Treatment of Acute Coronary Occlusion

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Introduction

During the past thirty years, the diagnosis of acute coronary occlusion has been greatly facilitated and its treatment has been greatly improved. The diagnosis has been improved by the routine use of 12 electrocardiographic leads and by the recognition of atypical symptoms. Numerous mild cases have, thereby, been discovered and a broader understanding of the disease has been reached. It should be emphasized that at the onset of the attack, possibly because the artery has not yet been completely occluded, the electrocardiogram may be normal or may show only slight changes. At this time, RS-T depression may occur, as in coronary insufficiency, instead of the expected RS-T elevation. The RS-T elevation may not appear for hours, and the Q-waves only later, at the end of one to two days. The treatment of the disease has been improved by several major therapeutic advances. These include oxygen therapy, a low calorie diet, the judicious use of digitalis and quinidine, the avoidance of such drugs as Adrenalin, nitroglycerin, camphor and strychnine, and the employment of anticoagulants. Recently, Levine has advocated the chair treatment of coronary occlusion.

In 1935, the mortality rate in our private patients, during the first attack, was 10 per cent. Today, it is less than 5 per cent, among such patients, and is only 15 to 20 per cent during all attacks, among ward patients.

Treatment

Pain: The intensity of the pain, which ushers in the attack, is not an accurate gauge of the severity of the attack. Not infrequently, a coronary occlusion which begins with very severe pain runs a remarkably smooth course after the pain has eased. It is essential to relieve the pain as soon as possible. The most effective drug for this purpose is morphine; if the pain is intense, morphine should be given intravenously in a dose of eight or 10 mg. In most cases, with less severe pain, the subcutaneous administration of morphine, as well as of Demerol, Dilaudid or Pantopon is effective. In our experience, however, Demerol is much less efficacious than morphine. In very rare cases intravenous sodium amytal may give at least temporary relief. Oxygen and aminophylline (by vein or suppository) may be used but only rarely are efficacious in relieving the pain.

Nitroglycerin does not relieve the pain of acute coronary occlusion but

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Read before the Atlanta Graduate Medical Assembly, February 23rd, 1955, Atlanta, Georgia.
This article appeared in the November 15, 1955 issue of the New York State Journal of Medicine.
is very effective in status anginosus, which represents a state of acute coronary insufficiency. It is distinguished from coronary occlusion by the finding of RS-T depression and/or T-wave inversion in the electrocardiogram. Reassurance, the free use of nitroglycerin and the administration of a narcotic, such as Dilaudid (1.5 to 2.5 mg.) three or four times a day, during a period of status anginosus often cause the pain to subside gradually and the patient may be asymptomatic for a long period. In other instances, however, status anginosus represents a premonitory phase of coronary occlusion. When the occlusion is complete, nitroglycerin no longer is effective and, indeed, is dangerous. Thus, nitroglycerin may be used not only therapeutically but also diagnostically.*

*An increase in serum transaminase activity may prove of value in determining the presence of significant myocardial infarction at the onset of an attack.

Hospital Vs. Home Care: As soon as the pain has been relieved, the advisability of hospitalization should be considered. If the conditions at home are satisfactory, the vast majority of patients can be treated at home, particularly since most attacks run a mild course following the initial pain. However, if the patient’s condition is serious, if the home environment is unsuitable and if nurses are not available, the patient may be removed to the hospital immediately by ambulance.

At the onset of the attack, the patient is usually apprehensive. He may even be terrified; for, like many others, he may believe that the development of a coronary occlusion spells the end of things physically, sexually, socially and financially. Therefore, it is important to allay his fears at the first feasible opportunity, by assuring him that the vast majority of patients make a good recovery and can resume a full, productive life.

Anticoagulants: Less than 10 years ago anticoagulants were introduced in the treatment of coronary occlusion. Their use soon became routine unless a specific contraindication was present. Early reports indicated that a significant diminution in embolic complications and a lower mortality rate resulted therefrom. Great credit is due these early workers, Wright and others, for anticoagulants have been of great value in patients seriously ill with coronary thrombosis. During the past few years, however, several authors have questioned the value of anticoagulants in coronary occlusion or have suggested that they were unnecessary in mild attacks. Russek and his co-workers found the mortality rate in good risk patients to be only 3.1 per cent, and the incidence of embolic phenomena to be only 0.8 per cent. They reasoned, therefore, that the use of anticoagulants can, at best, decrease the mortality rate only 1 per cent, and that such a possible improvement is scarcely worth seeking, in view of the danger of hemorrhage. On the other hand, it has been suggested that all patients should receive the benefit of anticoagulant drugs, since the apparently mild case may develop embolic phenomena, or may suddenly develop congestive failure or shock. In view of the infrequency of these complications in mild cases, however, this does not appear to be a wise course to follow. We have found that in the vast majority of attacks which are considered mild during the first day or two, this judgment proves to be correct. If there is any question in the physician’s mind at first, anticoagu-
lants may be administered for a day or two and then discontinued if the patient's course proves satisfactory. Naturally, should an indication for anticoagulant therapy arise at any time, it should be instituted immediately.

