AlTHOUGH the majority of emergencies of cardiac origin occur in patients with structural heart disease, many involve individuals without demonstrable organic lesions. Those affecting the latter are usually due to changes in rhythm. The most common of these are the paroxysmal supraventricular tachycardias.

Most cardiac emergencies are readily diagnosed by bedside examination, but occasionally, and especially in the presence of arrhythmias, the use of precision records such as the electrocardiogram is necessary to make an accurate diagnosis. It is, of course, essential to interpret correctly the nature of any emergency in order to institute proper treatment. In this article the methods used in recognizing the various emergencies will not be stressed and only the therapeutic procedures employed in their management will be discussed.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIAS

Auricular Tachycardia

This is the most common of all paroxysmal tachycardias and occurs most often in individuals who are free from structural heart disease. In such patients the paroxysms are usually of short duration and require little or no therapy. Despite the benign nature of these episodes, medical help is not infrequently sought, especially during the initial attacks, or for paroxysms which do not respond to simple measures which most patients learn to use after experiencing repeated attacks. Among the latter are the Valsalva procedure of attempted forced expiration with the glottis shut, prolonged holding of the breath, compression of the neck, and the assumption of various positions, such as lowering the head over the side of a couch, or twisting the head in extreme rotation to either side. Lastly, patients often induce vomiting by mechanical irritation of the pharynx or by drinking mustard diluted in water.

Although death is uncommon in adults during auricular paroxysmal tachycardia, even when organic heart disease is present, serious complications, including heart failure, may occasionally ensue. In infants and young children, however, heart failure and death may occur in the absence of recognizable heart disease. When structural heart disease is present, symptoms including dyspnea, substernal pain, vertigo and syncope may result. Persistence of this rapid arrhythmia may produce heart failure. In such circumstances it is imperative that the impaired heart be spared the excessive burden of the tachycardia by abolishing the abnormal mechanism as soon as possible.

Since most patients with paroxysmal tachycardia suffer from considerable apprehension during an attack, especially during the initial episodes, a sedative in the form of a rapidly acting barbiturate should be administered as promptly as possible. The patient should be placed at rest and reassured of the innocence of the attack. These measures alone will frequently produce reversion to normal rhythm.

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As a rule morphine and other opiates are contraindicated because of the danger of possible habituation. This is particularly true if the patient does not have underlying heart disease.

If the paroxysm still persists then other methods of therapy should be used. Among these are several physical procedures. Carotid sinus pressure is the one most often tried, and probably the most successful in abolishing paroxysmal tachycardia of auricular and nodal origin. Carotid pressure is most likely to be effective if the sinuses are massaged, trying first the right and then the left. This is best performed by pressing against the carotid sinus at the angle of the jaw, compressing the carotid posteriorly and medially against the vertebral bodies. Considerable pressure in the massaging is required. If this measure is not successful before medication, it is frequently effective in converting supraventricular tachycardias after the patient has been treated with one of the drugs which produce vagal stimulation. The latter include the digitalis substances and Mecholyt. Their uses will be described below. Ocular pressure is also a physical measure to produce vagal stimulation. It is not as effective and apparently is now less often used than carotid sinus massage. Occasionally damage to the eye may result from this treatment.

Although quinidin still seems to be the drug preferred by many clinicians to terminate auricular and nodal paroxysmal tachycardias, one of the rapidly acting glycosides of digitalis is being used more frequently as the drug of choice. In infants and young children who become critically ill in the presence of auricular paroxysmal tachycardias, intravenous administration of a digitalis preparation is not only effective but may actually be life-saving. Digitalis may be given by mouth in the usual digitalizing dosage if there is no urgency.

Ouabain, given intravenously, is probably the best preparation for the rapid conversion of auricular or nodal paroxysmal tachycardia. There is danger in employing this, or any other digitalis preparation, if the patient has received digitalis within the previous two weeks. The initial dose for adults is 0.5 mg. followed every hour by 0.1 mg. until the paroxysm ceases or a total of 1.0 mg. has been administered. Most patients obtain full therapeutic effect with 0.8 mg. of ouabain.

If ouabain is not available, lanatoside C (Cedilanid) may be administered intravenously. The initial dose is 0.8 mg., followed by 0.4 mg. if no favorable response is obtained in one hour. The majority of patients will respond to the initial dose of 0.8 mg. However, a total of 1.6 mg. of lanatoside C may be required. Digoxin, also given intravenously, is another preparation which may be employed for rapid digitalization in the management of auricular and nodal paroxysmal tachycardias. When administered intravenously the introductory dose of 0.5 mg. is followed every two hours by 0.25 mg. until full effect is obtained, or until a total of 1.5 mg. has been given.

