glucose and is not associated with convulsions in all cases despite its severity.[9,10] Pathologically, major differences can be found in the lungs and lymphoid tissue, which are grossly abnormal only in the aflatoxin-related disease.

The clear differentiation of these two disorders is a necessary prerequisite to the development of diagnostic criteria and perhaps of treatment. Until more specific biochemical parameters are identified the diagnosis of Reye's syndrome must rely on a number of clinical criteria which should at least include: (1) the presence of a progressive encephalopathy; (2) absence of known exposure to toxins or trauma; (3) normal cerebrospinal fluid (cells, culture, and protein); and (4) biochemical evidence of liver dysfunction without significant bilirubinaemia (either hyperammonaemia elevated twofold or prothrombin abnormality <70% of normal and evidence of elevation of liver enzymes >twofold). These biochemical parameters are clearly arbitrary but offer a reasonable chance of eliminating most toxic encephalopathies with hepatic involvement which are not Reye's syndrome. Further supportive evidence might be provided by the histological examination of the liver, but this may not be possible in patients with significant prothrombin abnormalities.

In order to avoid confusion, Reye's-like syndromes due to specific toxins should be identified as such. The use of the term toxic encephalopathy with fatty visceral changes due to a specific toxin, such as aflatoxin B_1, is preferable to the use of Reye's syndrome for disorders with identifiable aetiologies.—I am, etc.,

ELLEN KANG

St. Jude Children's Research Hospital, Memphis, Tennessee

1 Glasgow, A. M., Cotton, R. B., and Dhiensiri, K., *American Journal of Diseases of Children*, 1972, **124**, 827.
2 Glasgow, A. M., Cotton, R. B., Bourgeois, C. H., and Dhiensiri, K., *American Journal of Diseases of Children*, 1972, **124**, 834.
3 Glasgow, A. M., Cotton, R. B., and Dhiensiri, K., *American Journal of Diseases of Children*, 1973, **125**, 809.
4 Olson, L. C., *et al.*, *Pediatrics*, 1971, **47**, 707.
5 Bourgeois, C., *et al.*, *American Journal of Clinical Pathology*, 1971, **56**, 558.
6 Shank, R. C., and Bourgeois, C. H., Keschamras, N., and Chandavimol, P., *Food and Cosmetic Toxicology*, 1971, **9**, 501.
7 Bourgeois, C. A., *et al.*, *Journal of the Medical Association of Thailand*, 1969, **52**, 553.
8 Bourgeois, C. H., *Laboratory Investigation*, 1971, **24**, 206.
9 Reye, R. D. K., Morgan, G., and Baral, J., *Lancet*, 1963, **2**, 749.
10 Simpson, H., *Lancet*, 1966, **2**, 1274.

Suicide by Private Pilots

SIR,—In your leading article on this subject (2 February, p. 168) you pose the question whether aviation medical examiners should be required to explore in some depth the psychological aspects of private pilots.

There are, of course, international standards of psychological fitness as well as physical fitness which private pilots must meet before being given a flying licence, and the fact that accidents from medical causes are rare by comparison with the other causes shows that the system of medical supervision is reasonably effective. The aviation industry is more conscious of cost-effectiveness than most others, and the above background has led the International Civil Aviation Organization (the United Nations standard-setting body) to conclude that only relatively infrequent medical examinations of somewhat limited scope can be justified to meet the level of safety generally acceptable to the public—perhaps because private pilots more commonly kill only themselves in accidents.

Your timely leading article will remind aviation medical examiners—as we do at our annual seminars—of the background factors they should search for during routine medical examinations. I hope it will also remind doctors caring for patients under stress whom they know to be pilots that effective prevention of this type of accident lies almost entirely in their hands. The medical defence organizations are well aware of this problem and they or I would be happy to give suitably confidential advice on particular cases.—I am, etc.,

G. BENNETT
Chief Medical Officer, Civil Aviation Authority

London W.C.2

Cholestyramine and Diabetic and Post-vagotomy Diarrhoea

SIR,—We were interested in the letters from Dr. T. S. Low-Beer and Dr. J. G. Allan and others (22 December, p. 741) and would like to make some further comments.

We have now treated five patients with persistent post-vagotomy diarrhoea with cholestyramine, bowel habit returning to the "pre-vagotomy norm" in all subjects. These findings confirm those of Ayulo[1] and are of particular interest in view of the increase in bile acid excretion demonstrated in post-vagotomy subjects by Dr. Allan and his colleagues.[2,3]

Our theory of increased bile flow following vagotomy refers to patients in their natural feeding state and in whom acid and gastric contents are reaching the duodenum. Our assumption is based on the findings of Saburov,[4] who showed that in dogs introduction of hydrochloric acid into the duodenum caused increased bile secretion and that vagotomy led to an even greater intensification of secretion by the same quantity of acid. Secretin and possibly other hormones may be involved in this increased bile flow.

