Thromboembolic Complications Following So-called "Good Risk" Cases of Myocardial Infarction

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Thromboembolic complications frequently occur following myocardial infarction. Following the general acceptance of anticoagulant therapy for the treatment of myocardial infarction, there have been some authors who have recommended that this therapy be withheld from "mild cases," unless they develop thromboembolic complications. It is the belief of the present authors that this is not justified unless there are contraindications for anticoagulant treatment, provided that proper facilities are available. Summaries of experience with 14 so-called "good risk" cases of myocardial infarction who developed a total of 18 certain and 4 probable thromboembolic complications are presented. As a result, there were 4 major amputations in 3 patients, 1 of whom died. Other complications are discussed in detail.

It is a well known fact that thromboembolic complications frequently occur in patients who have suffered from myocardial infarction. In the series reported by the Committee on Anticoagulants of the American Heart Association, 26 per cent of the patients who received routine treatment without anticoagulants developed either one or more thromboembolic complications during the 6-week period of observation that followed the initial episode of coronary occlusion with infarction. In 19 additional series reported in the literature,1 an average of 18.9 per cent of patients who were not treated with anticoagulants developed thromboembolic complications.

Pathologic studies have demonstrated the high incidence of mural thrombi that develop in association with myocardial infarction.2,3 The figures published by different investigators have varied, the highest being 66.9 per cent reported by Garvin4 and 83 per cent in the series of Levine and Brown.5 Intracardiac thrombosis undoubtedly represents the main source of embolization, the formation of mural thrombi occurring most frequently in the left ventricle. Thrombosis of the veins of the pelvis and lower extremities is often the source of pulmonary emboli.6

Although thromboembolic complications usually develop during the first few weeks that follow an episode of myocardial infarction, they may also occur after months or even years of the attack. Levine7 has pointed out that mural thrombi may remain pocketed in the heart chambers for many years, and represent a continuous threat of embolization. Towbin,8 in a recent study, was able to demonstrate pathologic evidence of multiple cerebral embolism in 11 patients with chronic organic brain disease who had had old myocardial infarctions, in some cases several years prior to the development of cerebral embolism. All of them harbored intracardiac mural thrombi.

The value of anticoagulant therapy in the management of coronary thrombosis with myocardial infarction has been substantiated by many investigators. The results presented by the Committee on Anticoagulants of the American Heart Association1 and the data reported by others9-12 indicate that anticoagulant therapy markedly reduces the incidence of thromboembolic complications as well as the death rate of patients who have suffered from myocardial infarction.

After the Committee recommended that anticoagulant therapy be given to all patients with myocardial infarction provided no contra-
indications were present, several authors expressed doubts as to the advisability of instituting this form of therapy in every case of myocardial infarction. They have concluded that the selection of patients for anticoagulants should be based on certain prognostic criteria.

Russek and associates\textsuperscript{12-16} suggested that patients with myocardial infarction be classified as “good risk” or “poor risk” cases according to the signs and symptoms present on the day of admission to the hospital, and have proposed the criteria for such a classification. They consider the patient to be a “poor risk” case, when one or more of the following poor prognostic factors are present during the first 24 hours of hospitalization: (1) evidence of a previous myocardial infarction; (2) intractable pain; (3) extreme degree or persistence of shock; (4) significant enlargement of the heart; (5) gallop rhythm; (6) congestive heart failure; (7) atrial fibrillation or flutter, ventricular tachycardia, or intraventricular block; (8) diabetic acidosis, marked obesity, previous pulmonary embolism, varicosities in the lower extremities, thrombophlebitis (past or present), or other states predisposing to thrombosis. Contrariwise, a patient not presenting any of the signs mentioned above during the first 24 hours of hospitalization is considered a “good risk” case.

According to Russek’s report, the “good risk” cases have a low mortality rate as well as a low incidence of thromboembolic complications as compared with “poor risk” cases. Therefore, he does not consider the use of anticoagulant therapy indicated in those cases, since “the incidence of hemorrhagic complications and death due to anticoagulant drugs, even when prescribed by the most competent investigators, appears to outweigh any benefit which such therapy may confer in milder cases.”\textsuperscript{16} He concludes that anticoagulant therapy should be reserved for properly selected “poor risk” cases in which the mortality rate and the incidence of thromboembolism are markedly elevated. There have been several other workers who have expressed similar opinions.\textsuperscript{17, 18}

The Committee divided the cases of its series into good and poor risk groups, following criteria similar to and even more rigid than those of Russek and co-workers. Only 17 per cent of the cases could have been classified as “good risk” cases as contrasted with 47 per cent reported by Russek. Among the group of “good risk” cases of the Committee who were not receiving anticoagulant therapy, 23 per cent developed thromboembolic complications, including three deaths if an age of 80 or over is not defined as a poor risk criterion, and one death if the old age is considered to aggravate the prognosis. This is in sharp contrast with Russek’s report of less than 1 per cent of thromboembolic complications in his group of “good risk” patients.

