore, the experienced radiologist may at times be unable to distinguish a phrenic ampulla from an hiatal hernia. Pain due to "esophageal spasm" may be misinterpreted as angina. It is intermittent, spasmotic, unrelated to effort and is prone to occur under emotional tension and after dietary indiscretion. The pain of the "splenic flexure syndrome" is never substernal but is localized to the inframammary or precordial region and may be referred to the left arm. It may be exactly reproduced by distending the splenic flexure through a tube, and relief may be obtained following an enema.

**Congestive Heart Failure**

There is a widespread tendency to diagnose congestive heart failure without establishing the etiology. The common causes of heart disease leading to congestive failure are coronary arteriosclerosis, hypertension, rheumatic heart disease and congenital heart disease. In the absence of characteristic findings of these usual diseases, one should ask: (1) Are the symptoms and findings due to heart failure? (2) Is there pericardial or diffuse myocardial disease? (3) Is there an extracardiac cause of circulatory overloading such as acute nephritis or peripheral arteriovenous fistula? After establishing the etiologic type of heart disease, one should routinely search for a precipitating cause of the heart failure. This is of prime importance since success of therapy may depend on control of the precipitating factor. Infections, particularly respiratory, are the most common initiating and aggravating factors in congestive heart failure. Pulmonary embolism, acute rheumatic fever, acute myocardial infarction, cardiac arrhythmia, overloading with fluid and salt, bacterial endocarditis, thyrotoxicosis, hormone therapy, liver disease, renal disease and urinary retention are other important sources of aggravation. The presence of fever may be a clue to some of these since fever of more than 2 degrees does not occur with uncomplicated heart failure.

Congestive heart failure is often unrecognized in spite of a characteristic history of paroxysmal nocturnal dyspnea, since there may be no rales.
in the lungs several hours after such an episode. On the other hand, nocturnal hyperventilation due to anxiety may be misdiagnosed as congestive heart failure. It is not sufficiently recognized that hyperventilation may awaken a person from sound sleep. Ancillary symptoms suggesting hyperventilation are numbness and tingling of the lips, face or hands, giddiness or blacking out sensations, feeling of impending doom and chest oppression. Sighing respiration with inability to get a satisfying breath is a common manifestation between attacks. The hyperventilation syndrome is not uncommon in the person with organic heart disease.

It is often difficult to determine whether dyspnea is due to cardiac or pulmonary disease. The wheezing of bronchial and cardiac asthma may be indistinguishable. Each may be aggravated by effort and occur at night. Asthma appearing initially in middle life should be considered as a manifestation of heart failure until proven otherwise. The response to epinephrine and aminophylline do not differentiate the two conditions. The dyspnea resulting from bronchial asthma, emphysema, pulmonary fibrosis, and chronic pulmonary infection may be misinterpreted as heart failure. Whenever there is no clinical evidence of coronary, hypertensive or valvular heart disease and the heart size is normal, one should suspect a pulmonary origin of dyspnea and wheezing. At times, one must resort to a therapeutic trial of mercurial diuretics, digitalis and salt restriction to determine whether dyspnea is due to heart failure or to pulmonary disease. A weight loss of 4 pounds within 24 hours following a mercurial diuretic suggests congestive heart failure in the absence of renal or hepatic disease. This approach is particularly important where heart and lung disease coexist. In the management of patients with chronic lung disease, one should not neglect the use of antibiotics and bronchodilators.

One should not overlook the importance of anemia, obesity, thyrotoxicosis and metabolic acidosis as the sole cause or as an aggravating factor of dyspnea. It is not well appreciated that dyspnea is a frequent complaint in constrictive pericarditis and significant pericardial effusion. Edema is a late manifestation of heart failure. It should be recalled that 10 to 15 pounds of extracellular fluid may accumulate without pitting edema. Edema of the extremities may be due to obesity, varicose veins, old or recent phlebitis, cirrhosis of the liver, nephritis, or hypoproteinemia, and may be misinterpreted as evidence of congestive heart failure. Less common causes of edema such as lymphedema, myxedema, hormone imbalance due to menstruation, pregnancy and the menopause, and hormone therapy may be encountered. The edema of heart failure accumulates in the most dependent portions of the body. Thus one should not neglect to examine the presacral region of the bedridden patient. Another factor which determines the location of edema is tissue pressure. Such pressure may be low in the periorbital region, particularly in children, and the edema that occurs in this area may simulate renal disease. From a practical standpoint, one may exclude heart failure as a cause of edema if the heart size is normal by x-ray examination. Two possible exceptions to this rule are cor pulmonale and constrictive pericarditis.

