Summary

The value of radiotriolein in the diagnosis of steatorrhoea was assessed in 100 subjects by comparing the faecal radiotriolein excretion with concurrent faecal chemical fat determinations.

Sixty-seven cases did not have steatorrhoea as judged by a faecal chemical fat excretion below 7 g. daily. In these the upper limit of faecal radiotriolein excretion was 5% of the administered dose.

Thirty-three cases had steatorrhoea as judged by the faecal chemical fat excretion. Of these, 15 excreted more than 5% of the dose of radiotriolein. The remaining 18 excreted less than 5% and were not differentiated from the normal material.

Analysis of these results failed to explain the discrepancies. Comparison with similar studies has shown that some other workers have also found the radiotriolein test to be unreliable in the diagnosis of steatorrhoea.

If technical errors are the explanation of the false results, the practical value of the radiotriolein test is lessened.

Several possible defects and a few alternative applications of the radiotriolein test are discussed.

This study was assisted by the Tuberculosis Research Fund of the University of Sheffield. I am indebted to Professor A. W. Kay for invaluable advice and encouragement throughout; to the following consultants for allowing me to study their patients: Dr. H. P. Brody, Dr. C. E. Davies, Mr. F. J. P. O'Gorman, Dr. E. G. G. Rhind, Dr. E. W. Skipper, and Professor G. M. Wilson; to Dr. A. Jordan for faecal chemical fat determinations performed in his department; to Mrs. O. Fisher for guidance in radioisotope techniques; to Messrs. A. Bird and Sons Ltd. for supplies of "instant whip"; and to Miss R. Chalmers and Miss Z. Hinchliffe for technical assistance.

REFERENCES

- Annegers, J. H., Boutwell, J. H., and Ivy, A. C. (1948). *Gastroenterology*, **10**, 486.
 Balint, J., Pendower, J., and Ramsey, N. W. (1960). *Clin. Sci.*, **19**, 321.
 Beres, P., Wenger, J., and Kirsner, J. B. (1957). *Gastroenterology*, **32**, 1.
 Berkowitz, D., Sklaroff, D., Woldow, A., Jacobs, A. G., and Likoff, W. (1959). *Ann. intern. Med.*, **50**, 247.
 Berson, S. A., and Yalow, R. S. (1954). *J. clin. Invest.*, **33**, 1533.
 Chears, W. C., McGraw, B. H., Tyor, M. P., and Ruffin, J. M. (1958). *Sth. med. J. (Bgham, Ala.)*, **51**, 433.
 Clark, B., and Hübscher, G. (1960). *Nature (Lond.)*, **185**, 35.
 Comfort, M. W., Wollaeger, E. E., and Taylor, A. B. (1953). *Gastroenterology*, **23**, 155.
 Cooke, W. T., Thomas, G., Mangall, D., and Cross, H. (1953). *Clin. Sci.*, **12**, 223.
 Cox, A. G., and Hinchliffe, Z. (1961). *Gut*, **2**, 131.
 Fast, B. B., Wolfe, S. J., Stormont, J. M., and Davidson, C. S. (1959). *Gastroenterology*, **37**, 321.
 Fierst, S. M., Feldman, S. M., Solomon, N., and Langsam, A. (1958). *Ibid.*, **35**, 381.
 Frazer, A. C. (1959). *Proceedings of World Congress of Gastroenterology*, 1958, **1**, 619.
 Grossman, M. I., and Jordan, P. H. (1958). *Gastroenterology*, **34**, 892.
 Hall, G. V., George, E. P., and Hickie, J. B. (1959). *Aust. Ann. Med.*, **8**, 307.
 Jiménez-Díaz, C., Linazasoro, J. M., Marina, C., and Romeo, J. M. (1959). *Bull. Inst. med. Res. (Mad.)*, **12**, 41.
 Jones, R. V. (1960). *Brit. med. J.*, **2**, 1236.
 Krakower, A. (1934). *Amer. J. Physiol.*, **107**, 49.
 Lagerlöf, H. O. (1942). *Acta. med. scand.*, Suppl. 128, pp. 55-61.
 Lamberg, B.-A., Siurala, M., Turula, K., Hakkila, R., and Harjanne, A. (1960). *Scand. J. clin. Lab. Invest.*, **12**, 107.
 Lewis, G. T., and Partin, H. C. (1954). *J. Lab. clin. Med.*, **44**, 91.
 Lubran, M. (1960). *Lancet*, **2**, 907.
 — and Pearson, J. D. (1958). *J. clin. Path.*, **11**, 165.
 McClure, C. W., Huntsinger, M. E., and Fernald, A. T. (1934). *Amer. J. Physiol.*, **107**, 1.
 Mäkelä, T. E., Hakkila, R. L., and Hakkila, J. (1960). *Acta med. scand.*, **167**, 121.

