1-16). After the epidemic 89 (77%) had a four-fold titre rise; of those under 20 years of age 46 out of 50 had positive titres after the infection. Clinical illness occurred in over 50% of infected subjects.

Five years later a follow-up study was done using venous blood glucose tolerance test. Serum neutralizing antibody titres and two-hour plasma glucose levels were measured. Of the 136 persons under 25 years of age (M = 65, F = 71) the highest two-hour plasma glucose levels of 133 mg/100 ml. Persistence of positive titre levels was also documented since 77% of these subjects had a positive neutralizing antibody titre level.

Even though it was in the young diabetic that Dr. Gamble and his colleagues were able to show the strong association with positive Coxsackie B4 titres we could not show that Coxsackie B4 infection caused a single case of diabetes, though the attack rate in the 136 under 20 years of age at the time of the epidemic was extremely high.

—We are, etc.,

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MAX MILLER
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Serotonin and the Mesenteric Circulation

Sir,—Dr. I. M. Murray-Lyons and others (29 December, p. 770) reported some unusual cases of gangrene of the small intestine in patients with carcinoid tumours of the small bowel and with raised blood levels of 5-hydroxytryptamine (5-HT). They found proliferative changes in the elastic tissue in the wall of the mesenteric vessels. Anthony and Drury,1 reporting similar changes in the media of the arteries of patients with carcinoid tumours, suggested that these changes might be caused by substances produced by the tumours. We have been able to observe similar proliferative changes in the elastic tissue of cases with small bowel carcinoid, postulated that these substances are produced by the carcinoid tumour and contribute to the fibrosis and neovascularisation in some cases. This hypothesis has not been proved directly. The elastic lesions have not been reproduced experimentally and the theory is based on the well-known lesions in the arteries of patients with carcinoid tumours.

In a recent laboratory investigation in dogs, blood flow was measured with an electromagnetic flow meter simultaneously in the thoracic aorta, in the superior mesenteric artery (S.M.A.), and in the renal artery. An intravenous injection of 5-HT (0.03 mg/kg) caused a redistribution of blood flow, which was most marked in the S.M.A. (fig.). The reason for this specific sensitivity of the mesenteric vascular bed to 5-HT is not known. Nor can it be stated with certainty that the humoral mesenteric circulation responds to 5-HT in a similar manner. However, this observation does suggest an alternative hypothesis for the aetiology of the elastic changes in the mesenteric vessels—for a chronic increase in resistance in the mesenteric circulation. The sustained increased resistance causes the degenerative changes in the elastic in the branches of the S.M.A. that in turn contribute to ischaemic necrosis in some cases of small bowel carcinoid.—We are, etc.,

RAPPHEL ADAR
EDWIN W. SALZMAN
Department of Surgery, Beth Israel Hospital, Harvard Medical School, Boston, Massachusetts.

Serotonin Metabolism in Hepatic Encephalopathy

Sir,—We read with great interest the paper by Dr. A. J. Knell and others (23 March, p. 549) on serotonin metabolism in hepatic encephalopathy. Our interest in the metabolism of serotonin (5-HT) was aroused by a patient with cirrhosis of the liver who excreted large amounts of 5-HT and its precursor 5-hydroxytryptophan (5-HTP) in urine.1 The hepatic metabolism of 5-HT in experimental cirrhosis was characterized by decreased formation of sulphate conjugates, increased synthesis of glucuronide and conjugates,2,3 decreased uptake of 5-HT by the liver,4 and deranged enterohepatic circulation of metabolites of 5-HT.4 Metabolism of 5-HT was normal in the mucosal fluid of the small intestine and the serum albumin. When the cirrhosis was of long duration, however, the liver was unable to store and metabolize 5-HT.2 A raised concentration of 5-hydroxyindole acetic acid (5-HIAA) in cerebrospinal fluid might well be related to increased excretion of 5-HT and 5-HTP in urine. According to our preliminary observations the storage and metabolism of 5-HTP in the small intestine mucosa of cirrhotic rats is disturbed like the intestinal metabolism of 5-HT. Thus an increased efflux of 5-HTP into the portal circulation may result in an increased transfer of 5-HTP to the central nervous system, which might be misinterpreted as an increase of 5-HIAA concentration in cerebrospinal fluid in patients with hepatic encephalopathy.—We are, etc.,

PERTTI PENTIKAINEN
MATTI KELKI
Second Department of Medicine, Helsinki University Central Hospital, Finland.