Recently another digitalis-like substance, acetyl strophanthidin, has aroused interest but mainly from an experimental standpoint. Apparently this drug, when given intravenously, "causes an almost immediate effect on the ventricular rate of auricular fibrillation." Based on clinical work reported to date, it may prove to be of value in the treatment of acute left ventricular failure, where speed is essential, and in the management of auricular paroxysmal tachycardias. At present, there is not sufficient clinical experience to warrant accurate conclusions as to the indications or value of this drug.

Quinidine, while probably less effective than digitalis, is still preferred by many physicians, at least for preliminary trial, in the treatment of supraventricular tachycardia. When using this drug it is wise to give a test dose of 0.2 Gm. to rule out an idiosyncrasy, although true allergic reactions to quinidine are rarely encountered. After waiting one hour, quinidine sulfate, in doses of 0.4 Gm. to 0.6 Gm., should be given orally every two or three hours for five doses. If the attack has not terminated with this schedule of administration, increased dosages of 0.8 Gm. or 1.0 Gm. may be used in a similar manner for subsequent trials. If conversion has not occurred at these high levels it is best to discontinue quinidine, as success is unlikely. Procaine amide (Pronestyl) may be effective in the conversion of this arrhythmia. The
use and dosage of this drug will be discussed below under the treatment of ventricular tachycardia.

Another medication which is effective in the management of this arrhythmia is Mecholyl chloride (acetyl-beta-methyloholine chloride). It is given subcutaneously in doses of 20 to 40 mg. However, relatively few physicians use it because of the rather severe and unpleasant side reactions which invariably occur after the administration of this drug. Its use may be dangerous and is therefore contraindicated in the presence of allergic asthma, anginal syndrome, and following myocardial infarction. It should never be given unless atropine is available for immediate intravenous use if that serious reactions occur. Mecholyl must be administered only subcutaneously and furthermore, because of the fall in blood pressure along with other untoward effects, Mecholyl should be given with the patient in the recumbent position.

If unsuccessful alone, the above mentioned drugs are frequently effective if carotid pressure or other vagal stimulation is applied fifteen minutes to one-half hour after the medications have been given.

A number of therapeutic agents have been recommended for the treatment of paroxysmal tachycardias. None has proved to be superior to those which have been discussed. An old-fashioned remedy is used by many practitioners; namely syrup of ipecac in doses of 4 to 8 cc. This will frequently abolish supraventricular tachycardias because of the vagal effect of inducing nausea and vomiting.

**Paroxysmal Auricular Fibrillation**

Although this arrhythmia may occur in normal individuals it is most commonly observed in the presence of organic heart disease. The transient or paroxysmal type of auricular fibrillation is usually benign and may occur without the patient’s awareness. This is especially true when there are no structural changes in the heart. Paroxysmal auricular fibrillation is a relatively common complication of hyperthyroidism and acute infections, such as pneumonia, even in the absence of structural heart changes. In patients with organic heart disease, including those with rheumatic carditis, fibrillation may precipitate acute heart failure or embolization. It therefore requires emergency treatment.

As with paroxysmal tachycardia, the initial treatment should include physical and mental rest obtained by means of a sedative, preferably a barbituric acid derivative. Morphine may be required in those patients with organic heart disease in whom heart failure may be precipitated by the arrhythmia.

Quinidine is usually effective in converting paroxysmal auricular fibrillation to normal sinus rhythm, and is considered by most clinicians to be the drug of choice for this purpose, regardless of the underlying type of heart disease. However, in the presence of a recent myocardial infarct, caution must be exercised in the use of quinidine, particularly if heart failure or embolism has occurred. There is no doubt that anticoagulant therapy has made this problem somewhat easier, since, with this treatment, the tendency for the deposition of mural thrombi and therefore of embolism can probably be diminished. Most clinicians digitalize patients who show signs of heart failure or who have a rapid ventricular rate as a result of auricular fibrillation. Quinidine is subsequently introduced if reversion to normal sinus rhythm is desired. In the presence of a recent myocardial infarct, or mitral stenosis, or in any other heart disorder in which embolism is likely to occur, anticoagulant treatment is indicated. It is imperative that good laboratory control be available when anticoagulants are used. There is no obvious contraindication to the use of quinidine simultaneously with digitalization or following it. However, quinidine should never be employed in the presence of conduction defects or other evidence of digitalis intoxication. If there is no severe underlying heart disease and no factor which tends to perpetuate auricular fibrillation, such as hyperthyroidism or carditis, quinidine alone will usually restore normal sinus rhythm.