- Pimparkar, B. D., Tulskey, E. G., Kalser, M. H., and Bockus, H. L. (1960). *Brit. med. J.*, **2**, 894.
 Playoust, M. R., Wyatt, J. V., and Blackburn, C. R. B. (1959). *Aust. Ann. Med.*, **8**, 245.
 Reemtsma, K., Malm, J. R., and Barker, H. G. (1957). *Surgery*, **42**, 22.
 Ruffin, J. M., Shingleton, W. W., Baylin, G. J., Hymans, J. C., Isley, J. K., Sanders, A. P., and Sohmer, M. F. (1956). *New Engl. J. Med.*, **255**, 594.
 Rutenburg, A. M., Seligman, A. M., and Fine, J. (1949). *J. clin. Invest.*, **28**, 1110.
 Sammons, H. G., Vaughan, D. J., and Frazer, A. C. (1956). *Nature (Lond.)*, **177**, 237.
 Sandberg, H., Min, B. S., Feinberg, L., and Bellet, S. (1960). *Arch. intern. Med.*, **105**, 866.
 Stanley, M. M., and Thannhauser, S. J. (1949). *J. Lab. clin. Med.*, **34**, 1634.
 Turner, D. A. (1958). *Amer. J. dig. Dis.*, **3**, 594.
 van de Kamer, J. H., Huinink, H. ten B., and Weyers, H. A. (1949). *J. Biol. Chem.*, **177**, 347.
 van Handel, E., and Zilvermit, D. B. (1958). *J. Lab. clin. Med.*, **52**, 831.
 van Middlesworth, L. (1960). *Recent Progr. Hormone Res.*, **16**, 405.
 Veall, N., and Vetter, H. (1958). *Radioisotope Techniques in Clinical Research and Diagnosis*. Butterworth, London.
 Walker, W. F., Stewart, W. K., Morgan, H. G., and McKie, J. (1960). *Brit. med. J.*, **1**, 1403.
 Wollaeger, E. E., Comfort, M. W., and Osterberg, A. E. (1947). *Gastroenterology*, **9**, 272.
 — Lundberg, W. O., Chipault, J. R., and Mason, H. L. (1953). *Ibid.*, **24**, 422.

Medical Memoranda

Perforated Peptic Ulcer in Late Pregnancy

Peptic ulcer rarely occurs in pregnancy. This had probably been known to the astute physician since time immemorial, but Avery Jones's (1947) figures came to most as a surprise. He was unable to find a single case of peptic ulcer occurring among 10,000 pregnancies and 2,000 abortions reviewed. If peptic ulcer is rare, complications of peptic ulcer are, not unnaturally, exceedingly rare, and when Sandweiss *et al.* (1943) reviewed the world literature they were able to find only 13 recorded cases of complications of peptic ulcer occurring during pregnancy—nine perforations and four haemorrhages.

