local treatment is a prime necessity, and the apparent lack of any great interest in a controlled series, suggest that the benefits claimed may be due to the usual treatment, with perhaps a dash of enthusiasm.

The investigations reported here, in which all personal bias and normally varying factors were removed so far as was possible, have failed to substantiate the claims that the so-called "vitamin F" has any value in the treatment of atopic and infantile eczema. The findings of Vachon seem to be borne out, in that the ointment almost invariably irritated the eczematous skin.

Summary

A brief summary of the literature on the use of unsaturated fatty acids in the treatment of infantile and atopic eczema is followed by the details of a recent investigation.

The combined use of linoleic and linolenic acid ointment and capsules and the use of the capsules as an adjuvant to routine treatment were in no way preferable to the established methods of treatment.

My thanks are due to Dr. G. B. Dowling for allowing me to see and investigate his cases, and to the matron and nursing staff of the Goldie Leigh Hospital for their willing co-operation.

REFERENCES

Alechinsky, A. (1950). *Arch. belges Derm. Syph.*, **6**, 56.
Azerad, E., and Grupper, C. (1949a). *Sem. Hôp., Paris*, **25**, 684.
—— —— (1949b). *Gaz. méd. France*, **56**, 307.
Burr, G. O., and Burr, M. M. (1929). *J. biol. Chem.*, **82**, 345.
—— —— (1930). Ibid., **86**, 587.
—— —— and Miller, E. S. (1932). Ibid., **97**, 1.
Carletti, B., and Scalamogna, G. (1952). *Minerva pediat, Torino*, **4**, 76.
Charpy, L. (1948). *Bull. Soc. méd. Hôp. Paris*, **64**, 410.
Cornbleet, T. (1935). *Arch. Derm. Syph., Chicago*, **31**, 224.
Dubois, L. (1950) *Méd. et Hyg., Genève*, **8**, 388.
Faber, H. K., and Roberts, D. B. (1933). *J. Pediat.*, **3**, 78.
—— —— (1935). Ibid., **6**, 490.
Finnerud, C. W., Kesler, R. L., and Weise, H. F. (1941). *Arch. Derm. Syph., Chicago*, **44**, 849.
Finzi, M. (1951). *Policlinico, Sez. med.*, **58**, 408, 416.
Ginsberg, J. E., and Bernstein, C. (1937). *Arch. Derm. Syph., Chicago*, **36**, 1033.
Hansen, A. E. (1933). *Proc. Soc. exp. Biol., N.Y.*, **31**, 160, 161.
—— Knott, E. M., *et al.* (1947). *Amer. J. Dis. Child.*, **73**, 1.
Justin-Besançon, L., and Laroche, C. (1942). *Presse méd.*, **50**, 373.
Pätiälä, R. (1948). *Ann. Derm. Syph., Paris*, **8**, 146.
Taub, S. J., and Zakon, S. J. (1935). *J. Amer. med. Ass.*, **105**, 1675.
Touraine, A. (1949). *Concours méd.*, **71**, 739.
Vachon, R. (1948). *Bull. Soc. franç. Derm. Syph.*, **55**, 291. Quoted by Touraine (1949).

Medical Memoranda

Total Gastrectomy for Haematemesis

The object of this report is to point out the necessity for total gastrectomy in certain rare cases of haematemesis and to stress the value of oesophagoscopy in diagnosing the source of the haemorrhage.

Obvious bleeding occurs in some 20% of all peptic ulcers, and these form about 85% of all cases of haematemesis and melaena. The total mortality rate in the peptic ulcer group is roughly 8%, but in patients under 45 it is 2%. Death more often takes place from irreversible changes following prolonged anoxia than from acute anaemia, as necropsy nearly always shows the vessel occluded by clot. Hence the sheet anchor of treatment is blood transfusion, not only to prevent anoxia but also to enable the patient to withstand further haemorrhage by keeping the haemoglobin level about 80%. If this rule is followed the so-called critical periods on the third day or at any other time cease to exist, and surgery is indicated when replacement does not control the loss or if the loss continues or recurs for several days.

HAEMORRHAGE FROM FUNDUS OF STOMACH AND LOWER OESOPHAGUS

The importance of haemorrhage from the fundus of the stomach was first impressed upon me by a patient, known to be suffering from a chronic gastric ulcer for some years, who was admitted with severe haematemesis which did not respond satisfactorily to transfusion. A partial gastrectomy was successful, but the specimen showed that the bleeding was not from the known chronic ulcer but from a small acute ulcer high up the lesser curvature which had been included in the resected area by luck, not judgment. This acute ulcer was not palpable to the gloved finger, and I doubt if it would have been noticed on gastrotomy, which in other cases has proved to be of no value.

Further, the importance to the differential diagnosis of the site of the haemorrhage was brought home by another man, known to have a chronic gastric ulcer, who was admitted with severe haematemesis not controlled by transfusion. At partial gastrectomy cirrhosis of the liver was noted. He died five days later from continued bleeding from oesophageal varices. No evidence of haemorrhage from the ulcer was found. This error might have been prevented by an oesophagoscopic examination, in which it is simple to distinguish a normal oesophagus, a normal oesophagus with regurgitated blood in it, bleeding oesophageal varices, or a stomach distended by gastric haemorrhage. In fact, since then it has saved me from making the same mistake again.