We believe that the estimation of prognosis of a given case of myocardial infarction, based on the signs and symptoms present during the first 24 or 48 hours cannot be made with any degree of certainty. Halpern and co-workers\textsuperscript{19} recently analyzed a group of 107 patients who fulfilled the criteria for the diagnosis of acute myocardial infarction, and estimated the prognosis when the patient was first examined, and again in 24 and 48 hours. They found that in 29 per cent of their patients it was necessary to change the prognosis during the first 48 hours of hospitalization. Manchester\textsuperscript{20} has also attempted to classify 350 patients as “good” or “poor risk” cases. He found that it was necessary to change the category of 25 per cent of his “good risk” cases to “poor risk,” and he also noted that 33 per cent of his “poor risk” cases had completely uncomplicated recoveries. Many other workers in this country and abroad\textsuperscript{21-23} have agreed that it is impossible to determine with certainty the prognosis of an acute myocardial infarction during the first 24 or 48 hours of the attack.

At the International Conference on Blood Coagulation held in Switzerland in 1954, a panel was held on the problems of myocardial infarction. There was complete unanimity among the members that to base treatment on such an attempt to classify patients was not warranted, and that all patients suffering from myocardial infarction should receive anticoagulants, if adequate facilities were available and unless there were other contraindications.\textsuperscript{24} One of the difficulties in the classification of
a patient as "good" or "poor risk," following the criteria indicated by Russek and his group, is represented by the fact that no definitions have been given of what they consider as "significant enlargement of the heart," "intractable pain," and "other conditions predisposing to thrombosis." This vagueness, in some instances, makes it difficult to decide whether or not the patient should be considered as a "good risk" or "poor risk" case during the first 48 hours.

Moreover, even if a case is believed to be a mild form of coronary thrombosis, it becomes extremely hazardous to predict with certainty that the patient will do well and that no thromboembolic complications will arise. This is clearly demonstrated in patients who have sustained the so-called silent type of coronary occlusion, in which peripheral embolization may be the presenting symptom of myocardial infarction as in some of the cases reported by Lary and De Takats.\textsuperscript{25} It is also underscored by the rather frequent sudden death that occurs in patients who have had no previous evidence of cardiac disturbances.

The purpose of this paper is to report on thromboembolic complications occurring in the so-called mild or "good risk" cases of myocardial infarction. We have selected 14 patients who sustained mild attacks of myocardial infarction and fulfilled Russek's criteria of "good risk" cases. All of them were untreated or inadequately treated with anticoagulants, prior to their thromboembolic complications.

The case histories were selected from a group of patients with myocardial infarction seen at The New York Hospital and from the private practices of members of the Vascular Section of the Department of Medicine at The New York Hospital, Cornell University Medical School Center.

**Case Reports**

**Case 1** (S.R. 618371). A few hours prior to admission, a 69 year old man was picking up a kerosene can and moving it a few feet, when he suddenly noted the onset of sharp pain in the right hand. Shortly thereafter this pain extended up to the lower part of the forearm. Simultaneously, the hand became cold and functionless.

His past history, on close questioning, disclosed that he had developed slight precordial pressure on walking 2 blocks during the 3 months prior to admission. This "pressure" was of such a mild degree that he did not consider it seriously, nor did he go to his doctor.

At the time of admission, he had a blood pressure of 150/90, a pulse rate of 82 per minute, and a temperature of 99.6 F. The heart and lungs were unremarkable. On physical examination it was evident that the patient had an acute occlusion at the level of the brachial or the axillary artery. A chest film revealed some enlargement of the left ventricle. An electrocardiogram disclosed evidence of a recent infarction of the anterior wall.

The patient was treated with vasodilators and blocks of the brachial plexus. Anticoagulant therapy was not used in this case because shortly after admission the patient had a hematemesis of undetermined origin. Despite the therapy employed, the extremity became gangrenous and a midarm amputation had to be performed on the thirteenth hospital day. In the immediate postoperative state, a transitory episode of mental confusion and right facial weakness occurred, which cleared up shortly thereafter. The patient was followed in clinic for over a year, until January 1953.

