Chest Pain with Inverted T Waves, Predominantly in Precordial Leads, as the Only Electrocardiographic Abnormality

By Frank B. Cutts, M.D., Frank Merlino, M.D., and Frederic W. Easton, M.D.

Sixty-nine hospitalized patients, who gave a history of "coronary" type chest pain appearing at rest and lasting at least 15 minutes, and whose electrocardiographic changes were limited to deeply inverted T waves predominantly in the precordial leads, were selected from a large series of patients. The hospital course and electrocardiographic changes of these patients were studied. After an average follow-up period of 4 years, approximately half the patients were still able to work. Postmortem studies on 11 patients revealed extensive sclerosis of the coronary arteries in all.

A considerable number of patients without typical acute myocardial infarction have a "coronary" type of chest pain at rest, with only an inversion of the T waves, particularly in the precordial electrocardiograms. In this paper we seek to clarify some of the diagnostic and prognostic features of this group by an analysis of 69 hospitalized patients with this combination of symptoms and electrocardiographic findings.

Patients with this type of chest pain fall into a group that, with rather fuzzy borders, lies intermediate between typical angina pectoris and acute myocardial infarction. This syndrome has been variously labeled as "coronary failure," "acute coronary insufficiency," "intermediate coronary syndrome," and "prodomal symptoms in myocardial infarction." It is well recognized that patients with identical symptoms may demonstrate electrocardiograms clearly different from those with the precordial T-wave inversions described in this paper. There may be QRS abnormalities indicative of some degree of myocardial infarction; there may be a significant degree of S-T deviation, usually depression; and, indeed, the electrocardiogram may be entirely normal. Patients with these last 3 types of electrocardiograms are not considered here.

Methods

All electrocardiograms with multiple precordial leads, taken prior to January 1, 1955, and classified in our files under the headings "acute anterior myocardial infarction" and "abnormal precordial T waves," were scrutinized. During this period the electrocardiographic diagnosis of acute anterior myocardial infarction was made in 784 patients. Tracings with recognizable QRS abnormalities, including bundle-branch block, intraventricular block, and the pattern of marked left ventricular enlargement (abnormally tall R waves in V5 and V6 with maximal T-wave inversions in these same leads) were excluded. From this relatively large group were selected, by consulting the clinical records, only those patients who gave a history of substernal pain occurring at rest and lasting at least 15 minutes or longer. If hypertension was noted clinically, cases were included only if the electrocardiograms demonstrated considerable precordial T-wave inversion that had appeared since a previous record taken within 6 months, or if the T-wave inversions were shown to be transient by subsequent tracings. Patients on digitalis were excluded unless the T-wave inversions were clearly greater than could be reasonably attributed to this medication and were unaccompanied by significant sagging of the S-T intervals. Cases were omitted in which there was clinical evidence of pulmonary emboli, pericarditis, significant electrolyte disturbances, recent paroxysmal tachycardia, or myxedema. No instance of beriberi or the "juvenile pattern" of precordial T-wave abnormality was included.

Within these limitations we were left with 69 cases for analysis.

Results

Clinical Considerations. The pertinent clinical data are summarized in table 1. The ratio of males to females was somewhat less than the usual 3 or 4:1 noted in groups of patients with myocardial infarction. However, there were 14 males and only 1 female among those under 51 years of age; conversely, there were 8 females and only 2 males among those whose age was 71 or more. These figures clearly illustrate the tendency of coronary artery disease to become manifest in men at a younger age but our group...
Table 1.—Clinical Data

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>31-40</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>41-50</td>
<td>9</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>51-60</td>
<td>14</td>
<td>7</td>
<td>21</td>
</tr>
<tr>
<td>61-70</td>
<td>14</td>
<td>9</td>
<td>23</td>
</tr>
<tr>
<td>71-80</td>
<td>2</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>81-90</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>25</td>
<td>69</td>
</tr>
</tbody>
</table>

Number of patients | Per cent of total patients
Pre-existing angina pectoris | 34 | 49
Pre-existing hypertension | 37 | 54
Pre-existing diabetes mellitus | 6 | 9
Time of onset of chest pain
1 day or less | 25 | 36
2-7 days | 22 | 32
over 1 week | 22 | 32
Duration of chest pain
15 minutes | 14 | 20
15-30 minutes | 6 | 9
30-60 minutes | 9 | 13
over 1 hour | 35 | 51
unknown | 5 | 7
Fever (over 100°) | 12 | 17
WBC
over 10,000 | 29 | 43
10,000 or less | 39 | 57
Sed. rate (Wintrobe)
over 20 mm./hr. | 38 | 73
under 20 mm./hr. | 14 | 27

is too small to permit any other firm conclusions.

