Serum Glutamic-Oxalacetic Transaminase in Coronary Artery Disease
A Review of 201 Cases

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The results of serial glutamic-oxalacetic transaminase (GOT) assays in 201 cases in which coronary artery disease was suspected are correlated with the clinical, laboratory, and electrocardiographic changes. In 95 per cent of the cases diagnosed as myocardial infarction on the basis of clinical and electrocardiographic evidence there was an elevation of serum GOT. On the other hand, in 12 per cent of the cases in which clinical and electrocardiographic findings were negative or equivocal with regard to myocardial infarction, there was an elevated serum GOT. In 18 cases that were autopsied the correlation between acute myocardial necrosis and serum GOT elevation was 100 per cent.

While the diagnosis of acute myocardial infarction can in most instances be established by a correlation of clinical, laboratory, and electrocardiographic evidence, there is nevertheless a significant number of cases in which an unequivocal diagnosis is not possible. The elevation of serum levels of glutamic-oxalacetic transaminase (GOT) following myocardial infarction, reported first by LaDue, Wrobleski, and Karmen,1 and since amply confirmed in a number of laboratories,2-9 appears to offer a new objective test for establishing this diagnosis.

We have previously found, in cases of transmural myocardial infarction with clear-cut electrocardiographic evidence of myocardial necrosis, that the correlation between the electrocardiographic diagnosis and the elevation of glutamic-oxalacetic transaminase is better than 90 per cent.2-4 The present report describes our results in a series of 201 cases in which coronary artery disease was considered in the differential diagnosis and attempts an evaluation of the clinical application of this new diagnostic tool.

Methods and Material

In this series were included 201 patients with clinical or electrocardiographic evidence suggesting coronary artery disease at the time of hospitalization, although the final diagnosis was not infrequently absolved the coronary arteries. Blood samples were drawn for transaminase assay on at least 5 successive days after the onset of symptoms. In most patients serial electrocardiograms, erythrocyte sedimentation rates, and white blood cell counts were determined daily and rectal temperatures recorded every 4 hours during the first few days. All patients were seen and in most cases observed closely by 1 of the authors during their hospital stay.

At the conclusion of the study each clinical record was reviewed without knowledge of the transaminase levels. All cases were listed as positive or negative for acute myocardial infarction on the basis of the serial electrocardiograms, the history, white blood cell counts, erythrocyte sedimentation rates, body temperature, and the clinical course, with particular reference to the development of shock, left heart failure, and paroxysmal arrhythmias. Each case was assigned to 1 of the following groups on the basis of the electrocardiogram:

Group I. Acute Transmural Myocardial Infarction. Characterized by the development of Q-waves of 0.04 sec. or greater duration, with reciprocal ST-segment shifts and T-wave inversions.

Group II. Normal Electrocardiogram.

Group III. ST-T Changes. This group included

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myocardial ischemia, left ventricular hypertrophy and strain, digitalis effect, and subendocardial ischemia, defined as 2 mm. or more J-junction depression in the precordial leads.

Group IV. Masking Electrocardiograms. Due to bundle-branch block, previous infarction, Wolff-Parkinson-White syndrome, or pericarditis.

Serum GOT was determined by a modification of the method of Karmen, Wroblewski, and LaDue\textsuperscript{10} reported in detail elsewhere.\textsuperscript{11} Results are expressed as units per ml. of serum. The unit is defined as that amount of enzyme that causes optical density, at a wavelength of 340 m\( \mu \), to fall at the rate of 0.001 O.D. U./min. under the conditions of the assay at 25 C., with an effective light path of 1 cm. The Beckman DU spectrophotometer was used for the greater part of this study. However, the Bausch and Lomb Spectronic 20 Colorimeter, specially modified for use in this assay, was also found to be a satisfactory instrument. Analysis of the same sample on the 2 instruments checked within \( \pm 5 \) per cent. For routine use, especially when a controlled temperature bath is employed, the latter instrument is more convenient but somewhat less precise.\textsuperscript{11} Normal values ranged from 10 to 33 U./ml. serum.\textsuperscript{2} However, myocardial necrosis was not considered unless the transaminase value was 48 U./ml. or more.