This case represents a mild coronary thrombosis that remained undiagnosed owing to the paucity of symptoms presented by the patient before the thromboembolic complication took place. It illustrates the serious outcome that may sometimes occur in so-called mild myocardial infarctions.

**Case 2** (M.S. 696317). A 50 year old white woman was admitted to The New York Hospital on Nov. 5, 1954, complaining of numbness in the left leg of 9 hours' duration.

Six weeks prior to admission the patient began to develop occasional episodes of chest pain. The cardiac origin of this was not recognized at the beginning. She was treated with various medications, mostly sedatives, until finally, two and one-half weeks prior to admission, a diagnosis of myocardial infarction was confirmed and the patient was taken to another hospital. She was treated with bed rest and received some anticoagulant therapy over a period of 2 weeks. Two days prior to admission to The New York Hospital, after dicumarol therapy had been interrupted, the undilute prothrombin time was 17 seconds against a control of 15 seconds. This, of course, was not a protective level.

On arrival, she was complaining of cramps in the left leg and stated that the numbness had been present for 9 hours. On physical examination she had a blood pressure of 170/110, a pulse rate of 80 per minute, and a temperature of 99.3 F. The lungs were clear. The heart was not enlarged; the sounds and the rhythm were normal, and there were no murmurs. There were signs of arterial insufficiency in both legs, particularly in the left, where only the femoral pulse was present. The left leg was cool.
below the knee. An x-ray film of her chest was reported as being within normal limits. An electrocardiogram revealed evolutionary changes of a myocardial infarction of the anterior wall.

The patient was started again on anticoagulant therapy. However, gangrene developed and, later on, demarcated at the base of the left hallux. The patient was discharged on the sixty-ninth hospital day, to continue with anticoagulants on an ambulatory basis. She was followed in the Vascular Clinic. Early in March 1955, she developed self amputation of the toe. At present, she is being followed at the Vascular Clinic.

This patient had a mild attack of myocardial infarction and was treated with anticoagulants for only two weeks—too short a period of time. Two days after discontinuing dicumarol, when her prothrombin time had returned to normal, she suffered an embolus or thrombosis that produced gangrene of a toe, causing its subsequent amputation.

Case 3 (J.L. 703473). A 56 year old white man had a mild coronary occlusion on Dec. 24, 1954. He was treated at home with bed rest and papaverine. No anticoagulants were administered. A month later, after having had a bowel movement in a bedpan, he noted the sudden onset of pain in the left foot associated with coolness and numbness. Over the next few days he was given vasodilators and gradually started to ambulate, but he complained of pain in the anterior sole of the left foot on walking a few steps.

He was admitted to The New York Hospital on Feb. 15, 1955. On arrival his blood pressure was 120/88, pulse rate 92 per minute, and his temperature was 96.4 F. The heart was not enlarged. A normal sinus rhythm was present and no murmurs were heard. Examination of the lower extremities revealed signs of arterial insufficiency, more marked on the left side. Oscillometric readings were absent on the left leg and markedly reduced on the right. There were electrocardiographic changes of evolution of a myocardial infarction of the anterior wall.

The patient was placed in an oscillating bed and given Marcumar. He started to ambulate early in March and was discharged on March 12, 1955, very much relieved but without changes in the physical findings of his lower extremities.

This patient sustained a myocardial infarction of such mildness that it was not considered necessary to hospitalize him, and he was treated conservatively at home. Despite the mildness of the case, a month later he suffered an embolus to the terminal aorta, with subsequent fragmentation of the clot. Although no gangrene developed, the patient was left with arterial insufficiency of his lower extremities, secondary to the embolic complication.

Case 4 (E.F. 268730). A 46 year old white man was admitted on June 20, 1940, with gangrene of the right leg.

Seventeen days prior to admission, while shaving himself, this patient experienced the sudden onset of cramplike precordial pain, associated with mild dyspnea and aching in both arms. These symptoms subsided completely upon the administration of morphine. An electrocardiogram revealed evidence of a myocardial infarction, and the patient was advised by his private physician to rest in bed at home—but he was given no more specific therapy.

Eight days later he felt that his right leg was "swelling up." Pain developed in the leg and later it became anesthetic and functionless. Two and a half days later gangrene developed in the toes and extended to the entire foot and the lower leg.

Physical examination disclosed a well developed and well nourished white male, appearing chronically ill but not acutely distressed. Blood pressure was 135/84, pulse rate 80 per minute, and temperature 99.3 F. The heart, both clinically and on x-ray examination, was unremarkable. The abdomen was slightly distended. The gangrene was beginning to demarcate 5 cm. below the knee. The white blood cell count was 22,000, with shift to the left. An electrocardiogram confirmed the existence of a myocardial infarction of the anterior wall.