Quinidine sulfate, in doses of 0.4 Gm, given every two or three hours, will abolish most attacks of paroxysmal auricular fibrillation. If ineffectual after several doses, the dosage should be increased to 0.6 Gm. or higher. The
drug should be continued until conversion of the arrhythmia has occurred or symptoms of intoxication appear. Among these are tinnitus, diminished hearing, vomiting, diarrhea or allergic manifestations. It is advisable to give no more than five successive doses in a single 24 hour period. If the patient is vomiting, or if any other condition precludes the oral administration of quinidine sulfate, quinidine may be administered intramuscularly with an initial dose of 0.6 Gm. It is wise to take repeated electrocardiograms during parenteral quinidine administration and to be alert for the development of intraventricular or auriculoventricular conduction defects or other evidences of toxicity. Procaine amide (Pronestyl) may be effective in the conversion of this arrhythmia. The use and dosage of this drug will be discussed below under the treatment of ventricular tachycardia.

Quinacrine (atabrine) has recently been used in the treatment of paroxysmal auricular fibrillation. It is reported to be as effective as quinidine in this arrhythmia and apparently has succeeded in the conversion of auricular fibrillation in cases where quinidine has failed. This would suggest that it may be of use for those patients who do not respond to or who may be sensitive to quinidine. Further clinical evaluation of this drug in the treatment of auricular fibrillation is necessary.

**Paroxysmal Auricular Flutter**

Most patients with this rhythm have some type of structural heart disease although, as in paroxysmal auricular fibrillation, it is sometimes encountered in patients with normal hearts. Acute infections and hyperthyroidism are known to cause this arrhythmia.

In most cases of auricular flutter the patient is aware of the disturbance but is commonly in no great distress. However, heart failure may be present or may ensue, especially where the ventricular rate is rapid. While auricular flutter will occasionally respond to quinidine or to procaine amide, it is probably advisable to digitalize all patients with paroxysmal auricular flutter as soon as the arrhythmia is recognized. This is indicated particularly when there is a rapid ventricular rate, or when congestive failure is present or impending.

If flutter should occur in a patient with a recent myocardial infarct, and digitalis or quinidine is to be used, it is not necessary to employ anticoagulant therapy, as when auricular fibrillation occurs following infarction, since embolism rarely if ever takes place during flutter.

When acute heart failure is associated with flutter, ouabain, lanatoside C, or Digoxin should be given intravenously, provided the patient has not received a digitalis preparation during the previous 14 days. If heart failure is not present or is of mild degree, digitalization may be accomplished orally over a period of a day or so, using the whole leaf of digitalis, or whatever glycoside the clinician prefers. Digitalis therapy usually results in one of the following eventualities: the flutter may be converted to fibrillation and remain as such; fibrillation may then be converted to normal sinus rhythm with quinidine, usually after digitalis has first been discontinued; fibrillation may revert to sinus rhythm spontaneously after withdrawal of digitalis without the necessity of using quinidine; or flutter may persist despite digitalization. In the latter instance, quinidine should be tried, as described under treatment of auricular tachycardia.

**Ventricular Tachycardia**

Ventricular tachycardia occurs, as a rule, in the presence of various structural changes in the heart, especially during the course of myocardial infarction. It is one of the most serious complications which may occur following infarction. Occasionally it is caused by digitalis intoxication, particularly in a patient with a seriously impaired myocardium. Uncommonly it occurs in a normal heart.

The presence of this arrhythmia should be suspected in any patient with heart disease who has a sudden change of heart rate to 160 to 200 or more beats per minute and which is basically regular in rhythm. It is well to remember that it can occur in complete heart block and that it may lead to bouts of ventricular fibrillation which, with ventricular tachycardia, may be a cause of Adams-Stokes seizures.

