Mr. Westwood's subjects (Cases 14-22), who were not under psychiatric treatment. Thus, of the psychiatric group 11 out of 13 had sperm counts of 50 millions per ml. or more, while of the non-psychiatric group only two out of nine had counts over this level. There was little difference in volume, percentage of abnormal forms, or motility between the two groups, but differences in sperm density were reflected in an overall rating of "highly fertile" or "fertile" in 10 out of 13 psychiatric as against 2 out of 9 non-psychiatric cases.

Confirmation (from larger and more comprehensive surveys) of the striking difference in fertility between the two subgroups in our series would be of considerable importance in relation to theories of aetiology of homosexuality. Hypotheses readily come to mind in this context, but we think it inappropriate to list them at this stage in view of the small size of our study. The difference noted, if not fortuitous, may well be an artifact of the selection procedure. For example, some of the non-psychiatric subjects may have volunteered because they were already conscious or suspicious of genital abnormality, whereas psychiatric patients who chose to take part may have been influenced by quite different motives.

Summary

22 men who were predominantly or exclusively homosexual in orientation provided seminal specimens for analysis.

Of the 22, 8 were rated highly fertile, 4 fertile, 5 borderline, 3 subfertile, and 2 sterile, on the basis of the seminological findings.

The clinical diagnosis of "constitutional type" homosexuality was compatible with a high degree of fertility as judged seminologically.

The distribution of seminal analyses was similar in the group as a whole to that of a control group of 1,000 husbands of couples complaining of infertility.

Within the series there was a conspicuous difference between 13 patients under psychiatric treatment and 9 subjects drawn from a "normal" population. 10 out of 13 of the psychiatric, but 2 out of 9 of the non-psychiatric, group were rated as fertile or highly fertile. This is an interesting finding, but must be interpreted with great caution in view of the small size of the series and the method of selection.

We are indebted to Dr. J. L. Stafford and his colleagues and to Mr. Gordon Westwood for their collaboration. One of us (D. P.) acknowledges with thanks financial support from the Research Funds, St. George's Hospital.

REFERENCE

Raboch, J., and Nedoma, K. (1958). *Psychosom. Med.*, **20**, 55.

"Since the middle of the nineteenth century we have successfully dealt with most of the public-health problems associated with the early industrial revolution. Contaminated water supplies, non-existent or inadequate sewerage, bad housing are no longer the widespread health hazards they once were in our towns. But dirty air is one legacy of the past which is still very much with us, and is still a serious threat to health." (Sir KEITH JOSEPH, Parliamentary Secretary, Ministry of Housing and Local Government, at Harrogate Conference, National Society for Clean Air.)

LACTIC DEHYDROGENASE ACTIVITY IN ACUTE MYOCARDIAL INFARCTION

BY

J. KING, B.Sc., A.I.M.L.T.

Biochemist, Group Pathology Department,
Barrow-in-Furness

AND

A. P. B. WAIND, D.Sc., M.D., M.R.C.P.

Consultant Physician, North Lonsdale Hospital,
Barrow-in-Furness

The estimation of serum glutamic-oxalacetic transaminase (GO-T) has been used in the diagnosis of cardiac infarction since 1954 (LaDue *et al.*, 1954). Although this aid to diagnosis is now widely accepted, Dewar *et al.* (1958) have shown that false-negative results occur in 8.8% of cases. In those cases where the GO-T activity is raised, the increase is usually confined to a period of three to six days after the onset of pain (Wróblewski and LaDue, 1956).

The procedure for assay of serum GO-T activity by the techniques suited to routine work (Mohun and Cook, 1957; Reitman and Frankel, 1957; King, 1960a) is rather lengthy, and this fact, associated with the limitations previously mentioned, prompted an examination of the serum levels of other enzymes in cases of suspected cardiac infarction.

Hill and Levi (1954) demonstrated the presence of lactic dehydrogenase in human blood serum, and Wróblewski and LaDue (1955) observed an increase in activity similar to that of GO-T in cases of cardiac infarction. Wacker *et al.* (1956) confirmed this observation on 22 patients, as did Zimmerman and Weinstein (1956) on five patients in whom elevated enzyme activity up to two weeks after infarction was noted.

Snodgrass *et al.* (1959) repeated the work in great detail and showed in a large series of cases that cardiac infarction was invariably associated with elevation of lactic dehydrogenase level of serum. They considered such enzyme assay to be a determinate diagnostic measure in cardiac infarction.

Lactic dehydrogenase (LDH) is an enzyme, which, utilizing coenzyme 1 (DPN) as hydrogen carrier, shows its greatest activity in the reaction,

The present report deals with the serum levels of LDH compared with those of GO-T found in serial assays on a series of cases admitted to hospital with cardiac pain.