The existence of a limited reabsorptive capacity of the small intestine for bile salts is suggested by the fact that feeding pure bile salts for a few days induces severe diarrhoea.[5,6] Similarly Dr. Allan and his colleagues presented evidence of limitation of absorption of bile acids by the small gut after vagotomy. We presume that an increased volume of bile is expelled from the enlarged gall bladder, and as post-vagotomy bile contains a higher concentration of bile salts than before operation,[7] the overall quantity of bile salts presented to the small gut is increased.

Lastly, Dr. Low-Beer suggests that because the ^{14}C-glycocholate breathing test is normal diabetic diarrhoea is unlikely to be due to increased entry of bile salts into the colon. This test is based on the bacterial deconjugation of ^{14}C-glycine from cholic acid, the glycine-1-^{14}C being converted to carbon dioxide by bacterial and/or tissue enzymes. The test may be positive in bacterial overgrowth syndromes or if the enterohepatic circulation of bile salts is interrupted by ileal disease or resection, when both colonic and small-gut organisms may be involved. A positive result depends, among other things, on the presence of appropriate bacteria for a period of time long enough to allow the above process to occur. Fromm *et al.*[8] recorded four negative results in patients with ileal resection who had defective intestinal absorption of bile salts and they concluded that a negative result is likely to exclude bacterial proliferation in the small gut.

An alternative suggestion is that Dr. Low-Beer's negative results exclude "bacterial proliferation," particularly as no ileal disease has been demonstrated in diabetic diarrhoea. Bile acids, both conjugated and deconjugated, induce rapid emptying of the colon. This may not only reduce the time allowed for colonic organisms to split glycocholate but might also result in a decrease in the number of organisms in the large bowel.

We await with interest the results of direct measurement of faecal bile acids in patients with diabetic diarrhoea.—We are, etc.,

JOHN R. CONDON
M. I. SULEMAN
Y. S. FAN
M. D. MCKEOWN

St. Mary's Hospital, Eastbourne

1 Ayulo, J. A., *American Journal of Gastroenterology*, 1972, **57**, 207.
2 Allan, J. G., Gerskowitch, V. P., and Russell, R. I., *Gut*, 1973, **14**, 423.
3 Gerskowitch, V. P., Allan, J. G., and Russell, R. I., *British Journal of Surgery*, 1973, **60**, 912.
4 Saburov, G. E., *Sechenov Physiological Journal of the U.S.S.R.*, 1961, **47**, 685.
5 Hoffman, A. F., in *Handbook of Physiology* (section 6, vol. V), ed. C. F. Code, p. 2507. Washington, American Physiology Society, 1968.
6 Hardison, W. G. M., and Rosenberg, I. H., *New England Journal of Medicine*, 1967, **277**, 337.
7 Smith, D. C., Mackay, C., and McAllister, R. A., *Scottish Medical Journal*, 1973, **18**, 65.
8 Fromm, H., Thomas, P. J., and Hoffmann, A. F., *Gastroenterology*, 1973, **64**, 1077.

Limitations of Laparoscopy in Diagnosis of Gonococcal Salpingitis

SIR,—We have both recently treated patients with gonococcal salpingitis in whom diagnosis was missed at laparoscopy. Both were women in their late teens who presented as emergencies with pelvic pain.

In the first case, there was slight pyrexia, some tenderness in the right iliac fossa, and on vaginal examination some right adnexal tenderness. Preliminary laparoscopy showed a normal uterus, normal ovaries, and normal fallopian tubes. As a good view of the appendix was not obtained laparotomy was performed. The appendix, which looked quite normal, was removed. The fallopian tubes looked normal but gentle massage produced a bead of pus at the fimbriae, a swab from which grew gonococci. In the second case the sequence of events was almost identical except that there was no pyrexia.

We would conclude that gonococcal salpingitis cannot be excluded with certainty by inspection of the serosal surface of the fallopian tubes at laparoscopy and wonder how many patients are being sent home the day after laparoscopy with the assurance that serious pelvic disease has been excluded. We also wonder how many normal appendices are removed by general surgeons when the diagnosis was in fact early gonococcal salpingitis.—We are, etc.,

A. KENNEY
J. O. GREENHALF

Department of Obstetrics and Gynaecology, Westminster Hospital, London S.W.1

Br Med J: first published as 10.1136/bmj.1.5906.519-a on 16 March 1974. Downloaded from http://www.bmj.com/ on 9 April 2024 by guest. Protected by copyright.