adequate oxygenation for as long as 15 minutes if Boyle's machine and an endotracheal tube are not immediately available (Mozen *et al.*, 1956; Nickel and Gale, 1959). An emergency thoracotomy is much easier to perform than, say, an emergency tracheostomy, and once cardiac massage has been started and an airway established expert anaesthetic and surgical assistance can be awaited in comparative leisure.

Looking back on this case, we are aware that it was mismanaged in several ways, partly through ignorance and partly through lack of thought at the time. The heart was massaged incorrectly, against the ball of the thumb instead of against the chest wall or thenar eminence; the foot of the couch was not raised to aid cerebral blood flow; procainamide or quinidine should have been administered rather than digitalis; neither blood nor saline should have been given in view of the risk of cerebral oedema; and too large doses of pethidine and phenothiazine derivatives were given in the immediate post-operative period. Yet, in spite of these errors, in spite of a previous transmural infarct, and in spite of 40 minutes of cardiac massage with no detectable pulse, the patient survived, and survived, moreover, without apparent cerebral damage. This would suggest that cardiac massage does not demand ideal circumstances and expert knowledge to be successful, and reinforces our belief that it should become the accepted treatment in hospital practice of cardiac arrest after myocardial infarction in reasonably young and fit patients.

Summary

The case is described of a man who developed cardiac arrest after his second myocardial infarction, and was successfully resuscitated by cardiac massage in a hospital casualty department.

A distinction is drawn between cardiac arrest and cardiac failure, and the reason why resuscitation may be possible after arrest is discussed.

It is suggested that immediate cardiac massage should be the recognized treatment in hospital practice of cardiac arrest after myocardial infarction.

It is stressed that the opportunity for resuscitation after cardiac arrest will normally be presented to junior resident staff and that all house officers should therefore be acquainted with the principles of cardiac massage.

[ADDENDUM.—Ten months later the patient is in good health, with no dyspnoea or angina, and has returned to a light job with his previous employers.]

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THE ORAL GLUCOSE-TOLERANCE TEST IN TROPICAL MALABSORPTION SYNDROME

BY

K. T. RAJAN, M.B., B.S.

P. S. S. RAO, M.A.

I. PONNUSAMY, B.Sc.

AND

S. J. BAKER, M.D.

*The Wellcome Research Unit, Christian Medical
College Hospital, Vellore, South India*

Thaysen (1926) showed that when glucose was administered by mouth to patients suffering from idiopathic steatorrhoea the rise in blood-sugar levels was lower than would be expected in normal people. Subsequently the glucose-tolerance test has been widely used as an index of intestinal function in various disorders of the small intestine, and not least in the investigation of tropical sprue and tropical malabsorption syndrome (Serra, 1929; Thaysen and Norgaard, 1929; Fairley and Bromfield, 1932; Fairley, 1936; Frazer, 1950; Gardner, 1956; Baker, 1957).

A maximum rise of 40 mg./100 ml. in the blood sugar in the glucose-tolerance test (G.T.T.) is often taken as the limit of normal, any rise less than this being regarded as abnormally low or "flat." In studying subjects without any evidence of malabsorption we were struck with the frequent occurrence of a "flat" G.T.T. result as defined above, and therefore undertook a comparative study of G.T.T.s in patients with tropical malabsorption syndrome and in normal controls. This report presents the results of this study.

Materials and Methods

Subjects Studied.—Fifty consecutive cases of tropical malabsorption syndrome with steatorrhoea (a fat excretion of more than 5 g. a day on a 50-g. intake) were used for this study. These patients were all Indians, of differing social status, and with symptoms and signs covering the whole range of clinical pictures from the mildly affected almost symptomless cases to those with fully developed classical "sprue" (Baker, 1957). Twenty-four subjects living on a high-carbohydrate diet associated with rice-eating, in good health, and without any evidence of intestinal disease, were used as suitable controls.

Oral Glucose-tolerance Tests.—Subjects fasted for 10 hours before the test. An initial sample of venous blood was collected, and then the subjects drank 50 g. of glucose dissolved in 200 ml. of water. Thereafter blood samples were drawn at 30-minute intervals for a period of two hours. Blood-sugar levels were estimated by the method of Folin and Wu (1920) modified to give true glucose values. The "rise" in the G.T.T. was defined as the highest blood-sugar level recorded in the test (usually at half or one hour) minus the fasting level.

Results

Height of "Rise" in Glucose-tolerance Test.—As a group the patients with malabsorption syndrome have a lower rise of blood sugar above the fasting level than do

the controls (see Chart). The characteristics of the two groups are set out in Table I. The mean rise in the group of patients is 22.6 mg./100 ml., and in the control group 44.6 mg./100 ml. After obtaining the best

Height of maximum rise in blood-sugar levels in glucose-tolerance tests performed on patients with tropical malabsorption syndrome and normal controls, plotted against the level of the fasting blood sugar. The dotted lines at 25 mg. and 80 mg./100 ml. represent values referred to in the text.

estimate of the variance, the difference of these means was tested for significance, using the *t* distribution. The difference between the two groups is highly significant at the 0.1% level.