It is imperative that this rhythmic disturb-
ance be abolished as soon as possible because of the ever present possibility that ventricular tachycardia may become ventricular fibrillation with fatal outcome. Treatment consists of absolute rest, induced, if necessary, by morphine administered subcutaneously or, in emergency, intravenously. At present, quinidine still remains the drug of choice in terminating this arrhythmia. A dose of 0.6 Gm. should be given orally and repeated every two hours until the rhythm reverts to one of sinus origin or until there are evidences of toxicity. These may first be evident in electrocardiograms, which ideally should be taken before each successive dose. Electrocardiographic evidences of quinidine toxicity are conduction defects, chiefly with increase in QRS and the Q-T interval and lowering of the voltage. Recently several preparations (quinidine hydrochloride, quinidine dihydrochloride, quinidine lactate, quinidine gluconate, and quinidine sulfate) have become available which can be given intramuscularly without serious reactions. The recommended initial dose is 0.6 Gm. In desperate cases, quinidine may be used intravenously employing 0.6 Gm. diluted in 5 per cent glucose solution and given by the drip method. During quinidine administration, particularly when the drug is given parenterally, it is advisable to obtain serial electrocardiograms to follow the efficacy of treatment accurately and to watch for signs of toxicity. One authority has recently reported the intravenous route of administration to have been successful in 20 out of 31 attacks. Despite this favorable result we should like to emphasize great caution in the intravenous administration of any form of quinidine because serious or fatal reactions may occur. It would be well if this route were used only when all other agents have failed and only if the patient is in a critical state. A new drug has recently made its appearance for use in the control of ventricular tachycardia. This is procaine amide hydrochloride (Pronestyl hydrochloride). It appears to be sufficiently effective to offer promise. In a reported series, it failed in only two of 32 episodes of ventricular tachycardia.

Preliminary observations of our own and others indicate that procaine amide is also effective in the treatment of supraventricular arrhythmias. These include paroxysmal auricular and nodal tachycardias, auricular fibrillation, and auricular flutter. Procaine amide may be used as a substitute for quinidine; it may be tried in cases where quinidine treatment has been unsuccessful, or where there is intolerance to the latter drug. Like quinidine, procaine amide appears to be least effective when the arrhythmia has been of long standing.

Procaine amide may be employed both orally and intravenously; the latter route has been observed to cause a marked though transient fall in blood pressure. Nausea and vomiting have been noted occasionally as side effects but only where large doses are given orally. Clinical experience to date indicates procaine amide may be less toxic and more effective than quinidine in ventricular tachycardia. Evidence suggests that it is less dangerous when given intravenously than quinidine given by the same route. The dosage recommended for the administration of procaine amide is necessarily provisional at this time because of the limited number of cases which have been studied. The following dosage schedules are suggested: For oral use the drug is supplied in capsules of 250 mg. The oral route is advised with an initial dose of 1.25 Gm. This dose is frequently effective. If there has been no response and no evidence of toxicity, a second dose of 0.75 Gm. may be given in one hour. Further doses of 0.5 to 1.0 Gm. may be given at two to four hour intervals thereafter as required to terminate the aberrant rhythm. Procaine amide is supplied for parenteral (intravenous) use, in ampules of 10 cc., each cc. equivalent to 100 mg. The intravenous route is preferable only in patients who are unable to take oral medication or in acute emergencies, as when shock or failure are impending. The maximum rate of intravenous administration should be 200 mg. per minute, stopping the treatment when the rhythm reverts to normal, if there is any untoward effect or toxicity such as a fall in blood pressure, or when a total of 1 Gm. of procaine amide has been given. No more than 1 Gm. of the drug should be administered as a single intravenous dose. As in the use of parenteral quinidine it is helpful to take electrocardio-
grams, preferably with a direct-writing instrument, during intravenous administration. Procaine amide appears to have a quinidine-like action upon the heart. The toxic effects seen on the electrocardiogram are very similar to those of quinidine. They include prolongation of QRS and the Q-T interval and decrease in voltage. Rarely, ventricular tachycardia and ventricular fibrillation have resulted from procaine amide apparently because of too rapid administration or intravenous dosage in excess of 1 Gm.

In the treatment of ventricular tachycardias this new drug may make unnecessary trial with less effective agents, including magnesium sulfate, potassium chloride or Paredrine hydrobromide.

Although digitalis and its glycosides may, in toxic doses, produce ventricular tachycardia, the number of instances in which this occurs is probably small. The use of digitalis in the presence of ventricular tachycardia following myocardial infarction has recently been reviewed.12 Symptoms, which could have been interpreted as being due to digitalis intoxication in three patients with ventricular tachycardia and heart failure, disappeared in spite of the continued use of digitalis. The conclusions seem to indicate that digitalis may be used safely in the treatment of ventricular tachycardia when congestive heart failure is present. However, this requires further investigation before it can be completely accepted.

