Myocardial Infarction, Its Diagnosis and Treatment: Literature Review*

N. MALCOLM BALOTIN, Ph.D.
Philadelphia, Pennsylvania

Myocardial infarction is a pathological process established by a compromise in the blood supply to an area of myocardium of such severity that even with prolonged rest adequate oxygen cannot be obtained. In the United States there are approximately 600,000 to 800,000 persons each year suffering attacks from this disease. Even though in recent years greater facilitation in diagnosis and tremendous strides in therapy have reduced mortality during the first attack in private patients to 5 per cent or less, the overall annual mortality is 200,000. A large number of the deaths occur during the most productive years of life; many of those who survive suffer disabling complications. The figures for mortality and disabling complications can be decreased by a careful differential diagnosis of patients presenting chest pains and other symptoms characteristic of myocardial infarction and by individualization of therapeutic procedures.

Diagnosis

The diagnosis of acute myocardial infarction is based on the history, the clinical findings, and an evaluation of the electrocardiographic changes. There are many nonspecific signs which may be used as adjuncts.

A tentative diagnosis of myocardial infarction may be suggested in a patient presenting a severe, oppressive pain over the lower sternum or more extensively over the precordium or anterior chest which lasts for 30 minutes or longer and which is not relieved by rest or nitrites. Radiation of this pain to neck, shoulders, or arms confirms the likely origin in the heart. The pain builds up slowly and steadily to a plateau of maximal intensity. Anxiety and fear of impending doom mount rapidly as the pain continues. After several hours the pericardial inflammation resulting from ventricular necrosis may introduce a sharp, stabbing component to the pain. It is typically aggravated by respiration, emotion, swallowing, and may have a throbbing element synchronous with the heartbeat. Complications as shock and congestion of the lungs from acute left ventricular failure may increase the complexity of the symptoms; however, a rapid fall in blood pressure within twenty-four hours or even a more gradual fall in the next few days is interpreted as an important confirmatory sign. Fever, leukocytosis, and elevation of the erythrocyte sedimentation rate are clinical signs which support the diagnosis.

Serial changes in the electrocardiogram are of great value, but not essential, for the diagnosis. The electrocardiogram is considered characteristic of acute myocardial infarction when elevation of the RS-T segment and a deep Q wave are present in one or more leads, with progressive change from RS-T elevation to T-wave inversion in serial records. At

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the onset of the attack the electrocardiogram may be normal or may show only slight changes. At this time, RS-T depression may occur instead of the expected RS-T elevation, which may not appear for hours. The Q waves may appear even later, at the end of one or two days.

The diagnosis of myocardial infarction must be established by evidence of tissue deterioration. This is done most often with studies on glutamic-oxalacetic transaminase, the infarcted muscle releasing two to twenty times the normal serum value within 24 hours, returning to normal three to seven days thereafter. This test by itself is nonspecific because other body tissues release glutamic-oxalacetic transaminase in disease, e.g., liver, kidney, pancreas, and skeletal muscle. It must be used in conjunction with the studies outlined above. Measurements of serum transaminase can be done in most well-equipped hospital and clinical laboratories, the procedures being reasonably standardized and the necessary reagents commercially available.

Included in the differential diagnosis of myocardial infarction should be the following diseases:

1. Chest
   - angina pectoris
   - acute coronary insufficiency
   - pulmonary embolism
   - cardiac arrhythmias
   - acute pericarditis
   - spontaneous pneumothorax
   - dissecting (nonsyphilitic) aneurysm of the aorta
   - spontaneous interstitial emphysema of the lung
   - malinggersers familiar with the symptoms of myocardial infarction
   - other chest diseases—
     - syphilitic aortitis with aortic aneurysm
     - pneumonia
     - pleurisy
     - massive collapse of the lung
     - carcinoma of the lung
     - diaphragmatic or paraesophageal hernia
     - herpes zoster
     - costochondral arthritis
     - rupture of the costochondral junction
     - diseases of the spine or shoulder with referred pain to the anterior chest wall

2. Abdomen
   - acute indigestion
   - acute surgical abdominal disease—
     - perforated peptic ulcer
     - cholelithiasis
     - acute cholecystitis
     - acute pancreatitis
     - acute intestinal obstruction
     - acute appendicitis
     - biliary colic
     - sigmoid spasm
     - food poisoning
     - tabetic crisis
     - acute postoperative peritonitis
     - renal or ureteral colic with reflex ileus
     - and pain in abdomen or chest