Results

Group I. Acute Transmural Myocardial Infarction

Sixty patients showed the typical electrocardiographic changes of transmural myocardial infarction. The transaminase results in these cases are presented in some detail, since the interpretation of the data in the less well defined cases depends to a large extent upon the results noted in this group.

Characteristically, there was a sharp rise in serum GOT within the first 24 hours after onset of symptoms. Peak values, ranging from 50 to 375 U./ml., were reached on the average within 26 hours, with a range of 2 to 96 hours. The return to normal was equally characteristic but not so rapid, normal values being reached in most cases by the sixth day and in all cases by the eighth day from onset. Figure 1 shows the mean and the extreme GOT levels as a function of the number of days following infarction. The mean peak was 164 U./ml. While this value in women (206 U./ml.) was somewhat higher than that in men (145 U./ml.) the difference was not statistically significant (\( p > 5.0 \) per cent). Even though the elevations of enzyme persisted for 4 to 6 days in most cases, they returned to normal in some cases as early as the second or third postinfarction day. Transitory elevations of this type were more common in the group III cases discussed below.

Fifty-eight of the 60 cases in group I, all considered to have suffered myocardial infarction by our clinical criteria, showed elevation of the serum GOT, a positive correlation of 97 per cent. The following case is illustrative of the group.

H.S. (fig. 2), a 52-year-old man, without previous history of cardiovascular disease, was admitted to the hospital on April 13, 1955, following acute onset of substernal oppression and shortness of breath, which awakened him from sleep and continued until relieved by opiates. On admission the pulse was 88,
there were occasional premature ventricular contractions and gallop rhythm, and the blood pressure was 115/80. A few moist rales were heard at the left base and there was 1+ pretibial pitting edema. The patient was digitalized soon after admission. On the fourth hospital day the blood pressure dropped to 92/60 and vasopressor drugs were administered. The subsequent course was uncomplicated and the patient was discharged on the thirty-first hospital day.

As shown in figure 2, the serum GOT obtained 2 hours after onset was normal, but rose by the twenty-first hour to 375 U./ml. Normal values were restored by the fifth post-infarction day.

In table 1 an attempt has been made to relate the GOT results to changes in white blood cell count, erythrocyte sedimentation rate, and body temperature in all patients of group I. It will be noted that the elevation of GOT, in addition to its greater specificity, is generally more striking and hence less equivocal than changes in the other parameters. The peak in serum GOT level was usually reached well before the peak of the white blood cell count or erythrocyte sedimentation rate. It is of interest that carefully recorded body temperatures are shown to be extremely valuable, especially when the differential diagnosis lies between infarction and angina pectoris. The peak of the rectal temperature curve coincides most closely with the peak GOT.

Extension of a known infarct, while often inferred on purely clinical grounds, is frequently difficult or impossible to prove electrocardiographically. There were 5 cases of suspected extension in this group, 3 of which had no further electrocardiographic changes. All 5 showed a clear-cut secondary rise in GOT superimposed upon the usual curve. The following case is typical.

J.E. (fig. 3), a 54-year-old man with known hypertension of 5 years’ duration, was admitted on December 3, 1954, because of dull, aching pain along the inner aspects of both arms of 18 hours’ duration. The admission electrocardiogram showed a recent posterior infarct, and the initial GOT was markedly elevated. On the third hospital day the blood pressure fell from 160/110 to 92/68 and intravenous pressor drugs were started. A pericardial friction rub was heard from the third to fifth hospital days. On the fifth hospital day the patient became suddenly disoriented and combative, a reaction attributed at the time to opiates. Bibasilar rales were heard for the first time. No unusual changes in the evolving electrocardiographic pattern were noted. However, on the sixth day the serum GOT, which had returned almost to normal levels, rose dramatically to 270 U./ml. The remainder of the hospital course was uneventful.

