Acute Atypical Coronary Artery Insufficiency
Incidence and Clinical Course

By David Littmann, M. D. and J. H. Barr, Jr., M. D.

Not all patients with acute coronary thrombosis or inadequacy develop classic myocardial infarction. Almost 30 per cent of such patients were found to have atypical and apparently less extensive myocardial injury as estimated by the clinical and electrocardiographic findings. The morbidity as well as the mortality was of a lesser order and a somewhat different therapeutic regimen could be employed.

A CUTE atypical coronary disease is a clinical term applied in this communication to patients with manifestations of coronary insufficiency which are more severe than angina pectoris but which fail to satisfy the criteria for classical myocardial infarction. Included are cases of coronary failure, subendocardial necrosis, and small or obscure myocardial infarctions not demonstrable by present methods.

It is the object of this study to report the incidence of these conditions and to compare their clinical courses, complications and mortality with frank myocardial infarctions.

Material

The material consists of 207 consecutive patients admitted between July 1946 and June 1950 to the West Roxbury Veterans Administration Hospital† for symptoms arising out of acute coronary inadequacy. Cases were excluded in whom the acute attack occurred more than seven days before admission.

Classification of Material

Typical Myocardial Infarction. In order that it might be clearly separated from other forms of coronary disease, criteria were adopted for the diagnosis of unequivocal myocardial infarction.1 These included a history of protracted pain unaffected by effort or nitrites, elevations of temperature, elevated white blood count and sedimentation rate, and characteristic electrocardiographic stigmas. Simple T-wave inversion with or without S-T–segment depression was not considered certain evidence of myocardial infarction. Though isolated cases showing such changes may go on to infarction at a later date, the future evolution is ordinarily unpredictable. These cases were not, therefore, designated as myocardial infarction but were otherwise classified.

Since the concepts of coronary failure and of subendocardial necrosis without coronary occlusion are still controversial and possibly obscure, these will be briefly considered.

Coronary Failure. This term was applied by Blumgart and his associates2–4 to the clinical syndrome of acute coronary disease which is more than angina pectoris but less than infarction. They employed the word failure in much the same sense as in the terms cardiac failure or bank failure, implying inability to fulfill obligations completely or perform necessary functions adequately. This occurs in situations arising from disproportionate and unsatisfied demands upon an inelastic coronary system resulting from increased metabolic requirements, noxious influences or further coronary narrowing, occasionally occlusion. The actual process is considered to be prolonged but reversible myocardial ischemia. Myocardial infarction does not occur though this may follow at a later date. There is little or no fever, leukocytosis or accelerated sedimentation. The pain is more lasting and severe than angina pectoris and is unrelieved by rest.
or nitroglycerin. The blood pressure is maintained and no friction rubs develop. Electrocardiographic changes are said to be either absent, or slight and transient, similar to those seen in angina.

Only two patients of the 207 studied fulfilled these criteria for the diagnosis of coronary failure. There is reason to believe, however, that the limits of this condition, particularly those related to the electrocardiographic changes, may be more flexible than indicated. We have included in this classification patients with T-wave alterations of considerable duration which cleared during the period of hospitalization, ordinarily less than six weeks.

Subendocardial Necrosis (Infarction) without Coronary Occlusion. According to Master and his associates, this condition occurs commonly in individuals with sclerotic coronary vessels and exceptionally when these are normal. Any of a number of stimuli including emotion, hemorrhage, shock, sustained effort, tachycardia, toxic substances or further coronary narrowing leads to a state of myocardial hypoxia. If not promptly relieved this goes on to persistent cardiac pain and eventual necrosis of heart muscle. Destructive changes occur in papillary muscles and in the central myocardium. This localization is related to the character of the coronary vessels and to the ventricular pressure gradient. These normal phenomena are believed to reduce the blood flow to the deeper layers of the heart which are consequently rendered more liable to injury. The resultant subendocardial injury is diffuse or focal. The endocardial lining is spared, however, and no mural thrombosis occurs. The outer myocardium remains uninvolved and pericardial phenomena do not develop. The small total mass of necrotic muscle causes little or no fever, leukocytosis or increase in red cell sedimentation rate. There is no shock; the blood pressure either remains unchanged or rises while pain is present. The electrocardiographic changes are said to be diagnostic; they consist of S-T-segment depressions with or without T-wave inversions. The former, resulting from injury to the myocardium nearest the endocardium, is the exact opposite of the S-T elevations seen with pericarditis. No QRS changes are seen.