BRITISH MEDICAL JOURNAL 25 MAY 1974

Sir,—I was extremely interested in the letter by Dr. K. R. Greene and others (6 April, p. 54). Only three days before that date I was independently doing my first laparoscopic Pomeroy sterilization by virtually the same method as they described. I would confirm that it seems simple and safe and that a search of the literature suggests that it has not been tried previously. In this patient with a mobile uterus there would seem to be no need for two lower quadrant incisions because a single midline stab 2-3 cm above the symphysis pubis suffices. It would seem that the main hazards of this procedure are likely to be haemorrhage due to tearing of the mesentery of the Fallopian tube and, if one uses two stab incisions, accidental puncture of the inferior epigastric vessels by the operating cannula—another complication which should be avoidable if one remembers the surface markings of these vessels. Both complications should be excluded before withdrawing the laparoscope. Neither would be as serious as bowel burns and either would be an indication for laparotomy, for which consent should always have been obtained.

In due course I too hope to report a large series of patients having a laparoscopic Pomeroy sterilization. It would seem important to make an early and full assessment of a technique which requires only ordinary laparoscopy equipment, permits laparoscopic tubal ligation and transection, avoids the hazards of diathermy, and is surprisingly simple.—I am, etc.,

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Samaritan Hospital for Women, London N.W.1


Death during Dental Anaesthesia

Sir,—I have read with interest Dr. J. G. Bourne’s letter on this subject (16 March, p. 516), but I do not feel that he has adequately emphasized the fact that a young, healthy person died needlessly. The patient had an unrecognized faint during the intravenous induction of anaesthesia in spite of the presence of the cardiac sign of pallor. In spite of this pallor and while in the sitting position in the dental chair pure oxygen was given. The anaesthetist was administered by an operator anaesthetist, a practice which is not supported by the Society for the Advancement of Anaesthesia in Dentistry.1

While tragic errors of judgement have been made by most of us in the course of our professional careers we must realize that the concept of liberty is there to be routinely anaesthetize the dental patient in the supine position, but this is purely because of the trend in dental ergonomics. I have never shared Dr. Bourne’s anxiety about the sitting position and I never hesitate to use this position if the patient has, for example, a hiatus hernia or an abdominal
swallowing or expressed a preference for this posture. I do, however, maintain the lower limbs in the horizontal position.

A patient can just as readily faint in the horizontal as in the sitting position. Some time ago, I saw a young, healthy male patient who, while in the supine position, fainted following the insertion of the intravenous needle before any methohexital had been injected. She became unconscious, with marked pallor, a moist skin, and bradycardia. She did not regain consciousness until she was tilted into the Trendelenburg position and pure oxygen was administered. She was subsequently anaesthetized uneventfully. The importance of this is to recognize that faint immediately, whatever position the patient is in, and to treat it expeditiously.—I am, etc.,

W. N. ROLLASON

Department of Anaesthetics,
University of Aberdeen

1 S.A.A.D. Digest, 1974, 2, 103.

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**TABLE II—Primitive Reflexes Present in 40 Patients with Presenile Dementia and Parkinsonian Signs**

<table>
<thead>
<tr>
<th>Reflex</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glabella tap reflex</td>
<td>32</td>
</tr>
<tr>
<td>Raphe twitching reflex</td>
<td>29</td>
</tr>
<tr>
<td>Drop reflex</td>
<td>26</td>
</tr>
<tr>
<td>Palmo-mental reflex</td>
<td>21</td>
</tr>
</tbody>
</table>

This is quite distinct from Parkinson's disease in the early stages. As the two disorders progress, however, the overlap of their clinical features can on occasion be striking, though the severe tremor and flexion dystonia of axial muscles seen in Parkinsonism is not seen in those presenting with dementia. A further feature we have noticed is the singular failure of levodopa to influence the extrapyramidal disorder in either clinical context, and indeed profound depression, worsening of the dementia, and hallucinosis are more likely to occur at low dose levels (0.5 to 1.5 g/kg/hr).

