THE DIAGNOSIS AND TREATMENT OF CARDIAC EMERGENCIES

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Situations in which the prompt institution of proper treatment may be directly responsible for the saving of life probably are encountered more frequently in patients who have organic heart disease than in any other group of individuals presenting medical problems. A thorough understanding of the therapeutic measures available for use in cardiac emergencies, therefore, is of considerable importance. Fortunately, practically all of the measures of established value are of such a nature that one can be prepared at all times to use them.

ACUTE MYOCARDIAL INFARCTION

The most common cardiac emergency is acute coronary artery occlusion with infarction of the myocardium. The clinical picture of this condition is too well known to warrant detailed description. The pain is similar to that of angina pectoris, but is more severe and of longer duration. Symptoms of shock of mild to severe degree usually appear soon after the onset, and fever and leukocytosis generally develop within the first 24 hours. A pericardial friction rub may appear at any time during the first few days after the attack and may last for only a few hours or for several days. The electrocardiogram shows characteristic changes in practically every case if records are made at daily intervals and if both standard and precordial leads are used.

The first aim in the treatment of acute myocardial infarction is to relieve the pain. For this purpose aminophyllin is administered by intravenous injection, usually in a dose of 0.48 gm. in 20 cc. of solution. In certain patients this drug relieves the substernal pain almost immediately, but if it does not do so, it should be followed promptly by the hypodermic administration of morphine sulfate. The dose of this drug is usually ¼ grain, but whenever the pain is exceptionally severe one should not hesitate to administer ½ grain. Subsequent doses of ¼ grain should be given at intervals of approximately one-half hour if the distress continues unabated. Immediately after the administration of aminophyllin, and regardless of whether morphine sulfate is necessary, the patient should be given atropine sulfate by hypodermic injection. LeRoy and Snider[1] have demonstrated that sudden death after experimental ligation of a coronary artery is due to ventricular fibrillation, and that this arrhythmia is caused by vagal coronary constriction which, in turn, results in generalized myocardial ischemia. If the vagal vasoconstriction is reduced or abolished by adequate amounts of atropine,

the incidence of sudden death after coronary artery ligation is considerably reduced. In man the usual initial dose of atropine sulfate is 1/75th grain, and additional doses of 1/150th grain are given every six or eight hours during the first three or four days.

The patient should be placed in bed as soon as possible after the onset of symptoms and should not be disturbed by frequent examinations. Because of the shock and profuse perspiration which often are present, the body should be kept warm, and as soon as the patient is more comfortable and is free from nausea and vomiting, small amounts of fluids should be offered at frequent intervals. Stimulants, such as caffeine with sodium benzoate, are administered only if the systolic blood pressure falls below 80 mm. of mercury.

In the more severe attacks of myocardial infarction which are accompanied by cyanosis and intense dyspnea, oxygen should be administered as promptly as possible, either by means of an oxygen tent, nasal catheter, or a B. L. B. mask. This measure not only reduces the cyanosis and dyspnea, but also may lessen the intensity and shorten the duration of the pain.

After the pain and initial shock have been controlled, most patients require little medication. Atropine should be continued in the prescribed dosage, and it probably is best to administer adequate amounts of one of the xanthine preparations, preferably theobromine with sodium acetate in doses of 7½ grains four times a day. If the heart rate remains unduly elevated or if frequent premature beats develop, quinidine sulfate should be given in doses of 3 grains or 5 grains every three or four hours as a possible prophylactic measure against the development of ventricular paroxysmal tachycardia and ventricular fibrillation. Sedatives may be necessary in small divided doses during the day or at bedtime to control restlessness. Digitalis is administered only in the event of congestive myocardial failure, or when there is auricular fibrillation with a rapid ventricular rate. The diet should be simple and should be limited to a value of 800 or 1000 calories. If the bowels do not move spontaneously, enemas should not be administered until after the second or third day. The emphasis in treatment should be placed upon the necessity for absolute rest. The patient should be fed and should not be allowed to help in changing his position in bed for at least three weeks. The total period of rest in bed should be from six to eight weeks. The erythrocyte sedimentation rate is a helpful guide in this respect; rest is enforced until the rate shows a considerable return toward normal and reaches a stationary level. After the period of rest, the patient is permitted to be up for gradually increasing lengths of time daily, but is not allowed to return to his business activities for from three to twelve months, depending upon the severity of the attack.