Only those patients were included in this category who demonstrated the characteristic ST-T changes and who had relatively little or no fever or leukocytosis.

Although the terms coronary failure and subendocardial necrosis are intended by their proponents to designate the whole middle ground between angina pectoris and frank infarction, examples of both types are encountered at autopsy. Additionally, as suggested earlier, there may be a third subdivision which cannot be dignified with the diagnosis of classic myocardial infarction. This group, too, demonstrates slight fever, leukocytosis and accelerated sedimentation and shows no evidence of heart failure or peripheral shock. The pain lasts longer than 15 minutes and makes its initial appearance without relation to physical effort. The electrocardiogram tends to run a curious course. Often it is normal on admission, then, slowly over a period of days or weeks, when the patient is asymptomatic, symmetric T-wave inversions occur which persist indefinitely thereafter. Occasionally the record shows T-wave inversions initially which remain without significant change. There are no S-T-segment deviations. These patients are believed to have experienced small and obscurely located myocardial infarctions which are impossible to demonstrate. It might appear that no useful purpose is served by separating these from cases of frank, classic infarction. However, the uncertainty regarding the pathologic process together with the wide divergence of prognosis, clinical course and complications merits this division. For want of a more exact term these cases will be designated atypical myocardial infarction.

* The subendocardial myocardium is considered to have the poorest blood supply because it is nourished by the smallest coronary branches which are farthest from the source and have the least blood pressure. During systole the cavity pressure is identical with or somewhat greater than that in the aorta. This pressure diminishes in the middle layers of the heart and falls to zero at the epicardium. Accordingly, little or no blood flow goes on in the deeper layers of heart muscle during systole. It is restricted in the midzone but is uninhibited at the surface.
Results

The mortality for the entire group of 207 cases was 18.3 per cent. Excluding seven patients who failed to survive the first 24 hours, 15.5 per cent died during the three month period of observation.

Classic Myocardial Infarction. These patients were observed at the same time as the atypical forms and are presented only for purposes of statistical comparison. There were 148 patients in this category. The diagnosis was made when the clinical pattern coincided with the description given earlier. It was not made in the absence of significant Q waves in the electrocardiogram although at times these were difficult to demonstrate.

The mean age was 53 and the range 29 to 80 years. The mortality was 22.4 per cent. It was 19.7 per cent for the 142 patients who survived longer than 24 hours. There were approximately as many posterior as anterior wall infarctions with no significant difference between them in the rate of recovery. First infarctions fared much better than second or third. The mortality in the former was 10.7 per cent, in the latter 51.1 per cent. There were 11 patients in the fourth decade in whom the mortality was 18.1 per cent. In the fifth decade (34 cases) the death rate was 8.8 per cent. Except for this reversal the mortality increased progressively with the age.

The diagnosis of pulmonary infarction was made on 10 patients and suspected in one other. In two this complication was considered to be an important though not the sole cause of death. Both patients had multiple myocardial infarctions and were in congestive failure. Pulmonary infarction was thought to have contributed to death in a questionable but probably minor degree in two other patients. This diagnosis was made in six patients who survived. There were three instances of thrombophlebitis without pulmonary embolization. Except for one group of 25 consecutive patients who formed the basis for another study, most of the patients received prophylactic anticoagulants. In retrospect, however, it appears that in many instances, particularly during 1946 and 1947, inadequate prothrombin suppression was obtained. Despite this, the incidence of thromboembolism was low. In the group of 25 who received no anticoagulants there was one case of thrombophlebitis and none of pulmonary embolism.

Acute, Atypical Coronary Disease

There were 59 patients (28.5 per cent of the total) who failed to satisfy the criteria for classic myocardial infarction. Because of close similarities and overlapping criteria of the component members, considerable difficulty and probable inaccuracies were encountered in their classification. Accordingly, it was felt that the atypical cases could well be considered in a single group as well as separately.