**Complete Heart Block**

This rhythmic disturbance may cause no symptoms. However, with the occurrence of an Adams-Stokes seizure, emergency treatment often becomes imperative. Dizziness and syncope, with or without convulsions, are the main symptoms of the syndrome.

Since the Adams-Stokes syndrome may be initiated by ventricular tachycardia or by ventricular fibrillation or may be caused by complete ventricular asystole, differential diagnosis must be decided by the electrocardiogram. The treatment is different depending upon which of the mechanisms is responsible. If the attack is due to ventricular tachycardia or fibrillation, quinidine or procaine amide appear to be the drugs of choice, used as outlined previously.

If an Adams-Stokes seizure is the result of asystole of the ventricles, epinephrine, 1 cc. (1:1000 dilution), is the drug to be used. It should be administered by injection into the heart. This may be followed by ephedrine sulfate, 25 mg. subcutaneously every four hours. If ventricular standstill continues, Paredrine hydrobromide13 should be tried, also by injection into the heart. If cardiac standstill should occur while a patient is undergoing an operation, manual massage of the heart or direct electric stimulation of the ventricles of the exposed heart should be performed.14

If congestive heart failure occurs in the presence of complete heart block, digitalis should be administered. There is no contraindication to its use in this condition unless, of course, the arrhythmia is the result of digitalis intoxication.

**Congestive Heart Failure**

This condition is not infrequently an emergency in those instances where failure has appeared rapidly, or when a patient in congestive heart failure is in need of immediate surgical or obstetric care. Regardless of the type of heart disease, the basic principles of treatment are much the same. Management should be modified by the nature of the precipitating cause of the congestive heart failure. For example, if a myocardial infarct has induced heart failure it is considered advisable to use anticoagulant therapy, as well as such drugs as digitalis or one of its glycosides.

The greatest immediate benefit is accomplished by reducing the excessive burden carried by the impaired heart. This can be done by placing the patient at complete rest but not necessarily confined to bed. If there is considerable anxiety or pain, morphine should be administered subcutaneously or intravenously.

If there is no contraindication, particularly the use of digitalis within the previous two weeks, ouabain, Digoxin, or any rapidly acting digitalis preparation should be given intravenously, regardless of the heart rhythm. The initial dose of ouabain should be 0.5 mg., repeating 0.1 mg. every half to one hour or until therapeutic effect has been obtained, for a total of not more than 1 mg. Digoxin may be given with an initial dose of 0.5 mg., intravenously,
followed by 0.25 mg, every four hours until a favorable response or a maximum of 1.5 mg. have been given. When using lanatoside C intravenously the first dose is 0.8 mg. followed by 0.4 mg. every two hours if needed to obtain a therapeutic response. Total dosage should generally not exceed 2.4 mg.

**Acute Left Ventricular Failure**

This emergency occurs most commonly in patients with the hypertensive type of heart disease. Left ventricular failure may occur also during or after operation, particularly if saline infusions or transfusions have been administered rapidly or in large quantity to a patient with structural heart disease. The prominent symptom is paroxysmal dyspnea; cough is also common. This initial phase may progress to pulmonary edema; there is often expectoration of pink, frothy, watery sputum which may be raised in mouthfuls.

Morphine sulfate, 15 mg., combined with atropine sulfate, 0.6 mg., should immediately be administered hypodermically. If the urgency of the situation demands, morphine may be given intravenously. Nitroglycerin, when given early, seems to benefit some patients. The patient should be placed in Fowler’s position or allowed to sit in a chair, where he will frequently be more comfortable than in bed. Oxygen therapy should be started as soon as possible. It should be administered by means of an oxygen mask metered for positive pressure (Meter Mask) if available. With this method accurate control of pressure can be obtained by conducting the patient’s expired air through water in a bottle calibrated in centimeters. It is advisable to begin with a pressure of 5 cm. of water which is gradually reduced to 1 cm. A concentration of 40 to 60 per cent oxygen with a volume flow of 8 to 10 liters a minute should be employed. Although masks are comfortable for some patients, they are objectionable to others. When discomfort prevents the administration of oxygen by mask, a change should be made to a tent, since this is an effective means of administering 50 to 60 per cent oxygen. In order to obtain this concentration a maintenance flow of 10 to 12 liters per minute is necessary. Although a nasal catheter in the nasopharynx is as a rule more comfortable for long continued use, it is not as effective as the mask since the highest concentration possible by this method is only 40 per cent. A flow of 7 liters of oxygen a minute must be employed in order to maintain this concentration. Unfortunately, mouth breathing, which is quite prevalent, lowers the oxygen percentage in the air inhaled.