As the period of strict bed-rest in the treatment of coronary occlusion has been gradually shortened, not only in the mild but also in the more severe cases, the indication for anticoagulant therapy has further diminished since the major effect of anticoagulants is to reduce the incidence of phlebothrombosis, and early ambulation achieves the same end. In order to avoid phlebothrombosis, the patient should be urged to move his toes and legs and to take deep breaths early in the attack. Massage of the legs and the use of elastic stockings are beneficial.14

Anticoagulant drugs are indicated (1) when congestive failure or shock is present, (2) if there is evidence of or a history of phlebitis, (3) after a pulmonary embolus or infarction, and (4) following peripheral arterial embolism, with the exception of cerebral embolism. The age of the patient is not a factor in anticoagulant therapy; old age is not a contraindication to the use of anticoagulants. This is also true of pulmonary edema even if the sputum contains considerable blood.

Whether to use anticoagulants during the premonitory stage of acute coronary occlusion remains a moot question.1 15 16 Although some success has been claimed for them in preventing the completion of the occlusion,16 the data reported thus far appear insufficient and inadequately controlled. Our own results seem to indicate that anticoagulants neither prevent nor hasten the onset of coronary occlusion. At present, apparently no known treatment will prevent an impending attack.17 The premonitory stage of coronary occlusion cannot be differentiated from acute coronary insufficiency, and since the majority of attacks of coronary insufficiency subside spontaneously, the efficacy of anticoagulants in the premonitory stage of occlusion is, necessarily, difficult to evaluate; for there is no way of knowing whether the drugs prevented the completion of an impending occlusion or whether no occlusion was impending.

If anticoagulants are employed in acute coronary occlusion, it is wise to give 50-75 mg. heparin intravenously, every four to six hours, for the first day or two, or 200 mg. Depoheparin every 12 hours intramuscularly. Oral therapy with Dicumarol, Tromexan10 or Hedulin is instituted simultaneously, since their effect is not manifest for one to three days. The initial dose of Dicumarol is 300 mg., followed by 100 to 200 mg. the second day and smaller daily doses thereafter, depending upon the prothrombin time. The effect of Tromexan is apparent in 18 to 24 hours but the individual reaction and daily dose are more variable than with Dicumarol. The dose is five or six times that of Dicumarol. Hedulin also acts more rapidly than Dicumarol. The initial dose is 200 to 300 mg. and the maintenance dose 50 to 100 mg. Final evaluation of this drug is not yet possible.

It is scarcely necessary to emphasize the extreme importance of careful supervision during anticoagulant therapy for hemorrhage, and even death may occur. While the response of most patients to anticoagulants is as
expected, that of others is entirely unpredictable. An initial 300 mg. dose of Dicumarol has, in some cases, prolonged the prothrombin time to dangerous levels, either within a day or two, or only after three or four days. Occasionally, bleeding occurs when the prothrombin time is well within therapeutic levels, i.e., 40 to 50 per cent of the control. Evidence of bleeding should be looked for in the urine, in the skin and elsewhere. Hemorrhage may occur into serous cavities, including the pericardium. If bleeding occurs, vitamin K\(_2\) oxide (50 to 100 mg.) intravenously will return the prothrombin time to non-dangerous levels within several hours. In the event of serious bleeding, the transfusion of fresh blood or plasma alone is effective.

When anticoagulants are administered, they should be continued for approximately one week after the patient has been permitted out of bed, and longer if there has been evidence of peripheral phlebitis or pulmonary infarction. Long-term anticoagulant therapy has been employed for months or years following an attack, but the value of this procedure has not been demonstrated as yet.

Anticoagulants are contraindicated in non-specific pericarditis since they are apt to cause bleeding into the pericardium in this condition. For this reason, it is essential to differentiate non-specific pericarditis from coronary occlusion. This can usually be done by means of the electrocardiogram; while ST elevations occur in both diseases, Q-waves are absent in pericarditis and usually are present in coronary occlusion. It has even been suggested, from experience in one or two cases, that anticoagulants should not be employed in coronary occlusion when a definite pericardial friction rub is present. We doubt the wisdom of this course. Anticoagulants are contraindicated in patients with a history of bleeding tendency, ulcerative colitis, peptic ulcer, renal or hepatic disease, and, probably, in those who have had a cerebrovascular accident.

**Length of Bed Rest:** When acute coronary occlusion first became widely recognized, and its diagnosis was commonly established, complete and prolonged bed-rest was the cardinal principle in therapy, although only scant data substantiated the need for such treatment. In the nineteen twenties and thirties it was generally believed that it took at least six weeks for a scar to form in the infarcted area and that, therefore, the patient should remain in bed for that period. However, in the early forties, many physicians, including ourselves, questioned the need for so long a period of bed-rest and shortened it for various empirical reasons: (1) "Silent" coronary occlusions were discovered in many patients whose electrocardiograms were typical of a previous coronary occlusion, yet who gave no history of previous attack. And, although they had had no bed-rest, their cardiac function was good and no evidence of aneurysm of the left ventricle was found. (2) Patients with known acute coronary occlusion who refused to stay in bed or insisted, openly or surreptitiously, on going to the bathroom from the first day, also showed no harmful effects of their activity. (3) Many patients whose course during the coronary occlusion was mild developed great anxiety and other psychological tensions because
of the prolonged rest in bed. This merely aggravated an otherwise mild illness and, occasionally, produced a prolonged psychoneurosis. For these reasons patients with mild attacks were often permitted to use a commode after the first two to three days and to sit in a chair after two to three weeks.