Those who are not trained in the use of the common instruments for the visualization of their patients' orifices will find no difficulty in passing an appropriate-sized oesophagoscope if they will first pass a stomach tube, aspirate, and then pass the endoscope alongside it before its removal.

Treatment.—This must be governed by the general principles already mentioned. When surgery is indicated for gastric haemorrhage from acute ulceration it is no use temporizing by doing a gastro-enterostomy (as in my house-surgeon days) or a partial gastrectomy or ligation of gastric vessels (as is often done now). I know of deaths from continuing haemorrhage after these procedures have been carried out by competent surgeons and also of recovery when nothing has been done. Acute fundic ulceration is probably as common as is acute ulceration in any other part of the stomach, and unless at laparotomy the surgeon can determine the site of bleeding he is faced with only two logical procedures: nothing or total gastrectomy. The following case histories illustrate the problem.

CASE 1

An engineer aged 58 collapsed at work on March 16, 1952, and was admitted to Lodge Moor Hospital the next day as a case of "influenzal pneumonia." No cause for his collapse was found on admission, but 48 hours later he had a severe haematemesis. Blood transfusion was at once started, and he was transferred later in the day (March 19) to the City General Hospital. His pulse was 130 and blood pressure 105/60, and he was pale, cold, and clammy. Further transfusion was given, with improvement.

On March 21 he had further haematemesis and melaena in spite of a total of 8 pints (4.5 litres) of blood, so I was asked to see him with a view to surgery. His pulse was 84, blood pressure 85/55, Hb 8.8 g. (60%). He gave a history of several weeks of epigastric discomfort after food lasting an hour or so, and then complete freedom for several days before the next recurrence. I felt that the diagnosis of a chronic ulcer or carcinoma was very unlikely and suggested further conservative treatment.

However, on March 26 his condition deteriorated in spite of a total of 21 pints (11.9 litres) of blood, and laparotomy was advised.

First Operation.—On March 26, under intratracheal gas and oxygen, thiopentone, and D-tubocurarine chloride, a left upper paramedian incision was made. External exploration of the stomach and duodenum showed no lesion or evidence of scarring from an old lesion. The intestines were full of blood. The anterior stomach wall was incised from the lesser to the greater curvature roughly at its mid-point and the contents were aspirated. No fresh blood was present, but some adherent clot at the fundus was found. Nothing abnormal was seen or palpated though ungloved hands were used. The lower oesophagus also felt normal. The problem was total gastrectomy or nothing, and I chose the latter

course in the hope that the gastric haemorrhage, which was presumed to have come from the fundus, where adherent clot was found, would not recur.

The patient's condition improved for several days, though intermittent melaena occurred. On April 1 I passed an oesophagoscope into the stomach and found no evidence of bleeding or lesion in the oesophagus, but there was old clot in the stomach. Shortly after this he started to bleed severely, and as the consulting haematologist again found no evidence of blood dyscrasia I decided to do a total gastrectomy.

Second Operation.—On April 1, under a similar anaesthetic, the old incision was opened up and extended. There was a good deal of mild peritonitis due to the previous gastrotomy. Total gastrectomy was performed and a retrocolic jejunal loop was anastomosed to the oesophagus without entero-anastomosis (operative time two hours).

Recovery was uneventful and the patient was discharged on April 26 on an unrestricted diet. He had received 52½ pints (29.8 litres) of blood from March 19 to April 2. He was readmitted on February 3, 1953, with acute intestinal obstruction for which section of a solitary cord adhesion from the mesentery to the posterior abdominal wall was done. In June he was at work again, the blood counts, etc., being satisfactory.

Specimen.—The stomach showed about a dozen small erosions, three or four being in the pyloric region and the rest on the posterior wall of that part opposite to and above the oesophageal opening. The haemorrhage had occurred from the largest of these fundic lesions, which measured about 1 cm. in length and 2–3 mm. in width.

CASE 2

A crane-driver aged 48 was admitted to the City General Hospital on June 27, 1952, with severe haematemesis. His history was as follows: 1930, onset of symptoms of duodenal ulcer; 1941, pyloric obstruction—gastro-jejunostomy; 1947, severe haematemesis—14 pints (8 litres) blood transfused; 1950, further haematemesis—3½ pints (2 litres) blood transfused; 1951, high partial gastrectomy for two anastomotic ulcers. I first saw him at 9 p.m., after he had vomited 38 oz. (1 litre) of blood. His condition was improving under transfusion. The Hb was 62.5%, but, as it fell to 55% in the next twelve hours in spite of drip transfusion, I decided to operate without further delay though the rate of transfusion was increased.