The degree of elevation of GOT in group I of this series has not been useful in prognosis or as a basis for estimating the size of the infarct. The mean peak in the 12 cases coming to autopsy was 195 U./ml., only slightly higher than the over-all mean peak of 164 U./ml. The highest level observed in 3 cases with infarcts measuring less than 2 cm. in diameter was 191 U./ml., while in 9 cases with infarcts measuring over 2 cm. in diameter, it was 195 U./ml.
Group II. Normal Electrocardiograms

Eighteen cases showed no electrocardiographic abnormalities during their course. Six of the 18 were believed on clinical review to be instances of coronary artery disease with angina, in the absence of infarction. In 4 of the 6 cases, the GOT was normal but the remaining 2 had clear-cut elevations and a curve not unlike the group I cases. A summary of 1 of these cases follows.

A.M., a 33-year-old obese man, suffered 3 bouts of squeezing substernal pain just prior to admission, each lasting 10 min. and associated with perspiration, dizziness, and vomiting. Temperature, pulse, and blood pressure were normal and stable throughout the hospital course. The heart and lungs were normal. X-ray pictures of the chest and upper gastrointestinal tract were negative. There was no elevation of white blood cell count or erythrocyte sedimentation rate. The GOT, however, was 50, 80, 30, and 22 U./ml. on the first 4 hospital days. The electrocardiogram remained normal. No convincing diagnosis could be attached to this clinical episode.

Two additional cases in group II showed increased GOT levels. In 1, the final diagnosis was acute cholecystitis with lithiasis. There was no evidence of acute hepatocellular involvement. Although it is possible that the rise in enzyme was due to the gallbladder disease, it should be noted that this was the only elevation observed in 8 cases studied with acute cholecystitis, the majority of which also had cholelithiasis. The second patient, with a discharge diagnosis of postural hypotension, was admitted because of recurrent attacks of syncope without associated symptoms. There was an isolated GOT of 49 U. on the fifth hospital day.

In 14 of the 18 cases with normal electrocardiograms the GOT was also normal. Thus, in 78 per cent of the cases, the clinical appraisal and the GOT results agreed in ruling against infarction.

Group III. ST-T Changes

Eighty-two patients had electrocardiographic changes involving the S-T segment or T wave, or both. The electrocardiograms were interpreted as showing either myocardial ischemia, left ventricular hypertrophy and strain, digitalis effect, subendocardial ischemia, or combinations of these. In no instance was the electrocardiogram sufficiently characteristic to justify the interpretation of myocardial infarction.

In 26 of the 82 cases the clinical and electrocardiographic data supported the diagnosis of infarction; in 21 of these 26 the GOT was elevated. Two representative cases are summarized below.

M.H. (fig. 4), a 64-year-old woman with a history of occasional angina pectoris during the previous year, was admitted because of a severe episode of squeezing anterior chest pain lasting 1 hour. During the week prior to admission there had been more frequent bouts of angina. On admission the blood pressure was 135/85, the pulse 80 and regular. Fine moist rales were present at both lung bases. The electrocardiogram, which was normal a year before the present illness, showed ST-T changes of ischemia in leads I, aVL, and V4.

On admission the serum GOT was 90/ml., returning to normal by the third day. On the fourth hospital day the blood pressure dropped to 106/50. The erythrocyte sedimentation rate was 46 mm./hour on the fifth hospital day. Six months later the electrocardiogram showed a marked reduction in the height of the R-wave in V4.

H.W., a 56-year-old woman with a history of hypertension, frequent anginal episodes, and left heart failure, was admitted because of 3 days of persistent precordial and left arm pain. The admission blood pressure was 180/110. The erythrocyte sedimentation rate was 23 mm./hour, rising to 34 mm./hour during the first week. On the fourth hospital day pulmonary edema developed, requiring digital-
transaminase. By the second week the blood pressure had fallen to 100/70. The temperature remained normal.

The electrocardiogram showed no abnormalities during the first 2 weeks. However, in the third week T-wave inversion appeared and progressed into deeply coved and inverted T waves in the anterior precordial leads. At admission the GOT was strikingly elevated, 250 U./ml., and returned to normal on the third hospital day. In this case, as in the previous 1, the early return of GOT to normal suggests that infarction may have occurred 1 or 2 days prior to admission (fig. 1). It should be noted that the first evidence of infarction appeared 19 days after clinical and GOT indications of this infarction.