The greatest divergence of the atypical cases from patients with classic infarction was found in the death rate. Only four of the 59 or 6.8 per cent failed to recover. Discounting one patient who was moribund on entry and lived only a few hours, the mortality was 5.1 per cent. One patient who was desperately ill died on the second hospital day. Two other patients died unexpectedly. All of the deaths occurred within 48 hours of admission. Of the surviving 55 cases 11 proceeded to develop frank myocardial infarction following periods of three to 27 days after admission. Although it might be contended that this represented typical infarction from the start, it is considered improbable in view of the development of fresh symptoms in some of these patients and of the completely benign course in all.

Coronary Failure. There were 29 cases in this category. The ages ranged from 27 to 70 years with a mean of 46.4. This was significantly lower than for any other subdivision of acute coronary disease. With one exception, all the patients were less than 60 years of age while one half were under 50. The onset of symptoms occurred during activity in 21 instances, while in eight this took place during rest or sleep. There was a history of previous angina in 15 patients and of myocardial infarction in four. Eleven were experiencing their initial symptoms of acute coronary insufficiency. There were two deaths, a mortality of 6.9 per cent. There were no cases of thromboembolic disease.

With the exception of the two patients who
died, most members of this group were asymptomatic when first seen and remained so thereafter. This was true even when admission took place within two to three hours of the onset. Exceptionally, fleeting anginal twinges were noted during the period of observation. In two patients, however, troublesome angina continued for as long as three weeks. Neither cardiac failure nor peripheral shock occurred in any patient and friction rubs were not heard.

Fever over 99 F. occurred in nine patients; in two it exceeded 100 F. A temperature of 102 F. was seen in one patient who went on to develop classical myocardial infarction and 101.4 F. in another with pneumonitis. The remaining 20 cases were afebrile.

The white cell count varied considerably and appeared unrelated to other signs of illness. Counts were obtained from 5,000 to 17,000 and averaged 9,600. The erythrocyte sedimentation rate was even more variable and totally unpredictable. The electrocardiograms showed striking variations but all became normal in from one day to six weeks.

The two deaths are not easily explained, even in the one patient who was examined post mortem. They must be ascribed to acute arrhythmia or cardiac standstill. Both patients appeared relatively well but died suddenly and without warning. These deaths support the impression that though commonly benign and reversible, this syndrome is potentially hazardous. It may, of course, be purely fortuitous that both patients died at about the same time (within 48 hours of the onset). However, it is likely that such patients are most vulnerable during the first few days of the illness. It is, perhaps, well to assume that coronary occlusion has taken place. The muscle formerly supplied by the now useless vessel may not be infarcted but remains in a delicate, irritable and easily injured state for days or weeks thereafter, or until nutrition has improved through collateral channels. Accordingly, as it did in one patient, actual infarction can occur when the imbalance between coronary supply and cardiac demands is further exaggerated. In other instances this discrepancy may result in disturbances of rhythm which are incompatible with life.

Every effort is directed to keeping the heart at rest and lowering the demands upon it. This should include total bed rest with avoidance of straining or sudden, vigorous movements. Moderate sedation is indicated. The value of oxygen and of prophylactic quinidine is problematic but might, perhaps, be employed on theoretic grounds. There seems to be no place in the treatment of this syndrome for the use of anticoagulants. After 7 to 10 days greater freedom is allowed, but vigorous ambulation is delayed until the electrocardiogram has returned to normal. Use of the electrocardiogram as an index for physical activity is a moot point but appears justified where the patient is asymptomatic, afebrile and has no leukocytosis. Total ambulation is commonly permitted in from two to four weeks, generally in three weeks.

The following case reports illustrate patients of this group.

Case 1. A 38 year old electrician was admitted with a 10 day history of angina pectoris. The pain was first noted while he was engaged in moving his shop to a different location. It promptly subsided with rest and returned inconstantly during effort. On the morning of entry he noted a 20 minute episode of retrosternal squeezing pain which did not result from exertion.

