Technical methods

Oral butter fat test meal with serum nephelometry in suspected fat malabsorption

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There is a need for a simple test to exclude fat malabsorption so that the number of faecal fat estimations can be reduced, with increased justification for adequate preparation of the patient and more careful faecal collection. The Thorp micronephelometer was developed for the detection and investigation of hyperlipidaemia (Stone and Thorp, 1966). In the present study the micronephelometer has been used to measure changes in serum lipids following the standard oral butter fat test meal of Kabler, Atwood, and Schilling (1959), a satisfactory increase in serum lipids suggesting that there is no fat malabsorption.

Methods and Materials

Subjects were fasted overnight for not less than 10 hours. After a fasting sample of blood had been taken, they were given orange juice (about 50 ml), two or three rounds of toast with pure butter (0.5 g per kg body weight), and one or two cups of unsweetened coffee or tea, with milk if desired. All volunteers and patients found the breakfast palatable, a previous fat tolerance test having been found unpalatable (Penfold, 1967). A second sample of blood was taken after two hours. Initially a third blood sample was taken at three hours, since Gardner and Santiago (1966) found maximum serum chylomicron levels at two to three hours after a fat meal, but this third sample was abandoned when it was found that chylomicron clearance was well advanced by this time after the butter meal. Serum was diluted 1:10 with 0.85% saline. In 41 tests full estimations of both increases in chylomicra and in pre-beta-lipoprotein levels were carried out (Stone and Thorp, 1966). There was very little change in serum pre-beta-lipoprotein levels at two hours, and

the total LSI increase correlated directly with the serum chylomicron increase alone ($y = 0.925x, 0.912$, $r = 0.985$). A simpler, more rapid procedure was therefore used in 106 tests, including one reading on fasting diluted serum and a second reading two hours after the meal, results being expressed as increases in LSI units after two hours.

Forty-four control subjects with no obvious gastrointestinal disorders included male and female volunteers from the medical staff, nursing staff, laboratory technicians, and some patients, with ages ranging from 22 to 65 years. Sixty-two patients with gastrointestinal disorders included 36 patients under investigation for diarrhoea, ulcerative colitis, diverticular disease, and other disorders without overt malabsorption, 16 cases of coeliac disease treated with gluten-free diet, and one case not so treated, and 15 patients suffering from malabsorption. Faecal fat estimations on three-day collections of faeces, made with patients eating the ordinary hospital diet and without markers, were carried out by the method of van de Kamer, Huinink, and Weyers (1949), in parallel with the oral butter test on 30 patients. In a further five patients as the faecal weight after three days' collection was less than 200 g, no fat estimations were carried out. Collecting faeces for fat estimations was not possible in some other patients because of diarrhoea.

Results

When the two-hour increase in LSI units was plotted against the three-day faecal fat output in 30 patients (fig 1), a poor indirect correlation was found ($y = 41.3 - 0.6x, r = 0.33$; standard error of distribution = 0.19, $p > 0.05$). When faecal fat excretion exceeded 20 g per three days, no serum LSI increases exceeded 20 units in 13 patients.

In fig 2 the results of 106 LSI increases following butter fat test meals are shown. Two normal subjects with no overt gastrointestinal disorder had high initial serum LSI units after overnight fasting (146 and 196 LSI units respectively) with no subsequent increase after oral butter; the mean fasting value for the other 42 control subjects was $52.4 \pm 52.8$ (2 SD) LSI units.

Two-hour increases in serum LSI units were
found to be less than 20 in one treated coeliac disease patient and in one untreated patient, with increases of more than 20 LSI units in nine treated patients. In 21 patients with gastrointestinal disorders without clinical evidence of malabsorption, 19 patients had two-hour increases of 20 or more LSI units, while in the 30 patients suspected of suffering from malabsorption, eight out of 15 patients with a three-day faecal fat excretion of 15 g or less had two-hour increases of serum LSI units of less than 20, and 14 of 15 patients with a three-day faecal fat excretion of more than 15 g had two-hour increases of serum LSI units of 20 or less, with 12 patients having an increase of less than 10 units.

It appears that the butter meal can be used as a preliminary test before faecal fat estimation. If the butter fat meal is followed by a reduced serum lipid increase in a patient, it would then be justifiable to place that patient on a standard diet with a known fat content, for adequate markers to be given, and a careful three-day faecal fat estimation be carried out. Patients in whom a satisfactory increase in serum lipids of more than 20 LSI units at two hours was found would not require faecal fat estimations, unless otherwise clinically indicated.

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References


