Laparoscopy explosion hazards with nitrous oxide

SIR,—Professor J S Robinson and others (27 September, p 764 and 27 December, p 760) have suggested that the use of nitrous oxide as the inflating gas for laparoscopy is dangerous because of the risk of explosion. They suggest that hydrogen and methane, which are found in intestinal gas, will also be found in the abdominal cavity in concentrations great enough to form explosive mixtures with nitrous oxide.

We have taken gas samples from the abdominal cavity at the end of 12 laparoscopic procedures. The abdomen was inflated for an average time of 11 min (range 7-20 min). The samples were withdrawn into greased glass syringes closed by a polyethylene tap and analysed within 48 hours of collection. A standard mixture of methane and hydrogen in nitrogen was stored in a similar sampling syringe for 10 days. No change in composition occurred during storage, so it is unlikely that losses would have occurred from the samples before analysis was carried out. Analysis was by gas chromatography, with argon as carrier gas and a hot wire detector. This method can detect 20 ppm, by volume, of hydrogen and 100 ppm of methane in a 5-ml gas sample.

No methane was detected in any sample. In one sample hydrogen was detected at a concentration of 20 ppm. The lower explosion limit for hydrogen in nitrous oxide, at atmospheric pressure, is 5·5% by volume. This is about 2500 times greater than the concentration detected in this single case.

We would suggest that explosion is not a significant hazard in laparoscopy of short duration when nitrous oxide is used unless intestinal gas is released into the peritoneal cavity by puncture of the bowel wall.

We are grateful to Mr A Robertson, of the Institute of Occupational Medicine, and to Mr J Evans, of the National Coal Board, for the analysis of the gas samples.

G B DRUMMOND
D B SCOTT

Department of Anaesthetics,
Royal Infirmary,
Edinburgh

Endoscopic papillotomy

SIR,—I read with great interest the article by Professor M Classen and Dr L Safrany (15 November, p 371). One cannot but admire the digital dexterity of modern endoscopists, but I would like to comment on the dangers of duodenal perforation in endoscopic papillotomy.

The authors mention that the sphincter is of variable length and that this naturally cannot be estimated endoscopically, and pointed out that the longer the cut, the greater the risk of duodenal perforation and cite one instance of this in their series.

May I draw attention to some anatomical studies I carried out nearly 20 years ago, and subsequently published?[1] This investigation revealed that the sphincter length referred to as the thickened segment varied between 11 and 27 mm with a mean length of 16 mm in the series studied. At least 2 mm of this lies outside the duodenal wall, this portion having been called the sphincter of Boyden, and a further 2 mm lies level with the muscle of the duodenal wall. This means that the range of cut of a papillotomy which will not produce a duodenal fistula is 7-23 mm, with a mean length of 12 mm. If the authors are able to measure exactly the length of their cut, then the suggested length they advise of 10-15 mm is, in my view, too long and will inevitably lead to duodenal perforation in some cases.

I realise that the fact of the perforation will be revealed by extravasation of injected dye, indicating that open operation is required. However, I can also visualise a situation in which this could be missed at the time and open operation delayed. It would be a pity if such dexterous advances in endoscopy were discredited in the way that sphincterotomy, using a sphincterotome passed from above down the common bile duct, was some years ago and led to the more accurate method of dividing the sphincter transduodenally.

BERNARD H HAND

Department of Surgery,
Ipswich Hospital,
Ipswich, Suffolk

[1] Hand, B H, *British Journal of Surgery*, 1963, **50**, 486.

Phenytoin and serum cholesterol

SIR,—The article by Dr Risto Pelkonen and others (11 October, p 85) opens with the statement that "long-term treatment with phenytoin is often accompanied by various metabolic and endocrine abnormalities." Our experience at this clinic, in addition to a detailed search of the literature, does not substantiate this statement. Over the past 39 years we have prescribed phenytoin to at least 18 000 epileptic patients, many of whom have taken it daily for 10-35 years. During this time we have encountered clinical evidence of metabolic or endocrine disorders unequivocally due to phenytoin in only rare instances.