Physical Examination. The patient was well developed and nourished, lying quietly in bed and having no discomfort. The examination was entirely normal. The pulse was 88 and the blood pressure 128/82. There was no fever.

Laboratory Findings. The admission white blood count was 19,000. This was thought to be in error since a repeat determination 24 hours later was found to be 7,400. The sedimentation rate was 5 mm. per hour. The initial electrocardiogram (fig. 1) showed slight but definite S-T- and T-wave changes. On the following day the tracing had cleared and it remained normal thereafter.

Hospital Course. The patient was kept at bed rest and received no medication. He remained afebrile and asymptomatic during the entire hospital course of 24 days.

He returned for a follow-up examination one month later and reported himself free of symptoms. An exercise tolerance test was performed with caution and this was found to be strongly positive.

Case 2. A 48 year old stationary fireman employed at this hospital was admitted with a two
hour history of squeezing substernal pain which had occurred during exertion. During the four months prior to entry he had been troubled with indigestion which seemed to be related to the effort of shoveling coal immediately after lunch. This was always relieved by rest and did not return upon resumption of work. On the day of admission he noted the customary appearance of postprandial epigastric discomfort which differed from previous attacks in that it did not subside with rest.

Physical Examination. The patient, a large, obese, white man was comfortable on admission and the physical examination was entirely negative. The blood pressure was 120/70, the pulse was 88 and there was no fever.

Laboratory Findings. The only laboratory test performed was a corrected sedimentation rate of 18 mm. per hour. The initial electrocardiogram (fig. 2) showed negative T waves in all the precordial leads. On the following day T1 became inverted additionally. On the day of death T1 was diphasic while the precordial leads had become essentially normal.

Hospital Course. Following entry the patient was put to bed and sedated. He also received oxygen and was started on dicumarol. For the first 30 hours he appeared well and had no complaints. During the afternoon of the second hospital day, while in bed, he had a 15 minute episode of mild, squeezing retrosternal pain. At 3:00 a.m. on the morning of the third hospital day, while smoking a cigarette, he gasped once and died.

Postmortem Examination. The heart weighed 520 Gm. There were two partial occlusions of the left anterior descending artery, one 2 cm. from the origin and the other midway down its course along the anterior surface of the heart. The first was made up of gray, friable tissue which appeared recent. The second was yellow, recanalized, old and tough. There was no myocardial infarction by gross or microscopic examination.

Subendocardial Necrosis. This group consisted of 19 patients and was selected almost wholly on the basis of the electrocardiographic findings. It was surprising, therefore, that other details showed such close agreement. The greatest difference from the other classes was in the age which was significantly greater than the rest. The mean was 60 years with a range of 50 to 76 years. Activity of the patient at the onset of symptoms was found to be an unreliable diagnostic detail and could not be used in differential separation. Fewer than half of the group were active when pain first appeared. Severe anemia appeared to be the precipitating factor in three cases. Previous angina pectoris was present in 15 patients. A history of myocardial infarction was obtained in six. The patients appeared much more
seriously ill than those with coronary failure. Significant pulmonary or peripheral edema was apparent in four cases; varying degrees of shock were noted in six. Four patients had both shock and congestive failure and of these two succumbed, both within 48 hours of the onset. These were the only deaths.

Moderate fever and leukocytosis were seen in 12 patients and rather severe angina decubitus was present in three. Despite this, the mortality of 10.5 per cent was well below the rate in the same age group with classic infarction.* No friction rubs were heard and there were no thromboembolic phenomena.†

Six patients of this group proceeded to develop evidence of frank myocardial infarction during the period of hospitalization following intervals of 3 to 27 days. With one exception this took place without recurrence of symptoms and without S-T segment elevation. In these patients it appears likely that coronary thrombosis was present at the outset together with infarction of the deeper myocardial layers. Eventually this extended to involve the entire thickness. That this may actually be so was indicated by one of the two fatal cases that came to autopsy. Although in this instance widespread subendocardial infarction was observed, a fresh thrombosis of the left circumflex artery could also be identified. In three other cases examined post mortem* extensive subendocardial necrosis was unaccompanied by fresh coronary occlusion though widespread sclerosis and old closures could be seen.