About half the patients gave a history of pre-existing angina pectoris and an approximately equal number were observed at some time to have had hypertension, usually mild in degree or transient in duration. However, any patient observed at any time to have had a systolic blood pressure over 150 mm. or a diastolic pressure over 90 mm. was included under this heading. The onset of substernal pain appearing at rest occurred about equally 1 day or less, 2 to 7 days, or over 1 week prior to hospital admission. In about half the group the substernal pain was described as lasting over 1 hour. However, it often showed a tendency to wax and wane and seldom lasted over 4 hours. Moreover, the development of shock, gallop rhythm, or a pericardial friction rub was not observed, except with definite myocardial infarction, occurring later.

In 12 patients a temperature over 100 F. was noted during the first few days of hospitalization. Although this fever seldom lasted more than a day or 2 and did not exceed 102 F., it suggested the occurrence of some degree of myocardial necrosis. In all patients except 1, a white blood cell count was obtained and was elevated, rarely over 15,000, in somewhat less than half the group. When determined, the sedimentation rate was found to be elevated more often than not. These elevations of the sedimentation rate and the leukocyte count may indeed have reflected myocardial necrosis in some cases. The increase in the white blood cells was usually minor, however, and it has been demonstrated that angina pectoris alone will elevate the sedimentation rate.

Forty-seven patients received bishydroxycoumarin (Dicumarol) during their hospital stay. However, only 17 of these had a sufficient amount of the drug to constitute reasonably adequate treatment (prothrombin time determinations of 30 per cent or less on at least half the tests performed after the second day of Dicumarol administration). Although our cases are too few to permit any definite conclusions, we could detect no appreciable differences in the clinical course between the groups receiving “adequate,” “inadequate,” or no Dicumarol therapy. We have no data in this series concerning longer term Dicumarol therapy.

Electrocardiograms. In table 2 are summarized some of the electrocardiographic data. There was no evident relationship between the degree of precordial T-wave inversion and the clinical course. In slightly less than half the patients electrocardiograms were taken with sufficient frequency to permit some estimate of the duration of the precordial T-wave inversion. In 18 cases the T-wave had returned to normal by the end of 2 months. Conversely, in none of the 7 patients dying within the first year was a normal electrocardiogram obtained at any time. The last tracing available for each patient was normal in about one third of this series.

Follow-Up. The status of each patient when last seen or heard from is listed in table 3.
Eighteen patients recovered sufficiently to be without symptoms. The cases listed as "active," almost exactly half the total group, were working or able to do at least light work even though some had mild to moderate angina pectoris.

Deaths. During the follow-up period there were 24 deaths, of which 14 (20 per cent of the entire group) were known to be due to heart disease (table 4). Seven of the 14 cardiac deaths occurred during the first year. Thereafter the hazard of death from cancer (5 cases) approximated that from heart disease. In the 14 patients who died of heart disease, the incidence of angina, hypertension, and diabetes, the duration of pain, the degree of T-wave inversion, and the occurrence of fever, leukocytosis, and elevated sedimentation rates were about the same as in the entire series. However, in spite of the relatively high number of older women in the entire group, 11 of the 13 known to have had cardiac deaths were males, further illustrating the comparative vulnerability of men to this disease.

Autopsies. Eleven autopsies were obtained in the 24 patients who died. The most striking finding was the high degree of narrowing and atheromatosis of the coronary arteries (table 5). We had an opportunity to examine personally almost all of these hearts and can corroborate this finding. Three hearts revealed no myocardial lesion grossly, although 2 of these showed small focal areas of fibrosis microscopically. One heart showed streaks of fibrosis widely distributed throughout the midportion of the left ventricular myocardium, 9 years after the patient’s episode of pain. One heart exhibited a small healed posteroseptal infarct. The remaining 6 specimens all showed extensive infarcts, usually acute, that could clearly be related to episodes of prolonged pain occurring after the original episode for which they were included in this study. Of these 6 hearts, one had an old septal infarct, which
# Table 5.—Postmortem Findings