In this country two cases of perforated peptic ulcer in late pregnancy have been recorded during the past 13 years. James's (1948) case occurred at the 36th week, and ended satisfactorily for mother and child. The perforation was repaired, and the mother was delivered of a live child four days later. Both did well. Ten years later a case was described by Horwich (1958), occurring in the 32nd week. Conservative treatment was adopted, but this was not successful, a stillborn child being born the following day. Laparotomy was eventually necessary 60 hours after the onset of the perforation. The perforation was sutured, and after a very stormy convalescence the patient recovered.

The present case, in which twins were born the day after a partial gastrectomy, would appear to be unique.

CASE HISTORY

A married woman, aged 38, and 31 weeks pregnant, was admitted to the Maternity Department of Upton Hospital on January 7, 1961, as a case of suspected hyperemesis gravidarum. She had been vomiting persistently for three weeks, prior to which the pregnancy had been normal. A year previously she had had some abdominal pain, for which she had taken medicine; but she was not a known case of peptic ulcer, though there was a family history of it. In the early hours of the morning following her admission she began to complain of severe abdominal pain, and Dr. Ralph Evans, the house-surgeon, wisely suspected the possibility of a perforated peptic ulcer. When I saw

her she was extremely ill. She was only semiconscious, and looked toxic and dehydrated. A rigid and retracted epigastrium brought her large uterus into grotesque relief. There was little doubt regarding the diagnosis, and this was confirmed radiologically. At operation she was found to have an enormous perforation of such an extensive duodenal ulcer that I was in no doubt whatever that the correct procedure was a partial gastrectomy. Despite limited exposure and a horrid duodenum, the operation was not difficult. This was doubtless due to the excellence of my anaesthetist and assistant, but I should like to recommend very strongly a left-sided stance when faced with mobilization of a difficult duodenum: this is the only difficult step in any partial gastrectomy, and I always do these cases from the left, just as one stands on the right when mobilizing a spleen. Perhaps with me there is the added feature of familiarity. Having spent so many years standing on the left to assist other surgeons to do gastrectomies, one tends to remain there, like the cat who remains on the hearth even when the fire has gone out.

The patient went into labour next morning, and was delivered of live twins. Unfortunately one of these died, but the other survived. The patient herself made a quick recovery, and in less than a week was up and about and eating an almost normal diet. She remained in hospital a further two weeks, but this was only because she had no immediate home to which to go. Her rapid recovery was perhaps partly due to the natural resilience of pregnancy and partly to the patient's innate toughness—she was a native of the Shetland Islands!

I should like to thank Mr. Tom Ealand, under whose care the case was admitted, and Dr. Kimmet for an excellent anaesthetic in rather trying circumstances.

ROBIN BURKITT, M.A., M.D., F.R.C.S., F.R.C.S.I.,
Upton Hospital, Slough.

REFERENCES

- Horwich, M. (1958). *Brit. med. J.*, **2**, 145.
James, D. W. (1948). *Ibid.*, **2**, 74.
Jones, F. Avery (1947). *Ibid.*, **2**, 479.
Sandweiss, D. J., Podolsky, H. M., Salzstein, H. C., and Farberman, A. A. (1943). *Amer. J. Obstet. Gynec.*, **45**, 131.

Home-made Wine as a Cause of Lead-poisoning: Report of Case

Wine-making at home is a hobby enjoying a revived popularity and there are quite a number of publications on this subject. In these, details and advice are given on the container to be used for the fermentation, and from time to time one finds the reader encouraged to seek out in antique or junk shops large old crocks, which are regarded as being eminently suitable for the brew. However, it has been shown by Prior and Whitehead (1960) that some of these aged vessels are highly dangerous owing to the high lead content of their interior glazing, and an account is given here of another case of severe lead-poisoning from this cause. Although this is an uncommon cause of lead-poisoning in this country, it is still regarded as a serious problem in other countries (Beritić and Stahuljak, 1961).