There may be no liver enlargement in the adult with mild congestive heart failure, and ascites is a late manifestation. One exception is constrictive pericarditis which is characterized by the early appearance of liver enlargement and ascites. When ascites is due to congestive heart failure, the liver is invariably enlarged as contrasted to Laennec cirrhosis in which the liver may or may not be enlarged. Gaseous distension is a frequent complaint in association with hepatomegaly. Acute tenderness on palpation of the liver area suggests that enlargement has been rapid in development and of recent origin. Although liver engorgement may produce discomfort, one should not overlook peptic ulcer or other gastrointestinal disease as a cause of pain. Chronic cough or insomnia due to Cheyne-Stokes respiration may be predominant symptoms due to congestive heart failure and their significance may be overlooked.

There are certain physical signs particularly useful in the evaluation of the failing heart. Diastolic gallop rhythm is a frequent finding in the presence of myocardial insufficiency and
should be differentiated from the unimportant systolic gallop rhythm. At times a diastolic gallop can be better seen or palpated rather than heard. Pulsus alternans is another indication of myocardial insufficiency; it is commonly overlooked since sufficient care is not taken in determining the systolic pressure. Exercise may be necessary to bring out both diastolic gallop rhythm and pulsus alternans. The significance of pulmonary rales due to congestive heart failure is well known; however, it should be recalled that interstitial pulmonary congestion may be present without rales. In such cases x-ray study of the chest may reveal pulmonary congestion; this may prevent the unwary from making an erroneous diagnosis of the hypertensive syndrome. One should remember that rales may be due to causes other than heart failure such as chronic lung disease, hypostasis, pneumonia, atelectasis, aspiration, or to pulmonary embolism.

A careful study of the neck veins is often neglected. Although neck vein distension is common in congestive heart failure, pulmonary congestion may occur without obvious venous engorgement. The deep systolic jugular venous pulsation is usually misinterpreted as an arterial pulsation, and its significance is missed. The usual cause of such pulsation is relative tricuspid insufficiency resulting from chronic congestive heart failure. It is less commonly due to organic tricuspid insufficiency. This finding is important because in a large percentage of cases there may be no accompanying tricuspid murmur. One must find the optimum semirecumbent position in order to observe the systolic pulsation of the deep jugular veins. Light compression obliterates such a venous pulsation without altering the underlying arterial pulsation; furthermore, hepatic compression accentuates the venous overfilling.

Heart failure will be overlooked in children if one expects the manifestations usually seen in the adult. For example, there may be few rales in the dyspneic child, the liver enlarges quite early, and there may be minimal peripheral edema, even when ascites is present. The occurrence of periorbital edema may cause confusion with nephritis. Tachypnea is a more prominent feature than in the adult and is commonly attributed to pneumonia. Congestive heart failure is commonly precipitated by respiratory infection in the child with heart disease. Therapy should be directed towards treatment of both the infection and the heart failure.

When heart failure ceases to respond in the expected manner to adequate therapy, it is natural to assume that there is progressive deterioration of cardiac function. However, one should again consider certain extracardiac factors which may be responsible. Such things as electrolyte depletion, acute myocarditis, pulmonary embolism, pulmonary infection, thyrotoxicosis, renal and liver disease, and digitalis intoxication may be responsible.

Cardiac Arrhythmias

Paroxysmal auricular tachycardia is often unrecognized in spite of a characteristic history of sudden onset and cessation of rapid heart beat. The attack may be terminated by carotid sinus pressure in about one-third of the patients; however, this procedure is often overlooked or improperly performed. Carotid sinus pressure during the recording of an electrocardiogram may serve to identify the occasional case of sinus tachycardia simulating auricular tachycardia. The treatment of choice in the patient who does not respond to simple measures producing vagal stimulation is the administration of a rapidly acting digitalis preparation. Transient cardiac standstill or short runs of ventricular tachycardia are probably not uncommon at the termination of an attack, regardless of the type of therapy, and this does not preclude digitalis therapy for subsequent attacks. Simple sedation should not be overlooked in addition to the above measures.

Auricular fibrillation often occurs in the absence of demonstrable heart disease. Mitral stenosis, coronary disease and thyrotoxicosis are the commonly recognized organic causes of this arrhythmia. Less common causes which may be overlooked are pulmonary embolism, pulmonary infection, bronchial obstruction with atelectasis, constrictive pericarditis, acute pericarditis, digitalis intoxication, potassium depletion, active rheumatic fever, anesthesia, hypoxia and interatrial septal defect. There is
usually no problem in recognizing paroxysmal auricular fibrillation; however, aberration of the QRS complexes of the electrocardiogram may simulate ventricular tachycardia. The management is frequently improper. It is often forgotten that the aim of initial therapy is to slow the uncontrolled ventricular rate as rapidly as possible, and this can best be accomplished by rapid digitalization. Furthermore, paroxysmal auricular fibrillation is frequently reverted to sinus rhythm following such therapy, particularly when there is no demonstrable heart disease. When auricular fibrillation persists, quinidine can then be given. Despite the wave of enthusiasm for conversion of all cases of chronic auricular fibrillation, our personal experience justifies a word of warning. Such therapy is not without the small and unpredictable risk of major embolic episodes or sudden death, presumably due to ventricular fibrillation.