Great credit is due Levine,6, 21-23 Dock24 and Harrison25, 26 for pointing out that prolonged bed-rest in acute coronary occlusion and in heart failure is often unnecessary, and, indeed, may be harmful. It has been shown that recumbency tends to increase cardiac work by increasing the circulating blood volume.27, 28 In addition to the anxiety and depression it may produce, prolonged bed-rest leads to generalized loss of muscular and vascular tone, causes constipation and distention and predisposes to venous thrombosis. Conversely, chair treatment reduces these ill effects and lessens the need for prolonged anticoagulant therapy.

While many physicians now permit patients whose disease runs a mild course to sit in a chair after a shorter interval than before, Levine advocates the armchair treatment for all cases of acute coronary thrombosis, from the onset of the attack,6 and particularly for patients with definite congestive failure. The patient is lifted or helped back into bed. Levine has reported patients with severe left ventricular failure who did not respond to treatment as long as they were kept in bed but who improved when they were placed in a chair. Recent confirmatory favorable reports on the chair method of treatment have appeared.29, 30

It seems to us that the most important principle in the therapy of coronary thrombosis is to treat each patient individually. The physical and psychological state of each patient should determine how he can best be treated. The patient suffering from a mild attack may be treated liberally, with every expectation of a good recovery. If such a patient is unable to use a bedpan it may be desirable to permit him to use a bedside commode and to sit in a chair from the very start. Or, he may be kept in bed for one week and then be permitted to sit up in a chair. During the period of bed-rest the patient should be given a small enema every second or third day. Such a short stay in bed does not engender any of the harmful sequelae of prolonged bed-rest. In mild attacks our own inclination is to keep the patient in bed approximately a week unless we think that earlier chair treatment is necessary to raise morale.

Patients who are more seriously ill (congestive failure, tachycardia, a systolic blood pressure below 80 or actually a state of shock) should be kept in bed until the usual therapeutic measures have been tried. Efforts should be made to approximate the sitting position by using a hospital-type of bed or by placing nine inch blocks under the headposts. If a patient fails to improve in spite of these procedures, he may be placed in a chair. There should not be too long a delay before chair treatment is instituted in these cases. Much controlled experience is necessary to determine the efficacy of the "armchair" treatment. But indiscriminate prolonged bed-rest for all patients appears inadvisable.31

Ambulation and Return to Work: When the patient is permitted to walk about, depends to some extent upon when he sat up. In very mild cases,
we allow walking at the end of the second week or at the beginning of the third; usually the patient begins to walk the fourth week. Such patients may be ready to return to work within two or three months. In the more severe cases, rehabilitation is slower but many of these patients also can resume work in three to six months. Treatment must be individualized. We have found that four of every five patients are able to resume work following coronary occlusion and can lead productive lives for many years.

We do not depend upon the electrocardiogram as a criterion for determining the progress of the patient or for deciding when he may sit up, begin to walk or return to work. If his clinical course is satisfactory, the patient may get up and walk even though the electrocardiogram shows marked alterations. Nor do we always wait for the electrocardiogram to become stable. If the electrocardiogram returns to normal, the outlook is usually excellent but the patient may also do very well, during and after the attack, even if the electrocardiogram is markedly abnormal or shows the changes of ventricular aneurysm, i.e., large Q-waves, persistent ST elevation and deeply inverted T-waves. Nor do we place too much reliance upon the sedimentation time as a guiding factor in treatment. We have observed patients who were running a severe course of acute coronary occlusion, with little elevation of the sedimentation rate, and, on the other hand, patients with very high sedimentation rates whose course was mild. A woman of 63, for example, became asymptomatic and afebrile several days after the onset of the attack. Her first sedimentation rate was elevated to 95. After four weeks it was still 88 although she was ambulatory and felt well. We do not prolong the period of bed-rest because of an elevated sedimentation rate if the patient is doing well clinically. In some patients the sedimentation rate remains increased to 40 to 50 mm. for many months or even years following an occlusion, long after they have resumed working. The determination of the C reactive protein may prove more useful than the sedimentation rate. A normal ballistocardiogram in coronary occlusion usually indicates a good recovery, but the patient may be doing very well even when the ballistocardiogram is definitely abnormal. Of course, in people over 50 the ballistocardiogram may be abnormal in the absence of heart disease.