<table>
<thead>
<tr>
<th>Duration of chest pain</th>
<th>Interval between onset of chest pain and death</th>
<th>Heart weight (Gm.)</th>
<th>°Degree of coronary sclerosis</th>
<th>Heart muscle</th>
<th>Noncardiac findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 mins.</td>
<td>3 months</td>
<td>340</td>
<td>X</td>
<td>N+++</td>
<td>Old and recent anterior infarction, old posterior infarction</td>
</tr>
<tr>
<td>Several hours</td>
<td>6 years</td>
<td>360</td>
<td>N+++</td>
<td>N+++</td>
<td>Negative</td>
</tr>
<tr>
<td>Several hours</td>
<td>1 month</td>
<td>390</td>
<td>N+++</td>
<td>N+++</td>
<td>Negative</td>
</tr>
<tr>
<td>3 hours</td>
<td>2 weeks</td>
<td>430</td>
<td>N+++</td>
<td>N+++</td>
<td>Streaks of fibrosis in middle of wall of lt. ventricle</td>
</tr>
<tr>
<td>2 hours</td>
<td>9 years</td>
<td>350</td>
<td>N+++</td>
<td>N+++</td>
<td>Extensive acute infarction and some patchy fibrosis</td>
</tr>
<tr>
<td>1 hour</td>
<td>6 weeks</td>
<td>690</td>
<td>X</td>
<td>N+++</td>
<td>Extensive acute infarction and some patchy fibrosis</td>
</tr>
<tr>
<td>6 hours</td>
<td>5 years</td>
<td>460</td>
<td>N+++</td>
<td>N+++</td>
<td>Extensive acute infarction and some patchy fibrosis</td>
</tr>
<tr>
<td>4-5 hours</td>
<td>3 years</td>
<td>660</td>
<td>X</td>
<td>X</td>
<td>Old posterior myocardial infarction, patches of fibrosis anteriorly</td>
</tr>
<tr>
<td>Several hours</td>
<td>21 months</td>
<td>510</td>
<td>X</td>
<td>X</td>
<td>Recent anterior and old septal infarctions</td>
</tr>
<tr>
<td>3-4 hours</td>
<td>5+ years</td>
<td>400</td>
<td>X</td>
<td>N++</td>
<td>Extensive recent ant. infarction with rupture</td>
</tr>
<tr>
<td>Several hours</td>
<td>2 weeks</td>
<td>490</td>
<td>X</td>
<td>N++</td>
<td>Extensive recent ant. infarction with pericarditis and mural thrombus</td>
</tr>
</tbody>
</table>

* X = lumen occluded, N = lumen narrowed from 10-15 per cent (+) to 75-90 per cent (+++), LAD = left anterior descending coronary artery, LC = left circumflex artery, R = right coronary artery.

may have occurred at the time of the patient’s original chest pain, and another, in addition to the acute infarct, had patchy fibrosis microscopically, which may well have occurred with the initial episode of chest pain. The myocardial lesions were thus rather variable, and the one common denominator was quite clearly the high degree of coronary artery narrowing.

### Illustrative Cases

**Case no. 37.** D. T., a 47-year-old white man, was admitted to the Jane Brown Memorial Hospital on July 10, 1952. Four years previously he had de-
developed typical angina, which improved markedly on a low-fat diet and a subsequent weight loss of 20 pounds. He had no further cardiovascular symptoms until July 9, 1952, when he noted the sudden onset of dull substernal pain radiating into the left arm and lasting some 3 hours.

The temperature was 98.6° F., the pulse 80, and the blood pressure 120/70. The heart was normal in size, sounds, and rhythm. There was no evidence of congestive failure. An electrocardiogram (fig. 1) revealed no QRS or S-T abnormalities. T waves in aVL, however, were inverted, as were the terminal portions of the T waves in leads V₂ through V₅. Laboratory data included a negative urinalysis, sedimentation rate of 10 mm. per hour, white blood count of 10,600 with 74 per cent polymorphonuclear cells, and a blood cholesterol of 193 mg. per cent.

The patient was treated with rest and Dicumarol. After the second hospital day, 7 of 10 prothrombin activity determinations were 30 per cent or less. He remained afebrile and appeared to be recovering satisfactorily until the morning of July 24, 1952, when he awoke at 3:30 complaining of substernal discomfort that lasted about 2 hours and required 2 injections of meperidine for relief. Some 3 hours after the onset of pain, however, he suddenly died.