3. Other clinical syndromes
   - diabetic coma
   - sickle cell anemia during a hemolytic crisis
   - Addison's disease during a crisis

These conditions differ from myocardial infarction in their electrocardiographic pattern.
A MOST IMPORTANT DIFFERENTIAL DIAGNOSIS IS OUTLINED AS FOLLOWS

<table>
<thead>
<tr>
<th>Precipitating factors</th>
<th>Angina Pectoris</th>
<th>Acute Coronary Insufficiency</th>
<th>Myocardial Infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>effort, emotion, trauma, reflex from other viscera, tobacco, insulin, adrenalin, cold, eating</td>
<td>similar to angina pectoris, spontaneous acute conditions, anoxemia, hemorrhage, shock or fall</td>
<td>at rest or during routine activity</td>
<td></td>
</tr>
</tbody>
</table>

Pain: temporary relieved by nitro-glycerin

Circulation:
1. shock: none, may be present, falls, common
2. blood pressure: no change or rise, may be poor, fall, embryocardia, gallop, common (pericardial) rub, common
3. heart sounds: none, occasional, frequent present
4. arrhythmias: none, may be present, frequently absent
5. heart failure: none, frequent, abnormal
6. fever: absent, frequently normal
7. sedimentation rate: normal

Electrocardiogram:
1. usually no change, for several days, or weeks, no Q waves or RS-T elevation
2. evanescent RS-T depressions, changes for several days or weeks, no Q waves or RS-T elevation
3. RS-T depressions and T-wave changes for several days or weeks, I and III reciprocal leads, progressive pattern, often permanent

Biochemical studies:
1. serum transaminase: normal, increased 2 to 20 times, normal
2. serum aldolase: normal, increased
3. plasma isomerase: normal, increased
4. C-reactive protein: normal, elevated
5. serum lactic dehydrogenase activity: negative, positive, increased
6. Eosinopenia: none, present

Duration of incapacity: minutes to few hours, several hours or weeks, prolonged

Table I

Cardiac fluoroscopy, roentkymography and electrokymography give important information in the study of the patient with myocardial infarction. Observation of marked localized diminution or complete or partial reversal of pulsation of the left ventricular border is indicative of a circumscribed area of myocardial damage, usually as a result of previous infarct. Such findings may be observed in the absence of other typical clinical findings of previous myocardial infarction.
Treatment

The occurrence of an acute myocardial infarction often requires prompt decision. Even with incomplete knowledge and in the face of conflicting views the physician must act. An understanding of the sequence of pathological changes that occur and the potential changes in myocardial function are prerequisite for intelligent therapy. In brief, ischemic changes leading to necrosis occur in the muscle within the first few days after the onset of the myocardial infarction. During the second and third week healing processes set in, including fibrosis and the development of collateral circulation. Six weeks are required for most myocardial infarctions to be considered as healed. The severity of the attack depends on the state of the coronary circulation and integrity of the myocardium after infarction. Subsequent possible cardiovascular complications can be prevented or ameliorated by a wise approach in therapy.

Treatment is based on relief of pain and anxiety, rest for the heart during the healing phase, and rehabilitation of patient and family. Symptoms and complications are cared for as they arise. The chief hazard is overtreatment; routine recommendations of standard therapy should be discouraged. Individualization and attention to details are two of the cardinal elements of success.

Relief of pain is the primary objective in therapy. Morphine sulfate 10 to 15 mg. is given at the onset, subcutaneously if the pain is severe, intravenously if the pain is excruciating or unrelieved. Its euphoric action helps allay anxiety. After the initial injection of morphine it is frequently possible to change to meperidine (Demerol) hydrochloride 50 to 100 mg. subcutaneous and later methadone (Dolophine) hydrochloride, given orally in doses of 5 to 15 mg. every three to four hours. Restlessness or continued anxiety can often be partially controlled by phenobarbital 15 mg. four times a day, with larger barbiturate doses at night to insure sleep.

Hospitalization should be considered as soon as the pain has been relieved. If home conditions are satisfactory the majority of patients may remain there, as most attacks run a mild course following the initial pain. A serious condition or unsatisfactory home environment requires removal of the patient to a hospital, immediately, by ambulance.

