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doctors are possibly unaware that an estimate of 10% of all admissions to general wards have had an alcohol-dependency. Alcoholism is excluded from the curricula of medical schools. Few doctors therefore understand anything about alcoholism. Recovered alcoholics are fortunate people. Recovered medical alcoholics can greatly help by revealing their identity and assisting in information about this disease by enlightening their colleagues and making themselves available for consultation to sufferers from the disease. I hope personal experiences can be turned to good effect.—I am, etc.,

NEIL PANTON.

Porphyrinuria

Sir.—In Dr. Norman Gitlin's recent communication (11 January, p. 96), the most significant positive finding is the "strongly positive porphobilinogen". Also significant, in a negative sense, is the very modest abnormality in urinary porphyrin excretion. This would argue against any "acquired porphyria." On the other hand, it is hard to attach much significance to elevated urinary coproporphyrin in a patient with obvious alcoholic liver disease.

Can we be sure that the patient's pain and even the hyperlipaemia1 are due to "Zieve's syndrome" and not to acute intermittent or variegate porphyria (possibly precipitated by an alcoholic bout and/or inhalation)?2 What is badly needed in this case to put these factors in proper perspective is quantitation of urinary porphyrin levels (elevated in both acute intermittent and variegate) and faecal protoporphyrin (elevated in variegate).—I am, etc.,

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REFERENCES


Sudden Death in a Young Asthmatic

Sir,—As pointed out by Dr. J. Gregg and Dr. J. Batten (4 April, p. 29) the larninar arrangement of mucus in the medium-sized airways found post mortem in their patient suggests that hypersecretion had been occurring for some time before death. Moreover, the blockage may even have persisted from the time of the severe attack of asthma two months earlier. This raises the question whether the mechanical removal of such mucous plugs by bronchial lavage might not have been of prophylactic benefit. The difficulty in such cases is of course to know when the procedure is indicated.

One useful guide is the spasmodic cough from which many such patients suffer. This is often most troublesome and persistent, especially at night, the patient having great difficulty in raising even a small quantity of sputum, which is jelly-like, stringy, and extremely viscous. As a rule it contains no pathogenst but is teeming with eosinophils, often arranged in clumps. The picture reflects the situation within the medium-sized airways, and may be indicative of danger, particularly if increasing the steroid dose produces little or no improvement. As Leopold says, for socking long-hair pleural, laryngeal, or bronchial plugs, the plugs "speak in graphic fashion of a type of movement of the mucus and cell mass which is ineffective in bringing about bronchial clearance...in a severe attack not only is effective clearing of secretions impeded but the altered dynamics of breathing positively encourage the movement of aspiration"—that is, the intense inspiratory efforts made by the patient actually drag the viscid mucus outwards into the smaller peripheral airways. There would appear, therefore, to be good theoretical grounds for mechanical removal of the plugs not only in cases of status asthmaticus but in some cases of chronic wheezing where there is reason to suspect their presence. Fortunately the procedure in competent hands is a safe one,3 and though the effect is sometimes only temporary it does in a number of instances produce relief over a longer period. Perhaps it is noted significantly reducing the need for steroid therapy or the dose. The main difficulty is in deciding the exact indications, and here the type of cough and character of the sputum are important. The second useful indication, noted in the case reported by Drs. Gregg and Batten, is a fall in the peak expiratory flow rate from a previously recorded higher level. It is indeed advisable to take peak expiratory flow rate readings routinely in asthmatics at every outpatient visit.—We are, etc.,

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REFERENCES


2 Thompson, H. T., and Pryor, W. J., Lancet, 1964, ii, 792.


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Stomach Rupture Complicating Traumatic Diaphragmatic Hernia

Sir,—Gangrene or rupture of abdominal visceras in the thorax is a rare condition even complicating diaphragmatic hernia. We report here a case where the traumatic rupture of the diaphragm was followed a year later by strangulation and rupture of the dislocated stomach in the left thoracic cavity. The patient recovered after an emergency operation.

A 22-year-old man was admitted on 16 April 1968. One year before he had been treated in another hospital for a penetrating gunshot wound of the left thorax. On admission he gave a history of vague epigastric pain and intractable vomiting of 48 hours' duration. Examination revealed an acutely ill, undernourished, dehydrated man. Blood pressure 115/80, pulse 130, white blood cell count 11,000 cu. mm., haemoglobin 13 g./100 ml., blood urea, electrolytes, serum bilirubin, and thyroid tests normal. An electrocardiogram revealed sinus tachycardia. Chest x-ray examination demonstrated an enormous dilated stomach with a high fluid level in the left chest, the heart being displaced to the right. Treatment consisted of hydration, analgesia and respiratory support. On 18 April his condition deteriorated, he complained of constant severe chest pain, and vomited repeatedly tea-coloured gastric contents. Emergency laparotomy revealed he had perforated the third of the stomach only in the abdominal cavity, and the rest could not be delivered from the chest. A left thoracotomy was performed, and a large volume of gastric contents evacuated from the thoracic cavity. The wall of the stomach was not gangrenous, but a few livid