Methods and Materials

Serum GO-T was estimated by the method of King (1960a). LDH activity was assayed using a colorimetric technique. This technique and the values encountered in normal subjects have been described elsewhere (King, 1959, 1960b).

The clinical material consisted of 50 subjects admitted to the North Lonsdale Hospital with cardiac pain and electrocardiographic changes supporting a diagnosis of coronary thrombosis. Cases in which the electrocardiographic findings were equivocal, or were

obsured by previous infarction, or in which there was bundle-branch block, were excluded. Otherwise the series was a consecutive one. The ages of these subjects range from 38 to 76 years, and there were 12 females and 38 males. Blood specimens for enzyme estimation were obtained from the patients as soon as practicable after admission, and later specimens as required for prothrombin-time estimation in the control of anticoagulant therapy, or periods of up to three weeks after admission to hospital. In all more than 900 assays were performed on the specimens received.

Results

The series was divided into two groups.

Group 1

This group consisted of 32 patients who we considered had had unequivocal myocardial infarction. The criteria for inclusion were that there should have been a history of typical prolonged cardiac pain and electrocardiographic changes of the Q and T type, characteristic of acute transmural infarction.

In 29 of these cases the serum GO-T level was elevated for a period of from three to nine days, with an average of five days. In three subjects no abnormal increase in GO-T activity was detected, but these subjects were late admissions. Specimens for assay were not received until four to seven days after the onset of pain, and it may have been that the rise and fall of GO-T activity occurred before admission.

In every one of the 32 cases in this group the serum LDH activity was raised. The period of elevation ranged from a minimum of six to a maximum of more than 14 days, with an average of 11 days. The elevation of serum LDH activity lasted longer than that of GO-T in every patient except for one, who died on the ninth day, in whom both serum enzyme activities were grossly raised after extension of the infarction. Table I shows ages and sexes of this group with comparisons of duration of LDH and GO-T activity in the individual cases. Fig. 1 shows the serial enzyme levels encountered in this group.

Group 2

This group comprised 18 patients who gave a typical history of prolonged cardiac pain but in whom the electrocardiograph showed only T-wave changes, sometimes transiently in serial records. These patients were placed in a separate group because some slight doubt existed about whether actual muscle death had occurred. This group contained 4 females and 14 males aged from 46 to 71 years.

Serum LDH was elevated in all but two of these subjects, and, in one of

those, specimens for assay were not received until eight days after the presumed date of infarction. In the other case, however, specimens were received on the same day as the onset of pain. Serum GO-T showed no increase in activity in these cases or in a further three from whom specimens were received after two, five, and six days respectively. Table II and Fig. 2 summarize these results.

Discussion

A comparison between the clinical value of these two enzymes, based on the series of cases investigated, shows that LDH assay is at least as sensitive as that of GO-T. The group of 32 cases of undoubted infarction which produced 100% positive results using the LDH test is especially significant in this regard. In the whole series LDH assay yielded negative results in only two cases, one of which was a late admission to hospital. It would seem that LDH elevation occurs almost invariably at some period during the first 14 days after cardiac infarction, and in this regard our results are in

infarction, while after this interval LDH is the more sensitive index.

From a laboratory point of view LDH estimation is simpler to carry out than that of GO-T whatever the method used, and, taking less time, represents a considerable saving in laboratory resources.

Serum LDH elevations have been noted in various other disease states (Hsieh and Blumenthal, 1956), particularly in carcinoma (West and Zimmerman, 1958), anaemia (Zimmerman *et al.*, 1958), and leukaemia (Blanchaer *et al.*, 1958). We have also observed rises in mitral stenosis with heart failure, femoral-vein thrombosis with oedema, pneumonia, and cerebral thrombosis.

This formidable list would lend itself to endless false-positive results if it were suggested that estimation of LDH activity was a specific test for cardiac infarction. However, in its correct clinical setting—that is, in the investigation of cases of chest pain of cardiac type—the assay appears to have considerable value and certain advantages over enzyme assays at present accepted as useful.

Summary

A comparison is made between the serum lactic dehydrogenase and glutamic-oxalacetic transaminase activities in a series of 50 cases of cardiac infarction.

The advantages of the former assay in the investigation of this condition are discussed.

We thank Dr. J. E. Horrocks and Dr. J. Ward for encouragement and constructive criticism.