The hospital course was stormy. Two days after admission, the patient had a hematemesis of 240 ml. On June 29, 1940, he developed sudden pain in the left leg with clinical evidence of an acute arterial occlusion of the limb and subsequent development of gangrene. On the twenty-second hospital day, a right mid thigh amputation was performed, followed by amputation of the left leg at the same level on the fiftieth hospital day.

A month later, on September 14, 1940, he suddenly developed tachycardia, cough and bloody sputum. On examination of the chest, there was slight dullness to percussion, and rhonchi were heard in the right base. Small pleural effusions in the oblique fissures of both sides and some atelectasis of the right middle lobe were seen on x-ray study. The electrocardiographic findings were consistent with pulmonary embolism. Shortly thereafter, the blood urea nitrogen started to rise; the CO₂ began to drop, and the patient died on Sept. 27, 1940, 99 days after admission and 116 days following the onset of his illness.

This case clearly illustrates the fact that so-called mild myocardial infarction may be followed by thromboembolic complications that lead to a disastrous outcome. A man without a previous history of heart disease developed a mild attack of myocardial infarction. A week later he suffered a series of embolizations to his legs and his lungs, with the development of gangrene that required bilateral amputation of his lower extremities, and he finally died almost four months after the coronary occlusion.

Case 5 (F.P. 75041). A 58 year old white man was admitted to Doctors' Hospital on July 16, 1952,
after having noted the sudden onset of numbness and coldness of his right foot.

A week prior to admission, after a transcontinental motor trip, the patient complained of having some general fatigue without any evidence of cardiac symptoms. For this reason he remained in bed for a day, but was able to resume his normal activities later on. There was no previous history of cardiovascular disease.

On physical examination he was found to have a blood pressure of 140/80, a pulse rate of 110 per minute, and a temperature of 100.5 °F. His heart was not enlarged, the rhythm was regular and there were no murmurs. Pulses and oscillometric readings were absent on the right thigh above the knee and were diminished in the left ankle. An electrocardiogram revealed that the patient had sustained what appeared to be a recent myocardial infarction of the posterior wall.

Within three hours of admission, a right femoral embolectomy was performed by Dr. Jere Lord. While a large clot was removed, it was clear that terminal vessels of the leg had been occluded by fragments of the embolus. Subsequently anticoagulant therapy was initiated. However, in spite of careful management, the leg became gangrenous, and a low thigh amputation was carried out on the thirteenth hospital day. The patient had an uneventful postoperative course and was discharged on September 17, 1952.

This case represents a typical silent type of myocardial infarction with peripheral embolization and loss of a limb.

Case 6 (F.S. 76815). In October 1934, a 45 year old man was admitted to The New York Hospital. A week before he had developed pain in the right chest, exacerbated by deep inspirations. The pain subsided rather quickly, but soreness in the right shoulder persisted for several days. Forty-eight hours following the onset of his illness, he coughed up small amounts of clotted blood. Shortly thereafter the patient experienced dysuria, and red blood cells were demonstrated in the urine.

The patient was referred to the hospital by his private physician. On admission he appeared moderately acutely ill. He had a temperature of 102 °F., and the blood pressure was 140/90. Examination of the lungs revealed signs of consolidation in the right base. Heart sounds were of good quality and the rate was 130 per minute. There was a soft, low-pitched systolic murmur inside the apex and a questionable apical gallop. The laboratory studies showed a leukocytosis of 10,000. X-ray films were consistent with the diagnosis of pulmonary infarction; there was haziness of the right middle lobe; the heart shadow was slightly increased, and there was an enlarged aortic area. An electrocardiogram showed small Q waves in lead I, and deep Q waves and inverted T waves in lead IV. The patient recovered and was discharged on the twenty-second hospital day.

In 1943, at the age of 54, he was admitted to the hospital with a cerebrovascular accident that left him with a right hemiplegia. Three years later he went into congestive heart failure with atrial fibrillation and finally died in 1948.

At autopsy the heart weighed 600 Gm. There was evidence of old scarring. The right atrium contained large mural thrombi. There was an old 3-centimeter indurated area of fibrosis in the right lower lobe, corresponding to the healed pulmonary infarction. Multiple infarcts were found in the kidneys and the spleen.

This patient had a mild myocardial infarction at the age of 45, and a few days later he developed a clinical picture suggestive of pulmonary infarction. The autopsy many years later demonstrated old scarring in the myocardium and an area of fibrosis in the right lung, corresponding to an old pulmonary infarction.