Operation.—On March 28, under gas and oxygen, thiopentone, and D-tubocurarine anaesthesia, oesophagoscopy was carried out. The oesophagus was normal, but there was blood in the stomach. A left postero-lateral thoracotomy was done with resection of the eighth rib. Total gastrectomy was performed with considerable difficulty in 3 hours 10 minutes. Unfortunately, the patient died some 30 hours later, but the cause of the haemorrhage was acute ulceration of the fundus of the stomach.

JUDSON T. CHESTERMAN, F.R.C.S., F.A.C.S., M.R.C.P.

Two Cases of Ovarian Cyst in Childhood

The deceptive presentation of these cases—one as retention of urine and the other as appendicitis—is of clinical interest.

CASE 1

In October, 1950, a girl aged 3½ was admitted to hospital with acute retention of urine. A week previously she had had a sore throat and diarrhoea with much mucus in the stools; this cleared up after three days, but two days later she began to have frequency, and micturition was apparently difficult and painful. The urinary symptoms increased in severity and culminated in acute retention.

On examination of the abdomen a smooth rounded cystic swelling was found rising out of the pelvis half-way up to the umbilicus. This was taken to be the distended bladder. The external urethral orifice appeared normal, there was no fever or any evidence of a neurological lesion, and an *x*-ray film of the abdomen and pelvis showed nothing abnormal. With difficulty a catheter was passed and only a few millilitres of normal urine was obtained.

Under a general anaesthetic the cystic swelling previously described, which had been mistaken for the urinary bladder, became disimpacted from the pelvis, and at a subsequent laparotomy was shown to be a large multilocular teratoma of the left ovary, measuring 11 by 6.5 cm. It was removed without difficulty.

The mechanism of the production of urinary retention was possibly the same as that which occurs in retroflexion of the gravid uterus, whereby the urethra is compressed and elongated. The bladder, normally an abdominal viscus in young children, is easily displaced and compressed anteriorly by the impacted ovarian cyst. Disimpaction under anaesthesia allowed the bladder to empty easily and no further dysuria was experienced. Urinary disturbances occur not uncommonly in cases of ovarian cyst in children, but the occurrence of acute retention seems to be quite rare.

CASE 2

In June, 1951, a girl of 4 years 9 months was admitted to hospital with abdominal pain and vomiting. She had had three attacks of constipation and abdominal pain lasting two to three days in the past nine months. The present attack began three days before admission, with vomiting and lower abdominal pain. She had been constipated for three days and had been irritable and had a poor appetite. There were no urinary symptoms.

On examination she was seen to be flushed, with mild pyrexia and tachycardia. There was a tender rounded swelling arising out of the pelvis and extending 2 in. (5 cm.) above the symphysis pubis; slight muscular guarding was present over it. Rectal examination revealed a tender swelling palpable through the anterior wall of the rectum. A catheter was passed readily and 2 oz. (57 ml.) of urine was withdrawn. A diagnosis of pelvic abscess or cyst was made. Under general anaesthesia the swelling emerged from the pelvis and was freely mobile in the abdomen; it was evidently a cyst. The subsequent laparotomy revealed a right-sided unilocular ovarian cyst which had undergone torsion twice in a clockwise direction. It was removed. The left tube, the ovary, and the uterus were normal. Convalescence was uneventful, and the patient was discharged on the eighth day. On section a simple ovarian cyst with haemorrhage into the ovary was reported.

COMMENT

Study of reported cases shows that simple or multilocular cysts of the ovary account for one-quarter to one-third of all ovarian neoplasms in girls under 15 years, while granulosa-cell tumours produce symptoms of precocious puberty, and teratomas can sometimes be recognized by *x*-ray examination; ovarian cysts may remain unrecognized for a long time. Lower abdominal pain, distension, constipation, and frequency may occur. Torsion leading to acute symptoms may be the first indication of the presence of an ovarian cyst, and differential diagnosis from acute appendicitis may be very difficult; in fact, many series of reported cases show that this is the commonest pre-operative diagnosis, particularly as most cysts are on the right side. Steel (1931) reported 25 cases of twisted ovarian cysts diagnosed pre-operatively as appendicitis.

The most useful procedure to help in diagnosis is probably rectal examination, this fact being emphasized in many reports; it was of great assistance in these cases.

We are indebted to the late Mr. J. O. D. Wade, Professor A. G. Watkins, and Mr. R. J. Williams for permission to publish these cases.

P. T. BRAY, M.B., B.Sc., M.R.C.P., D.C.H.,
Lecturer in Paediatrics, Welsh National School of Medicine.
L. P. THOMAS, M.B., B.Sc., F.R.C.S.,
Senior Surgical Registrar, Cardiff Royal Infirmary.

BIBLIOGRAPHY

Gross, R. E. (1940). *Amer. J. Dis. Child.*, **59**, 579.
Mayo, C. W., and Butsch, W. L. (1938). *Minn. Med.*, **21**, 256.
Steel, W. A. (1931). *British Medical Journal*, **2**, 798.
Witzberger, C. M., and Agerty, H. A. (1937). *Arch. Pediat.*, **54**, 339.