induction until the patient left the recovery area. Student’s t test was used to compare variances and mean values and the Chi-squared test to compare the frequency of arrhythmias between the two groups.

There were no significant differences between the two groups with regard to age, weight, duration of anaesthesia, arterial pressure or heart rate. The values for $P_{E_{CO_2}}$ minute volume and respiratory rate are shown in table I. There were no significant differences between the means of these values for the two groups. The maximum increase in $P_{E_{CO_2}}$ when comparing the values at the end of insufflation with those at the start of the operation in any patient was 1.33 kPa. Eighteen patients showed a decrease in $P_{E_{CO_2}}$ during insufflation.

### Table I. End tidal carbon dioxide tension, minute volume and respiratory rates immediately before insufflation compared with those immediately after removal of the laparoscope

<table>
<thead>
<tr>
<th></th>
<th>$P_{E_{CO_2}}$ (kPa)</th>
<th>Minute volume (litre min$^{-1}$)</th>
<th>Respiratory rate (b.p.m.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group E</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before insufflation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SEM)</td>
<td>6.6 (0.15)</td>
<td>3.94 (0.34)</td>
<td>16 (0.69)</td>
</tr>
<tr>
<td>Range</td>
<td>5.6-7.8</td>
<td>2.1-8.1</td>
<td>10-23</td>
</tr>
<tr>
<td>End of insufflation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SEM)</td>
<td>6.6 (0.15)</td>
<td>4.61 (0.37)</td>
<td>19 (1.22)</td>
</tr>
<tr>
<td>Range</td>
<td>5.3-7.7</td>
<td>2.2-8.8</td>
<td>11-35</td>
</tr>
<tr>
<td><strong>Group I</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before insufflation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SEM)</td>
<td>6.2 (0.18)</td>
<td>3.87 (0.29)</td>
<td>18 (1.22)</td>
</tr>
<tr>
<td>Range</td>
<td>4.8-8.0</td>
<td>2.2-6.3</td>
<td>9-26</td>
</tr>
<tr>
<td>End of insufflation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SEM)</td>
<td>6.2 (0.19)</td>
<td>5.12 (0.37)</td>
<td>20 (1.40)</td>
</tr>
<tr>
<td>Range</td>
<td>4.3-8.1</td>
<td>2.8-9.9</td>
<td>12-34</td>
</tr>
</tbody>
</table>

Six patients developed an arrhythmia (either unifocal ectopic or bigemini) during the course of the study. Three occurred before induction and one patient developed bigemini during intubation. All four of these patients reverted to sinus rhythm before insufflation. Arrhythmia during insufflation occurred in none of the patients receiving enflurane and in two of the patients receiving isoflurane. This difference was not significant. No arrhythmia occurred in any patient after the carbon dioxide had been removed from the abdomen.

Laparoscopy for routine procedures may be very short in duration, for example as short as 13 min in our study. Even with the newer non-depolarizing blockers atracurium and vecuronium, rapid and complete antagonism after such a period may be difficult. Careful monitoring of neuromuscular function is important because simple clinical evaluations of residual blockade may be misleading (Jones, 1985). In studies of postoperative morbidity following outpatient anaesthesia involving neuromuscular blockade there were instances of double vision which may have been caused by residual blockade (Collins, 1984). Therefore an anaesthetic technique which avoids the use of non-depolarizing agents may be advantageous. Also, some surgeons prefer the degree of muscle tone provided by a non-paralysed patient when inserting the laparoscope.

The authors believe that an anaesthetic technique of spontaneous ventilation with enflurane or isoflurane is satisfactory for routine laparoscopy provided that the intra-abdominal pressure does not exceed 25 mm Hg.

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### OXYGEN LOSS FROM ROTAMETER

Sir,—I would like to report a dangerous fault which recently occurred in a Boyle-type anaesthetic machine, which led to a selective oxygen loss from the inspired gas mixture.

Shortly after a patient had been connected via a Bain circuit to a Penlon Nuffield ventilator, cyanosis was noted. A rapid assessment of the position of the tracheal tube, ventilation of the lungs and the anaesthetic machine (the rotameters showing a concentration of only 12%) revealed no fault. However, an oxygen analyser showed a concentration of only 12%. A replacement machine was installed and the patient’s condition improved immediately. The operation continued uneventfully and the patient showed no ill-effects following the procedure.

Subsequent examination of the faulty machine showed an axial tear in the upper rubber grommet of the oxygen rotamer. Despite the machine having been tested (a “single hose” test) before the case, this defect had not been apparent, nor had it been prevented by servicing carried out during the previous week. Clinical observation, confirmed by an oxygen analyser, averted a serious outcome. A pressure leak test, as described by Page (1977) might have brought the fault to light had it been utilized.