Case 7 (M.M. 407972). A 62 year old man was admitted to The New York Hospital on December 21, 1950, with a diagnosis of optic neuritis, which was treated with ACTH. The patient had had no symptoms related to his cardiovascular system. Five days after admission a routine electrocardiogram revealed evidence of a myocardial infarction of the anterior wall. Physical examination disclosed a blood pressure of 135/78 and a pulse rate of 80 per minute. The heart sounds were normal, and no murmurs were heard. An x-ray film of the chest showed the heart to be of normal size, with elongation and tortuosity of the aorta. Subsequent electrocardiograms showed evolutionary changes of coronary occlusion. No anticoagulants were given.

Twenty days after admission the patient developed chest pain not related to effort, breathing or coughing. Rales were found in both bases, particularly in the left, and an x-ray film of the chest showed slight changes in the left costophrenic angle. A diagnosis of pulmonary infarction was entertained. Six days following this episode he suddenly complained of numbness in the left foot. Physical examination showed coldness of the foot, absent pulses, and negative oscillometric readings below the knee. In addition, he had a transitory loss of consciousness that might well have represented a small embolization to his brain. A spinal puncture revealed normal findings. Following this, the patient received anticoagulant therapy for six weeks and recovered satisfactorily.

This case illustrates the development of multiple thromboembolic complications, (lung, leg and probably brain), in a man who had sustained an asymptomatic myocardial infarction, which would be classified as a "good risk" case.

Case 8 (K.P. 419489). A 50 year old white man was admitted to The New York Hospital on Au-
August 14, 1945. Two days prior to admission, while fighting a fire, he inhaled smoke and shortly thereafter experienced pain in the chest radiating to the back and the arms, and associated with some nausea, sweating and weakness. The pain subsided after several hours.

On admission, the blood pressure was found to be 120/70 and the pulse rate to be 110 per minute. Examination of the lungs showed a few fine moist rales at the right base. The heart was not enlarged; the rhythm was regular and there were no murmurs. A chest film was within normal limits. An electrocardiogram disclosed evidence of acute myocardial infarction.

The initial hospital course was unremarkable until the tenth day, when he suddenly complained of severe pain and burning sensation in the left leg. Two days later the right leg also became painful and the pain spread up to the groins. There was evidence on physical examination that he had sustained a saddle embolism with fragmentation of the clot and involvement particularly of the left leg. He recovered and left the hospital on Nov. 1, 1945, with arterial insufficiency in both legs, to be followed in the Vascular Clinic.

Eight years later, in 1953, he was readmitted to the hospital and died of primary carcinoma of the liver. At autopsy, the heart weighed 390 Gm. There was an old, 4 by 6 cm., left apical zone of infarction limited by a thin layer of myocardium. There were also dilatation of the left ventricle and a small thrombus in the same chamber.

This represents another case of arterial insufficiency of the lower extremities secondary to peripheral embolization, following what was initially a very mild infarction. It is interesting to observe that the autopsy demonstrated mural thrombi in the left ventricle eight years after the infarction had occurred.

Case 9 (A.P. 385558). A 40 year old man was admitted to The New York Hospital on May 12, 1944. Ten hours prior to admission, following sexual intercourse, he developed severe pain behind the sternum, radiating posteriorly to the back and the shoulders. The pain was associated with sweating and subsided in an hour, although a slight precordial discomfort persisted intermittently until arrival at the hospital.

The past history was unremarkable except that he had rheumatic fever at the age of 10. Physical examination revealed a well nourished, well developed, healthy looking man, complaining of retrosternal distress. Vital signs were temperature, 102.2 F.; pulse rate, 110 per minute; blood pressure, 134/98; lungs were clear. Examination of the heart revealed a normal sinus rhythm. The point of maximum impulse was found 8 cm. outside the midsternal line in the fourth left intercostal space. There was a soft, apical systolic murmur. A friction rub appeared the day after admission, and persisted, on and off, for four days. An electrocardiogram disclosed evidence of coronary occlusion with infarction of the anterior wall. A portable chest film suggested some enlargement of the heart, particularly of the left ventricle.

On the fifth hospital day, when he appeared to be doing well, he developed fever and signs of consolidation of the right base, occurring without chest pain or hemoptysis. The course of the fever was not altered by the use of sulfadiazine. The possibility of a pulmonary infarct or pneumonia was considered. Two weeks later the patient suddenly developed lumbar pain that persisted for several days but gradually subsided. The pain was associated with albuminuria, hematuria and fever. A diagnosis of renal infarction was suggested. He finally recovered and, at the time of discharge on July 6, 1944, the signs of pulmonary consolidation had disappeared and the urine was clear.