Oxygen should be administered to patients exhibiting any of the following conditions:

- cyanosis
- shock
- acute pulmonary edema
- severe and persistent cardiac pain
- congestive failure
- certain cardiac arrhythmias
- a sharp fall in blood pressure
- a rising heart rate
- marked leukocytosis
- high fever
- Cheyne-Stokes respiration not induced by drugs

It is administered as a 50 per cent mixture, 12 to 14 liters per minute, in a cooled tent, for 48 hours to several weeks, depending on need; it may, on occasion, be the crucial factor in saving life.

Relatively few patients with acute myocardial infarction die at the onset
of their illness, indicating that survival of the remainder of these patients depends on what complications resulting from their myocardial damage or temporary impairment of circulatory dynamics may develop. The important complications which can be considered potentially preventable causes of death are shock, serious arrhythmia, thromboembolic phenomena and pulmonary edema.

Shock in some degree is developed in approximately one-half of the patients who survive the onset of an acute myocardial infarction, the mortality rate in this complication varying directly with its severity and duration. Blood transfusions have been disappointing, excessive infusion leading to pulmonary edema or congestive failure or both. The most effective treatment is with vasopressor drugs, the best of which is levarterenol (Levophed) bitartrate. This agent raises the blood pressure to respectable levels and promptly eliminates the symptoms of shock. Undesirable side reactions, as myocardial stimulation, are lacking in levarterenol. The drug is administered by intravenous drip, 4 mg. per liter, in a 5 per cent glucose solution, at the rate of 20 to 30 drops per minute. If the desired blood pressure is not maintained (100 mm. Hg systolic in previously normotensive patients, 120 mm. Hg systolic in previously hypertensive patients) then the drug concentration is increased; a faster rate of infusion is undesirable because it excessively increases body fluid. When discontinuing the levarterenol a slow drip of 5 per cent glucose solution is substituted to prevent a possible hypotensive recurrence. Levarterenol is intensively irritating if extruded from a vein, causing tissue sloughs; extra precautions should be taken during its administration. Phenylephrine (Neosynephrine) hydrochloride 5 mg. subcutaneously or intravenously every 15 minutes or less, or mephentermine (Wyamine) sulfate 15 mg. intravenously or subcutaneously, or in intravenous drip containing 35 to 70 mg. in 100 cc. of 5 per cent glucose solution, can be used initially until levarterenol can be instituted.

Serious arrhythmias may develop during the course of myocardial infarction in the form of auricular or ventricular premature beats, auricular flutter or fibrillation and auricular or ventricular tachycardias. Unless the patient has a past history of an arrhythmia it is not advisable to administer drugs prophylactically to counteract such irritability. Quinidine is a cardiac depressant with many side affects. This drug should be given only if premature beats are present, its dosage being 0.2-0.4 gm. every six hours. Pronestyl (procainamide) hydrochloride may be substituted for quinidine, 0.25-0.50 gm. administered every six hours. If the arrhythmia is auricular fibrillation or flutter, digitalis is more appropriate as an initial measure. The belief that digitalis in the presence of a myocardial infarct will favor rupture of the myocardium is erroneous.

Paroxysmal ventricular tachycardia presents a special problem in that it cannot be long sustained in patients with myocardial infarction without fatal result; it produces severe cardiac and circulatory stress. Quinidine orally every two hours in increasing dosage of 0.4 gm., 0.6 gm., 0.8 gm. to obtain conversion is given if the general condition of the patient continues to be satisfactory. If the patient's condition is deteriorating, intravenous procainamide hydrochloride diluted in 5 per cent glucose solu-
tion is given at the rate of 50 to 100 mg. per minute until the rhythm reverts to normal or worsens appreciably, the maximum dose being 1 gm. Since intravenous procainamide may cause a considerable lowering of blood pressure, levarterenol may be administered simultaneously.2

Complete heart block with Stokes-Adams syndrome is rare,13 but if this medical emergency threatens ephedrine sulfate 25 to 50 mg. orally every three to four hours or, if necessary, 1 : 100,000 epinephrine hydrochloride by slow intravenous injection or epinephrine subcutaneously or intramuscularly may be administered.11