If the patient has not had any digitalis in the previous two weeks, ouabain, lanatoside C, Digoxin, or any other rapidly acting preparation, should be given intravenously. The dosage to be used has been described. However, if the patient is to undergo an emergency procedure such as an operation or an obstetric delivery, it is advisable to supplement and maintain the effects produced by a rapidly dissipated parenteral glycoside with the simultaneous administration of an oral digitalis preparation. One advantage of this procedure is that rapid and complete digitalization is accomplished within 6 to 12 hours. The other is that it dispenses with the necessity of administering a digitalis preparation at repeated intervals during the first 24 hours. By this method, maintenance dosage may be started 24, or if necessary, 48 hours after initial digitalization which is performed as follows: a single dose of one of the rapidly acting glycosides is given parenterally. Simultaneously, a dose of one of the more slowly acting digitalis preparations is given orally, in a dosage depending on the estimated weight of the patient. Experience with this procedure has been most satisfactory, employing ouabain in a dose of 0.5 Gm., intravenously, and the whole leaf tablet of digitalis (New York Heart Association Preparation, U.S.P. XIII) orally, in amounts ranging from 0.4 Gm. to 0.8 Gm., depending on body weight: 0.4 Gm. of digitalis leaf for weights to 125 pounds, 0.6 Gm. for weights of 125 to 175 pounds, and 0.8 Gm. for patients who weigh 175 pounds or more.

If any reason a digitalis preparation cannot be used, aminophylline, 0.24 Gm., should be administered intravenously. It should be given slowly to avoid such reactions as headache, vertigo, palpitation, and possibly precordial pain or substernal oppression. It is best not to give this medication intramuscularly.
because of the intense local irritation which it usually causes. Suppositories of aminophylline are sometimes helpful as a substitute for, or as a supplement to, intravenous administration. The suppositories are supplied in 0.24 and 0.48 Gm. sizes. They are particularly useful in that they can be used by the patient at the onset of a bout of paroxysmal dyspnea and sometimes lessen the severity of the attack.

“Bloodless phlebotomy” may be an effective adjunct to treatment if pulmonary edema is accompanied by venous engorgement. The purpose of this procedure is to utilize the peripheral venous system as a reservoir, decrease the circulating blood volume, and thereby diminish the venous return to the heart. It is accomplished by applying tourniquets, preferably blood pressure cuffs, to all four extremities. Three of the cuffs are inflated at a time to a pressure slightly higher than the level of the patient’s diastolic pressure. Release of each cuff is done in rotation every fifteen minutes to permit re-establishment of adequate blood flow. If this, and the measures outlined above, have not been successful in relieving the acute failure, phlebotomy, with the rapid withdrawal of at least 500 cc. of blood, should be performed.

Since the maintenance of an edema-free state is important, particularly in paroxysmal dyspnea or pulmonary edema, a mercurial diuretic should be administered intramuscularly or subcutaneously shortly after the measures described above have been initiated. A new mercurial diuretic, Thiomerin, which may be administered subcutaneously, intramuscularly, or intravenously, has been employed with excellent results. Based on ease of administration, degree of diuresis, and local tolerance, it is now considered by many to be the diuretic of choice.

**Shock in Myocardial Infarction**

Shock may be a complication of myocardial infarction. Its development is of very serious prognostic significance, as it is one of the main causes of death in the initial period following infarction.

The fall in blood pressure, which is often noted as part of a coronary attack, does not of itself constitute shock, even when systolic pressures of 90 mm. Hg or lower are present. Congestive failure, which not uncommonly occurs as a consequence of myocardial infarction, and which may be present concomitantly, should also not be confused with this condition. Shock is identified by the following characteristic findings: The patient may be restless and apprehensive but he is more often in a semi-comatose or comatose state. There is extreme pallor or cyanosis and the skin is cold and moist. The peripheral pulses are weak, threadlike or undetectable. The systolic pressure is usually 90 mm. Hg, or lower, and the pulse pressure is small; in patients who have previously manifested hypertension, the systolic pressure may be considerably higher than this figure.

Therapy should be based upon an understanding of the pathologic physiology which is responsible for the condition. Unfortunately, fundamental data concerning these mechanisms are inadequate and incomplete. There remains considerable controversy as to whether the primary mechanisms are cardiogenic or due to factors related to the vascular system. Because this process is not completely understood, plans of management of shock following myocardial infarction vary from the practice of “judicious neglect” to the use of a conglomeration of drugs and procedures based upon varied theoretic concepts.