ADVANCED CONGESTIVE HEART FAILURE

The second most common type of cardiac emergency is encountered in patients who have congestive heart failure and are first seen after their condition has become critical. In situations of this kind the patient usually has experienced increasingly severe symptoms for days or even weeks, and examination reveals an exhausted, apprehensive individual in extreme respiratory discomfort and often in a condition bordering upon shock. Orthopnea is present, and there is cyanosis, engorgement of the jugular veins, an enlarged, tender liver, and extensive peripheral edema. Hydrothorax and ascites may be present, and in many patients there is repeated vomiting. The cardiac rhythm may be regular or irregular; the most common type of arrhythmia observed is auricular fibrillation with a very rapid ventricular rate and a large radial pulse deficit.

In an emergency of this kind the first indication is for the administration of digitalis, and because a delay of even a few hours in obtaining the therapeutic effect of the drug may mean the difference between a fatal and a successful outcome, intravenous administration is necessary. It is important to remember in this connection that when digitalis action is urgently needed, one must not rely upon intramuscular injection. There are several preparations of digitalis which are marketed in ampules for intravenous use. Unfortunately, these preparations differ considerably in potency among themselves, and to a somewhat lesser extent, in different lots of the same product. It is therefore advisable that one become familiar with the clinical action of a single preparation and confine himself to the use of that product. For several of the preparations on the market, an initial intravenous dose of 10 cc. is suitable, and the same amount may be given again if necessary after an interval of four hours. After this, it is generally advisable to complete the process of digitalization either by oral administration of the drug, if vomiting has ceased, or by intramuscular injection.

Strophanthin may be given intravenously in place of one of the digitalis preparations. This drug, however, has no definite advantage over digitalis and is dangerous when given in large doses. The initial dose should be not more than 0.5 mg. Additional doses of 0.1 mg. may be given if necessary at intervals of four hours until a total of not more than 1 mg. has been administered. It is, of course, important to ascertain that patients to whom digitalis or strophanthin is given intravenously have not received digitalis during the preceding two weeks.

The administration of digitalis or strophanthin may produce remarkably prompt improvement in the patient who is critically ill with congestive heart failure, particularly when auricular fibrillation is present. In patients with auricular fibrillation, slowing of the ventricular

rate usually is noted within five minutes after administration of the drug, and within thirty minutes there may be complete cessation of vomiting and great diminution in the degree of dyspnea. Strophanthin attains its maximum effect in approximately one hour and the digitalis preparations in about two hours after intravenous injection.

In addition to receiving digitalis or strophanthin intravenously, patients who are desperately ill with congestive failure should be given morphine sulfate promptly by hypodermic injection. This drug depresses the respiratory and higher cerebral centers and relieves the patient's dyspnea and his anxiety and apprehension. Not only should morphine be administered when the patient is first seen, but a second injection, usually of ¼ grain, also should be given the same evening to insure a comfortable night's rest. Several hours' sleep frequently results in striking improvement in the general condition and morale of the patient.

Advanced congestive failure often is attended by the accumulation of large amounts of fluid in the serous cavities of the body. Extensive hydrothorax may be present on one or both sides and, by compressing the lung, may be responsible for a considerable part of the reduction in vital capacity and resultant dyspnea. It is important, therefore, that the fluid be removed as completely as possible soon after the initial administration of digitalis and morphine. Less frequently, ascites is present in sufficient amounts to interfere with the movement of the diaphragm and to contribute to the degree of dyspnea. Under such circumstances, abdominal paracentesis is indicated.

In patients with myocardial failure, the peripheral venous pressure is increased approximately in proportion to the degree of decompensation. In severe failure the jugular veins may be engorged to the angle of the jaw even with the patient well propped up in bed. Venesection with the removal of 350 to 600 cc. of blood may result in prompt improvement in cases of this kind and should be employed whenever the institution of digitalis therapy and the other measures mentioned previously fail to produce satisfactory improvement. This procedure directly reduces venous congestion and diminishes the degree of dilatation of the heart. It is desirable to measure the venous pressure during the removal of the blood and to continue the bleeding until the pressure has been reduced to within the upper limits of normal. For practical purposes, however, a reliable guide is furnished by observation of the jugular veins, venesection being continued until jugular distention has been relieved. In favorable cases the venous pressure remains low after having been reduced by venesection, but in unfavorable cases peripheral venous congestion promptly returns.

CARDIAC ASTHMA AND ACUTE PULMONARY EDEMA

Cardiac asthma is a form of paroxysmal dyspnea which occurs in patients who have serious organic heart disease. Its onset occasionally gives the first warning of a damaged heart, but more often the patient has experienced dyspnea or anginal pain on effort for some time before the first seizure. The attacks develop rapidly and are characterized by asthmatic breathing with both inspiratory and expiratory difficulty, orthopnea, and a sense of suffocation. The paroxysms may last from several minutes to a few hours, and death may occur during the attack.