Auricular flutter is usually associated with organic heart disease; it is more disturbing to the patient because of wide variations in rate occasioned by variable A-V block, and is more resistant to therapy. The treatment of choice is the cautious administration of digitalis to full therapeutic levels. This produces slowing of the ventricular rate by increasing A-V block or by causing auricular fibrillation which may subsequently revert spontaneously to sinus rhythm. Quinidine may be useful in those who do not respond to digitalis, but such therapy is less often successful than in cases of auricular fibrillation.

Ventricular tachycardia may be confused with auricular tachycardia with bundle-branch block or aberrant conduction and at times differentiation may be impossible. The rate may vary slightly over a period of time but does not respond to carotid sinus pressure. The use of an esophageal lead often serves to identify the rhythm by demonstrating independent auricular and ventricular rhythms. When the rhythm cannot be definitely identified as supraventricular, it should be treated as ventricular tachycardia with quinidine or procaine amide. It is important to remember that the drugs used in treating ventricular tachycardia may also produce this rhythm in toxic doses.

Syncope

While syncope is not a common symptom of heart disease, it may be a disturbing complaint, and when accompanied by convulsions may be mistaken for epilepsy. Stokes-Adams attacks are one of the more important causes of transient unconsciousness. They are more often associated with coronary disease but are not uncommon in the presence of aortic stenosis. It should be remembered that episodes may occur with a normal electrocardiogram and normal P-R interval between attacks. While the majority of attacks are due to cardiac standstill, they may be due to ventricular tachycardia or to ventricular fibrillation. Syncope may occur during exertion in the patient with advanced aortic regurgitation or with aortic stenosis. Excessive ventricular rates during paroxysmal tachycardia or the transient standstill at the end of an attack may be responsible for unconsciousness. The onset of a cerebrovascular accident may be confused with syncope of cardiac origin. Points of help in differentiation are: flushing, slow stertorous respiration, preceding symptoms such as headache, weakness or paresthesias of the lips or extremity, neurologic signs, or loss of memory for recent events. It is now apparent that thrombosis of an atherosclerotic carotid artery is a common cause of cerebral vascular accident with or without associated syncope. Other causes of syncope which should not be neglected are: hypperirritable carotid sinus reflex, emotionally induced vasovagal attacks, postural hypotension, cough syncope, spontaneous hypoglycemia, hyperventilation syndrome, pulmonary embolism, dissecting aneurysm and epilepsy.

Rheumatic Heart Disease

Rheumatic fever in early childhood manifests itself largely in the form of carditis and not polyarthritis; it may be readily overlooked. Rheumatic fever is more common in the age group between 5 and 15, but it is an error to assume that it does not occur at an earlier age. The first suspicion of the disease may be aroused by the development of a systolic murmur at the apex in the child with a history of repeated sore throats. While joint symptoms
may be present in about half of all children, they may be vague and indefinite. Rheumatic fever is the most common cause of heart failure in childhood, and under such circumstances a significant systolic murmur is present at the apex. The predominant apical location of murmurs serves to differentiate rheumatic from congenital lesions in which the murmurs are largely basal or midsternal in location. It is often assumed that a diastolic rumble at the apex indicates mitral stenosis; however, such a murmur is not uncommon in acute rheumatic fever and it may disappear as activity subsides. It should be recalled that a diastolic apical rumble may also occur with such congenital lesions as patent ductus arteriosus or large ventricular or atrial septal defects. Recent experience indicates that the majority of patients thought to have Lutembacher’s syndrome have only an interstitial septal defect. An error is made in assuming that rheumatic fever is in inactive when the sedimentation rate is normal, since a normal rate may occur in the presence of congestive heart failure. Contrary to prevailing impression, a single electrocardiogram is of limited usefulness in the recognition of rheumatic fever. In addition to first degree A-V block, nodal rhythm and interference dissociation may occur in acute rheumatic fever. It should not be forgotten that a number of the common childhood diseases may produce a prolonged P-R interval. Despite the adequate evidence that the recurrence of rheumatic fever may be significantly diminished by long-term prophylaxis with penicillin and sulfia drugs, this type of therapy is being constantly neglected. Every effort should be made to treat pharyngitis or sore throat in the rheumatic subject promptly with the appropriate chemotherapy. The common error is the failure to continue chemotherapy for at least 7 to 10 days in order to eradicate the streptococci. Furthermore, the rheumatic subject should be protected by adequate chemotherapy while hospitalized on an open ward, since the frequency of respiratory infection is so great. It is a mistake to expect cure of rheumatic fever from the administration of cortisone or corticotropin (ACTH) since available evidence lends no support for such a view. It is apparent that the hormones do not prevent the development of valvular damage and their chief usefulness lies in tiding the patient over a critical period.

