Ostrow *et al.* (1956) quote one case of Fiedler's myocarditis in which the S.G.O.T. was elevated as one would expect, and Chesler (1958) reports elevated levels in diphtheritic myocarditis. Nydick *et al.* (1955a, 1955b) give an interesting account of its elevation in acute rheumatic carditis. The latter at any rate cannot often be confused with myocardial infarction, and no such difficulty arose in the present series.

Summary

Serial daily estimations of serum glutamic oxalacetic transaminase (S.G.O.T.) for at least 10 days have been made in 34 acute episodes of myocardial infarction occurring in 28 patients. The results have been compared with changes in erythrocyte sedimentation rate (E.S.R.), temperature, and electrocardiogram.

Estimations of S.G.O.T. are most useful (a) when the electrocardiogram is obscured by previous myocardial infarction, arrhythmias, bundle-branch block, left ventricular strain, and digitalis; (b) when extension of myocardial infarction is suspected; and (c) to distinguish between acute coronary insufficiency and myocardial infarction.

There was no rise in S.G.O.T. in 8.8% of the cases of fresh myocardial infarction.

The maximum elevation of S.G.O.T. roughly corresponds with the electrocardiographic estimation of the size of the infarct only if other causes of a raised transaminase can be excluded. The most important of these in myocardial infarction is centrilobular necrosis of the liver secondary to cardiac failure.

In small infarctions serial estimations of S.G.O.T. may show a characteristic curve even though the peak does not exceed 40 units.

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SERUM TRANSAMINASE: ITS CLINICAL USE IN DIAGNOSIS AND PROGNOSIS

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Human serum contains several enzymes concerned with the reaction of transamination. The one most commonly determined is serum glutamic oxalacetic transaminase (SGO-T), which catalyses the reversible reaction:

Similarly serum glutamic pyruvic transaminase (SGP-T) catalyses the reaction:

Cohen and Hekhuis (1941) showed that transaminase was in highest concentration in cardiac and skeletal muscle. LaDue *et al.* (1954) also found it to be present in the brain, liver, and kidney in lower concentrations.

The serum transaminase level (SGO-T) has been found to be elevated following myocardial damage, hepato-cellular necrosis, pancreatitis, trauma to skeletal muscle, myositis (including dermatomyositis), active rheumatic carditis, and any process associated with active damage to skeletal or cardiac muscle (LaDue and Wróblewski, 1955; Wróblewski and LaDue, 1956; Ratner and Sacks, 1957).

The curve of serial serum transaminase estimations would appear to be characteristic in cases of myocardial infarction, and it has been suggested that a characteristic curve occurring within the normal range of values may be found in some cases of angina (Chinsky *et al.*, 1956) or associated with a very small infarct (Ratner and Sacks, 1957). These authors also found that the serum transaminase level remains normal in patients with cerebrovascular damage, acute or chronic infections, acute phlebitis, uraemia, neoplastic, allergic, or metabolic diseases, unless associated with damage to skeletal or heart muscle. Further, there appears to be no apparent correlation with age, sex, colour, weight, leucocyte count, blood sedimentation rate, the presence or absence of fever, heart failure or shock, or the use of anticoagulant drugs or cortisone.

The above evidence suggests that serial transaminase readings are a possible aid to diagnosis in cases of doubtful myocardial infarction and might prove to be related to the actual size of infarct and, because of this, a possible guide to prognosis. We felt, therefore, that a small series of cases investigating these points was justified.

Technique Employed.—The technique we used was that devised by Frankel and Reitman (1957). In this method the amount of keto-acid formed in one hour is determined colorimetrically by the formation of

a highly coloured "hydrazone," using a spectrophotometer or photoelectric absorptiometer in the range 500 to 520 $m\mu$.

Results

Serum transaminase levels were estimated in 31 cases of myocardial infarction and the typical findings in several cases are described below.

(47) *Case 1.*—Woman, aged 69. Angina three weeks; eight hours' central chest pain. Serum transaminase: 24 SGO-T units 8 hours after onset, 156 units at 32 hours, 27 units at 4 days, 18 units at 8 days. E.C.G.: (a) posterior infarct eight hours after onset; (b) much improved six weeks later.