In all but a few cases cardiac asthma is due to failure of a left ventricle which has been damaged previously as the result of hypertension, coronary artery sclerosis, or aortic valve disease. The seizures in these cases usually occur at night, although occasionally they are induced by exertion. Because of relative weakness of the left ventricle, an increased amount of blood gradually accumulates in the pulmonary vessels during sleep in the recumbent position. The vital capacity, which is already diminished, is further reduced as the degree of pulmonary congestion increases, and all that is now needed to initiate the attack of cardiac asthma is some factor which acts as a trigger mechanism. Cough, Cheyne-Stokes respiration, noise, disturbing dreams, and the urinary reflex most commonly supply this factor. The patient wakens with respiratory distress and is forced to sit up or stand in order to breathe. Asthmatic breathing develops, and as the attack progresses, acute pulmonary edema may supervene.

In a much smaller group of patients cardiac asthma results from advanced mitral stenosis without myocardial failure. A series of such cases has been studied by McGinn and White[2] who point out that, in contrast to attacks resulting from failure of the left ventricle, the seizures in uncomplicated mitral stenosis usually are precipitated by exertion, emotional upsets, or paroxysmal tachycardia. When the heart rate is accelerated by any of these factors, the hypertrophied right ventricle expels blood into the pulmonary circulation more rapidly than it can pass through the narrowed mitral orifice. Acute pulmonary congestion develops and produces a paroxysm of cardiac asthma.

The most important measure in the treatment of the attack of cardiac asthma due to left ventricular failure is the intravenous administration of aminophyllin, usually in a dose of 0.48 gm. in 20 cc. of solution. This may produce prompt and lasting improvement. The beneficial effect of the preparation has been attributed principally to its action on the coronary circulation,[3] but the drug also causes a diminution in the degree of bronchial spasm.[4] It is known that bronchial spasm is present during the paroxysm of cardiac asthma and undoubtedly contributes importantly to the degree of dyspnea. It may

be, therefore, that the beneficial effect of aminophyllin is due as much to its effect on the bronchial musculature as to its action on the coronary arteries. If the drug does not prove sufficiently beneficial, it should be followed promptly by the hypodermic administration of morphine sulfate, ¼ grain, which should be repeated if the patient is not improved within 15 or 20 minutes. Venesection, with the removal of 350 to 500 cc. of blood should be carried out in patients in whom the venous pressure is elevated and may prove to be a very helpful measure. An effect similar to that of venesection may be obtained by applying blood pressure cuffs to the four extremities and inflating them to a pressure just above diastolic blood pressure. The administration of oxygen by means of a tent, mask, or nasal catheter is also of great value and should be instituted whenever aminophyllin and morphine do not give sufficient relief.

When cardiac asthma progresses to acute pulmonary edema in spite of the above measures, either strophanthin or a suitable preparation of digitalis should be given by intravenous injection. It is, of course, essential to ascertain that these patients have not received digitalis previously.

The treatment of cardiac asthma due to uncomplicated mitral stenosis is similar to that employed for attacks resulting from left ventricular failure. When the seizures are due to paroxysmal auricular fibrillation, rapid digitalization should be carried out, and when the precipitating factor is paroxysmal tachycardia, suitable measures for this condition must be employed.

A patient who has experienced an attack of cardiac asthma due to failure of the left ventricle should be treated as any other individual who presents evidence of impaired myocardial reserve. Complete digitalization and the subsequent administration of daily maintenance doses of the drug are indicated in individuals who have had but mild attacks, and this may suffice to prevent the recurrence of paroxysms. In those who have suffered more severe attacks, a period of absolute rest is advisable and should be followed by strict limitation of activity. Restriction of fluids, a diet low in sodium chloride content, and the administration of diuretic drugs also are valuable measures. At times, the intravenous injection of hypertonic glucose solution (50 cc. of a 50 per cent solution daily for several days) helps to reduce the frequency of the attacks. Because cardiac asthma due to left ventricular failure usually occurs at night and because the onset of the seizure is favored by the recumbent position, the patient should be instructed to sleep well propped up in bed. Sedatives should be used with caution since their depressant effect upon the respiratory center may favor rather than hinder the development of attacks.

In cardiac asthma due to uncomplicated mitral stenosis, digitalis is seldom effective in preventing the recurrence of attacks except when the paroxysms are induced by auricular paroxysmal tachycardia. Diuretics also are of little value, and therefore it is of great importance that these patients avoid exertion and emotional upsets which may precipitate an attack. Sedatives should be given daily in divided doses to those patients who display evidence of emotional instability.