Heart Failure: Not uncommonly coronary occlusion sets in dramatically as an attack of pulmonary edema. The treatment of this condition is the same as if coronary occlusion were not present. Morphine intravenously or intramuscularly is usually efficacious but intravenous aminophyllin and strophanthin may be necessary. Aminophyllin must be administered very slowly, the dose being 0.5 mg. The initial dose of strophanthin K is 0.25 mg. Injection of 0.1 mg. may be repeated every hour until 1 mg. has been given in 24 hours. Oxygen under positive pressure, rotating tourniquets or phlebotomy if shock is not present, and an intravenous mercurial diuretic may be helpful. Occasionally, the inhalation of alcohol vapor has been found to be effective. In patients with pulmonary edema anticoagulant therapy should be instituted even if the sputum is bloody.
In the persistent type of congestive failure, in coronary occlusion, the usual treatment is given. However, particular care is required to avoid digitalis intoxication which is more dangerous in the presence of acute infarction. It is possible that the infarcted myocardium is more irritable and more sensitive to digitalis, for many of the attacks of ventricular tachycardia encountered in coronary occlusion have followed the administration of digitalis. Aged patients are more sensitive to digitalis than the young, often requiring half the average dose or less. The earliest symptoms of digitalis overdosage are usually weakness, anorexia and abdominal pain. These appear before nausea, vomiting and diarrhea occur. Premature beats and any other type of arrhythmia may develop. Potassium is the most effective antidote for this condition. A convenient method of administering it is a liquid oral preparation, such as “Potassium Triplex,” but it may have to be given intravenously.

In addition to the use of digitalis, the salt intake should be restricted and mercurial diuretics given. If the degree of failure is not severe, an oral diuretic such as Neohydrin or Diamox is often beneficial. Two or three tablets of Neohydrin are given daily. The dose of Diamox is one tablet (250 mg.) daily four or five times a week.

Shock: Mild transitory states of shock are very common at the onset of coronary occlusion. Sometimes, the shock is profound and persistent. In the past this has augured a fatal outcome in 80 or 90 per cent of the cases. However, several vasopressor drugs are now available and are occasionally effective if used early.37-40 Among them are (1) Norepinephrine (Levophed), (2) Mephentermine (Wyamine), (3) Methoxamine (Vasoxyl), and (4) Neosynephrine. Recently Aramine has been studied in normal subjects.41

Norepinephrine must be given intravenously. The initial dose is four mg. per liter of five per cent glucose in water. This may be increased to eight, 16 or even 32 mg. if necessary, until the systolic blood pressure rises to 100 to 110. The advantages of Norepinephrine are the ability to regulate the height of the blood pressure by varying the dose and the speed of injection, the rapid disappearance of its effect and the absence of undesirable side actions, such as myocardial stimulation. During its use the blood pressure should be recorded frequently, not only to determine whether the dose is adequate but also to avoid an excessive rise in blood pressure. If congestive failure is present, the amount of intravenous fluid administered with the Levophed should not be excessive. The dose of Mephentermine (Wyamine) is 15 to 30 mg., given intravenously or intramuscularly, and repeated as often as indicated. The dose of Methoxamine (Vasoxyl) is 20 mg., intravenously or intramuscularly. The dose of Neosynephrine is one mg. intravenously or five to 10 mg. intramuscularly. The sooner the vasopressor drugs are employed, the better is the outlook. Unfortunately, however, even if the blood pressure is raised, only some of the patients recover.

The use of ordinary plasma and blood infusion or of intra-arterial transfusion has not proved effective in shock following coronary occlu-
sion. When there is an element of congestive failure associated with shock, intravenous strophanthin or other digitalis preparations may be effective. In the absence of failure, digitalis may be harmful in shock and should be avoided.

**Arrhythmias:** All types of arrhythmias are common in coronary occlusion. We do not use quinidine routinely to prevent them. Premature beats, atrial and nodal tachycardia and atrial fibrillation and flutter are often transitory. If they do not remit quickly, or if any degree of shock or heart failure is present, treatment is indicated. For frequent premature beats, quinidine or Pronestyl is given. Atrial or nodal tachycardia often responds to carotid sinus or eyeball pressure; if it does not, Neosynephrine, 0.5 mg. may be administered intravenously, particularly if the blood pressure is low. If Neosynephrine is effective, digitalis is given parenterally or orally. If necessary quinidine or Pronestyl may be given orally or intramuscularly.

Ventricular tachycardia should be treated promptly with either quinidine or Pronestyl, administered orally or intramuscularly. If these drugs are ineffective by these routes, Pronestyl may be given intravenously, if well diluted and injected slowly. Since intravenous Pronestyl is apt to cause a considerable drop in blood pressure, Levophed may be administered simultaneously to maintain the blood pressure or Neosynephrine given as soon as indicated.