The murmur of mitral stenosis may be missed unless the patient is examined in the left lateral position after exercise with the bell of the stethoscope. At times a split or reduplicated mitral first sound may be misinterpreted as a presystolic murmur. The absence of a mid-diastolic rumble in such a case virtually eliminates the possibility of mitral stenosis. The murmur of mitral stenosis may be obscured by paroxysmal auricular tachycardia, auricular fibrillation or pulmonary edema and it is important to re-examine the patient after these have been corrected. With the advent of mitral commissurotomy, it has become apparent that the murmur of tricuspid insufficiency may be transmitted towards the apex and be confused as the murmur of mitral insufficiency. In the patient with mitral stenosis, it may be impossible to differentiate the murmur of aortic insufficiency from the murmur of relative pulmonary insufficiency, although when the intensity of the murmur varies from time to time the latter lesion is more likely.

The most common valvular heart disease after age 50 is calcific aortic stenosis. It is missed with surprising regularity. The murmur is usually heard in the aortic area and is transmitted to the neck. The murmur is often attributed to dilatation of the aorta due to atherosclerosis or hypertension. It may be obscured or heard only in the neck in the presence of emphysema. It may diminish during acute heart failure, shock or during cardiac arrhythmia. The murmur is rough in quality but need not be loud. A thrill is present in about one-third of the cases. The murmur may be heard with maximum intensity at the apex or to the left of the sternum for years before it is recognized as arising at the aortic area. When the murmur is heard at the precordial region it may have a musical quality. The aortic second sound is normal in approximately half of all cases, and a small pulse pressure is a very late and infrequent sign. A marked degree of left ventricular hypertrophy with strain pattern in the electrocardiogram in the absence of hypertension should lead one to suspect aortic
stenosis. Unfortunately, many diagnoses are missed because a careful search for aortic valve calcification is neglected; however, the absence of calcification does not exclude the lesion. The association of angina pectoris and syncope demands a careful search for aortic stenosis. Unexplained fever in the elderly is occasionally found to be due to bacterial endocarditis superimposed on the aortic valve.

**Bacterial Endocarditis**

The commonly expected findings of petechiae, splenomegaly, hematuria and leukocytosis occur in less than one-half of cases of bacterial endocarditis. Unexplained fever of a week's duration is sufficient grounds for strongly suspecting bacterial endocarditis in the patient with valvular or congenital heart disease and demands blood cultures. On the other hand, an occasional case of bacterial endocarditis has been observed in which there was no fever. An uncommon presenting manifestation of bacterial endocarditis is anemia.

The prior administration of antibiotics may produce bacteriostasis, and positive blood cultures may be obtained with difficulty. Sensitivity tests are useful in determining the relative resistance of organisms but the conventional tests measure bacteriostatic, not bactericidal, effect. Thus one may be misled by a report of marked sensitivity in vitro and yet the in vivo effect may be inadequate for cure. There is a general tendency to use inadequate dosage which may hasten the emergence of resistant strains. It is probably unwise to use less than 10 million units penicillin daily initially while awaiting positive cultures or the results of sensitivity tests. The concomitant use of streptomycin is indicated, irrespective of the sensitivity of the organism. The alarming frequency of infections due to resistant strains of staphylococci is noteworthy, and such cases may require extremely large doses of penicillin for cure (60-100 million units) or the simultaneous use of several antibiotics including erythromycin.

**Congenital Heart Disease**

Space will not permit a detailed discussion of the common errors made in each of the numerous types of congenital heart disease. Therefore, only a few of the common mistakes are enumerated.

The parent's history regarding cyanosis at birth may be misleading. Mild degrees of arterial oxygen unsaturation may be overlooked and with the passage of time more obvious cyanosis may be apparent. For example, an infant with tetralogy of Fallot may not appear cyanotic at birth, and persistent cyanosis may not be noted until after several months. All cyanosis is not due to congenital heart disease but may be due to chronic lung disease or pulmonary arteriovenous fistula. There is still much confusion regarding the proper interpretation of murmurs. The mere presence of a murmur does not necessarily indicate heart disease. Indeed slight murmurs may be heard in most perfectly normal children. If one hears a murmur which is definitely abnormal, the physician may be able to state that heart disease is present but should not forget that many different defects can produce similar murmurs. For example, a similar basal systolic murmur may be produced by patent ductus, pulmonic stenosis, interatrial septal defect and interventricular septal defect. The absence of murmurs does not rule out congenital heart disease. For instance, endocardial fibrosis, tricuspid atresia, interatrial septal defect, and pulmonary atresia at times may be present without murmurs. In the very young, it may be difficult to localize a murmur because in the thin, small chest, murmurs may be widely transmitted.