Two cases of this group are of special interest. Both were patients with severe pernicious anemia who were admitted for complaints arising out of acute coronary inadequacy. Though seriously ill and showing evidence of

* Two recent cases not included in this report.
both congestive failure and peripheral shock, both recovered following blood transfusion, antianemic therapy and cardiac supportive measures.

We have come to associate this condition with rather widespread coronary disease in older people. It is regarded with considerable respect but with a survival expectancy which is better than with frank, transmural myocardial infarction. Spreading infarction may be anticipated in every case but cannot be individually predicted. Where this takes place the subsequent course is relatively benign, possibly because it occurs under ideal circumstances in a heart that has been well conditioned. Both fatalities occurred within the first 48 hours and were anticipated at the outset.

No unusual therapeutic measures were employed in this group except for blood transfusion in the anemic cases. Though thromboembolic phenomena were not seen, anticoagulants were employed largely because of the advanced age of most of these patients and the presence of congestive failure. Wherever possible, ambulation was urged as soon as the temperature and blood counts had remained normal for 7 to 10 days.

Except for some lessening of the S-T segment depression the electrocardiogram frequently retained its abnormal configuration. In five patients, however, the tracing reverted completely to normal.

Only one patient of this group is known to have died after leaving the hospital. This occurred within three months following discharge.

Case 3. A 73 year old Spanish War veteran was brought to the hospital in an almost moribund state. One year earlier he began to experience dyspnea and angina of effort. The pain increased in frequency and duration until it was present most of the time. Dyspnea also increased and edema of the legs appeared. There had been loss of an undetermined amount of weight. The patient was unaware of any pallor.

Examination revealed a well developed, pale, chronically and acutely ill old man. There was evidence of congestive failure with distended neck veins, enlarged liver, pulmonary and peripheral edema. The heart was enlarged. The blood pressure was 100/65, the pulse 110 and the temperature 99 F.

The hemoglobin was 4 Gm., white blood cell count 7,800 and the nonprotein nitrogen 85 mg. per cent. The urine contained albumin, granular casts, numerous white and occasional red blood cells. The electrocardiogram showed widespread S-T depressions and T-wave inversions.

Shortly after admission the patient's condition became critical. The pulse rose to 120 while the blood pressure receded to 70/40. Since it was apparent that anemia was an important factor in the total picture, he was transfused with packed red cells, a total of 4 units in the first 48 hours. He also received digitalis and mercurial diuretics. Despite his desperate state the patient responded well to therapy. Following the fourth transfusion evidence of congestive failure waned, pain disappeared and the blood pressure stabilized. Bone marrow studies were consistent with pernicious anemia. The response to the usual antianemic measures was equivocal, however, and the red blood cell count never rose above 3.6 million cells. The greatest hemoglobin level was 10.7 Gm.

Despite the relative absence of symptoms, the electrocardiogram (fig. 3) first showed extensive improvement then went on to further change. Five days after admission the findings were those of an anteroseptal myocardial infarction.

The patient was released on the forty-sixth hospital day, having been asymptomatic for three weeks. There was no evidence of failure and no angina pectoris. He was still without complaints when seen four months later.

Atypical Myocardial Infarction. There were 11 patients in this category with no mortality. It is, perhaps, the most controversial group, with symptoms and findings most nearly resembling those of mild or small myocardial infarctions. The main point of divergence is the electrocardiogram which simulates the findings of inflammatory heart disease more nearly than those of myocardial infarction. Though it would appear that the injury was anteriorly located in all instances, friction rubs were not encountered except in one patient after he developed a frank transmural infarction. It is suggested that these are instances of small, isolated areas of necrosis which cannot be satisfactorily demonstrated but which, in some instances, enlarge to involve the total thickness of the ventricular wall. As indicated earlier, it is well to separate these patients from the remainder because they carry such a...
low incidence of morbidity and, as implied by this series, no mortality.

The average age of 47 years was lower than in classical infarction by 6.3 years. The small number of patients, however, makes the difference questionably significant. The range was 33 to 57 years. There was a history of angina pectoris in four. None had experienced myocardial infarction and seven were having their initial cardiac symptoms. No patient exhibited shock or cardiac failure but 10 of the 11 cases had fever to 99.6 F. The white blood count varied from 7,700 to 19,200 with an average of 12,200. The sedimentation rate was accelerated in every instance.