The entire curve was available for mathematical treatment in 14 of the 21 group III cases with elevated GOT. The average curve showed a peak rise to 102 U./ml. at 30 hours and returned to normal at 64 hours. It is apparent that the patients with only ST and T changes had a lower and more transient elevation in comparison with the group I cases.

The GOT was normal in 5 of the 26 cases that were considered positive for myocardial infarction by the clinical and electrocardiographic appraisal. In 2 of the 5 cases so diagnosed there is no explanation for the discrepancy. However, 3 of the 5 patients came to autopsy and in none was there pathologic evidence of myocardial infarction. The data concerning 1 of these patients are included in some detail.

K.M. (fig. 5), a 59-year-old woman with known hypertension of 5 years' duration, was admitted on December 2, 1954, because of malignant esophageal obstruction requiring surgical relief. The patient was receiving antihypertensive therapy with reserpine at the time of admission. During induction of anesthesia severe shock developed with unobtainable blood pressure. With intravenous pressor medication recovery ensued but surgery was postponed. During this episode the electrocardiogram showed deeply coved and inverted T waves in leads I, II, aVL, and all precordial leads. These changes persisted for 19 days, showing slight evolution toward normality. At no time was the serum GOT elevated.

On the nineteenth day an esophagectomy and esophagogastronomy were successfully performed. During surgery all T waves became upright once again. The postoperative course was stormy, the patient developing an esophago-bronchopleurocutaneous fistula. On the twenty-first postoperative day inverted T waves appeared in V₁–V₄ and became upright 15 days later. The patient was discharged on the thirty-seventh postoperative day. Five months later she was admitted to the Clinical Center, National Institutes of Health, because of progression of the malignancy and died on July 13, 1955. Autopsy was performed by Dr. Louis Thomas and the following details were taken from his report:

"The heart weighed 340 Gm. There was slight left ventricular hypertrophy and marked dilatation of the right atrium and ventricle. This acute cor pulmonale was caused by thrombotic and tumorous occlusion of many small pulmonary vessels. . . . The coronary arteries were carefully examined and found to have thin, collapsed walls with empty lumens. No atheromatous plaques were seen. The myocardium was reddish-brown and firm. No gross areas of fibrosis or hemorrhage were seen.

"Microscopically . . . the small intramuscular branches of the coronary arteries were slightly thickened due to perivascular fibrosis. None of these small vessels was occluded by thrombi. . . . No areas of hemorrhage or acute necrosis of cardiac muscle were seen. One microscopic area of fibrosis in the myocardium was seen. This zone of scarring was approximately 2 mm. in greatest diameter and around its periphery were a few hemosiderinophages. No acutely necrotic or degenerated myocardium was seen around this old microscopic focus of myocardial necrosis."

The single minute area of fibrosis was considered inadequate to explain the acute hypotension and extensive electrocardiographic changes recorded. It is more likely that the peripheral collapse was due to the anesthesia and reserpine. Coakley, Alpert, and Boling have observed circulatory collapse during anesthesia in patients receiving reserpine.¹²

The clinical and electrocardiographic appraisal was considered to be negative for

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**Fig. 5.** Results in a patient developing severe myocardial ischemia during induction of anesthesia without elevation of serum GOT (group III).
acute myocardial infarction in 56 of the 82 cases in group III. The GOT was normal and in agreement in 45 of the 56 cases. Of the 11 cases with elevated GOT, 6 were found to have noncardiac causes, such as pulmonary infarction and acute pancreatitis. The clinical appraisal was proved incorrect in 2 of the 11 cases in which autopsy revealed recent myocardial infarction. In the remaining 3 cases the disagreement between the clinical diagnosis and the transaminase level was unresolved.

In summary, there were 16 cases in which the clinical diagnosis and the GOT value were in disagreement. However, in 5 of the 16 cases autopsy proved the “GOT diagnosis” to be correct. Of the remaining 11 cases, 2 had normal GOT values, but were considered clinically positive for infarction. Nine patients had elevated GOT and were considered clinically negative for infarction. Although in 6 of the 9 cases, the source of the elevated GOT was determined to be extracardiac, all 11 cases are included as “errors” in GOT diagnosis, giving an over-all positive correlation of 86.6 per cent. With exclusion of the 6 cases having extracardiac causes for GOT elevation the positive correlation becomes 93.9 per cent.