The murmur of patent ductus may be heard only in systole during the first few years of life, and it may be impossible to differentiate it from a normal murmur. At an increased age the murmur of patent ductus is usually continuous, but one must not forget that there are other causes of continuous murmurs. The most common continuous murmur is due to a normal venous hum, and abnormal continuous murmurs can be heard in aortic septal defect, pulmonary A-V fistula, collateral circulation in truncus arteriosus or pseudotruncus and anomalous pulmonary venous drainage. A diastolic rumble at the apex may be heard in patients with patent ductus arteriosus, interatrial and interventricular septal defect. One of
the difficulties in the recognition of coarctation is the difficulty of obtaining the blood pressure or pulses in the normal newborn. Patients with pure pulmonic stenosis are not cyanotic, contrary to a widespread erroneous impression. The usual patient with patent ductus is not cyanotic, but on rare occasions when there is a reversed shunt, cyanosis can occur. Electrocardiographic evidence of right ventricular hypertrophy can occur in the latter situation, whereas the usual ductus has a normal electrocardiogram. The occurrence of either a normal axis or left axis deviation in the cyanotic child is a clue to tricuspid atresia. Occasionally the findings of a patient with a high interventricular septal defect may simulate those of an interatrial septal defect and either may simulate an Eisenmenger's complex in early life. Von Gierke's disease is extremely rare, and a more common cause of heart failure in early life in the absence of significant murmurs is endocardial fibrosis. A large heart in a cyanotic patient is more common in transposition of the great vessels than in tetralogy of Fallot. Rales in the lungs and a hacking cough suggest anomalies accompanied by increased pulmonary blood flow. Despite careful examination, an occasional young child with clinical findings consistent with a tetralogy will be found to have complete transposition or pseudotruncus. The importance of adequate fluoroscopic examination is still not appreciated. It is essential to determine whether the pulmonary vascular markings are increased and whether the secondary pulmonary arterial branches pulsate, since this indicates increased flow to the lungs. The opposite findings of decreased vascular markings without pulsations indicate decreased pulmonary blood flow. Often a definite diagnosis cannot be established in the very young until a satisfactory fluoroscopic examination can be carried out with good cooperation of the patient. The left branch of the pulmonary artery will pulsate in an occasional patient with decreased pulmonary flow and cause great confusion.

It is a mistake to assume that all cases of congenital heart disease need to have cardiac catheterization or angiocardiographic studies. The usual case does not. At times, however, these procedures are necessary and must be carried out.

We are now observing cases of congenital heart disease at very young ages when the diagnostic problems are greater. As long as cardiac function is adequate, there should be no great panic as far as the doctor or parents are concerned. As the child grows older the type of defect may become obvious. If not, additional diagnostic procedures can be carried out with greater facility and safety. On the other hand, all steps toward making a diagnosis, including operation, should be carried out in a child who is dying of what is suspected of being a correctible congenital defect.

**Pericardial Disease**

The pain of idiopathic pericarditis may be misinterpreted as pain due to myocardial infarction or to simple "pleurisy." The characteristic aggravation by respiration and change of position from onset serves to differentiate it from the pain of myocardial infarction. The presence of fever and friction rub at or shortly after onset suggests benign pericarditis rather than myocardial infarction. The occurrence of precordial pain aggravated by breathing several days after onset of myocardial infarction may be due to pericarditis or less commonly to pulmonary embolism and should not be misinterpreted as further myocardial infarction.

Rheumatic pericarditis may or may not be accompanied by pain; the majority have clinical evidence of associated myocarditis. The widespread use of antibiotics has resulted in a decrease in pyogenic pericarditis to such an extent that it may not be considered in differential diagnosis. It is usually secondary to pulmonary infection, and the source may be readily overlooked or masked by antibiotic administration. One should not neglect the occurrence of pericarditis as a manifestation of systemic disease such as collagen disease, leukemia, lymphoma, bronchogenic carcinoma or other neoplastic disease. Tuberculous pericarditis characteristically has an insidious onset, but it is a mistake to think that it cannot begin abruptly with chest pain. Protracted fever, night sweats and weight loss are common. It may at times appear as unexplained
fever or congestive failure. It is common in the absence of parenchymal lung disease and is almost invariably associated with a normal leukocyte count and a positive tuberculin skin test. Although massive pericardial effusion is common, tuberculous pericarditis can occur without detectable effusion, and a pericardial rub may be present even in the presence of considerable effusion. It is usually impossible to differentiate clinically or by x-ray examination pericardial effusion from generalized cardiac dilatation. This is particularly true where there is diffuse muscle disease such as Fiedler's myocarditis, amyloid or collagen disease. The best means of differentiation is by pericardial tap. Tubercle bacilli may be recovered by culture of the pericardial fluid in only about one-half of cases.18 Unfortunately, cultures may require some weeks and specific therapy should be promptly instituted where clinical evidence favors a tuberculous etiology. Bloody pericardial effusion is commonly due to tuberculosis or neoplastic disease but may occur with any type of pericardial inflammation, including idiopathic pericarditis.13 In cases of obscure etiology, pericardial biopsy may furnish a clue to the diagnosis.