Two patients in this group had recurrence of symptoms and went on to develop evidence of classical infarction. Electrocardiographic evidence of transmural necrosis occurred in two others who remained asymptomatic. There was no thromboembolic disease despite inadequate prophylaxis in many of these patients. Ambulation was begun early except where extension occurred. Ordinarily such patients were released when the electrocardiogram had stabilized. This occurred in from three to five weeks. In some instances the electrocardiogram remained abnormal and unchanged throughout the period of observation. In two patients gradual and complete return to normal took place within two years.

No one of this group has died or developed evidence of new acute coronary disease in up to four years of observation. One patient has had mild congestive failure during the past two years.

The following cases are considered to be characteristic of the group.

Case 4. A 48 year old letter carrier entered the hospital following a 12 hour episode of intermittent chest pain. While at a movie the day before entry he became dizzy and vomited. A short time later, after a meal, he was seized with a severe, knife-like pain in the left upper chest which persisted until relieved by morphine. On the morning of admission he was awakened by a similar pain which remained until after entry.

Physical Examination. The blood pressure was 105/60, the pulse 70 and the temperature 101 F.

The patient did not appear ill and the examination was negative except for evidence of sinusitis.

Laboratory Findings. The white count was 12,750 initially and 9,250 on the fifth day. On the sixth day the sedimentation rate was 40 mm. in one hour. Four electrocardiograms (fig. 4) obtained during the first four days were all normal. On the fifth day T-wave changes appeared and one week later T1, T2 and T3 were all symmetrically inverted.

Hospital Course. The patient was placed at bed rest and sedated as required. The temperature fell to normal on the fifth day. While there were no further complaints of chest pain the patient continued to have spells of weakness, faintness, nausea and trembling for the first two weeks. His recovery was otherwise uneventful and he was released on the forty-seventh day. At no time was there any friction rub or other evidence of pericardial reaction.

The patient was seen three months after discharge following a minor constricting anterior chest pain. The electrocardiogram at this time had returned almost to normal and he was not readmitted. When seen sporadically over a period of 18 months he continued to complain of chest pain. An extensive functional overlay made it difficult to determine if he was actually experiencing angina pectoris.

Case 9. A 48 year old transit system foreman was hospitalized with a complaint of retrosternal pain of 48 hours duration. For a number of years there had been symptoms of duodenal ulcer. He had otherwise been entirely well until two days prior to admission when he noted the onset of severe, squeezing chest pain while hurrying for a street car. This subsided after 20 minutes. He was able to work but the pain recurred later the same day and on the morning of admission. On each occasion it was precipitated by effort and lasted for one hour.

Physical Examination. The blood pressure was 120/80, pulse 64 and the temperature was normal. The patient was a moderately obese white male in no distress. The examination was entirely normal.

Laboratory Findings. The white blood count was 8,000 and the sedimentation rate 15 mm. per hour. The initial electrocardiogram (fig. 5) was normal except for a low T1.

Hospital Course. The patient was placed at bed rest and on an ulcer regimen. He was given nitrates and sedation as needed. During the first week he complained of occasional retrosternal distress which responded to nitroglycerin. On the second hospital day the electrocardiogram exhibited a negative T1 though the precordial leads were normal. On the seventh day while lying quietly in bed the patient became aware of a retrosternal pain which increased in severity and lasted four hours. It was unrelieved by nitrates. Within 24 hours the white blood count rose to 15,000 and the sedimentation rate to 34 mm. in one hour. Two days later the electrocardiogram showed small Q waves in leads III and aVr and within 10 days the findings of posterolateral infarction were unmistakable. There was no further pain or fever and the remaining 37 days of the hospital course were uneventful.

The patient was seen three months after discharge. There had been a single transient attack of anginal pain shortly after leaving the hospital; otherwise he had remained free of symptoms.