A-V block occurs not infrequently when the right coronary artery is occluded and the diaphragmatic surface of the left ventricle is infarcted. Partial A-V block usually requires no treatment but atropine may abolish it. Complete A-V block was at one time considered a very ominous finding but our recent experience has been much more favorable. The danger of complete A-V block lies in the development of Stokes-Adams seizures. They may result from asystole or ventricular fibrillation or both. When the seizures are caused by asystole, adrenalin is administered subcutaneously or, if necessary, intravenously in very small doses. Sublingual Isuprel, 10 mg. may also relieve the attack. Recently, Zoll has devised a machine, called the "Pacemaker," for external electric stimulation of the heart in cases of asystole. He has reported good results from its use. If the Stokes-Adams attacks are caused by ventricular fibrillation, Isuprel should be employed; quinidine and Pronestyl are contraindicated since they may completely depress the ventricle. If asystole and ventricular fibrillation alternate in the same patient, Isuprel and the "Pacemaker" should be used. In recurrent Stokes-Adams attacks, secondary to complete heart block, adrenalin or Isuprel may be repeated as often as necessary and the "Pacemaker" may be applied for long periods.

**Diet:** The low calorie diet has greatly improved the outlook during the attack of coronary occlusion. It decreases the work of the heart and prevents gastro-cardiac reflexes. During the first few days or weeks, 800 to 1200 calories daily suffice. Thereafter, the intake may be gradually increased.
Nausea and Vomiting: These symptoms are often very disturbing and may be serious. They may be prevented by abstinence from fruit juices, cold milk and spicy foods. Dramamine (50 mg.), Thorazine (10 to 25 mg.) or Marezine (25 to 50 mg.) given orally, intramuscularly or by suppository, is the most effective treatment. Sips of charged water and ginger ale may also be helpful. Fluids by mouth should be restricted and solids given. Since nausea and vomiting may be caused by digitalis, morphine, amphetamine, ammonium chloride or quinidine, these drugs should be temporarily discontinued. If the nausea and vomiting persist, however, administration of the drugs may be resumed, since congestive failure alone may cause nausea and vomiting, which may disappear only after further digitalization.

Particularly, if the patient has received much morphine, severe constipation and distention may set in. A cathartic should be given at night and an enema in the morning, if necessary. The patient must be warned not to strain at stool. In the presence of distention, fruit juices and cold milk should be avoided. Intramuscular prostigmine may be required.

Hiccough: Hiccough is usually frightening to the patient and it is extremely important to reassure him over and over again. If this is done, the hiccough almost always subsides. If it persists, various remedial procedures may be employed, all of which have proved successful at some time: rebreathing into a paper bag, Carbogen (five or seven per cent CO₂ and O₂ inhalations), chlorpromazine (Thorazine), quinidine intramuscularly, niacin or atropine intravenously, inhalation of amyl nitrite, ethyl chloride spray along the diaphragm, ether anesthesia or ether intramuscularly, gastric lavage and stellate ganglion block. In rare cases, unilateral phrenicectomy or phrenic nerve crush is required.

Penicillin: We administer this drug if pulmonary congestion or other signs of congestive failure are present or if the temperature is elevated, i.e., above 102° F.

Alcohol and Tobacco: Whiskey should not be given in acute coronary occlusion since it may increase the pulse rate. Smoking should be prohibited.

Cortisone and ACTH: Although these hormones have sometimes been found helpful experimentally, they are of no value clinically and may even be dangerous.

SUMMARY

The diagnosis of acute coronary occlusion has been facilitated and its treatment improved.

At the onset of the attack the electrocardiogram may be normal or show only slight changes.

The major therapeutic advances include oxygen administration, a low calorie diet, the judicious use of digitalis and quinidine, the avoidance of such drugs as Adrenalin and nitroglycerin, and the employment of anticoagulant and pressor drugs. Therapy must be individualized, particularly in reference to the time chair treatment is begun, the time of ambulation, and the time for returning to work.

The majority of patients can be treated at home.
The pain which ushers in the attack should be relieved immediately. For this purpose, morphine is most efficacious. Nitroglycerin is not effective, and may be dangerous.

The patient's anxiety must be allayed; he should be reassured that he, like the vast majority of patients, will make a good recovery and resume a full, productive life.

Indiscriminate prolonged bed-rest for all patients is inadvisable. The period of bed-rest is determined by the physical and psychic state of each patient. Some mild cases are permitted to be in a chair on the second or third day; others, after a week. Early chair treatment has definite advantages.

In the average mild case, the patient begins to walk during the fourth week.

The routine use of anticoagulants is unnecessary. They should be employed in patients with heart failure or shock, and in those who develop peripheral phlebitis, peripheral arterial embolism, or pulmonary embolism. When anticoagulants are administered, the patient should be under close supervision.

We believe that anticoagulants, administered during the premonitory phase of coronary thrombosis, are inefficacious; they neither prevent nor hasten the progress of the thrombosis.

Anticoagulants are contraindicated in patients with a history of bleeding tendency, ulcerative colitis, peptic ulcer, renal or hepatic disease, or a cerebrovascular accident.

It is important to differentiate coronary occlusion from non-specific pericarditis, since anticoagulants appear to be harmful in this condition.

The electrocardiogram should not be used as a criterion for determining the progress of the patient and the time when he may sit up, begin to walk, or return to work. Neither should undue reliance be placed upon the sedimentation rate as a guiding factor in treatment.

The treatment of heart failure in coronary occlusion is the same as if coronary occlusion were not present, but special care is necessary to prevent digitalis intoxication.