Heart failure due to constrictive pericarditis may go unrecognized for long periods of time. It should always be considered when the cause of increased venous pressure, hepatomegaly, ascites and edema cannot be readily explained. This is particularly true when there is no orthopnea, and tricuspid valve disease can be eliminated. It is not generally appreciated that dyspnea is the most common symptom and may occur with or without pulmonary congestion. Pulmonary edema is rare except in the small group in which the constriction is localized predominantly to the left ventricle and auriculoventricular groove. The overwhelming majority of cases show a normal or an enlarged cardiac silhouette by x-ray visualization rather than the classic small heart.14 In a few instances systolic murmurs may be heard at the apex and a mid-diastolic third heart sound is common. The presence of such sounds may cause confusion with rheumatic heart disease. Calcium may be demonstrated in the pericardium in about one-half of the cases with x-ray films taken in the lateral projection. Auricular fibrillation, low amplitude of the complexes and nonspecific T-wave abnormalities may be demonstrated in the electrocardiogram. Right axis deviation is occasionally noted and suggests that the constriction is confined largely to the left side of the heart. A characteristic pressure curve may be obtained by catheterization of the right ventricle18 and may serve to differentiate constrictive pericarditis from diffuse muscle disease.

**Pulmonary Embolism**

Pulmonary embolism remains largely unrecognized. It is one of the most important events that can happen in the life of a patient with heart disease. It is most destructive to cardiac reserve and may precipitate heart failure where none existed before, or cause a marked increase in heart failure already present. It should always be suspected when congestive heart failure does not respond to customary therapy. It may produce coronary insufficiency or myocardial infarction in the patient with coronary sclerosis on the one hand and on the other may be misinterpreted as myocardial infarction because of clinical and electrocardiographic similarities. It is not appreciated that pulmonary embolism may cause paroxysmal arrhythmias, particularly auricular fibrillation. The greatest error in recognition results from demanding too much to establish the diagnosis. Thus signs of consolidation in the lung, hemoptysis, positive Homan's sign, persistent calf tenderness occur in perhaps less than half of the cases, and if one awaits the appearance of these the opportunity for early therapy will be missed. In addition to the classic findings, clues to recognition which may be overlooked are: (1) acute pleuritis, (2) syncope, (3) episodes of weakness and sweating, (4) tachycardia out of proportion to fever, (5) paroxysmal auricular fibrillation, (6) refractory heart failure, and (7) repeated attacks of what are thought to be coronary occlusion occurring close together.

**Electrocardiogram**

In spite of the avalanche of literature on electrocardiography the number of pitfalls has
not decreased. Assuming that one interprets electrocardiograms correctly, errors are made when the limitations of electrocardiography are not appreciated. The greatest error in the use of the electrocardiogram is the failure to realize that 75 per cent of patients with angina pectoris due to coronary atherosclerosis have normal records.16 The use of the exercise tolerance test brings forth new problems since minor S-T segment changes are common with tachycardia, particularly in the anxious person. It is in this group with atypical chest pain that a search may be so strenuously pursued for objective evidence of disease. The views of Scherf in this regard are pertinent; he disregards changes unless they are marked and unequivocal.17 It is not commonly appreciated that digitalized patients with normal resting electrocardiograms occasionally develop the S-T force characteristic of digitalis effect after exercise, and simulate the subendocardial injury of a positive exercise test.

The greatest use of the electrocardiogram is the recognition of myocardial infarction. It is particularly useful where definite QRS, S-T and T-wave abnormalities are present. Common errors encountered in this area are as follows: it is not generally appreciated that about 15 per cent of patients with myocardial infarction will demonstrate no electrocardiographic abnormalities. All too often only a single tracing is recorded, and it is not uncommon for electrocardiographic abnormalities to appear several days after an episode of chest pain. At times S-T and T-wave abnormalities are misinterpreted as being due to myocardial infarction, whereas, they may actually be due to pericarditis. A large number of myocardial infarcts produce only T-wave abnormalities. It is here that a large number of mistakes are made. The physician may be so rigid in his electrocardiographic criteria for infarction that he refuses to accept the fact that myocardial infarction can occur with only T-wave changes. At the opposite extreme, others become so alarmed with all T-wave changes that they interpret unimportant findings as grave diagnostic signs. Left bundle-branch block may obscure the QRS findings of myocardial infarction, and this has led to confusion, forcing some to state that infarct cannot be diagnosed if left bundle branch block is present. This is not invariably true since certain S-T and T abnormalities may offer a clue to infarct even in the presence of such conduction disturbance.

It is generally recognized that the current of injury may subside following myocardial infarction. At times, however, marked S-T segment shift may persist for months or years and does not indicate new injury. It is also generally appreciated that ischemic effects of myocardial infarction may subside after several weeks or months. However, it is not common knowledge that definite QRS changes may vanish after months or years, leaving the electrocardiogram completely normal.