CASE REPORT

The patient, a gardener aged 52, was admitted to hospital as a surgical abdominal emergency, ? intestinal obstruction. He gave a history of loss of appetite for one month and constipation for one week before admission. He had vomited a few times during the three days before we saw him, and had then developed some colicky lower-abdominal pain.

On examination he was rather dull and listless, and said he felt weak. Temperature 99° F. (37.2° C.). Pulse 100.

Tongue dry and furred. Gums showed no significant abnormality. Abdomen very obese, tenderness in the right iliac fossa and centrally. Liver and spleen not palpable. Rectal examination negative. Blood-pressure 140/80. Heart normal. The other systems were clinically normal.

It was decided to prepare the patient for operation for possible intestinal obstruction as a straight x-ray film of the abdomen was thought to suggest small-bowel obstruction. However, a routine blood count showed: haemoglobin, 62% (9.05 g./100 ml.); white blood count, 13,000/c.mm. (polymorphs 68%, lymphocytes 26%, monocytes 6%). Film: the red cells showed anisocytosis and marked polychromasia and punctate basophilia; some late normoblasts were seen. The blood picture was very suggestive of a haemolytic anaemia. Further investigations showed: direct Coombs test negative; red cells negative for the presence of Heinz bodies; red-cell fragility within normal limits; serum bilirubin 1.9 mg./100 ml.; E.S.R. 12 mm. in 1 hour.

The possibility that this might be a case of lead-poisoning was discussed with the surgeons, and the operation was postponed while further investigations were carried out. The patient would not admit to having used or been in contact with any lead-containing compounds in his work or elsewhere, and at this time only admitted to moderate drinking of draught cider (proprietary brands, not home-made). The urine gave strongly positive qualitative tests for both coproporphyrins and uroporphyrin. A specimen of urine submitted for lead content was found to contain 0.3 mg./litre—that is, about ten times the quantity normally present in unexposed persons, and the amount found in persons with clinical symptoms of lead-poisoning.

A firm diagnosis of lead-poisoning was now made, and treatment with calcium disodium versenate was begun, 2 g. daily for seven days. A more intensive search was made for the source of the lead, but investigation of garden sprays used by the patient, greenhouse paint, domestic water and garden standpipe water, and cider from the draught-cider cask all proved negative for any significant amounts of lead. The patient responded well to treatment, and three weeks after admission his haemoglobin had risen to 86%, though the red cells still showed some punctate basophilia. He was symptom-free and much brighter. On being questioned once more with regard to his habits and hobbies he mentioned the fact that he made wine at home, but denied using a lead-containing vessel. However, as he was ready for discharge, arrangements were made to obtain some of the remains of the wine which he had been drinking, and also to examine the container he had used for the brew.

The wine he had been drinking for some time before his admission to hospital was damson wine prepared by himself more than a year before his admission to hospital. He had made 5 gallons (22.8 litres) of this wine and had consumed nearly all of it in the months before his admission, at the rate of 2–3 pints (1.1–1.7 litres) a week. Fortunately he had a little wine and sediment left in one bottle, so a sample of this was analysed for lead content. If there had been any doubts about the diagnosis, these were certainly dispelled by finding a concentration of 120 mg./litre.

The wine had been prepared in a large old bread crock which had been given to him by his employer, in whose family's possession it had been for at least 70 years. The fruit had been placed in the crock and then covered with boiling water. It was then left for ten days, after which the liquor was separated and replaced in the crock, then sugar and yeast were added, and the whole brew was allowed to ferment for about two to three months. After completion of the fermentation it was bottled.

The patient was informed of our findings and advised not to use this crock again for wine-making and wisely broke the crock up, but, unfortunately, before we had had an opportunity to examine it whole; however, we managed to obtain a few pieces. Some of the glaze was scraped off these and the scrapings were placed in very dilute hydrochloric acid at 37° C. overnight. The resultant solution gave very strong standard analytical tests for lead. The crock