(21) *Case 2.*—Man, aged 59. Angina two years; eight hours' central chest pain. Serum transaminase: 216 SGO-T units 56 hours after onset, 64 units at 4 days, 54 units at 6 days, 32 units at 7 days. E.C.G.: antero-lateral infarct 80 hours after onset.

(3) *Case 3.*—Man, aged 63. Hypertension; angina one week; four hours' central chest pain. Serum transaminase: 184 SGO-T units 12 hours after onset, 103 units at 36 hours, 49 units at 60 hours. E.C.G.: (a) postero-lateral infarct 24 hours after onset; (b) unchanged seven days later.

FIG. 1.—Case 4. Serum transaminase readings.

These cases show the typical transiently raised transaminase values in myocardial infarction, the diagnosis in each case being confirmed clinically and on electrocardiography.

(8) *Case 4.*—Man, aged 49. Angina one year; five hours' central chest pain. Serum transaminase: 23 SGO-T units 6 hours after onset, 160 units at 24 hours, 102 units at 54 hours, 30 units at 6 days (Fig. 1). E.C.G.: normal bar Q wave V_{4+7} 48 hours after onset; antero-septal infarct 10 days after onset (Figs. 2 and 3).

FIG. 2.—Case 4. E.C.G. 48 hours after onset.

FIG. 3.—Case 4. E.C.G. 10 days after onset.

(44) *Case 5.*—Man, aged 62. Chronic bronchitis; cor pulmonale; jaundice (painless). ? Cardiac cirrhosis. Serum transaminase: 460 SGO-T units day after admission. E.C.G.: left bundle-branch block day of admission. Serum bilirubin, 11.3 mg./100 ml. (total). Necropsy: Enlarged heart; right ventricular hypertrophy; extensive recent septal infarct involving anterior wall of both ventricles; liver showed cardiac cirrhosis.

(17) *Case 6.*—Man, aged 77. Prostatic carcinoma with metastases. Atrial fibrillation. Serum transaminase: 40 SGO-T units 3 days after admission, 30 units at 5 days, 86 units at 6 days, 77 units at 7 days. E.C.G.: auricular fibrillation and right bundle-branch block three days after admission (Fig. 4). Necropsy: Terminal bronchopneumonia; carcinoma of prostate with metastases; recent occlusion of circumflex coronary artery.

(12) *Case 7.*—Man, aged 61. Three days' central chest pain. Serum transaminase: 430 SGO-T units at 4 days after onset, 550 units at 5 days. E.C.G.: left bundle-branch block five days after onset. Died. Necropsy not obtained.

Case 4 appears to demonstrate transaminase levels confirmatory of recent infarction before fully diagnostic electrocardiographic changes had occurred. In Case 5 the transaminase level again agreed with the proved diagnosis in the absence of confirmatory E.C.G. changes. The raised serum bilirubin level might be expected to cause a rise in transaminase values due to the high transaminase content of bile. However, the results in this case were consistent with the proved diagnosis at necropsy.

Case 6 was also proved at necropsy despite non-confirmatory electrocardiography and in the presence of carcinomatosis.

Case 7 reveals apparently diagnostically raised transaminase values, whereas the electrocardiographic changes are merely suggestive. Although permission for necropsy was not obtained there is felt to be no doubt on purely clinical grounds about the diagnosis.

Cases 4 to 7, therefore, are instances in which transaminase levels proved of greater diagnostic aid than initial electrocardiography.

(15) *Case 8.*—Man aged 76. Diabetic; sudden collapse and death in two hours; no prior history of cardiac pain. Serum transaminase: 36 SGO-T units at onset of collapse. E.C.G.: postero-lateral infarct three days before collapse. Necropsy: fibrotic myocardium; 2-cm. thrombus in right coronary artery proximal part several (? 10) days old, distal part quite fresh.

FIG. 4.—Case 6. E.C.G. 3 days after admission.

(28) *Case 9.*—Woman, aged 76. Hypertensive "collapse" 10 days previously. Serum transaminase: 20 SGO-T units 10 days after collapse. E.C.G.: antero-lateral infarct six days after collapse; much improved 13 days after collapse. Recovered.

Cases 8 and 9 illustrate the need for transaminase levels to be estimated at the appropriate times after infarction if they are to prove of help in assessment. Thus in Case 8 the estimation was too soon after the terminal infarction to reveal any change, and in Case 9 the time interval of 10 days after infarction is too long. The optimum times after infarction appear to be from 24 hours to five days.