This middle-aged man sustained a mild infarction and developed two thromboembolic complications over a period of three weeks after the onset of his illness. The initial chest pain was severe but subsided in an hour. Thereafter, a mild precordial discomfort persisted intermittently for several hours. Severe pain of one hour's duration would not usually be considered as intractable. If it were to be so interpreted, this patient would be considered as a "poor risk" case. This illustrates the difficulty in applying this specific point of the prognostic criteria that have been suggested.

Case 10 (M.P.M. 639901). A 52 year old hospital orderly was admitted to The New York Hospital on July 7, 1954. During the four months prior to admission, the patient had developed several episodes of left anterior chest pain on exertion. The pain was always relieved by rest. Seventy-two hours prior to admission he was seized with a severe aching sensation in the lower sternal area, associated with some nausea and sweating. The symptoms quickly subsided. On the day of admission he again complained of chest pain, this time radiating to the back and spreading down the left arm.

Physical examination revealed a mildly obese, moderately acutely ill, white man. Blood pressure was 130/90, pulse rate 76 per minute, and temperature 102.2 F. The heart was displaced to the left with the point of maximum impulse in the fifth intercostal space, 2 centimeters outside the midclavicular line. The heart sounds were normal, and there were no murmurs. Fine rales at the bases were present according to one observer. Slight tenderness was found in the right calf, and the dorsalis pedis pulse was absent on that side. An x-ray film of his chest was reported as showing a "right midlung density that represents either disk atelectasis or pulmonary infarct." The electrocardiogram showed evidence of a recent myocardial infarction of the posterior wall. Evolutionary changes were demonstrated in subsequent tracings. The patient was
started on dicumarol therapy upon admission, and was discharged on the forty-third hospital day.

He was then referred to the Vascular Clinic, complaining of intermittent claudication on walking two city blocks. Physical examination showed evidence of arterial insufficiency of both legs, (mostly of the right), with absent oscillometric deflections and pulses below the knee.

This patient never had symptoms suggestive of arterial insufficiency of the lower extremities prior to the onset of this illness. Tenderness in the right calf, with an absent dorsalis pedis pulse on that side, was discovered on admission. When he left the hospital, marked intermittent claudication was present, and there were physical findings of impaired arterial circulation of both legs that were not present previously. It seems likely that he had a saddle embolus that disintegrated and descended into the smaller arteries of his legs. Anticoagulant therapy was instituted shortly after admission to prevent the propagation of these clots and to prevent additional thromboembolic complications.

Case 11 (R.M.M. 651703). A woman, aged 69, was admitted to the hospital on March 15, 1953, having had two bouts of hemoptysis during the 24 hours prior to admission. These were associated with moderate tightness in the lower sternal area and difficulty in breathing. On arrival, she expectorated bright, red blood and again complained of retrosternal tightness. Her past history was of interest in that 15 years earlier she had had a small hemoptysis of unknown etiology.

Physical examination revealed her lying quietly in bed without significant discomfort. Temperature on admission was 99.5 F., and a low grade fever persisted for over a week. Respirations were 28 per minute, pulse rate was 84 and blood pressure was 170/110. The lungs showed tactile fremitus and rales at the right base. She had a normal sinus rhythm with occasional premature beats. The heart was moderately enlarged, both clinically and on x-ray examination, and there was a faint apical systolic murmur. The second aortic sound was louder than the second pulmonic sound. A roentgenogram of her chest failed to reveal any pulmonary disease. The heart shadow was slightly increased in size, and there was elongation and calcification of the aorta. Laboratory analysis included a white cell count that showed a leukoerytosis of 13,000, with slight deviation to the left.

A bronchoscopy performed on the fifth hospital day was entirely negative. The patient left the hospital on the ninth day. An electrocardiogram made after the patient had been discharged showed evidence of recent coronary occlusion with infarction.

The symptoms and physical findings in this case suggest the possibility that this patient had a pulmonary infarction that could not be clearly differentiated on x-ray films. This occurred in a patient who sustained a recent silent coronary occlusion, demonstrated by evolutionary changes of myocardial infarction in the tracing.

Case 12 (E.C.). This patient was seen in the office of one of us (I.S.W.) on April 12, 1955. Two months prior to his visit he had developed pain in the left shoulder, radiating down the left arm. Oral medica
tion and diathermy did not help him and the pain persisted at the time of his visit to the office. One week after its onset, he developed pain in the left leg on walking, with a resultant intermittent claudication at a distance of about 200 feet. At no time had he had pain in the chest or in the retrosternal area. He had not noticed any relation of the pain to exertion.