ACUTE COR PULMONALE

Sudden embolic occlusion of the pulmonary artery or its primary branches may be immediately fatal or may cause severe dyspnea associated with substernal oppression and the rapid development of a state of shock. If the patient survives the onset of the attack, evidence of prompt dilatation and failure of the chambers of the right side of the heart develops. McGinn and White[5] have termed this cardiac disturbance "the acute cor pulmonale." They reported nine cases of acute cor pulmonale and described the clinical and electrocardiographic features which differentiate the condition from acute myocardial infarction. The most important clinical features are an increased pulsation palpable in the left second intercostal space adjacent to the sternum, accentuation of the pulmonary second sound, the frequent occurrence of gallop rhythm over the pulmonary area, the occasional presence of a friction rub in the second and third interspaces adjacent to the sternum, and, unless the patient is in deep shock, engorged and distended neck veins in the absence of signs of passive congestion in the lung bases.

The medical measures employed in the treatment of acute cor pulmonale due to pulmonary embolism consist of the administration of oxygen by tent, mask, or nasal catheter, and the intravenous administration of papaverine hydrochloride, ¼ grain, or atropine sulfate 1/100 grain. Morphine may be necessary for the control of pleural pain. Surgical removal of the embolus has been attempted, and in rare instances the operation has been successful.

DISTURBANCES OF CARDIAC RHYTHM

Certain disturbances of cardiac rhythm are sufficiently important to be classified as cardiac emergencies, while others are of little significance, although they may produce symptoms that alarm the patient. Ventricular paroxysmal tachycardia is an example of the first type of disturbance, and auricular paroxysmal tachycardia belongs to the second group. Ventricular paroxysmal tachycardia is a relatively uncommon condition which usually is due to serious organic heart disease. It occurs most commonly as a complication of myocardial infarction and may be a forerunner of ventricular fibrillation. Occasionally, however, it is encountered in the absence of any evidence of organic heart disease. In

patients who have coronary artery disease, the paroxysm of tachycardia may be attended by serious collapse and occasionally by the rapid development of acute pulmonary edema. Levine[6] has pointed out that in many cases ventricular paroxysmal tachycardia may be recognized by clinical means alone. Carotid sinus or ocular pressure and breath-holding do not affect the heart rate in this condition as they do in auricular paroxysmal tachycardia and auricular flutter. Furthermore, if one listens carefully to the heart, an occasional slight irregularity in rhythm will be noted, and the intensity of the first heart sound will vary from time to time. The one drug of established value in the treatment of this type of tachycardia is quinidine sulfate administered by mouth. Usually an initial dose of 3 grains is given, and this is followed at intervals of two hours by additional doses of 6 grains each until sinus rhythm is reestablished. Morphine may be necessary to relieve dyspnea, and the occurrence of acute pulmonary edema calls for the administration of oxygen.

Paroxysmal auricular flutter seldom occurs in the absence of organic heart disease. When the ventricular rate is elevated to 160 beats per minute or more, the condition may be responsible for the rapid development of congestive heart failure. In those cases in which the ventricular rhythm is regular and the rate between 120 and 200 beats per minute, the disturbance must be differentiated from sinus tachycardia and from auricular paroxysmal tachycardia. The electrocardiogram affords the most precise means of establishing the diagnosis, but the arrhythmia can often be recognized without instrumental aid. Careful inspection of the venous pulsations in the neck may clearly reveal two or more auricular pulsations to each ventricular wave. In auricular flutter the ventricular rate remains constant within very narrow limits and is not appreciably affected by exercise. This is in contrast to the variability of the rate in sinus tachycardia, but does not aid in distinguishing the condition from auricular paroxysmal tachycardia. Pressure upon the carotid sinus may cause temporary slowing of the ventricular rate in auricular flutter, or it may produce an abrupt standstill of the heart for a variable length of time, followed by resumption of the original rate. The first of these responses may be obtained in patients with sinus tachycardia, but the second does not occur. In auricular paroxysmal tachycardia pressure upon the carotid sinus either produces an abrupt reversion to normal rhythm, or has no effect at all. The ventricular rhythm in auricular flutter may be irregular because of variations in the degree of auriculoventricular block, and at times the irregularity is sufficiently marked to suggest auricular fibrillation. Careful auscultation usually reveals an underlying dominant rhythm, however, and this distinguishes the condition from auricular fibrillation.

Although the administration of quinidine sulfate may convert auricular flutter to sinus rhythm, the preferred treatment for the arrhythmia consists of rapid digitalization. In successful cases digitalis converts auricular flutter to auricular fibrillation, and discontinuance of the drug may then be followed by spontaneous resumption of normal sinus rhythm.