Our review of the group III cases emphasizes that firm conclusions regarding myocardial necrosis based on ST-T changes alone are rarely possible. The only electrocardiographic pattern that appeared to be reliably related to infarction when interpreted in the light of the transaminase results was the appearance of a persistent depression of 2 mm. or more of the J-junction in the precordial leads.

**Group IV. Masking Electrocardiograms**

There were 41 patients in the group with “masking” electrocardiograms, that is, electrocardiographic patterns that may conceal or be confused with the pattern of myocardial infarction. In 34 of 41 cases the clinical diagnosis and the “GOT diagnosis” were in agreement, a correlation of 84 per cent.

Fourteen of the 41 cases had bundle-branch block and in 11 of these a diagnosis of myocardial infarction was made on clinical grounds. Ten of the 11 had distinct elevations of the enzyme. A representative example is the patient D.H.

D.H. (fig. 6), a 69-year-old man, was admitted on February 10, 1955 because of dull anterior chest pain of 4 hours’ duration associated with dizziness, weakness, vomiting, and cold clammy sweat. On admission the blood pressure was 96/62 and pulse was 84. There were frequent premature ventricular contractions, and a systolic gallop was heard. The initial electrocardiogram was interpreted as right bundle-branch block with S-T changes indicative of an acute myocardial process. A repeat electrocardiogram 7 hours later showed only occasional premature ventricular contractions, the bundle-branch block and S-T changes having disappeared. All subsequent electrocardiograms showed only left bundle-branch block. The GOT curve was characteristic of that seen in acute transmural infarction.

One case with bundle-branch block was diagnosed clinically as myocardial infarction but showed no GOT elevation. In 3 cases with bundle-branch block the clinical review was negative for infarction and none of these patients had elevated GOT levels.

Of 22 cases with residual electrocardiographic changes from previous infarction 9 were determined on clinical grounds to be positive for infarction. Six of the 9 had high serum GOT and 3 were normal. The remaining 13 were not considered to be infarcts on the basis of clinical review. In 10 of the 13, serum GOT was normal and in 3 clearly abnormal. Two of the 3 died during their hospital stay but did not come to autopsy. The third patient had signs and
symptoms suggestive of pulmonary infarction, which may have been the cause of the elevated GOT.

Four patients had the clinical findings and S-T and T changes of pericarditis. All had normal GOT levels throughout the serial study. Three other cases of pericarditis in which isolated determinations were made also had normal GOT. Included in this group were patients with uremic pericarditis, idiopathic pericarditis, and metastatic involvement of the pericardium.

A patient with Wolff-Parkinson-White syndrome was included in this group because of a presenting complaint of palpitation and persistent substernal pressing pain. There was no elevation of GOT at any time and the electrocardiogram, which resembled that of posterior myocardial infarction during the first few hospital days, later showed the typical Wolff-Parkinson-White pattern.

Table 2 presents a summary of this study in terms of the correlations in each electrocardiographic group between the transaminase results and the clinical-electrocardiographic

**Table 2.—Summary of Clinical-ECG vs. GOT Correlations in Diagnosis of Myocardial Infarction**

<table>
<thead>
<tr>
<th>ECG Group</th>
<th>Clinical evaluation</th>
<th>GOT</th>
<th>% Correlation Clinical vs. GOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I: Acute transmural infarction</td>
<td>60</td>
<td>0</td>
<td>58 2* 97</td>
</tr>
<tr>
<td>II: Normal</td>
<td>18</td>
<td>0</td>
<td>18  4* 78</td>
</tr>
<tr>
<td>III: LVH and S, myocardial ischemia, digitalis effect, sub-endocardial ischemia</td>
<td>82</td>
<td>22  48 32 50 86</td>
<td></td>
</tr>
<tr>
<td>IV: BBB, WPW, pericarditis, old infarct</td>
<td>41</td>
<td>17  17 21 20 83</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>201</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Indicate the cases in which GOT and Clinical-ECG evaluation were in conflict.