The very high incidence of hypercholesterolaemia (82%) reported by these investigators in a small group of patients who received phenytoin for periods up to 12 months is also inconsistent with our experience in a much larger series over many more years. All of our patients receiving phenytoin undergo complete blood chemistry examinations and we have not observed a prevalence of elevated serum cholesterol values in excess of that expected in the general population. It is of interest to note that in experimental animals the administration of phenytoin was associated with a decrease or no significant change in the blood cholesterol level[1] and a marked decrease in dermal lipids, including cholesterol.[2]

From their finding that nine of 11 patients exhibited increases in serum cholesterol concentration ranging from 6 to 48%, Dr Pelkonen and his colleagues conclude that "an increase in serum cholesterol may be regarded as an untoward effect of long-term phenytoin treatment because it increases the risk of coronary heart disease." I do not believe that the results of their relatively brief investigation in such a small group of patients warrants such a dogmatic proclamation. In a letter to the Editor (12 April 1975, p 87) Professor V Lindén stated that "experienced neurologists in Norway say that they have not seen a single chronic epileptic die of myocardial infarction." Our findings are consistent with the Norwegian observations. We have followed thousands of epileptic patients, most of whose anticonvulsant drug regimens included phenytoin, throughout the middle years of life and are also impressed with the exceedingly low incidence of coronary heart disease in this group.

We would be interested to hear whether other physicians who have treated large numbers of epileptic patients for many years have observed a similarly low incidence of myocardial infarction.

SAMUEL LIVINGSTON

Epilepsy Clinic,
Johns Hopkins Hospital,
Baltimore, Maryland

[1] Chung, A C, *Journal of Atherosclerosis Research*, 1967, **7**, 373.
[2] Nakamura, K, and Masuda, Y, *Archives internationales de pharmacodynamie et de thérapie*, 1966, **162**, 255.

Uterine hypertonus after induction of labour with prostaglandin E_2 tablets

SIR,—We read with interest the report by Mr A Alaily and Mr G A Morewood (27 December, p 731) on the use of oral prostaglandin tablets for the induction of labour. In their series of 100 cases they report "absence of overstimulation and adverse effects on fetomaternal wellbeing." Using an identical induction regimen for favourable multigravidae, we have recently encountered a case complicated by severe uterine hypertonus occurring late in the first stage of labour.

The patient was a 22-year-old multigravida. Her first pregnancy had ended in a spontaneous, first-trimester, complete abortion. The second pregnancy had proceeded to term with a normal delivery. In her third pregnancy labour was induced at eight days past term, with an inducability rating of 7 as assessed by Bishop's method.[1] A forewater amniotomy was performed, followed 30 min later by an initial dose of 0·5 mg of dinoprostone (prostaglandin E_2) by mouth. Approximately one hour later weak uterine contractions were occurring at 10-min intervals and a second dose of 0·5 mg of prostaglandin E_2 was given. The patient rapidly progressed into established labour with uterine contractions every 2 min. Their duration was 65-70 s and at this time there was no increase in the resting tone of the uterus. She was given 100 mg of pethidine by intramuscular injection. The third dose of prostaglandin E_2 was withheld because of uterine hyperactivity, as recorded by the cardiotocograph. The fetal heart rate remained within normal limits, with occasional type I decelerations.

Forty minutes after the onset of uterine hyperactivity and 3½ hours after amniotomy the uterus became hypertonic. A vaginal examination was performed and the cervix was found to be 8 cm dilated with the fetal head 1 cm below the ischial spines. A fetal bradycardia of 90/min was noted at this stage, and preparations were made to expedite delivery. Vacuum extraction was considered to be the method of choice. However, the patient rapidly achieved full dilatation of the cervix and a low forceps delivery followed. The infant's Apgar score at birth was 9. The duration of the hypertonic phase was 20 min and the induction-delivery interval was 3 hours 50 minutes.

We conclude that uterine hyperstimulation can occur with this regimen. The value of continuous cardiotocography for all patients receiving uterine stimulants cannot be overestimated.

JOHN E FELMINGHAM
M C OAKLEY
R D ATLAY

Sefton General Hospital,
Liverpool

[1] Bishop, E H, *Obstetrics and Gynecology*, 1964, **24**, 266.

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