The following are the most accepted measures in treatment: Elevation of the foot of the bed is of some value but is contraindicated if congestive failure is also present. High concentrations of oxygen administered by mask or tent should always be instituted as soon as possible. Artificial heating units should not be employed since burns may be easily produced in the presence of poor peripheral circulation and diminished sensory perception. Also, excessive sweating, due to external heat, may cause an additional loss of needed body fluids. The patient should, however, be covered with blankets sufficient to keep him comfortably warm. Bandaging the lower extremities from ankle to mid thigh is thought by some to be of value. The use of the vasoconstrictor drugs is apparently increasing, although the question of their indication is controversial. These medications include Neosynephrine, ephedrine,
Paredrine, and possibly norepinephrine. Paredrine is given in dosage of 10 to 20 mg. intramuscularly, or 5 to 10 mg. intravenously. The dosage of Neosynephrine is 5 mg. subcutaneously, or a maximum of 0.3 mg. may be given intravenously in dilute solution.

Morphine is indicated only if the patient is extremely restless, anxious, or in considerable pain. Otherwise it is contraindicated because the opiates depress pulmonary as well as tissue respiration and thereby tend to increase the degree of anoxia which is frequently present.

It is known that if shock following myocardial infarction is treated with these measures alone, the mortality is still about 80 per cent. In view of this high mortality, more dynamic forms of therapy, if proved effective and not potentially too dangerous, would seem to be indicated. Until there is adequate understanding of the causative factors in this form of shock, we can only be guided in its treatment by experimental and clinical results following trial with various therapeutic measures.

Investigative studies on dogs appear to indicate that the early transfusion treatment of shock following coronary occlusion will improve failing myocardial contraction of the ischemic area and thereby probably increase cardiac output. This work seems to justify the use of emergency measures directed at rapidly relieving acute shock following myocardial infarction, principally with the use of transfusions of blood or plasma and perhaps also with vasoconstrictor drugs. Since hemoconcentration is frequently present, plasma transfusions may be preferable.

In recent years, some clinicians have advised the use of transfusions in this form of shock. Reported clinical experiences with intravenous transfusion in the treatment of shock following myocardial infarction indicate that, while intravenous transfusions are not as yet of proved value, they have usually resulted in at least transient improvement. Where the treatment has failed it has appeared that larger, more frequent, or more rapidly administered transfusions might have reversed the shock picture or prevented recurrence. It has also been noted rather surprisingly that heart failure has rarely been produced by transfusions employed in this condition. Occasionally, indeed, where some degree of failure has been present at the time the transfusion was started, the signs have cleared following treatment. On the basis of experience thus far, it appears that the use of transfusions following myocardial infarction is only definitely contraindicated when a high venous pressure, or marked pulmonary edema, is present.

Experimental studies and preliminary clinical trials indicate that intra-arterial transfusions may be superior to intravenous transfusions in the treatment of this type of shock. Theoretic advantages of the intra-arterial, as compared with the intravenous route, are that intra-arterial transfusions effect a more prompt increase in blood pressure and probably of circulating blood volume. Consequently, there should be less danger of immediate overloading of the heart than with the use of larger intravenous transfusions. Experimental work on dogs in shock has shown rapid retrograde perfusion of renal, coronary and cerebral arteries following arterial transfusion. In view of these considerations the use of intra-arterial transfusions of blood or plasma in the treatment of "coronary shock" warrants further investigation and evaluation.

SHOCK FOLLOWING PULMONARY EMBOLISM

Another form of shock which may be considered a cardiac emergency is that which frequently follows pulmonary embolism. Pulmonary emboli usually come from thrombi in the deep veins of the lower extremities or from the veins of the pelvis in surgical and obstetric patients. Symptoms are variable but in typical cases the patient suddenly becomes acutely dyspneic and cyanotic, is restless and anxious, complains of severe substernal oppression, and often there is rapid progress to shock and death. Differential diagnosis between pulmonary embolism and acute myocardial infarction may be difficult. History, x-ray, and especially the electrocardiogram are helpful in the diagnosis. Unfortunately, myocardial infarction is known to occur secondary to pulmonary embolism, in which circumstance evidences of both conditions will coexist. Prognosis of pulmonary embolism depends chiefly upon the size of the
embolus, the severity of secondary reflex effects, and the promptness with which treatment directed at the relief of these factors is instituted.