Right ventricular hypertrophy as determined electrocardiographically is normal in the newborn and persists for a variable period, perhaps one to two years. Accordingly, abnormal right ventricular hypertrophy cannot be diagnosed until this period has elapsed. Once this period has passed, the electrocardiogram is fairly reliable in indicating right ventricular hypertrophy due to congenital heart disease. It does not aid in determining the etiology of the right ventricular hypertrophy since the right ventricular hypertrophy produced by pulmonary stenosis, pulmonary stenosis with patent foramen ovale, interatrial septal defect, tetralogy of Fallot, transposition of the great vessels and Eisenmenger's complex appears the same. On the other hand, considerable right ventricular hypertrophy may be present due to acquired disease, such as emphysema or mitral stenosis, and the electrocardiogram may remain normal.

Much left ventricular hypertrophy can be present and the electrocardiogram may remain normal. When the mean QRS vector is directed leftward and posteriorly, left ventricular hypertrophy may be suspected when the QRS magnitude is increased. Care must be taken in making such a diagnosis, since the person with a thin chest may have an increased QRS magnitude in the chest leads simply because the recording electrode is nearer the source of electrical energy. When the QRS vector is directed to the left and posteriorly and the mean T vector is oppositely directed, left ventricular hypertrophy may be suspected with or without
increased QRS voltage. This criteria is also fallible since various conditions producing left ventricular ischemia may produce similar findings. The electrocardiogram offers no clue to the cause of left ventricular hypertrophy, since similar findings result from aortic stenosis, aortic regurgitation, essential hypertension and coarctation. The findings of left ventricular hypertrophy in a cyanotic infant or child usually indicates tricuspid atresia.

A patent ductus is usually associated with a normal electrocardiogram. Left ventricular hypertrophy as determined by the electrocardiogram may occasionally occur, but right ventricular hypertrophy is quite rare. Interventricular septal defect is occasionally associated with large biphasic complexes in lead I suggestive of biventricular hypertrophy. Right ventricular hypertrophy may occasionally occur when the interventricular septal defect is high and physiologically simulates an interatrial septal defect.

Right bundle-branch block is usually due to coronary disease but also is produced by interatrial septal defect, pulmonary embolism, and myocarditis. It must never be forgotten that such a conduction defect may be congenital in origin and completely unimportant. Left bundle-branch block is usually due to coronary disease or myocarditis. It may obscure the QRS abnormality of myocardial infarct unless the septum is involved. S-T and T-wave abnormalities due to myocardial infarct may be observed, however, when the infarct is recent.

Pulmonary embolism frequently produces sinus tachycardia without other electrocardiographic abnormalities. At times the nonspecific findings of auricular fibrillation, subendocardial injury, right bundle-branch block, anterior myocardial ischemia are observed. Right ventricular conduction defect may be produced and under certain circumstances has been misinterpreted as posterior myocardial infarction.

During the last year the following misconceptions regarding the electrocardiogram have been encountered:

1. Wrong concepts about congestive heart failure and the electrocardiogram. The electrocardiogram gives no information about the pumping action of the heart.
2. Misconceptions about digitalis and the electrocardiogram. There are no electrocardiographic findings that indicate when a patient needs digitalis. Indeed one should not depend on the electrocardiogram to judge whether a patient needs digitalis, how much digitalis he has had, or if he is fully digitalized. One person may be fully digitalized and the electrocardiogram may remain normal while another subject's electrocardiogram may show the characteristic S-T and T findings due to digitalis after a fraction of the usual digitalizing dose. There is evidence also to indicate that digitalis effect may occur more readily when the heart is abnormal, particularly when there is ventricular hypertrophy. One should never forget that clinical digitalis toxicity can occur without evidence of toxicity in the electrocardiogram, and that electrocardiographic evidence of toxicity can occur without clinical symptoms. Digitalis interferes with the standard exercise test.

3. Misconceptions of establishing etiology from the electrocardiogram. In general, one cannot determine the etiology of heart disease by inspecting the electrocardiogram. For example, clinical data is needed to determine why there is right ventricular hypertrophy.

4. The false concept that treatment can be determined by inspecting an electrocardiogram. There is nothing about electrocardiography that dictates therapy. This is even true when arrhythmias are clearly diagnosed, since the individual who studied only the electrocardiogram would be unaware of previous therapy. There is no treatment, and none is needed, for certain purely electrocardiographic abnormalities such as ventricular ischemia and bundle block.

5. A wrong conception concerning the determination of prognosis from the electrocardiogram. The prognosis of a patient with an electrocardiographic abnormality is that of the underlying disease producing the abnormality. For example, the subject with right bundle block due to a congenital anomaly of the conduction system should live a normal life span whereas the patient with similar right bundle-branch block together with Fiedler's myocarditis has a very poor outlook.

6. The misconception that a normal electrocardiogram indicates a normal heart. As indi-
cated, most errors are in this general area. The prime example of this pitfall is spot-lighted when one recalls that 75 per cent of the electrocardiograms are normal in patients with angina pectoris due to coronary atherosclerosis.

7. A misconception that an abnormal electrocardiogram indicates heart disease. The problem here is failure to appreciate the normal range of any biologic information.

8. The faulty concept that the electrocardiogram always solves a complicated rhythm. Although the electrocardiogram is an indispensable aid to the diagnosis of cardiac arrhythmias, it is not uncommon to see complicated arrhythmias that are very difficult to solve, and at times multiple explanations may be plausible.