Discussion

Individual consideration of patients and the treatment of each case according to its own merits are prime ingredients of good medical practice. Such methods, however, have not been widely employed in dealing with "acute coronaries." Although there is general agreement that patients with acute coronary disease vary widely, treatment has become formalized and stereotyped. Except in some desperate situations which may call for unorthodox measures, most patients are treated alike. In recent years there has been a growing tendency to earlier amputation of patients with myocardial infarction. Such methods, though probably of value in most instances may, however, be detrimental in some. It appears eminently reasonable that a 65 year old patient experiencing his second myocardial infarction (mortality 51 per cent) should be kept in bed longer than a 45 year old patient having his first cardiac injury (mortality 8.8 per cent). Similarly, the almost universal acceptance of anticoagulants has led to the routine use of potentially hazardous drugs in many patients who statistically would not be expected to have thrombembolic phenomena. Such complications are extraordinarily rare in the relatively young patients who are not debilitated, have no obvious peripheral vascular disease, are not in congestive failure and who are encouraged to move around in bed. Yet, just such people are treated with potent anticoagulants and subjected to daily venipuncture together with the attendant discomfort and expense.

Identification and individualization of coronary types should, therefore, serve an exceedingly useful purpose. Those known to carry a low mortality and a trifling incidence of complications, especially the atypical forms, may be spared the discomfort, cost and even dangers of long-term hospitalization and treatment. Con-
versely, where the findings point to more serious disease, longer bed rest and the protection of anticoagulants should be in order. It is of some interest that the degree of cardiac neurosis with which most "coronary" patients leave the hospital is in direct proportion to the length of confinement.

Separation of the atypical forms from the classic and differentiation between them is made largely on the basis of the electrocardiographic interpretation. Currently, the status of this invaluable tool is paradoxic. Clinicians are well aware of the extraordinary accuracy with which this device demonstrates myocardial disease. Despite this, when the electrocardiogram fails to confirm an "obvious clinical impression" it is often dismissed as just another possibly nonspecific laboratory test. As a result, numerous patients with radiculitis or hiatus hernia have been prevailed upon to spend six weeks in bed for "coronary thrombosis or spasm." None denies the positive electrocardiographic findings in myocardial infarction. It is when the trace fails to confirm that it is discarded. This curious attitude stems from former three- or four-weeks when anteroseptal infarctions were occasionally missed. Such is no longer the case but earlier experiences served to discredit the procedure and this unjustified view has persisted into the present. For such reasons many clinicians are unwilling to make differential separation of the subdivisions of coronary disease by means of the electrocardiogram and be guided in their treatment by this means. Such an attitude is at variance with the statistical results and should be discarded. It is just as unreasonable to regard a case of coronary failure as myocardial infarction as it would be to confound a sprain with a fracture. The surgeon depends on the expertly made roentgenogram for the diagnosis of fracture. The properly made and interpreted electrocardiogram is little, if at all, less specific.

It is suggested, therefore, that when the findings are less than those of frank, classic, transmural myocardial infarction, an attempt should be made to obtain a more accurate and definite diagnosis. The subsequent treatment may then be modified to fit the illness.

Conclusions

1. Analysis was made of 207 consecutive cases of acute coronary artery insufficiency to determine the incidence of atypical forms not characteristically coronary occlusion with myocardial infarction.

2. Fifty-nine cases or 28.5 per cent were separated from the total and considered together as instances of acute atypical coronary artery disease.

3. Further subdivision of this group was accomplished largely on the basis of electrocardiographic differences.

4. There were 29 cases of coronary failure. There were 19 cases of subendocardial necrosis though not necessarily without coronary occlusion. There were 11 cases of atypical (small, obscure) myocardial infarction.

5. The mortality, complications and clinical course of the patients in these subdivisions are tabulated and discussed.

6. Atypical coronary disease is shown to consist of a more benign group of conditions than classic myocardial infarction. Recommendations are made regarding length of hospitalization and therapy.

REFERENCES


Acute Atypical Coronary Artery Insufficiency: Incidence and Clinical Course
DAVID LITTMANN and J. H. BARR, JR.

Circulation. 1952;5:189-200
doi: 10.1161/01.CIR.5.2.189

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1952 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/5/2/189

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/