(7) *Case 10.*—Woman, aged 55. Depressive; hypertension; five hours' epigastric pain. Serum transaminase: 83 SGO-T units 48 hours after onset, 47 units at 3 days, 16 units at 4 days. E.C.G.s taken 48 hours and 10 days after onset are shown in Figs. 5 and 6.

FIG. 5.—Case 10. E.C.G. 2 days after onset.

deaths reveal an equivalent average figure of 576 SGO-T units. These figures are not altogether accurate, as in a few of the recovered cases the transaminase levels

If serum transaminase levels prove a reliable guide the test will be of extreme value in this type of case, where the infarct is presumably small, in that a more definite prognosis may be given. A negative result (normal transaminase levels) would be of equal value. This was the only case of this type in the present series.

Of the 31 cases of myocardial infarction, 23 occurred clinically within seven days prior to serum transaminase estimation being obtained. Of the 23 patients with acute infarctions, 16 recovered and 7 died (see Table). The 16 recoveries reveal an average peak transaminase value of 111 SGO-T units. The

FIG. 6.—Case 10. E.C.G. 10 days after onset.

Results in 23 Cases with Acute Infarctions

Case No.	Observed Serum Transaminase Peak Values	Outcome	Necropsy
2	216 units 56 hours after onset	Recovered	
3	184 " 12 " admission	"	
4	160 " 24 " onset	"	
5	460 " 1 day after admission	Died	Extensive infarct of both ventricles
6	86 " 6 days " "	"	Recent circumflex artery occlusion
7	550 " 5 days after onset	"	No necropsy
8	36 " at the time of infarct	"	Old and recent infarcts
10	83 " 48 hours after onset	Recovered	
12	200 " 36 " "	"	
13	61 " 78 " "	"	
14	120 " 12 " "	"	
15	73 " 5 days after admission	"	
16	43 " 24 hours after onset	"	
17	129 " 12 " "	"	
18	41 " 7 days " "	"	
19	95 " 3 " "	"	
20	900 " 3 " "	Died	No necropsy
21	156 " 32 hours " "	Recovered	
22	24 " 4 days " "	"	
23	280 " 48 hours " "	Died	Extensive infarct
24	114 " 33 " "	Recovered	
25	75 " 48 " "	"	
26	1,720 " 48 " admission	Died	No necropsy

obtained were not necessarily the highest that actually occurred. Similarly in the fatal group, one case reveals a low value as it was too soon after infarction to expect a rise, and one patient with infarction died from bronchopneumonia and carcinomatosis rather than the infarct, and consequently the low value obtained agrees with the necropsy findings of a small infarct.

These points would possibly cause a higher average figure in the recoveries, but also would increase the figure in fatal cases, and it is felt, therefore, that the difference is probably significant.

The average time of peak transaminase level relative to the time of infarction was estimated from cases where serial transaminase levels were known. Where the time of infarction was in doubt it has been accepted as from the moment of admission. The average figure so obtained was 62 hours after infarction in the 18 suitable cases.

Serum transaminase estimations were carried out both during and for several days after attacks of angina in appropriate cases. On each occasion the transaminase value was found to be within the normal range. On several occasions, in two cases of old myocardial infarction with recurrent attacks of angina, we found transaminase value of help in distinguishing prolonged anginal pain from that due to further myocardial infarction, the transaminase values correlating well with subsequent electrocardiography and clinical progress.

In this series the overall average age of the patients with myocardial infarction was 60 years. The average age of the males was 58 years, and of the females 67.

Discussion

It is appreciated that the majority of cases of myocardial infarction can be diagnosed on clinical grounds, but a group remains in which the diagnosis is initially in doubt.

This small series of cases confirms reports in America and this country that recent myocardial infarction can be detected in some cases by a typical rise and fall in serum transaminase values before diagnostic electrocardiographic changes have occurred. The correlation of degree of rise in serum transaminase level with size of infarct and immediate outcome suggests that transaminase values of over 250 units are of serious portent and such cases are unlikely to recover.

The importance of time of transaminase estimation after infarction was well shown in several cases. It is suggested that transaminase values should be estimated at 24-hourly intervals from the time of infarction for at least four and preferably seven days, or until the peak value is passed, if full information is to be obtained from the results.