**Errors in Therapy**

**Digitalis**

Much of the confusion regarding digitalis therapy exists because of the multitude of available preparations. One should master the use of an oral, intramuscular and an intravenous preparation rather than resorting to a variety of preparations. Confusion still exists in determining when a patient is adequately digitalized. This can only be determined by trial and error by giving increased increments of digitalis until a therapeutic response is obtained or minor toxic symptoms develop. In our experience, 0.2 mg. of digitoxin is too much for the average patient, and over a period of time may result in toxicity. When auricular fibrillation is present, the apex rate during mild effort should be used as a guide to adequate digitalization, since the ventricular rate may be normal at rest and unduly accelerated with activity. The presence of ventricular premature beats does not contraindicate digitalis in the patient with heart failure who has not been digitalized; they may disappear following administration of the drug. The presence of myocarditis or myocardial infarction does not preclude digitalis administration; however, it should be used with greater caution. In the presence of myocarditis, thyrotoxicosis, and pulmonary embolism, one cannot determine the dose by the pulse response but must choose an arbitrary dose according to the patient’s weight. The young child cannot relate the symptoms of digitalis toxicity and the therapeutic response may not be as clearly defined as in the adult. Accordingly, an average dose based on previous experience must be used.4

Digitalis intoxication is commonly overlooked. This is partly due to the lack of appreciation that there may be only a single toxic symptom, and that toxicity may occur without significant electrocardiographic changes. The most common toxic symptom due to digitalis is anorexia, and this may be particularly overlooked when digitalization was instituted at some prior time. The accompanying weight loss over a period of time may be so severe that malignancy may be suspected. Nausea, vomiting, diarrhea, abdominal discomfort and pain, visual disturbances and psychosis may occur and are readily recognized as toxic manifestations. Several days may be required after discontinuing the drug before toxic symptoms subside. Vigorous diuresis of the edematous patient by mercurial diuretics may result in digitalis intoxication. This is due to the loss of potassium as well as to possible mobilization of digitalis. Pulsus bigeminus due to ventricular ectopic beats is the most common arrhythmia due to digitalis. One should not forget that any type of cardiac arrhythmia may result from digitalis therapy. When the pulse rate increases rather than decreases after digitalis, one should be certain that ventricular tachycardia is not present before further drug is given. The loss of potassium by diuresis, vomiting, diarrhea or intubation may precipitate cardiac arrhythmia, particularly in the digitalized patient, even though the serum potassium be within the normal range. Paroxysmal auricular tachycardia with A-V block is particularly prone to occur under such circumstances.15

The administration of potassium may control the disturbances. A rare patient can tolerate only small amounts of digitalis; it usually helps very little to change types or brands of digitalis.

**Nitroglycerin**

One of the most common errors in the management of the patient with angina pectoris is the overcautious attitude of the physician regarding the use of nitroglycerin. Patients should be instructed to take the drug promptly and as frequently as necessary. The family must also be instructed in order to avoid undue anxiety.
The initial administration of nitroglycerin should be under observation and the drug given in a dosage of 0.3 mg. to avoid disagreeable side effects that may discourage the patient from using it subsequently. The patient should be urged to use the drug in anticipation of events which frequently bring on pain, for example, just prior to meals or exertion, before bathing, before retiring, prior to intercourse, upon first awakening and prior to situations involving increased emotional stress.

Mercurial Diuretics

Mercurial diuretics are indicated when congestive heart failure is not satisfactorily controlled by digitalis and sodium restriction. These drugs are indicated when there are distressing symptoms that would ordinarily require many hours or days to relieve with digitalis and diet alone. For example, paroxysmal nocturnal dyspnea in a nonedematous patient is an indication for mercurial diuretics. In the patient with congestive heart failure, a record of the weight should be kept, and if an abrupt gain of three pounds occurs, a mercurial diuretic should be administered. Often it may be desirable to administer the drug at regular intervals to prevent the recurrence of congestive phenomena. There may be unjustified reluctance to use mercurial diuretics when albumin and red cells are found in the urine; indeed such therapy may result in clearing of these findings when due to heart failure. Even the presence of chronic renal disease does not preclude their use, although enjoining caution. The nonprotein nitrogen may be elevated to 80 mg. per 100 cc. due to heart failure alone and administration of these drugs may reduce it. It is not common knowledge that chills and fever may rarely follow the injection of a mercurial diuretic. When there is a failure of expected diuresis after an injection, one should suspect electrolyte depletion, particularly when there are symptoms of weakness, drowsiness, muscle cramps, anorexia, nausea and vomiting.

REFERENCES


Errors in the Recognition and Treatment of Heart Disease
R. BRUCE LOGUE and J. WILLIS HURST

Circulation. 1954;10:920-932
doi: 10.1161/01.CIR.10.6.920
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1954 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/10/6/920.citation