These findings suggest the possibility that the cerebral atrophy may be a part of the primary disorder in both Alzheimer's and Parkinson's diseases; that in the late stages there may therefore exist a supranuclear disorder of basal ganglia function. This may be important in modulating neurotransmission in both dopaminergic and neuropeptide granules at a lower level, as a result of the neuronal depletions and atrophy of the cortex. The lack of therapeutic response to levodopa and the "hypersensitivity" to this drug in those patients with the tetrads of features described are explicable on this basis.—I am, etc.,

JOHN PEARE

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Hull

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**TABLE I—Parkinsonian Signs Present in 40 Patients Presenting with Presenile Dementia**

<table>
<thead>
<tr>
<th>Sign</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial Masking</td>
<td>30</td>
</tr>
<tr>
<td>Akinesia</td>
<td>26</td>
</tr>
<tr>
<td>Rigidity</td>
<td>22</td>
</tr>
<tr>
<td>Tremor</td>
<td>42</td>
</tr>
<tr>
<td>Glabella tap reflex</td>
<td>32</td>
</tr>
</tbody>
</table>

or more primitive reflexes were elicited in 32 (80%) of them (table II). Cortical atrophy or ventricular dilatation was seen at air encephalography in 92% patients, as would be expected in Alzheimer's disease.

I would emphasize that the clinical picture in those presenting with presenile dementia

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Ten healthy male students aged 19 to 25 volunteered for the experiment. Each received ethanol 1.5 g/kg as a 20% aqueous solution which he drank at a constant rate over the 4 hours. The experiments were started at 6 p.m. By the next morning all the ethanol had been eliminated and most of the subjects had a more or less severe hangover. Each also participated in another experimental session during which he drank water only. Each subject served as his own control. Blood samples for determining levels of luteinizing hormone (LH) and testosterone were taken at the start of drinking and at 4, 8, 12, 15, and 20 hours thereafter. Concentrations were estimated by radioimmunoassay.

During the period of acute intoxication there were no changes in plasma testosterone concentration, but during the hangover (12-20 hours after the start of drinking) the testosterone level was only slightly below what it was during the control period (table). In addition, the decrease seemed to correlate with the intensity of the hangover. The decrease was not due to inhibited LH secretion because plasma LH concentration was not decreased by ethanol. On the contrary, there was a slight increase in LH level during the hangover.

Though the biochemical mechanism and physiological significance of the decrease in plasma testosterone concentration during the post-alcoholic phase are not yet known we wish to report this finding, because it indicates that ethanol can alter the metabolism of biologically active sex hormones without causing any liver damage. If the testosterone level is constantly low during chronic alcohol intake this may explain some of the endocrine abnormalities found in chronic alcoholics.—We are, etc.,

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**Pseudotumours of the Orbit**

Sir,—I was interested to read the leading article (6 April, p. 5) on pseudotumours of the orbit. One cause of pseudotumour is in the elderly, temporal or giant-cell arteritis, was unmentioned. I reviewed 43 cases of temporal arteritis and found three that had presented with proptosis and chemosis (in one case bilateral) sufficient to warrant the term pseudotumour of the orbit. Diplopia occurred in two of the cases and one of these developed unilateral blindness. In all three cases there was marked disablment and severe orbital pain.

If pseudotumour of the orbit presents in a patient over the age of 60-65 years temporal arteritis should be considered and other manifestations, especially glaucoma, skin, and radicular artery, sought. A E.S.R. may be