In differential diagnosis, too, the transaminase levels may be of help. On several occasions we found it useful in assessing whether severe cardiac pain was due to prolonged angina pectoris or actual infarction, the transaminase results correlating in each case correctly with electrocardiographic findings and subsequent clinical progress.

No diagnostic rise in transaminase levels was detected in two cases of pulmonary infarction, two cases of gall-bladder dysfunction, two cerebral catastrophes, and six cases of pneumonia.

It is of interest that one case of active rheumatic pericarditis (not included in this series) revealed a transiently raised serum transaminase level. This suggests concurrent myocardial damage of a temporary nature, and it will be of interest in the future to ascertain whether serum transaminase levels will help in assessing activity in cases of rheumatic carditis where this is in doubt.

In carrying out these estimations of serum transaminase levels the method of Frankel and Reitman (1957) was found to have several inherent disadvantages regarding cost and technique. These seem to be overcome in the method described by King (1958), and the simpler technique and its cheapness should place it well within the scope of all routine laboratories.

Summary

Serum transaminase estimations in a small number of cases of myocardial infarction are related to clinical, electrocardiographic, and necropsy findings. The test's possible value and advantages in early differential diagnosis and prognosis are discussed.

This series reveals close correlation between peak transaminase levels, size of infarct, and immediate outcome.

Evidence is presented which shows that cases in which transaminase values rise above 250 SGO-T units do not usually recover.

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Rank Precision Industries (B.A.F.) Ltd. has issued a leaflet describing the films and filmstrips on hospital and medical training in the G.B. Film Library. The leaflet lists 44 films and 33 filmstrips which are available from the library. It gives brief details of their length, number of reels or frames, hire rates, and purchase prices. Handbooks containing essential information for the guidance of teachers or film users are supplied with the films and filmstrips. Films are available for either outright purchase or hire; viewing copies will be supplied free of charge on application. Filmstrips are for outright purchase only, but may be previewed under a 14-days "sale or return" approval scheme. Further details from the G.B. Film Library, Aintree Road, Perivale, Greenford, Middlesex.

SCURVY FOLLOWING BILATERAL ADRENALECTOMY

REPORT OF TWO CASES

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During the convalescent period of two patients who had undergone bilateral adrenalectomy and oophorectomy for advanced breast cancer it was noticed that they developed petechial skin haemorrhages on the arms and legs. One of these patients also complained of bleeding gums. A diagnosis of scurvy was made and the vitamin-C saturation test was carried out. In the first patient only 21 mg. of the vitamin appeared in the urine, following an intravenous injection of 1,000 mg. of ascorbic acid, and in the second case only 31 mg. was excreted. The average amount of ascorbic acid excreted during this test in a normal healthy individual varies between 300 and 500 mg.

These findings stimulated us to investigate two other patients in the ward who had recently had their adrenals and ovaries removed, and in both cases the test showed a vitamin-C deficiency. Further patients were investigated and of nine who were subjected to the test similar deficiencies were found in seven. The remaining two patients had been operated upon several months previously; both had progressed well, had good appetites, and had increased in weight, and in them the values were 370 and 570 mg. respectively. The average amount of vitamin C excreted in the seven patients (average age 53 years) in whom the figure was low was 85 mg.

A control series of tests was carried out on nine patients who had recently undergone operations such as radical mastectomy, cholecystectomy, appendicectomy, and colectomy. The average amount of vitamin C excreted by them was 300 mg., their average age being 49 years. None of these patients had been given vitamin C therapeutically.

Although there is some doubt about the reliability of the vitamin-C saturation test, these figures seemed significant and led us to believe that patients who had both adrenals and ovaries removed might be deficient in vitamin C, especially in the early post-operative period.

We record the two case histories of scurvy following bilateral adrenalectomy and oophorectomy, and discuss the possible cause of this, together with the relationship of vitamin C to the adrenal glands.

Case 1

A housewife aged 37 was admitted to hospital on June 17, 1958. Nine months earlier she had noticed a swelling in the left breast which had ulcerated through the skin. She complained of loss of energy, tiredness, and poor appetite. On examination she had a foul-smelling, fungating cancer of the left breast, with an infected carcinomatous ulcer in the left axilla. She was pale and listless, with oedema of the legs and sacrum. A blood count showed a haemoglobin level of 22%, and 16,600 white blood cells, of which 95%