Physical examination revealed a blood pressure of 140/88. The lungs were clear. The heart was in normal sinus rhythm, with sounds of fair quality; no murmurs were heard. The liver edge was palpable 2 fingerbreadths below the costal margin. Examination of the extremities showed the left foot to be cooler than the right. There was 1+ rubor on dependency and 1+ pallor on elevation of the left foot. In the right leg the femoral pulse was felt, but the popliteal, dorsalis pedis and posterior tibial pulsations were absent. Oscillometric readings showed marked diminution in the left leg as compared with the right. An electrocardiogram indicated evidence of a myocardial infarction that had occurred in the recent past, from which the patient was apparently recovering.

This patient had a mild myocardial infarction that was silent insofar as retrosternal pain is concerned. The marked diminution of the circulation of his left leg was probably produced by an embolus, although the possibility of a coincidental occlusion from a thrombosis cannot be completely ruled out.

Case 13 (O.M. 527530). A 40 year old woman was admitted to the hospital on Dec. 10, 1948.

Seven days prior to admission, she awoke with a dull, aching pain and a sense of pressure over the lower anterior chest. The symptoms gradually subsided in 2 or 3 hours. She went to work that day and complained of some light-headedness. On the next day the patient awoke with squeezing lower anterior chest pain that was relieved with an injection, but she noted the persistence of a dull, aching sensation the rest of the day. Two days later she developed severe shooting pain in the right flank, spreading anteriorly to the groin, associated with desire to urinate, nausea and vomiting. The pain lasted five hours. Over the next few days she complained of difficulty in urinating.

The family history disclosed that her mother had been diabetic. She was also a suspected diabetic 17 years prior to admission, but since then she had not shown evidence of diabetes. Her weight was about 190 pounds.

On admission she was found to have a blood pres-
sure of 142/88. The pulse rate was 84 per minute, and the temperature was 99.5 F. The heart was in normal sinus rhythm, with sounds of fair quality; no murmurs were heard. There was some tenderness in the right costovertebral angle. An x-ray film of the chest was within normal limits. The electrocardiogram showed evolutionary changes of a myocardial infarction of the anterior wall. A urinalysis revealed 2+ albumin and 2+ glucose. No red cells were demonstrated.

In view of the possible renal infarction, she was started on dicumarol, and was finally discharged on the thirty-fifth hospital day.

This patient was a suspected diabetic many years prior to admission. Nevertheless, the diagnosis was not proved, although it is possible that she has a latent diabetes. The history clearly reveals that she had a mild myocardial infarction that was followed two days later by probable embolization to her right kidney, with fairly severe manifestations.

Case 14* H.G. A 60 year old lawyer, 6 feet 2 inches tall and weighing 235½ pounds. For many years he had physical examinations at frequent intervals. He enjoyed excellent health until the present illness. In recent years he had followed a low cholesterol diet.

In March 1955, he experienced shortness of breath on mild exertion, such as walking from his house to his car. He also developed pain in his chest, especially on deep respiration. He sought medical advice and was told that he had virus pneumonia. The symptoms increased. He went to bed and developed a slight fever. After a week in bed he noticed pain in his left calf. He was then told that he had phlebitis and was ordered to stay in bed with hot packs and with the foot of his bed elevated. Suddenly, on April 8, the right leg became cold, white, and numb. The left leg also became cold, but less so than the right. Dr. Foley saw him in consultation at the Lawrence Hospital in Bronxville, New York.

On examination it was found that he was pulseless below the waist. The large and small left superficial saphenous systems were involved in a phlebitic process. It was thought that the patient had suffered a saddle embolus, and that the probable origin of the embolus was from the heart. An electrocardiogram showed an extensive anterolateral infarction. The patient was placed on an oscillating bed and anticoagulants were started. His course was very rough.

Several days later he developed atrial fibrillation. This responded to digitalis therapy. After 3 weeks in bed he was ambulated. From then on his course was excellent. Collateral circulation developed in the legs. The femoral pulse returned to the left groin, but remained absent on the right side. His walking distance had gradually increased, and at present he is able to play nine holes of golf.

When last examined on September 5, 1955, his oscillometric readings were as follows:

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<th>Location</th>
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<th>Left</th>
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In retrospect, it appeared that this patient had developed a mild myocardial infarction as the initial phase of his illness. While resting in bed he developed phlebitis and, later, a saddle embolus, which occurred secondary to a mural thrombus in the left side of his heart. The infarction gave such mild symptoms that it was not recognized nor suspected by the physicians attending him.