A low calorie diet is important, for it diminishes the work of the heart, prevents gastrocardiac reflexes, and reduces weight in the obese.

Constipation and distention must be prevented. Cold milk and fruit juices should be avoided and laxatives used.

Nausea and vomiting are benefited by the oral or intramuscular administration of anti-motion sickness drugs.

Early treatment of shock is essential. The vasopressor drugs are helpful, if administered early. If congestive failure is a factor, strophanthin or digitalis should be given.

Pulmonary edema requires immediate treatment with morphine. If necessary, aminophylline, strophanthin, mercurials, oxygen under pressure, and rotating tourniquets or phlebotomy are employed. The inhalation of alcohol vapor is occasionally efficacious.
Arrhythmias occur frequently and often remit. The indications for the treatment of each type are discussed and the details outlined. Quinidine is not used routinely.

Hiccough may be a serious problem. Reassurance of the patient is most important. Adequate sedation should be used. Numerous successful therapeuetic measures are available.

Cortisone and ACTH are not efficacious in coronary occlusion.

Antibiotics are administered if pulmonary congestion or signs of congestive heart failure are present, or if the temperature is above 102° F.

Whiskey should not be given in acute coronary occlusion, since it may increase the pulse rate. Smoking should be prohibited.

The prognosis in coronary occlusion has greatly improved during the past 30 years. In private practice, the mortality rate during the first attack is now five per cent or less. Most patients can be rehabilitated within two or three months. The vast majority makes a fair or good recovery, more than half make an excellent functional recovery. Four out of five return to work.

RESUMEN

El diagnóstico de la oclusión coronaria aguda, se ha facilitado y su tratamiento ha mejorado.

Al principio del ataque el electrocardiograma puede ser normal o sólo mostrar ligeros cambios.

Los adelantos terapéuticos más importantes incluyen administración de oxígeno, alimentación baja en calorías, el uso juicioso de la digital y de la quinidina, el evitar drogas tales como la adrenalina y la nitroglicerina y el empleo de drogas anticoagulantes y las que actúan sobre la presión. El tratamiento debe ser individualizado en particular en lo relativo al tiempo en que el enfermo puede ser colocado en silla; el tiempo de empezar la demabulación y el del regreso al trabajo.

La mayoría de los enfermos pueden tratarse a domicilio.

El dolor con que se inicia el ataque debe ser aliviado inmediatamente, para lo que la morfina es lo más eficaz. La nitroglicerina no es eficaz y puede ser peligrosa. La ansiedad del enfermo debe ser calmada; deben dárselle seguridades de que él como la gran mayoría de los enfermos, tendrá una recuperación rápida y podrá volver a una vida completamente productiva.

No es aconsejable el prolongado reposo en cama sin distinciones.

El periodo de reposo en cama se determina por el estado psíquico y físico del enfermo en cada caso. Algunos casos moderados pueden permitirse el pasar a la silla en el segundo o tercer día; otros después de una semana. El pasarlo al reposo en sillón pronto puede tener ventajas definidas.

En el caso moderado medio, el enfermo empieza a caminar durante la cuarta semana.

El uso rutinario de los anticoagulantes no es necesario. Deben emplearse en enfermos con desfallecimiento cardiaco o shock y en los que presentan flebitis periférica, embolia arterial periférica, o embolia pulmonar. Cuando
se administran los anticoagulantes el enfermo debe estar bajo vigilancia estrecha.

Creemos que los anticoagulantes administrados durante la fase pre-monitoria de la trombosis coronaria son ineeficaces; ni previenen ni apresuran la evolución de la trombosis.

Los anticoagulantes están contraindicados en los enfermos con antecedentes de tendencia hemorrágica, colitis ulcerosa, úlcera péptica, enfermedad renal o hepática, o accidente cerebrovascular.

Es importante diferenciar la oclusión coronaria de la pericarditis no específica ya que los anticoagulantes parecen ser dañosos en esta afección.

El electrocardiograma no debe usarse como criterio para determinar la evolución del enfermo y el tiempo cuando debe sentarse, empezar a caminar o regresar al trabajo. Tampoco debe tenerse demasiada confianza en la sedimentación globular como guía del tratamiento.

El tratamiento del desfallecimiento cardíaco en la oclusión coronaria es el mismo que si la oclusión no se tuviera presente pero debe tenerse especial cuidado en evitar la intoxicación digitálica.

Es importante la dieta baja en calorías porque disminuye el trabajo del corazón, evita los reflejos gastrocardíacos y reduce el peso de los obesos. Deben prevenirse la distensión y el estreñimiento.

La leche fría y los jugos de frutas deben evitarse y deben darse laxantes. La náusea y el vómito mejoran por el uso oral o intramuscular de medicamentos contra el mareo.

El tratamiento inmediato del shock es esencial. Las drogas vasopresoras son útiles si se administran pronto. Si hay el factor de insuficiencia congestiva deben darse estrofantine o digital.

El edema pulmonar debe tratarse con mofina. Si es necesario deben usarse la aminofilina, estrofantine, mercuriales, oxígeno a presión, torotiquetes o flebotomía han de emplearse. La inhalación de vapores de alcohol es eficaz a veces.