Level of Fasting Blood Sugar.—As a group the patients with malabsorption syndrome have a lower fasting blood-sugar level than do the controls (see Chart). The mean fasting blood sugar of the patients is 72.4 mg./100 ml., while in the control group it is 93.8 mg./100 ml. The characteristics of the

TABLE I.—Maximum Rise in Venous Blood Sugar Above Fasting Level in the Two Groups

	Patients	Controls
No. of cases	50	24
Range of blood-sugar rise (mg./100 ml.)	4–65	11–111
Median (mg./100 ml.)	20.00	40.50
Mean $\pm$ standard error (mg./100 ml.)	22.61 $\pm$ 2.09	44.56 $\pm$ 4.86

TABLE II.—Fasting Blood-sugar Levels in the Two Groups

	Patients	Controls
No. of cases	50	24
Range of fasting level (mg./100 ml.)	40–109	75–114
Median (mg./100 ml.)	74	96
Mean $\pm$ standard error (mg./100 ml.)	72.38 $\pm$ 2.03	93.85 $\pm$ 2.51

Discussion

Comparison with Results of Other Workers

Numerous factors are known to influence the results of a G.T.T., such as the site of blood-sampling, the diet of the individual prior to the test, age, exercise, etc. It is therefore difficult to compare the results obtained by different investigators.

Thaysen and Norgaard (1929) defined a "flat" result as one where the rise in blood sugar after the administration of 50 g. of glucose was 40 mg./100 ml. or less. They found that 5% of normal people and 50% of people with "idiopathic steatorrhoea" had a "flat" G.T.T. according to this definition. This figure of 40 mg./100 ml. has often been taken as the dividing line between normal and abnormal by subsequent workers.

It is evident that our figures are quite different from those of Thaysen and Norgaard (1929), since 50% of our normals have a rise of 40 mg./100 ml. or less. One of the chief reasons for this difference is probably that we estimated glucose in venous blood, whereas Thaysen

and Norgaard used capillary blood. Hagedorn (1921) and subsequently others—for example, Cavett and Seljeskog (1933)—have shown that the use of venous blood for blood-sugar estimations results in a slightly lower fasting level and a flatter curve in the G.T.T.

Watson (1938) made a study of G.T.T.s in 83 normal people, using venous blood for the estimations. He found a mean rise of 42.9 mg./100 ml., which agrees fairly closely with our figure of 44.6 mg./100 ml. in the control group. Gardner (1956), also using venous blood for the estimations, found that 45% of apparently normal subjects had a rise in blood sugar of 40 mg./100 ml. or less in the G.T.T. A similar incidence of "flat" curves (with a maximum rise of 40 mg./100 ml. or less) has been reported by Moyer and Womack (1950) in apparently normal people in Texas. These figures are in agreement with our finding of 50% of normal subjects with a rise of 40 mg./100 ml. or less.

Statistical analysis of our results shows that there is a highly significant difference between the height of the mean rise in the two groups—patients and controls. However, when using the G.T.T. as a diagnostic aid in a given patient the distinction is not clear-cut (see Chart). From our results, and the work of others quoted above, it is clear that for venous samples the level of 40 mg./100 ml. is not a useful dividing line. We have now adopted the figure of 25 mg./100 ml. suggested by Gardner (1956)—a rise of blood sugar of 25 mg./100 ml. or less in the G.T.T. being considered abnormally flat. Thus defined, a "flat" curve was present in 16% of our normal subjects, and in 66% of our patients with tropical malabsorption syndrome. Our figure of 16% of normal people with a rise of 25 mg./100 ml. or less is comparable with the figures found by Gardner (1956) and by Moyer and Womack (1950) (Table III).

The four apparently normal subjects who had a rise of only 25 mg. or less were investigated further, but did not show any steatorrhoea or radiological changes in the small intestine. This, however, does not rule out the possibility that they may have had some abnormality

TABLE III.—Comparison of the Percentage of Normal Individuals with Maximum Rise in the G.T.T. of 40 mg./100 ml. or under, and 25 mg./100 ml. or under, in the Present Series, and in the Series of Gardner (1956) and Moyer and Womack (1950)

	Present	Gardner	Moyer
Rise of 40 mg./100 ml. or under	50	45	40
" 25 " " "	16	22	10

of their intestine preventing proper absorption of glucose, since absorptive defects can occur in the absence of steatorrhoea (Baker, 1958) and of radiological changes (Paterson and Baker, 1958).

Gardner (1956) investigated 21 patients suffering from malabsorption syndrome in Puerto Rico and found that all of them had an abnormal G.T.T., whereas only 66% of our patients had an abnormal G.T.T. (a rise of less than 25 mg./100 ml.).