Thromboembolic complications in acute myocardial infarction are effectively reduced by anticoagulant therapy.11, 13, 15 Immediate effects may be obtained with heparin sodium 50 to 75 mg. intravenous or intramuscular every four to six hours, regulating this dosage schedule so that the clotting time is prolonged to not more than twice the normal time at the end of four hours. Blood for the prothrombin test should not be drawn within four hours of heparin administration because of some hypoprothrombinemic effect of this agent. Heparin has the asset that protamine sulfate 50 to 100 mg. intravenous is an excellent and immediately effective antidote, neutralizing the circulating heparin and promptly returning the coagulation mechanism to normal. Simultaneous with heparin administrationbishydroxycoumarin (Dicumarol) can be given orally, regulating the maintenance dose so that the prothrombin is 20 to 30 per cent of normal. When this is achieved, usually within 48 to 72 hours, heparin can be omitted. Anticoagulant therapy should be continued until the patient is ambulatory. Anticoagulants should be employed in most patients with acute myocardial infarction, noting the following contraindications to or indications for caution:15

- gastrointestinal bleeding
- peptic ulcer
- recent fracture or bone fusion operation
- recent prostatectomy
- recent central nervous system operation
- increased capillary fragility
- thrombocytopenia
- renal insufficiency
- severe hypertension
- possible cerebral hemorrhage
- pregnancy
- subacute bacterial endocarditis
- liver disease (cirrhosis or hepatitis)
- congestive heart failure
- depletion of vitamin K stores or prothrombin reserves—
  - cachexia
  - oral antibiotic therapy
  - parenteral feeding
  - malnutrition
- history of bleeding with previous anticoagulant therapy
- inadequate or undependable laboratory facilities for
  - prothrombin determination
- immediate postpartum or postoperative period
- nonspecific pericarditis2
- ulcerative colitis2

Pulmonary edema is a common occurrence in patients with myocardial infarction. It is treated as if myocardial infarction were not present.2, 13 The patient is elevated to a sitting position in bed. Morphine sulfate 30 mg. hypodermically or intravenous is usually efficacious but intravenous aminophyllin and strophanthsin may be necessary. Aminophyllin 0.5 gm. is admin-

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istered slowly. The initial dose of strophanthidin K is 0.25 mg.; 0.1 mg. injections may be repeated every hour until 1 mg. has been given in 24 hours. Oxygen under positive pressure, tourniquets on all four extremities, leaving one cuff loose and rotating every 15 minutes, phlebotomy if shock is not present, and an intravenous mercurial diuretic may be helpful. Occasionally the inhalation of alcohol vapor 20 to 30 per cent to reduce foaming in pulmonary edema has been effective. These patients should receive anticoagulant therapy even if the sputum is bloody.

Minor disturbances are often quite upsetting to the patient and may even lead to serious consequences. Nausea and vomiting may be prevented by abstinence from fruit juices, cold milk, spicy foods and treated with dimenhydrinate (Dramamine) 50 mg., chlorpromazine (Thorazine) 10 to 25 mg., or cyclizine hydrochloride (Marezine) 25 to 50 mg. orally, intramuscularly or by suppository.2 Hiccough will almost always subside with constant reassurance. If not, remedial procedures as chlorpromazine, ethyl chloride spray along the diaphragm, and atropine sulfate intravenous have often proved successful.

During the period of bed rest the patient need not be so restricted as to caution against wiggling the fingers, moving the arms, or turning on the side with help.2 The upper segment of a hospital bed can be raised so that complete recumbency with the attendant slight increase in the work of the heart is avoided. The patient should feed himself as soon as he is able. Constipation may be present because of the effect of morphine, lack of activity, and limited food intake. Mineral oil or milk of magnesia at night, assisted by a small saline enema in the morning, may initiate movements after the early period has passed and prevent straining at the stool.3 Use of the bedpan often requires much physical effort and mental stress. Careful lifting of the patient onto a bedside commode or nearby bathroom commode is saving in cardiac effort and is usually considerably more acceptable. Gentle foot and leg exercises are advisable to prevent stasis of blood in extremities. Patients suffering mild attacks of myocardial infarction are kept in bed approximately one week unless it is felt that sitting up in a chair earlier than this period is necessary to raise morale.2

In more severe attacks the patient remains in bed for longer periods of time.13 During the third and fourth weeks increased movement of the extremities in bed is allowed. While sitting, the patient may engage in occupational therapy within his capability, read newspapers and magazines that he can manage by himself. Letter-writing may be permitted, as well as an increasing number of short visits with friends. During the fifth and sixth weeks standing leads progressively to walking on the level and the beginning of stair-climbing.