Auricular paroxysmal tachycardia is encountered much more often in individuals who have normal hearts than in those who present evidence of organic heart disease. Although the patient may be greatly disturbed by weakness, light-headedness, and palpitation incident to the attack, the tachycardia seldom produces evidence of coronary or myocardial insufficiency. Occasionally, however, in the presence of organic heart disease, the patient may experience severe substernal pain or develop symptoms and signs of congestive myocardial failure. The paroxysms usually begin and terminate suddenly and are characterized by a perfectly regular rhythm, generally with a rate between 160 and 200 beats per minute. There are a number of simple procedures that often are effective in abruptly terminating the attack. The most reliable of these are carotid sinus or ocular pressure and breath-holding. When these measures fail, drug therapy should be employed, and the most effective preparation is mecholyl by subcutaneous injection. The usual initial dose of this drug for adults is 20 mg., and a second dose of 20 mg. may be given, if necessary, after 20 or 30 minutes. In many cases normal heart rhythm will be reestablished within 15 minutes after the first injection, and before administering a second dose, it is best to massage the site of the injection and repeat the carotid sinus or ocular pressure, or breath-holding. A syringe containing atropine sulfate, 1/100 grain, should be prepared before the first dose is administered, so that this drug can be given by intravenous injection if mecholyl causes disagreeable or alarming symptoms of excessive parasympathetic stimulation. Mecholyl should not be employed in individuals who have bronchial asthma or coronary artery disease. If the drug does not control the tachycardia, quinidine sulfate may be given by mouth in the manner outlined for the treatment of ventricular paroxysmal tachycardia. If this also fails, moderately rapid digitalization should be instituted and may be effective in terminating the tachycardia.

The higher grades of auriculoventricular block, and particularly complete auriculoventricular dissociation, may be complicated by Adams-Stokes attacks due to temporary standstill of the ventricles. The seizures are characterized by dizziness, syncope, or convulsions, depending upon the duration of the ventricular asystole. Adams-Stokes attacks are not common, but individuals in whom they occur are liable to have repeated seizures. The actual attacks are of such short duration that

they seldom require treatment, and therapy therefore is directed toward preventing their recurrence. Occasionally, however, the standstill of the ventricles is of such duration as to necessitate the intracardiac injection of epinephrine, and this procedure may be directly responsible for the saving of life. The most effective drugs for preventing recurrent attacks are epinephrine (0.3 cc. to 1.0 cc. of the 1:1000 solution) by intramuscular injection every three or four hours and ephedrine sulfate (⅜ grain to ½ grain) by mouth three or four times in 24 hours.

ACUTE CARDIAC COMPRESSION

Acute compression of the heart results from the rapid accumulation of serous exudate, pus, or blood in the pericardial sac. In the usual case of serous or sero-fibrinous pericardial effusion, the amount of fluid present is not sufficiently large to embarrass the circulation importantly, and pericardial paracentesis therefore is not necessary. Occasionally, however, an effusion becomes so extensive that the superior and inferior vena cavae are compressed and the return flow of blood to the right auricle is interfered with. Unless this excessive intrapericardial pressure is relieved, the condition will rapidly prove fatal. The most important indications that a pericardial effusion is attaining dangerous proportions are orthopnea, cyanosis, greatly elevated venous pressure, and a rapid fall in arterial blood pressure with a small pulse pressure. The development of these symptoms and signs indicates the need for paracentesis with the removal of as much fluid as possible without producing pain, cough, or faintness. The procedure is carried out under novocaine anesthesia, and the needle usually is introduced either in the left fifth intercostal space just within the outer border of dulness, or in the right fourth intercostal space just inside the right border of dulness.

Purulent pericarditis is an unusual condition which generally occurs as a complication of pneumococcus pneumonia or staphylococcus osteomyelitis. The development of signs of pericardial effusion and acute compression of the heart in these conditions should be interpreted as an indication for immediate paracentesis. The discovery that the effusion is purulent calls, in turn, for surgical drainage.

Hemopericardium with resultant acute cardiac tamponade may result from a number of conditions, but the only form amenable to treatment is that due to penetrating wounds of the heart. The treatment is, of course, surgical, but a successful operation depends directly upon prompt recognition of the condition.

SUMMARY

The most frequently encountered cardiac emergencies are acute myocardial infarction, far advanced congestive heart failure, cardiac

asthma, and certain disturbances of heart rhythm. Less common emergencies consist of acute compression of the heart due to the rapid accumulation of blood, pus, and serous exudate in the pericardium, and acute cor pulmonale. The diagnosis and treatment of each of these conditions have been discussed.

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