LVH = Left ventricular hypotrophy and strain.
BBB = Bundle-branch block.
WPW = Wolff-Parkinson-White syndrome.

**Table 3.—Summary of Autopsy Correlation**

<table>
<thead>
<tr>
<th>Myocardial necrosis at autopsy</th>
<th>No. cases</th>
<th>Clinical-ECG appraisal</th>
<th>GOT results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+</td>
<td>-</td>
<td>% correlation</td>
</tr>
<tr>
<td>Present</td>
<td>13</td>
<td>11</td>
<td>2 85 13 0 100</td>
</tr>
<tr>
<td>Absent</td>
<td>5*</td>
<td>2 3 60 0 5 100</td>
<td></td>
</tr>
</tbody>
</table>

* In one of these cases both Clinical-ECG appraisal and “GOT diagnosis” were negative but at autopsy 4 months later several old infarcted areas were found that could not be dated.

diagnosis with respect to infarction. A full discussion of each group has already been presented. It will be noted that for the complete series of 201 cases there is a final positive correlation of 88 per cent.

In this group of 201 patients, there were 24 deaths and 18 autopsies. Among the 18 autopsyed patients, 2 were considered to be clinically positive for infarction, the electrocardiogram showing serial T-wave inversions. However, GOT levels were normal, and no infarction was demonstrated by gross and microscopic examination of the myocardium. In 2 other cases considered clinically negative for infarction but showing elevated GOT levels, large areas of acute myocardial infarction were found at autopsy. In 3 of the 18 cases electrocardiographic changes of the S-T and T type were present, but the episode studied was listed as negative for myocardial infarction. The serum GOT levels were normal, and at autopsy no acute myocardial infarcts were found. In the remaining 11 cases GOT was elevated, all were considered positive for infarction, and in 10 acute infarcts were found at autopsy. The 1 discrepancy turned out to be nonspecific myocarditis (Fiedler’s), with diffuse rather than circumscribed myocardial necrosis.

Table 3 summarizes the autopsy experiences in this study. It serves to emphasize that in every instance the “GOT diagnosis” and the pathologic diagnosis regarding myocardial necrosis were in agreement.

**Pulmonary Embolization**

Serum GOT was studied in 15 cases diagnosed as pulmonary embolization by clinical.
roentgenographic, and electrocardiographic evidence. Eight of the 15 had consistently normal GOT. In 7 of the 15 the values were increased but none rose higher than 85 U./ml. In contrast to the sharp early rise observed in myocardial infarction, the rise in most of these cases did not occur until the fourth to the sixth day after onset of symptoms, often just preceding or concomitant with the appearance of icterus. The cause of the elevation is not clear but may be attributed to the pulmonary necrosis, hemolytic reaction, hepatic dysfunction, or a combination of these.

The elevation of GOT in approximately ¾ of the cases of pulmonary embolism coincident with the appearance of jaundice in several suggests that the serum enzyme will be elevated only in those cases that go on to pulmonary infarction. This possibility merits further investigation.

**DISCUSSION**

In order to evaluate a new diagnostic procedure it is necessary to have an unequivocal means for establishing the true diagnosis. A clinical-pathologic correlation is, of course, the ultimate basis for evaluation. In the 18 cases of this series coming to autopsy, 12 showed recent myocardial infarction and all had an elevated serum GOT in the period immediately following onset. The high levels observed in the autopsied case of Fiedler’s myocarditis indicate that any form of acute myocardial necrosis may release this enzyme into the serum. That this is indeed the mechanism for the observed elevations of GOT in the serum is shown by the studies of Nydick, Wroblewski, and LaDue. These workers found that normal dog heart muscle contains as much as 465,000 U. of GOT/Gm., whereas infarcted areas contained only 5,000 to 33,000 U./Gm.