REFERENCES

- Blanchaer, M. C., Green, P. T., Maclean, J. P., and Hollenberg, M. J. (1958). *Blood*, **13**, 245.
Dewar, H. A., Rowell, N. R., and Smith, A. J. (1958). *Brit. med. J.*, **2**, 1121.
Hill, B. R., and Levi, C. (1954). *Cancer Res.*, **14**, 513.
Hsieh, K. M., and Blumenthal, H. T. (1956). *Proc. Soc. exp. Biol. (N.Y.)*, **91**, 626.
King, J. (1959). *J. med. Lab. Technol.*, **16**, 265.
— (1960a). *Ibid.*, **17**, 1.
— (1960b). *Ibid.*, **17**, 89.
LaDue, J. S., Wróblewski, F., and Karmen, A. (1954). *Science*, **120**, 497.
Mohun, A. F., and Cook, I. J. Y. (1957). *J. clin. Path.*, **10**, 394.
Reitman, S., and Frankel, S. (1957). *Amer. J. clin. Path.*, **28**, 56.
Snodgrass, P. J., Wacker, W. E. C., Eppinger, E. C., and Vallee, B. L. (1959). *New Engl. J. Med.*, **261**, 1259.
Wacker, W. E. C., Ulmer, D. D., and Vallee, B. L. (1956). *Ibid.*, **255**, 449.
West, M., and Zimmerman, H. J. (1958). *A.M.A. Arch. intern. Med.*, **102**, 103.
Wróblewski, F., and LaDue, J. S. (1955). *Proc. Soc. exp. Biol. (N.Y.)*, **90**, 210.
— (1956). *Ibid.*, **91**, 569.
Zimmerman, H. J., and Weinstein, H. G. (1956). *J. Lab. clin. Med.*, **48**, 607.
— West, M., and Heller, P. (1958). *A.M.A. Arch. intern. Med.*, **102**, 1115.

A new monthly publication, *Abstracts of Japanese Medicine*, is now being published by the Excerpta Medica Foundation with the aid of a grant from the Public Health Service, National Institutes of Health, U.S. Department of Health, Education, and Welfare. The chairman of the Japanese editorial board is Professor T. YOSHIDA, dean of the faculty of medicine, University of Tokyo. The publication includes a list, translated into English, of the periodicals covered and indicates those which are published in the English language. Details may be obtained from the Excerpta Medica Foundation, 119-23 Herengracht, Amsterdam-C., The Netherlands, or 56 Grosvenor Hill, Mayfair, London W.1.

TABLE I.—Serum GO-T and LDH Activity in 32 Patients with Electrocardiographic Evidence of Myocardial Infarction

Case No.	Sex and Age	Day First Specimen Received	Duration of GO-T Elevation	Duration of LDH Elevation
1	M 56	1	5	14
2	F 60	1	5	10
3	F 67	4	<4	11
4	M 59	2	8	Till death on 11th day
5	M 64	1	4	8
6	M 55	1	5	13
7	M 41	2	4	7
8	F 76	2	5	9
9	F 70	2	4	6
10	M 55	1	4	10
11	F 72	5	5	12
12	M 53	3	5	12
13	M 46	3	3	10
14	M 65	1	5	12
15	F 71	2	5	13
16	M 38	2	5	14
17	M 68	1	7	14
18	M 55	3	6	11
19	M 54	1	6	12
20	M 48	4	5	11
21	M 57	2	3	7
22	M 57	3	Till death on 9th day	
23	M 52	1	5	8
24	F 53	7	<7	14
25	F 68	3	5	10
26	M 64	3	4	6
27	M 60	5	<5	14
28	M 55	1	4	8
29	M 51	2	5	8
30	M 53	2	5	9
31	M 54	6	6	11
32	M 68	2	3	10

keeping with those reported by Snodgrass *et al.* (1959). Serum GO-T activity, on the other hand, was normal in these two cases mentioned and in a further six subjects.

The elevation of serum LDH activity was always prolonged beyond that of GO-T, and this might well be useful in the investigation of cases of myocardial infarction admitted late to hospital. The scattergrams demonstrate this point. They also show that GO-T is relatively more elevated during the 48 hours after

TABLE II.—Serum GO-T and LDH Activity in 18 Patients with Probable Myocardial Infarction

Case No.	Sex and Age	Day First Specimen Received	Duration of GO-T Elevation	Duration of LDH Elevation
1	M 71	3	5	11
2	M 60	2	4	10
3	M 62	1	4	8
4	M 60	5	<5	9
5	M 69	6	<6	13
6	M 69	8	<8	<8
7	M 59	2	5	9
8	F 58	1	Till death on 5th day	
9	M 69	1	3	7
10	M 64	2	4	8
11	M 60	1	3	5
12	M 49	1	3	6
13	F 46	1	4	6
14	F 67	2	4	10
15	M 59	1	None	None
16	M 53	1	6	>9
17	F 61	1	5	11
18	M 65	2	<2	4