Laparoscopy Hazard

SIR,—Mr. H. G. E. Arthure (21 November, p. 492) and Mr. M. J. Muldoon (2 January, p. 51) are quite rightly concerned with the avoidance of a high intra-abdominal pressure during laparoscopy.

The possible danger of compression of the inferior vena cava will probably only occur when the intra-abdominal pressure is very much raised.

Hodgson *et al.*¹ found that with intra-abdominal pressures of 15-20 mm Hg the central venous pressure was in fact raised, and in some cases the cardiac output was thus raised. They suggest that at this pressure blood may be transferred to the vena cava by mild compression of the abdominal organs. Therefore a pressure of 15-20 mm Hg may even be beneficial to the circulation. An excessive pressure might occlude the vena cava.

However, Alexander *et al.*² have shown that at this pressure of 15-20 mm Hg there is always elevation of the diaphragm, which will produce alterations in pulmonary mechanics. It is well recognized that all patients under general anaesthesia during laparoscopy should be given a muscle relaxant and be ventilated. This technique allows the abdominal wall to balloon out with the minimum of gas pressure, thus facilitating the examination while keeping the pressure within acceptable limits and maintaining adequate pulmonary ventilation.

It is regrettably not so readily accepted that the intra-abdominal pressure during laparoscopy in the conscious patient is very much less controlled and may easily become excessive. Even a well-sedated patient will need a moderately high intra-abdominal pressure to balloon out the abdominal wall in order to achieve adequate vision. The elevation of the diaphragm so produced will distress the patient and cause dyspnoea. The abdominal wall will thus tighten and so increase the pressure still further. This hazardous situation is difficult to avoid in the conscious patient. The more debilitated the patient is, the more hazardous will be the situation.

I therefore believe that laparoscopy should always be performed in the controlled conditions given by general anaesthesia, in conjunction with intermittent positive pressure ventilation and a muscle relaxant, and should rarely, if ever, be performed on the conscious patient.—I am, etc.,

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¹ Hodgson, C., McClelland, R. M. A. and Newton, J. R., *Anaesthesia*, 1970, 25, 382.

² Alexander, G. D., Noe, F. E., and Brown, E. M., *Anesthesia and Analgesia: Current Researches*, 1969, 48, 14.

Phenacetin and Papillary Necrosis

SIR.—In their article, "Papillary Necrosis in Rats Caused by Aspirin and Aspirin-containing Mixtures" (5 September, p.559), Dr. R. S. Nanra and Dr. Priscilla Kincaid-Smith "suggest that aspirin, and not phenacetin, may be the major factor in analgesic nephropathy in patients taking A.P.C. mixtures." Their conclusion is based on their observation that the same percentage of rats fed aspirin (36.8%) developed papillary necrosis as rats fed A.P.C. (aspirin, phenacetin, and caffeine) mixtures. However, they do not state specifically what figure they were using as the base for the A.P.C. group to compare with the aspirin groups. If one assumes that they were refer-

ring to the overall percentage that can be calculated from Table III, then their percentage of papillary necrosis in A.P.C. (high and low purity) and aspirin and phenacetin-fed rats is 45%, a figure which is somewhat less than the percentage published by Saker and Kincaid-Smith in 1969.¹ Using this figure, along with the previously published observation that "phenacetin alone in the same dose over nine months failed to cause any renal damage,"² perhaps their conclusion is reasonable.

It does not seem reasonable, however, that in computing their figure of 36.8% for papillary necrosis in aspirin-fed rats the data concerning the aspirin plus caffeine-fed animals be ignored. Given their hypothesis, Dr. Nanra and Dr. Kincaid-Smith should include these latter data in calculating the percentage of aspirin-fed rats developing papillary necrosis. If this is done the data of Table III yield a percentage of 27.6 for the aspirin and aspirin and caffeine-fed rats.