Aetiology of Flat Glucose-tolerance Test in Malabsorption Syndrome

Numerous theories have been advanced to account for the flat G.T.T. in patients with malabsorption syndrome. Delay in gastric emptying has been held responsible (May and McCreary, 1940), but it has been shown by Frazer *et al.* (1952) that if glucose solution

is administered direct into the duodenum the result of the G.T.T. is unchanged. Excessive secretion of mucus (Frazer *et al.*, 1952), decreased intestinal motility, bacterial invasion of the small intestine (Herter, 1909), and faulty phosphorylation (Verzár and McDougall, 1936) have all been blamed for impaired glucose absorption. However, none of these theories appear to offer a really satisfactory explanation of observed phenomena. Not all patients with evidence of excessive mucus production, as judged by the flocculation of barium sulphate in a barium-meal study, have a flat G.T.T. (Paterson and Baker, 1958, Fig. 3B). Conversely, as shown by the following case history, not all patients with steatorrhoea and a flat G.T.T. show radiological evidence of excessive mucus production.

A man aged 27 complained of diarrhoea for the previous 10 weeks. On a fat balance with an intake of 50 g. a day he excreted an average of 13 g. of fat a day. A G.T.T. carried out on two occasions showed a maximum rise of 7 and 10 mg./100 ml., respectively. Barium-meal examination was performed, using, first, a non-flocculating medium, and subsequently ordinary barium sulphate suspension, and on each occasion the results were entirely normal.

If decreased motility were the cause of the flat G.T.T., then all patients with decreased motility from any cause should have a flat G.T.T., but, as the following case history shows, this is not always the case.

A married woman aged 37 complained of vague upper abdominal pain, associated with flatulence, and alternate bouts of "diarrhoea and constipation." Clinical examination revealed nothing abnormal except some gaseous abdominal distension. A G.T.T. showed a maximum rise of 80 mg./100 ml. Barium-meal examination showed considerable delay in the passage of the barium through the small intestine, the mouth-to-ileum time being three and a half hours.

That it is not always due to bacterial invasion is suggested by the work of Anderson and Langford (1958), who found little evidence of bacterial invasion of the upper intestine in children with coeliac disease. Also in three patients with malabsorption syndrome not included in this series, who had flat G.T.T.s and who were submitted to laparotomy for other reasons, the upper small intestine was studied by the direct sampling method of Cregan and Hayward (1953) and found in each case to be sterile (Baker and Achaya, 1961).

A defect of phosphorylation would not explain the defective absorption of other substances—for example, urea—which frequently occurs in association with a flat G.T.T. (Frazer *et al.*, 1952).

A further possible reason for a flat G.T.T. might be the rapid removal of glucose from the blood-stream. The blood-sugar curves obtained after intravenous administration of glucose in the sprue syndrome have usually been stated to be normal (Ross, 1936; Fairley, 1936). However, Thaysen (1932) reported that in a case of non-tropical sprue there was an increased tolerance to intravenously administered glucose. Preliminary investigations carried out in this unit show that, at least in some cases, the intravenous G.T.T. is flat as well as the oral test (Pulimood *et al.*, 1961). Should this be confirmed by further study, it will suggest that there are factors involved in the production of the flat G.T.T. other than those which might just interfere with absorption.

Low Fasting Blood Sugar

The finding of a statistically significant lower fasting blood sugar in the group of patients as compared with

the control group is of interest. This is further suggestive evidence that there is a more profound disturbance of carbohydrate metabolism than can be explained merely by interference with absorptive mechanisms. Inspection of the results published by Gardner (1956) also reveals a lower fasting level in his group of patients than in his control group.

The low fasting blood sugar in the malabsorption group may be used as a further aid to the interpretation of G.T.T. results in individual patients. In this series a fasting blood sugar (venous blood) of 80 mg./100 ml. or less was found in only 8% of normal people but in 76% of people with tropical malabsorption syndrome (see Chart).

Summary

Oral glucose-tolerance tests, using samples of venous blood for the glucose estimations, have been carried out on 50 patients with tropical malabsorption syndrome and 24 controls, and the results compared statistically.

The mean maximum rise in blood sugar in the tropical malabsorption group (22.6 mg./100 ml.) was significantly less than that in the control group (44.6 mg./100 ml.). A maximum rise of 25 mg./100 ml. or less was found in 16% of the control group, and in 66% of the tropical malabsorption group.

The fasting blood sugar was significantly less in the tropical malabsorption group (72.4 mg./100 ml.) than in the control group (93.8 mg./100 ml.). A fasting blood sugar of 80 mg./100 ml. or less was found in 8% of the control group and in 76% of the tropical malabsorption group.

Both fasting blood-sugar levels and the height of the blood-sugar rise should be taken into consideration in interpreting glucose-tolerance tests in cases of possible malabsorption syndrome.

The results are compared with those of other workers, and possible explanations for the findings are discussed.

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