At necropsy, the heart weighed 430 Gm. Serial sections through the myocardium were negative on gross inspection. The coronary arteries revealed 3 to 4 plus sclerosis with markedly narrowed, but not occluded lumina. Sections through the anterior left ventricle revealed several scattered, irregularly shaped, focal areas that were pale-staining and composed of remnants of myocardial fibers and small amounts of granulation tissue with congested capillaries and small numbers of round cells. These areas were surrounded by myocardial fibers of normal appearance. The coronary vessels revealed large atheromatous plaques that reduced the lumina to 10 to 20 per cent of normal. No subendothelial hemorrhages were seen (figs. 2 and 3).

Case no. 39. J. B., a 64-year-old white man, was admitted to the Rhode Island Hospital on January 3, 1942. Previously, while walking home, he suddenly experienced epigastric and lower substernal oppression accompanied by dyspnea, perspiration, and collapse. His pain lasted 2 to 3 hours.

Physical examination revealed a cyanotic, obese man breathing rapidly with audible wheezing. Temperature was 98°F., pulse 120, respirations 35, blood pressure 158/80. Rales and rhonchi were present bilaterally. The heart was slightly enlarged, with normal sounds. For the first 3 days his temperature reached 101°F. rectally. He made an uneventful recovery and was discharged after 17 days. Electrocardiograms (fig. 4) showed only T-wave changes with a maximum inversion of 17 mm. in lead CF₄ on January 5, 1942. Other laboratory data included a negative urinalysis, a blood glucose of 104 mg. per cent and a blood urea nitrogen of 21 mg. per cent. On the third hospital day, a white blood count was 11,200 with 81 per cent polymorphonuclear cells.

During 1943 the patient suffered 2 minor cerebrovascular accidents. In 1948 he was re-admitted because of polyuria, polydipsia, and weakness of both legs. With control of his newly discovered diabetes he improved. Examination of the heart was negative. The electrocardiogram was now normal (fig. 4).

In 1950 he re-entered the Rhode Island Hospital because of mild hematemesis. X-ray studies showed a duodenal ulcer. On April 30, 1951, he was hospitalized for the last time because of urinary incontinence. Since his previous hospital admission he had been digitalized. He denied angina but claimed some palpitation, exertional dyspnea, and occasional ankle swelling. Heart examination showed a grade III harsh precordial systolic murmur; by x-ray the heart measured 12.9 cm. and the intrathoracic diameter was 25.1 cm. He died of urinary sepsis 5
days after cystoscopy. Electrocardiograms (fig. 4) showed low, but upright T waves in V₄ to V₆.

Autopsy revealed the primary cause of death to be urethral rupture with pelvic abscess formation. The heart was normal grossly except for 4 plus coronary sclerosis and streaks of fibrosis in the mid-wall of the left ventricle. The heart weighed 350 Gm. Microscopically small bands of fibrosis between myocardial fibers were noted.

**DISCUSSION**

Three aspects of the problem presented by the type of patient under consideration here may merit further comment.

**Prognosis.** Some authors³, ¹⁰ have commented on the relative benignity of this syndrome. For example, Barker¹¹ described similar cases and stated that the prognosis is invariably good. However, Levy¹² pointed out the considerable hazard of sudden death in patients with similar symptoms and electrocardiographic findings, and Mounsey⁴ indicated that at times this clinical picture may represent the prodromal phase of acute myocardial infarction. Since 7 of our patients died within 1 year after the onset of this syndrome, we are inclined to be less sanguine than some regarding the short-term prognosis. After the first year the hazard of death from heart disease seems to diminish appreciably.

**Pathologic Considerations.** In an excellent study leavened by the lighter touch, Pruitt and co-workers² considered the clinical and pathologic implications of deeply inverted T waves in the midprecordial leads. They found that patients selected only on the basis of this type of electrocardiogram showed a high incidence of severe coronary insufficiency or myocardial infarction. In their 9 autopsied cases healed subendocardial infarction was found in 8. Although all 11 of our autopsied patients showed advanced narrowing of the coronary arteries, in only 3 instances were grossly recognizable healed infarcts evident in multiple sections of the heart, cut transversely in the plane of the atrioventricular groove. Since many, if not all, of our surviving patients presumably have a similar advanced degree of coronary artery narrowing (3 of our autopsied cases died of causes other than heart disease), one is compelled to marvel at the efficacy of the intercoronary anastomotic circulation¹³ that seems to develop as the subjects recover or improve.