Las arritmias se presentan a menudo y frecuentemente remiten. Las indicaciones para el tratamiento de cada forma se discuten y los detalles se proporcionan. La quinidina no se usa de manera rutinaria. El hipó puede ser un problema serio. El dar confianza al enfermo es lo más importante. Se usará sedación adecuada. Hay numerosas medidas terapéuticas.

La cortisona y la ACTH no son eficaces en la oclusión coronaria.

Se administrarán antibióticos si la congestión pulmonar o signos de insuficiencia congestiva se presentan o si la temperatura asciende arriba de 102 F.

No debe darse whisky en la oclusión aguda puesto que aumenta la frecuencia del pulso. Debe prohibirse fumar.

El pronóstico en la oclusión coronaria ha mejorado grandemente durante los últimos treinta años. En la práctica privada la mortalidad durante el primer ataque es de 5 por ciento a menos.

La mayoría de los enfermos pueden rehabilitarse dentro de dos o tres meses. La gran mayoría logran una recuperación bastante o buena; más
de la mitad la hacen excelente recuperación funcional. Cuatro de cada cinco, regresan al trabajo.

RESUME

Le diagnostic d’occlusion coronarienne aiguë se fait maintenant plus facilement et son traitement a été amélioré.

Au début de l’attaque, l’électrocardiogramme peut être normal, ou montrer seulement des altérations légères.

Les éléments thérapeutiques essentiels comprennent l’administration d’oxygène, un régime pauvre en calories, l’emploi judicieux de digitaline et de quininide, le rejet de produits tels que l’adrénaline et la nitroglycérine, et l’utilisation de produits anticoagulants et de toniques vasculaires. Le traitement doit varier en fonction du malade, surtout en ce qui concerne le moment du lever, de la promenade, et de la reprise du travail.

La majorité des malades peut être traitée à domicile.

La douleur qui apparaît lors de l’attaque devrait être soulagée immédiatement. Dans ce but, la morphine est le médicament le plus efficace. La nitroglycérine n’est guère active et peut être dangereuse.

L’anxiété du malade doit être calmée ; on doit lui donner l’assurance que, comme la grande majorité des malades, il guérira lui-même et reprendra une vie pleine et productive.

Le repos prolongé au lit sans discrimination pour tous les malades n’est pas souhaitable. La période de repos intégral au lit est déterminée par l’état physique et psychique de chaque malade. On permet aux cas bénins de s’asseoir sur une chaise le second ou le troisième jour ; dans les autres après une semaine. Le traitement consistant à faire lever précocement le malade a des avantages précis.

Dans le cas bénin moyen, le malade commence à marcher au cours de la quatrième semaine.

L’utilisation systématique de produits anticoagulants n’est pas indispensable. Ils devraient n’être employés que chez les malades atteints d’arrêt cardiaque ou de shock, et chez ceux chez lesquels se développe une phlébite périphérique, une embolie artérielle périphérique, ou une embolie pulmonaire. Quand les anticoagulants sont administrés, le malade doit être soumis à une étroite surveillance.

Nous estimons que les anticoagulants, administrés durant la phase pré-montoire de la thrombose coronarienne sont inefficaces ; ils n’arrêtent ni ne hâtent le progrès de la thrombose.

Les anticoagulants sont contre-indiqués chez les malades qui ont tendance à l’hémorragie, à la colite ulcéreuse, à l’ulcère peptique, aux affections rénales ou hépatiques, ou à un accident cérébrovasculaire.

Il est important de différencier l’occlusion coronarienne de la péricardite non spécifique, depuis que l’on sait que les anticoagulants peuvent être nuisibles dans ce dernier cas.

L’électrocardiogramme ne devrait pas être utilisé comme critère pour déterminer le progrès du malade et le temps au bout duquel on peut l’autoriser à se lever, puis à marcher, puis à reprendre le travail. Pas plus
qu'on ne doit accorder une confiance excessive au taux de sédimentation comme facteur déterminant dans le traitement.

Le traitement de l'arrêt cardiaque dans l'occlusion coronarienne est le même que s'il n'existait pas d'occlusion coronarienne, mais on doit faire très attention à empêcher l'intoxication par la digitaline.

Le régime pauvre en calories est important, car il diminue le travail du cœur, empêche les réflexes gastro-cardiaques, et réduit le poids chez les obèses.

La constipation et la distension colique doivent être évitées. Il faut proscrire le lait froid et les jus de fruits et on peut utiliser les laxatifs.

La nausée et les vomissements sont améliorés par l'administration des produits par voie buccale ou intramusculaire.

Le traitement précoce du shock est capital. Les toniques vasculaires sont utiles, s'ils sont administrés précocément. S'il existe un élément de congestion par insuffisance cardiaque, la strophanthine ou la digitaline doivent être prescrites.

L'oedème pulmonaire demande un traitement immédiat par la morphine. Si nécessaire, l'aminophylline, la strophanthine, les produits mercuriels, l'oxygène sous pression et la saignée par phlébotomie doivent être utilisés. L'inhalation de vapeur d'alcool est éventuellement efficace.

