must be cast on the ability of the ACCR to detect or exclude pancreatitis in patients with acute abdominal pain.

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¹ Durr, H K, *et al*, *New England Journal of Medicine*, 1977, **296**, 635.

Stiff-neck syndrome

SIR,—During the past three months, six female members of a hospital staff of approximately 1600—three nurses, two domestics, and one administrator—have presented with a similar clinical picture. This consisted of the sharp onset of severe and persistent pain in the cervical region, which was rigidly held in the erect central position and resisted the least movement, active and passive, in any direction, the pain being greatly augmented by such attempts. With one exception resolution was complete within a few days and the impression gained was that this was spontaneous rather than attributable to the local application of heat and the collar supplied. One recurrence has taken place, of identical nature and duration. Systemic upset, pyrexia, other abnormal physical signs, and radiological changes were all absent.

Although chest pains have not infrequently been reported among the staff during this period, no example suggestive of Bornholm disease has been encountered nor has inquiry in the medical wards yielded information of its presence or that of the stiff-neck syndrome among the patients.

The clustering of the cases raises the possibility of an infective aetiology. It would be of interest to learn of experience of like nature.

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Suffocation and sudden infant death

SIR,—I wish to report the case of a 10-week-old male infant who was brought to the accident and emergency department of this hospital last November.

He was born by normal delivery to a healthy gravida 2 mother after an uneventful pregnancy and had previously appeared normal and healthy. On this afternoon he was put outside in his pram wrapped in an infant's sleeping bag with the hood loosely drawn around his head. When his mother next looked she found him to be white, rigid, and not breathing. The nylon lining of the sleeping bag was damp and stuck across his nose and mouth. He started to breathe when she turned him upside down and slapped his back. On arrival at the department he was pale and breathing with difficulty and had generally increased tone, especially in the right arm. He was given oxygen by face mask and rapidly became completely normal. Chest x-ray showed no evidence of inhalation and he has since been quite well.

The sleeping bag is made of two layers of closely woven nylon with a safety lining between. Adults who have experimented find that, *when wet*, the nylon sticks to the face and is quite impossible to breathe through. Although the child may have had a convulsion, either initially or as a result of the anoxia,

we were concerned that the way in which he was wrapped might have led to a fatal outcome. In recent years suffocation in clothes and bedding has largely been discounted as a cause of sudden unexpected death in infancy, but this incident suggests that it should still be considered.

The baby did not struggle or cry out, and if he had died one wonders what evidence would have remained to explain death. This case underlines the importance of trying to construct an exact picture of all the circumstances surrounding any unexplained death in infancy as a number of factors, some of them innocent and harmless in themselves, can prove overwhelming in combination.

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Krukenberg's chopsticks

SIR,—I was delighted to see your leading article on Krukenberg's chopsticks (21 January, p 129), and this is not because of any personal experience in the performance of this operation. Those of us who have had the privilege of working for a short time in the Orthopaedic Hospital in Bangladesh have all seen the quite remarkable results of this procedure as carried out by Ronald Garst in Dacca. Once you have felt the force of the pincer grip achieved by one of his patients, you do not come back a second time.

Over many years in Britain we have all been grilled against the place of this operative procedure for severe forearm injuries, but Garst's success with many patients in Bangladesh is a tribute both to him and to the value which this operation may have at the present time in many places in the world.

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Ink caps and alcohol

SIR,—As a native of Switzerland, where edible fungi are much sought after and highly prized, I would like to add my support to Dr A P Radford's defence of *Coprinus comatus* (14 January, p 112).

This fungus, aptly known as the "shaggy ink cap" or "lawyer's wig," should not be confused with its close relative *C. atramentarius*, which has a smooth cap. Drs M J Caley and R A Clark (24-31 December, p 1633) draw attention to a serious interaction between *C. atramentarius*, which they describe as an edible fungus, and alcohol. In Europe symptoms due to this type of interaction are well known and are generally considered to be the result of acetaldehyde formation during detoxication of alcohol. It has been claimed that the fungus contains disulfiram,¹ but other workers have denied this.² It is worth noting, however, that despite the widespread culinary use of wild fungi in many areas of Europe, reports of serious cardiovascular symptoms due to such interaction are extremely uncommon. Mild hypotension, sometimes with alarming orthostatic features, may occur and has been reported elsewhere,³ but cardiac arrhythmias are very rarely seen.

Although many textbooks describe *C. atramentarius* as an edible fungus, such interactions as can follow its ingestion together

with alcohol (even if the latter is taken many hours later) cause it to be widely viewed with suspicion. *C. comatus*, however, is entirely free from this unpleasant complication and is not only safely edible but delicious.

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¹ Simandl, J, and Franc, J, *Chemie Listy*, 1956, **50**, 1862.

² Wier, J K, and Tyler, V E, *Journal of the American Pharmaceutical Association* (Scientific edn), 1960, **49**, 426.

³ Buck, R W, *New England Journal of Medicine*, 1961, **265**, 681.

Treatment of hepatic osteomalacia

SIR,—The careful study by Dr R G Long and others of four patients with biliary cirrhosis (14 January, p 75) illustrates well the problems of hepatic osteodystrophy. It also raises one or two questions, particularly the authors' interpretation of the results of 1,25-dihydroxycholecalciferol ($1,25-(\text{OH})_2\text{D}_3$) therapy.

While their patients had clinical bone disease the extent of osteomalacia was uncertain: their x-rays indicated osteoporosis and not osteomalacia; previous fractures either had healed or were healing, suggesting adequate vitamin D activity; metabolic myopathy may be obscure in patients near to death; and virtually all biochemical indices of osteomalacia, without evidence of secondary hyperparathyroidism and including calcium absorption and phosphorus excretion, were normal. It must be hard to be sure of clinical improvement following $1,25-(\text{OH})_2\text{D}_3$ treatment when the second biopsy specimens were obtained post mortem in two subjects. Nevertheless, if one accepts significance of histological changes following $1,25-(\text{OH})_2\text{D}_3$ and of the stated clinical improvement the possible reasons need to be more carefully considered.

The authors' data suggest that patients with biliary cirrhosis may have an increased requirement for vitamin D above that necessary to overcome malabsorption of the vitamin. But they fail to provide grounds for concluding that either 1α -hydroxylation is at fault or that vitamin D_2 is inferior to vitamin D_3 . Many other possibilities are at least as likely. In particular there is good evidence that deficiency of active metabolites may result from increased urinary loss in this disease.¹ Years of parenteral vitamin D therapy in the authors' patients had failed in two of them to raise plasma 25-hydroxyvitamin D (25-OHD) levels above $11.4\text{ }\mu\text{g/l}$ (misprinted as pg/l in the paper); higher levels might be expected following oral therapy in patients without malabsorption.² One might expect similar urinary loss of other active metabolites. Furthermore, comparison of different parenteral routes of vitamin D administration as a means of raising plasma 25-OHD levels suggests that availability of vitamin D following injection of an oily bolus directly into storage organs (muscle and adipose tissue) may be limited.³ (Defective hepatic 25 -hydroxylation of vitamin D, which would also explain inappropriately low 25-OHD levels, has been largely excluded by recent work.⁴) Whatever circulating activity is maintained in the face of increased loss and restricted availability, immediate substitution of $1,25-(\text{OH})_2\text{D}_3$ will simply be additive and tells little of the nature of any "resistance".

Additional mechanisms which are theoretically neither more nor less likely than the