The electrocardiogram is not depended upon 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.2 If the clinical course is satisfactory the patient may get up and walk even though the electrocardiogram shows marked alterations. Nor is too much reliance placed upon the sedimentation rate as a guiding factor in treatment.

A planned dietary regimen seems worthwhile in these patients.2, 12 Lowering fat intake may retard or even reverse arteriosclerotic processes,
MYOCARDIAL INFARCTION

decreasing the likelihood of future complications. Dieting decreases the work of the heart and prevents gastro-cardiac reflexes. During the first few days or weeks 700 to 1200 calories suffice; thereafter the intake may be gradually increased. Fried foods, the fatty parts of meat, cream, creamed soups, and thick gravy are omitted. Butter may be used in moderation, lean meats, eggs, and ordinary cheese are allowed in limited amounts. Consumption of skimmed milk, cottage cheese, green and yellow vegetables is encouraged. A moderate dieting regimen of this nature can be followed without great hardship or difficulty.

Regarding tobacco there is no proof that it aggravates coronary atherosclerosis while it appeals to people all over the world. The physician should carefully evaluate the importance of this restriction in a person who is already limited in so many ways. The same applies to the moderate use of alcohol.¹¹

The vast majority of patients surviving myocardial infarctions make a fair or good recovery; over half of the patients make an excellent functional recovery.² Four out of every five patients are able to resume work following coronary occlusion and can lead productive lives for many years. Very mild cases may be ready to return to work within two to three months, more severe cases within three to six months.

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Addendum: At Jefferson Medical College Hospital, the prothrombin level is kept at 10 to 20 per cent.

SUMMARY

In patients suffering an attack of myocardial infarction the history and symptoms are often sufficient to make a tentative diagnosis. Serial changes in the electrocardiogram are valuable but not essential. The diagnosis must be established by evidence of muscle necrosis.

The chief hazard in treatment is excessive therapy. Individualization and attention to details are two of the cardinal elements for success. Symptoms and complications should, in general, be treated as they arise. The chief complications which are potentially preventable causes of death are shock, serious arrhythmia, thromboembolic phenomena, and pulmonary edema.

A vast majority of these patients make a fair to good recovery and can lead productive lives for many years.

RESUMEN

En los enfermos que sufren un ataque de infarto del miocardio, los síntomas y la historia clínica a menudo bastan para hacer un diagnóstico preliminar. Los cambios en la serie de electrocardiogramas son valiosos pero no esenciales. El diagnóstico debe establecerse por la evidencia de la necrosis muscular.

El principal riesgo en la terapéutica es el tratamiento excesivo.

Son elementos cardinales para el buen resultado la individualización y la atención a los detalles. En general, los síntomas y las complicaciones deben tratarse conforme se presenten. Las principales complicaciones que son posibles de prevenirse como causas de muerte son el shock, la arritmia grave, fenómenos tromboembólicos, y el edema pulmonar.

Una amplia mayoría de estos enfermos se recuperan bastante bien o bien y pueden llevar una vida productiva por muchos años.

RESUME

Chez des malades atteints d'infarctus du myocarde, l'histoire et les symptômes sont souvent suffisants pour permettre une tentative de diagnostic. Des al tétrations répétées sur l'électrocardiogramme sont valables mais pas essentielles. Le diagnostic doit être établi par la preuve d'une nécrose musculaire.

Le principal danger du traitement est réalisé par une thérapeutique excessive. L'individualisation et l'attention donnée aux détails sont deux des principaux éléments de succès. Les symptômes et les complications devraient, en général, être traités des qu'ils apparaissent. Les principales complications qui sont des causes cependant évitables de la mort sont le shock, l'arythmie grave, les phénomènes thrombo-emboliques, et l'œdème pulmonaire.

Une grande majorité de ces malades obtinrent une guérison convenable ou bonne, et purent mener une vie active pendant plusieurs années.
N. MALCOLM BALOTIN

ZUSAMMENFASSUNG

Bei Patienten die an einem Anfall von Heroinfaekt leiden, reichen oft Vorgeschichte und Symptome aus, um eine vorläufige Diagnose zu stellen. Serienveränderungen im Elektrocardiogramm sind von Wert, aber nicht wesentlich. Die Diagnose muss begründet werden durch Anzeichen von Muskelnekrose.


REFERENCES