Five cases at autopsy showed no evidence of recent myocardial infarction and in none of these was GOT increased. In 1 case there were several old infarcts and it could not be determined if 1 of these had occurred at the time the patient was studied 4 months prior to death, at which time the GOT was negative. Hence, in at least 17 of the 18 cases the “GOT diagnosis” was corroborated by autopsy findings with reference to myocardial necrosis. Also of interest are the 4 autopsied cases in which the clinical-electrocardiographic appraisal was proved incorrect. In all 4 the “GOT diagnosis” was in agreement with the pathologic findings.

With transmural myocardial infarction (group I) the electrocardiographic changes are considered pathognomonic and hence the correlation in this group is probably equivalent to histopathologic proof. We have previously reported high serum GOT in 22 of 24 cases of transmural infarction. A total of 60 such cases has been studied to date and 58 have been confirmed by the enzyme test. These findings firmly establish the association of increased serum GOT with myocardial necrosis. LaDue and Wroblewski have reported 50 cases of transmural infarction, 49 of which had elevated serum GOT. These results in transmural infarction, taken together with the autopsy results in the present series, appear to justify certain conclusions in the interpretation of those cases with equivocal or nondiagnostic electrocardiographic changes. It would seem reasonable to infer that, in the absence of an alternative explanation, the presence of a high serum GOT with a typical curve of evolution supported by either clinical or electrocardiographic evidence of active coronary artery disease justifies the diagnosis of myocardial infarction. If this inference is supported by further studies the very real value of serum GOT in differential diagnosis is clear.

The increase in serum GOT after infarction is transient. In most cases in this series it persisted for 4 to 6 days but in some, especially those with only ST-T wave changes in the electrocardiogram, it was elevated for only 2 or 3 days. This is in agreement with the results of LaDue and Wroblewski, who reported return to normal by the seventh day in all cases and by the third day in 20 per cent of the cases. A single test will most likely be positive if performed 24 to 48 hours after onset of symptoms. However, it is evident that a series of at least 3, and preferably 5, daily determinations is more informative. The rapid rise and almost equally rapid fall in level is characteristic. It will establish in borderline cases that a given value obtained on the first
or second day was indeed above the normal for that individual.

It has been reported\textsuperscript{13, 14} that the increase in serum GOT corresponds approximately with the size of the infarcted area. In the few autopsied cases in this series no correlation of this type was noted. However, the rise and fall of GOT during the first few days is so rapid that it is possible to miss the peak concentration in the serum. Further experience is necessary to evaluate the use of serum GOT in estimating the extent of cardiac damage.

Serum GOT promises to be particularly useful in cases like those in group IV, where the electrocardiographic picture is masked or distorted by a previous infarction or by left bundle-branch block. The test should also be of value in the differential diagnosis of pericarditis, since to date all patients with pericarditis, uncomplicated by myocarditis, have had normal values. In addition, Glassner and associates\textsuperscript{18} have shown that severe experimental pericarditis in dogs is not associated with elevated serum GOT. Another area in which the test may be helpful is in the diagnosis of extension of a myocardial infarct, which not infrequently fails to declare itself in the electrocardiogram.

It is well recognized that myocardial infarction can occur without the characteristic Q wave in the electrocardiogram. In subendocardial infarction the electrocardiographic picture is fairly well defined. It should be noted that all 4 cases in this series presenting this picture and clinically believed to have had myocardial infarction had abnormal serum GOT. The present findings confirm that certain cases with only S-T and T changes in the electrocardiogram actually have acute myocardial necrosis. Other workers have also noted GOT elevations in patients with S-T and T changes only.\textsuperscript{7, 8, 14} As more experience accumulates in the use of this test, it should be possible to improve diagnostic accuracy in establishing the presence or absence of myocardial necrosis. Of course, the ultimate validity of the GOT findings in these cases must await the accumulation of extensive clinical-pathologic correlations.