A comparison of this percentage with that A.P.C. and/or aspirin and phenacetin-fed rats, then, would not permit one to conclude that aspirin is the "major factor" in analgesic nephropathy, since the difference between 45% and 27.6% may be significant. Some additional "major factor" must be involved. Nanra and Kincaid-Smith's own data would suggest that the factor might be phenacetin. It would seem that one reasonable conclusion to be drawn from the authors' work is that the nephropathy of analgesic abuse is the result of the combined effect of two drugs, aspirin and phenacetin. Such a conclusion is also consistent with the work of Bluemle and Goldberg,² which suggested that papillary necrosis from analgesic abuse may be related to high papillary concentrations of APAP (*N*-acetyl-*p*-aminophenol)—the major metabolic product of phenacetin. In our opinion it is still too early to absolve the phenacetin of A.P.C. mixtures of its probably major role in the pathogenesis of papillary necrosis induced by analgesic abuse.—We are, etc.,

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¹ Saker, B. M., and Kincaid-Smith, P., *British Medical Journal*, 1969, 1, 161.

² Bluemle, L. W., and Goldberg, M., *The Journal of Clinical Investigation*, 1968, 47, 2507.

Childhood Enuresis

SIR.—There are several points in Dr. Roy Meadow's article (26 December, p. 787) with which I disagree. He appears to condone, if not actually recommend, that children should be lifted and potted at 10 p.m. while not fully awakened. This procedure trains the child to pass urine while sleeping—that is, to be an enuretic. I considered that potting a sleeping child was a major contributing factor in 28.7% of 1,000 cases of enuresis.¹

Dr. Meadow does not give references for the controlled trials of the tricyclic antidepressants. I quoted a number of papers showing they were not superior to a placebo in the treatment of enuresis. The tricyclic antidepressants are of more use

than a placebo only when the patient is depressed. Not all enuretics are depressed.

I cannot accept his theory that after using the buzzer "the child is conditioned to putting up with the sensation of a full bladder . . .". My conclusion based on 359 children was that the buzzer works because the child knows that if he uses it correctly he will not have a wet bed. It is most important that the child should get out of bed after the buzzer rings when there is only a damp patch, not bigger than a saucer. If the bell does not wake the child sufficiently quickly for this the mother must sleep with him and see that he gets out in time; otherwise the bed will be really wet and the child will not gain the confidence which is essential for success in this or any other method of treatment.

As a general practitioner who also runs a local authority enuresis clinic I do not agree that enuresis is best managed by the family doctor, as not many have the time, which is so necessary for the proper management of these cases.—I am, etc.,

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¹ White, M. S., *Medical Officer*, 1968, 120, 151.

Atrial Myxomas

SIR,—I was surprised that your leading article (9 January, p. 65) on atrial myxomas made no mention of the role of ultrasound in their diagnosis. Ultrasonic mitral echograms are now an established part of cardiac investigation. They are atraumatic and have proved to be reliable in the diagnosis of left atrial myxomas.—I am, etc.,

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Secretion of Parathyroid Hormone by a Renal Adenocarcinoma

SIR.—Perhaps it is no coincidence that the case investigated by Dr. R. M. Buckle and others (19 December, p.725) and one reported in 1965¹ both had renal carcinomas and hypercalcaemia, corrected by removal of the neoplasms. Chronic irritation from long-continuing hypercalcaemia might have been responsible for the tumours. The earlier case had systemic sarcoidosis, and surprisingly metastases were not found at necropsy, six years after operation. Instead, there was a parathyroid adenoma and hypoplasia of the remaining three glands. Longstanding skin pigmentation and adrenal hypoplasia (attributed to prolonged steroid treatment) were also of interest. Parathyroid antibodies were recently reported, and they may be produced by sarcoidosis by a mechanism similar to sarcoid hyperthyroidism.² In this context an over-active antibody response might later provoke formation of parathyroid adenomas. Indeed, Dr. Buckle's patient may possibly have sarcoidosis and an adenoma, because both patients became euparathyroid following operation. Conceivably the tumours, not concentrating the blood parathyroid hormone, might in