L'arythmie survient fréquemment et souvent finit par disparaître. Les indications du traitement des malades de ce type sont discutées et les détails suggérés. La quinidine n'est pas utilisée d'une façon systématique.

Le hoquet peut être un problème grave. Le fait de rassurer le malade est le plus important. On devrait utiliser des médications qui apportent la sédation immédiate. On dispose de nombreux moyens de traitement satisfaisants.

La cortisone et l'A.C.T.H. ne sont pas efficaces dans l'occlusion coronarienne.

Les antibiotiques sont administrés s'il existe une congestion pulmonaire, ou des signes d'insuffisance cardiaque congestive, ou si la température est au-dessus de 39°.

On ne devrait pas autoriser la boisson de whisky dans l'occlusion coronarienne aiguë, car il peut en résulter une augmentation du pouls. L'usage des cigarettes doit être interdit.

Le pronostic de l'occlusion coronarienne a été grandement amélioré ces trente dernières années. En pratique privée, le taux de mortalité durant la première attaque est maintenant de 5% ou moins. La plupart des malades peuvent être rendus à la vie active en deux à trois mois. La grande majorité d'entre eux peut avoir une guérison moyenne ou bonne, plus de la moitié peut retrouver un état fonctionnel excellent. Les 4/5 peuvent reprendre leur travail.

ZUSAMMENFASSUNG

Die Diagnose des akuten Koronar-Verschlusses ist erleichtert und seine Behandlung verbessert worden.

Zu Beginn de anfalls kann das Elektrocardiogramm normal sein oder nur leichte Veränderungen zeigen.


Dem Angstgefühl des Patienten muss Erleichterung verschafft werden. Man soll ihm fest versichern, dass er sich, wie der grösste Teil solcher Kranken, gut erholen und seine volle produktive Tätigkeit wird wieder aufnehmen können.


In einem durchschnittlich leichten Fall beginnt der Kranke während der 4. Woche mit Spaziergängen.


Wir sind der Ansicht, dass Antikoagulantien wirksungslos sind, wenn sie während der Prodromal-Phase der Koronar-Thrombose gegeben werden; weder verhindern sie, noch beschleunigen sie das Fortschreiten der Thrombose.

Antikoagulantien sind kontraindiziert bei Patienten mit anamnestischen Angaben über Blutungsbereitschaft, Colitis ulcerosa, Magengeschwür, Nierenoder Lebererkrankung oder einer Schädigung der Hirngefäße.

Von Wichtigkeit ist es, einen Koronar-Verschluss abzugrenzen von einer unspezifischen Pericarditis, die bei diesem Zustandsbild Antikoagulantien schädlich erscheinen.

Das Elektrokardiogramm sollte nicht als ein Kriterium dienen zur Bestimmung der Besserung des Befundes des Patienten und des Zeitpunktes an dem er aufsitzen, mit Spaziergängen beginnen, oder an seine Arbeit zurückkehren kann. Ebenso wenig sollte übermässiges Vertrauen auf die Blutsenkungsgeschwindigkeit gesetzt werden, als einem leitenden Faktor in der Behandlung.

Die Behandlung des Versagens des Herzens beim Koronar-Verschluss bleibt die gleiche wie wenn ein Koronar-Verschluss nicht bestünde, jedoch
ist besondere Vorsorge notwendig, um eine Digitalis-Vergiftung zu verhindern.

Eine kalorienarme Diät ist wichtig, denn sie verringert die Herzarbeit, verhindert gastro-cardiale Reflexe und vermindert das Gewicht bei Fettleibigkeit.


Gegen Übelkeit und Erbrechen geht man mit oralen oder intramuskulären Gaben von Mitteln gegen Bewegungskrankheiten vor.

Frühzeitige Behandlung des Schocks ist wesentlich. Die Blutdrucksteigerung mit Mitteln sind eine gute Hilfe, wenn sie frühzeitig gegeben werden. Spielt ein Schlaganfall eine Rolle, soll Strophantin oder Digitalis gegeben werden.


Kortison und ACTH haben keine Wirkung bei Koronar-Verschluss.

Antibiotika gebracht man, falle eine Lungenstauung oder Zeichen eines Herzversagens durch Stauung vorliegen oder die Temperatur über 39° anzeigt.

Whisky sollte beim akuten Koronar-Verschluss nicht gegeben werden, weil dadurch eine Pulsbeschleunigung zustande kommen kann. Das Rauchen muss verboten werden.

Die Prognose des Koronar-Verschlusses hat sich im Laufe der vergangenen 30 Jahre erheblich gebessert. In der Privatpraxis beträgt die Sterblichkeitsziffer während der ersten Attacke jetzt 5% oder weniger. Die meisten Patienten können innerhalb von 2 oder 3 Monaten genesen. Der größte Teil erholt sich leidlich oder gut, mehr als die Hälfte erholt sich funktional ausgezeichnet. 4 von 5 nahmen ihre Arbeit wieder auf.

Complete reference list appears in reprint from New York State Journal of Medicine.