In the treatment of this condition, oxygen should be given as soon as available. Morphine, combined with atropine, is administered if the patient is acutely dyspneic and is suffering from pain or anxiety. Vasodilator drugs are probably of considerable value in the presence of reflex vasospasm, but they should not be given if shock has developed. These medications include papaverine, 0.06 Gm., aminophylline, 0.48 Gm., and atropine, 0.6 mg., which are given by the intravenous route.

Anticoagulants have become one of the most important factors in reducing mortality and morbidity following pulmonary embolism. They are given to prevent propagation of thrombus in the pulmonary vessels and in an effort to control further embolization from the primary site. Heparin, because of its rapid action, should be given as early as possible. It is best administered intravenously in doses of 75 to 100 mg. which are repeated at six hour intervals until dicumarol or one of the more slowly acting anticoagulants, given simultaneously, has become fully effective. Proper laboratory control is, of course, essential. If further embolization occurs despite adequate anticoagulant therapy, bilateral femoral vein ligation and even ligation of the inferior vena cava may be indicated.

It should be kept in mind that pulmonary embolism can usually be prevented by the use of anticoagulants and the other prophylactic measures which should be employed in conditions where thromboembolic phenomena are apt to occur.

Cardiac Tamponade

Although this condition is not common it constitutes a serious emergency, which, if not treated promptly and properly, will invariably lead to death. The diagnosis of tamponade is missed with sufficient frequency to warrant a brief discussion to aid in its recognition.

Acute tamponade of the heart may occur following injury to the heart, to the pericardium, or to the great vessels. It results commonly from stab or gunshot wounds, but it may also complicate infections of the pericardium if there is considerable effusion. This is especially liable to occur in acute suppurative pericarditis. Rare, and invariably fatal, causes of tamponade are rupture of the heart following myocardial infarction, and retrograde progression of a so-called dissecting aneurysm of the aorta.

Patients with wounds and tamponade of the heart may be restless or in profound shock when first seen. The pulse is usually weak, thready, or imperceptible. Classically it is paradoxical in type. The blood pressure is commonly very low or unobtainable. The veins of the neck are distended. Heart sounds are generally distant or inaudible.

In the treatment of this condition aspiration of the pericardial space is usually indicated as an emergency measure to relieve tamponade. Early aspiration is also necessary because this blood clots rather rapidly and can be easily removed only soon after bleeding occurs. Fortunately, in many instances the bleeding does not recur and subsequent operation is not necessary. However, if aspiration is not successful, or if signs of tamponade recur, open operation with suturing of the incised myocardium is indicated.

An aid in therapy which may help to prevent circulatory failure in acute pericardial tamponade is the rapid administration of intravenous infusions of saline, or of other intravenous solutions. The increase in venous pressure produced by this means will frequently overcome the resistance to the return of the blood to the right heart caused by tamponade. This increase may be sufficient to permit improvement in cardiac filling, and therefore of cardiac output. The result of this treatment is largely dependent upon whether the pericardial sac is able to stretch further. Intravenous infusion alone may aid in maintaining the circulation of the patient with tamponade. At least this appears to be a supportive measure which may be a valuable adjunct to aspiration or surgery.

A useful routine has been suggested in the treatment of cardiac tamponade following stab wounds of the heart. The patient is given an infusion of saline solution and fluoroscoped immediately. If there are clinical and x-ray signs of tamponade, aspiration is performed
immediately. The patient then receives a transfusion. If aspiration is unsuccessful or if the signs of pericardial tamponade recur after a successful aspiration, the heart is saturated.

Cardiac tamponade may also result from empyema secondary to acute supplicative pericarditis, or from large effusions such as are found in tuberculous pericarditis. Tamponade due to these causes produces circulatory failure of the same type as that observed in patients with stab wounds of the heart. It is treated by aspiration or by surgery with open drainage if aspiration is inadequate. Penicillin or other antibiotics may be of value when instilled directly into the pericardial sac in supplicative pericarditis following initial aspiration or surgical drainage. This is usually supplemented by systemic antibiotic therapy.

REFERENCES

24 Personal observations.
The Management of Acute Cardiac Emergencies
CLARENCE E. DE LA CHAPELLE and O. ALAN ROSE

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An erratum has been published regarding this article. Please see the attached page for:
/content/5/2/x.full.pdf
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Erratum: In the paper entitled "The Management of Acute Cardiac Emergencies," by Clarence E. de la Chapelle and O. Alan Rose (Circulation 4: 764, 1951), an error in the dosage of intravenous ouabain appeared on page 770. The dosage stated is 0.5 Gm.; the correct dosage is 0.5 mg.