**Discussion**

From these case presentations it is clear that all these patients could have been classified initially as "good risk" cases, or mild forms of myocardial infarction. Complete absence of the classical signs and symptoms was characteristic of two cases (7 and 11); in 2 others (1 and 5), the only complaints that could indicate the development of the infarction were represented by mild fatigue and by slight precordial pressure on exertion. Some of the patients (cases 2 and 12) developed symptoms of such a mild nature that their cardiac origin was not recognized at the beginning, and the patients were treated for a while with diathermy and sedatives. In a few, the pain was of moderate or severe intensity, but subsided promptly, either spontaneously or by the effect of morphine. In one case (9), although the pain receded, intermittent precordial discomfort persisted for several hours, and stressed the difficulty of deciding whether symptoms of this sort should be considered as "intractable pain," since there is confusion regarding a proper definition of the term. The importance of this is obvious if one wishes to classify a patient as a "good" or "poor risk" case.

The heart showed some enlargement that we did not consider significant in 4 of our 14 patients. Here again, we have no precise definition of what should be considered "significant enlargement of the heart," one of the points mentioned by Russek in his criteria for classification of patients in "poor" and "good risk" groups.

These 14 so-called "good risk" cases of myo-

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* From the Lawrence Hospital, Bronxville, N. Y. Introduced through the courtesy of Dr. William T. Foley.
cardiac infarction developed a total of 18 certain and 4 probable thromboembolic complications. Of four patients who sustained silent infarctions, or attacks of such mildness that the symptoms were unrecognized as being of cardiac origin, two (cases 1 and 5) developed thromboembolic complications as the presenting symptom of the clinical picture, and both of them suffered an amputation of a limb.

There were 4 major amputations in 3 patients (cases 1, 4 and 5), one of whom (case 4) finally died after a series of thromboembolic phenomena. One minor amputation was performed in another person (case 2), in whom anticoagulants were used inadequately and for too short a period of time. In this patient, the embolism occurred when she was taken off anticoagulants and her prothrombin time had virtually returned to normal. Six patients (cases 3, 7, 8, 10, 12, and 14) developed arterial insufficiency of the lower extremities as a consequence of embolization to the terminal aorta or peripheral vessels.

Although thromboembolic complications are not frequent in the majority of "good risk" cases of myocardial infarction, they occur occasionally and may lead to disastrous results that can even be fatal, as illustrated by case 4.

As can be seen from the case histories of those patients (cases 7, 8, 9, and 10) who were admitted to the hospital shortly after the myocardial infarction had occurred and before the onset of thromboembolic complications, the absence of serious signs during the first 48 hours of hospitalization did not exclude them from developing thromboembolic complications.

In the past, the question of the risk of hemorrhagic complications from the use of anticoagulants has quite justifiably given rise to concern. This risk has steadily decreased as physicians and technicians have become more familiar with this form of therapy. Severe hemorrhage and rupture of the myocardium have been much more common in serious cases of myocardial infarction. Both these complications have been very rare in mild cases. An important added factor of safety has been the increasing use of Vitamin K₁, which acts within a few hours when administered either orally or parenterally. Ten to 50 mg. of this substance will control most elevated prothrombin-time levels, and the dose may be repeated if necessary. While treatment must be meticulous, the risk of hemorrhage no longer constitutes a serious deterrent, if the indications for anticoagulant therapy are clear.

**Conclusions**

This and other experience leads us to conclude that serious thromboembolic complications occur in initially mild cases, and that the prognosis in myocardial infarction can never be predicted with certainty during the first 48 hours of hospitalization. It has been demonstrated that anticoagulants markedly reduce the incidence of this type of complication, and we hold that if no contraindications are present, patients of this type should be given the benefit of anticoagulant therapy. It does not appear justified to wait until thromboembolic complications occur, which may be serious, as these cases illustrate, before beginning the administration of anticoagulant therapy.

**Summario in Interlingua**

Complicationes thrombo-embolic occurrentemente post infarcimento myocardial. Le uso de therapia anticoagulante in infarcimento myocardial es generalmente acceptate, sed certe autores ha recommendate abstention ab iste therapia in "leve casos" excepte quando illos disveloppa complicaciones thrombo-embolic. Le presente autores opina que isto non es justificate, providite que il non ha altere contraindicaciones a providite que le requirite facilitates es disponibile. Es presentate summarios de 14 casos "a prospectos favorable" de infarcimento myocardial in que il habeva 18 certe e 4 probable complicaciones thromboembolic. Ie resultato eseva le necessitate de 4 major amputations in 3 patientes. Un de illos moriva. Altere complicaciones es discutite in detalio.

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