Agress and associates\textsuperscript{16} failed to find an elevated GOT following experimental pul-

<table>
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<tr>
<th>Table 4.—Transaminase Value in Other Diseases</th>
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<tbody>
<tr>
<td><strong>GOT elevated in:</strong></td>
</tr>
<tr>
<td>1. Myocarditis, acute</td>
</tr>
<tr>
<td>2. Active hepatocellular damage</td>
</tr>
<tr>
<td>3. Acute pancreatitis</td>
</tr>
<tr>
<td>4. Pulmonary infarction</td>
</tr>
<tr>
<td>5. Hemolytic crisis</td>
</tr>
<tr>
<td>6. Extensive crushing injuries or burns</td>
</tr>
<tr>
<td>7. Surgery</td>
</tr>
<tr>
<td><strong>GOT normal in:</strong></td>
</tr>
<tr>
<td>1. Pericarditis</td>
</tr>
<tr>
<td>2. Angina pectoris or coronary insufficiency</td>
</tr>
<tr>
<td>3. Malignant disease</td>
</tr>
<tr>
<td>4. Infectious diseases</td>
</tr>
<tr>
<td>5. Rheumatic fever</td>
</tr>
<tr>
<td>6. Acute cholecystitis</td>
</tr>
<tr>
<td>7. Perforated peptic ulcer</td>
</tr>
<tr>
<td>8. Arthritis, rheumatoid and hypertrophic</td>
</tr>
</tbody>
</table>

monary infarction in dogs, but these animals were observed for only 40 hours. We have observed a delayed rise of the enzyme in human subjects. It usually occurs the fourth day after onset of symptoms and rarely exceeds 65 U./ml. The curve is thus quite different from that seen following myocardial infarction. We have detected such elevations only in those cases of pulmonary embolization associated with pulmonary infarction.

There are other pathologic conditions besides acute myocardial infarction in which GOT is elevated. These are listed in table 4. The myocarditis may be of rheumatic or nonspecific etiology. The curve of GOT in liver disease generally does not show the rapid rise and fall characteristic of myocardial infarction. Injuries to skeletal muscle must be fairly extensive to cause elevation. A patient with a crush injury and 27 fractures had a serum GOT of 355 U./ml.\textsuperscript{17} GOT has been normal in those conditions listed in table 4 when no active hepatocellular disease is concomitantly present.

**Summary**

The results of serial serum glutamic-oxalacetic transaminase (GOT) assays in 201 cases in which coronary artery disease was suspected are reported. The final positive correlation between the clinical-electrocardiographic diagnosis and the "GOT diagnosis" with respect to acute myocardial infarction was 88 per cent.

In 18 cases coming to autopsy the correlation between elevated serum GOT and recent myocardial necrosis was 100 per cent. Similar elevations were observed in 97 per cent of 60 cases with transmural infarction.
If the clinical and electrocardiographic appraisal for infarction is accepted as the basis of reference, 95 per cent of all cases diagnosed as infarction had elevated serum GOT.

In 18 of 201 cases in which the clinical and electrocardiographic diagnosis was uncertain or negative for infarction there was elevation of serum GOT. Six of these 18 were found to be of noncardiac origin.

It is concluded that the determination of serum GOT provides an additional useful method for the diagnosis of myocardial infarction.

**Sammario in Interlingua**

Es reportate le resultatos de serial essayos de transaminase glutamic-oxalacetic (TGO) del sero in 201 casos con suspicion de morbo de arteria coronari. Le final correlation positive inter le diagnose clinico-electrocardiographic de infarimento myocardial e le diagnose a TGO de ille condition esseva 88 pro cento.

In 18 casos autopsiate, le correlation inter elevate TGO del sero e recente necrosis myocardial esseva 100 pro cento. Simile elevations esseva observate in 97 pro cento de 60 casos con infarimento transmural.

Si on accepta le constatation clinico e electrocardiographic de infarimento, 95 pro cento del casos diagnosticate como infarimento mostrava elevate nivellos de TGO seral.

In 18 del casos il habeva elevation de TGO seral in association con diagnoses clinico e electrocardiographic de indecision o negativitate pro infarimento. In sex de iste 18 casos, le condition del patients esseva reducite a origins noncardiac.

Nos conclude que le determination de TGO seral provide un utile methodo additional pro le diagnose de infarimento myocardial.

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**References**


Serum Glutamic-Oxalacetic Transaminase in Coronary Artery Disease: A Review of 201 Cases

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