**Anticoagulant Therapy.** Some investigators⁸.
Fig. 3. Case no. 37. Photomicrograph of section from the left ventricle (X 400). The irregular, roughly elliptical, area in the center of the field represents focal necrosis of muscle cells without much loss of residual stroma. Some granulation tissue and small numbers of round cells are present. The surrounding myocardium is normal.

9, 14 recommend the use of anticoagulants in the type of cases considered here; others3, 4 do not believe they are indicated. Our own data are not very helpful on this point. However, since only 2 of our patients died during their original hospitalization and of these 1 was on adequate Dicumarol therapy, the immediate value of anticoagulants in this type of case remains uncertain. Certainly this type of therapy does not appear to lessen the hazard of sudden death nor does it prevent myocardial infarction with any great assurance.15 As indicated in table 4, since the hazard of subsequent myocardial infarction is appreciably greater in the first year after the acute episode of pain, anticoagulant therapy, if undertaken, should probably be prolonged, perhaps following the recommendations of Thompson,8 who continues treatment until the electrocardiogram is normal and angina, if present, is no worse than before the acute episode.

Summary

This study is concerned primarily with the clinical course of 69 hospitalized patients with chest pain at rest and with normal electrocardiograms except for deep inversion of the T waves, particularly in precordial leads.

In a small number of cases there was clinical evidence of a minor degree of myocardial necrosis. More commonly such evidence was lacking. In about one third of the entire group, the electrocardiogram returned to a normal configuration. After an average follow-up period of about 4 years, half the total group were either working or were active in their homes and communities.

There were 24 deaths, of which 14 were
known to be due to heart disease, chiefly myocardial infarction. Seven of the cardiac deaths occurred in the first year of follow-up. Eleven patients came to autopsy. In all instances there was a high degree of atheromatous narrowing of the coronary arteries. Grossly visible myocardial lesions, which could plausibly be correlated with the episodes of pain under consideration here, were usually absent. In a few instances small healed infarcts were noted.

We are rather skeptical of the value of Dicumarol in the type of case under discussion, but our data permit no definite conclusions. If

Fig. 4. Case no. 39. Electrocardiograms: January 5, 1942. Note the normal QRS complexes and the deep inversion of the T waves in the precordial leads. December 22, 1948. The electrocardiogram is now normal. May 2, 1951. Taken 6 days before death, QRS complexes are unchanged and the T waves, although lower, are not beyond normal limits.
used, it should probably be continued for several months.

ACKNOWLEDGMENT

We are indebted to Dr. George F. Meissner, Associate Pathologist, Rhode Island Hospital, for taking the photomicrographs shown in figures 2 and 3, and for assistance in their interpretation.

SUMMARIO IN INTERLINGUA

Iste studio es concernite primarimente con le curso clinic de 69 patientes hospitalisate con dolores thoracice in stato de reposo e con electrocardiogrammamas normal a parte le presentia de un profunde inversion del undas T, particularmente in derivaciones precordial.

Un parve numero de casos exhibiva signos clinic de minor grados de necrosis myocardial. Plus communemente, tal signos eseva absent. In circa un tertio del gruppo, le electrocardiogrammas retornava a configurationes normal. Post un periodo medie de 4 annos de observation consecutivi, un medietate del gruppo total travaliava o eseva active in lor domiciliios e communitates.

Occurreva 24 mortes. Dece-quatro de istos eseva cognoscite mente causate per morbo cardiac, principalmente infartos myocardial. Septe del mortes cardiac occurreva durante le prime anno del observation ulterior. Dece-un patientes eseva necropsiate. In omne iste casos, un alte grado de restriction atheromatose del arterias coronari eseva constatate. Lesiones myocardial de visibilitate macroscopiche que se correlacionava plausiblemente con le episodios de dolor hic considerate eseva usualmente absent. In un basso numero de casos, parve e curate infartos eseva notate.

Nos es satis sceptic quanto al valor de Dicumarol in le typo de caso sub discussion, sed nostre datos non permitte le formulation de conclusiones definite. Si usate, il es probabile que le droga deberea esser continuate durante plure menses.

REFERENCES

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