Similar occurrences have been reported in this Journal by Bishop, Lerick and Hodgson (1967), Gupta and Varshneya (1975), and from this hospital by Powell (1981). In subsequent correspondence, Rendell-Baker (1981) recommended that the oxygen flowmeter be relocated to the right-hand side next to the flowmeter manifold outlet. This could be done in situ at little expense, and with the re-education of users.

### REFERENCES


May we suggest that manufacturers should consider this design fault, and also alter the replacement schedule for flowmeter grommets? In the meantime, all anaesthetists should be aware of this potential problem. Moreover, this incident illustrates the value of an oxygen analyser in supplementary clinical observation.

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E. Walsh
Bristol

REFERENCES

TREATMENT OF NAUSEA WITH EXTRADURAL DROPERIDOL

Sir,—Following the report on the use of extradural droperidol (Bach et al., 1985) I would like to present some further clinical observations following its use in two patients.

A 57-year-old man presented with bilateral thoracic metastases, mild ascites and pain in the rib cage. Previously, a biopsy of a Virchow’s node showed adenocarcinoma, primary site unknown. A thoracic extradural catheter was placed at T10/11 and a twice daily dose of morphine, 12 and 8 mg, was given until a dose of 30 mg, 8-hourly, produced adequate analgesia. One week later the patient developed nausea and hiccups that were unresponsive to chlorpromazine by mouth. Droperidol was given, mixed together with the morphine, in a twice daily dose of 1.25 and 2.5 mg. Within 1 h of the first dose of droperidol the nausea disappeared, and the hiccups ceased gradually over the next 24 h. The patient remained mobile and could eat normally without signs of sedation or hypotension. The dose of droperidol remained unchanged, although the morphine requirements increased in the terminal stage of his disease, without the return of symptoms. He died 8 weeks after the start of the treatment with morphine–droperidol.

The second patient, a 67-year-old man, presented with left-sided intractable thoraco-abdominal pain, corresponding to the T6-11 dermatome. Two years previously he underwent a left nephrectomy for adenocarcinoma of the kidney. He had developed nausea and hiccups that were unresponsive to chlorpromazine by mouth. Droperidol was given, mixed with the morphine, in a twice daily dose of 1.25 and 2.5 mg. Within 1 h of the first dose of droperidol the nausea disappeared, and the hiccups ceased gradually over the next 24 h. The patient remained mobile and could eat normally without signs of sedation or hypotension. The dose of droperidol remained unchanged, although the morphine requirements increased in the terminal stage of his disease, without the return of symptoms. He died 8 weeks after the start of the treatment with morphine–droperidol.

The second patient, a 67-year-old man, presented with left-sided intractable thoraco-abdominal pain, corresponding to the T6-11 dermatome. Two years previously he underwent a left nephrectomy for adenocarcinoma of the kidney. He had been treated for 1 month before admission with chlorpromazine and ketobemidone by mouth, with decreasing effect. Computed tomography showed enlargement of the pre-aortic lymph glands and a mass invading the retroperitoneal space. A biopsy of a Virchow’s node showed adenocarcinoma, primary site unknown. A thoracic extradural catheter was placed at L1/2 and morphine 1.25 mg in the morning and 2.5 mg at evening. Following the extradural the nausea abated, but the effect was short-lived. During the following 2 days the droperidol was increased until doses of 5 mg, 5 mg and 2.5 mg, at 8-hourly intervals, relieved the nausea. The patient continued in full activity and showed no signs of cerebral sedation, or hypotension. The patient died 7 weeks later and, in spite of increasing morphine requirements, the nausea did not return.

The aetiology of the nausea is, of course, speculative and several possibilities exist. Potentiation of the analgesia, although apparently adequate at the time, may have resulted in the disappearance of the symptoms. The nausea may have been a side effect of the extradural morphine per se. A further possibility is that the nausea and, in the first patient, hiccups were a result of tumour spread within the abdominal cavity.

Droperidol is a neuroleptic drug with a predilection for certain areas of the brain known to be rich in dopaminergic synapses. It is thought to exert its antiemetic effect by affecting the emetic trigger zone. The absence of behavioural effects and the lack of alpha-adrenergic blockade suggest an antiemetic site of action at the spinal level, possibly by interfering with transmission of noxious impulses in the spinal cord.

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Kongsberg

REFERENCE

BLOOD VISCOSITY

Sir,—The study of blood viscosity and its modification is of interest in anaesthesia (Gordon and Ravin, 1978).

Viscosity is a physical property of fluids; blood has a “non-Newtonian” behaviour (its viscosity is not constant, changing inversely with the rate of flow), while plasma has “Newtonian” behaviour (constant viscosity). Factors having some influence on blood viscosity are: haematocrit, plasma proteins (basically, fibrinogen and globulins). The influence of platelets and leucocytes is less important—at an intercellular level the aggregation and deformability of red cells are the most important factors (Gosling, 1984).

The measurement of whole blood viscosity does not present a defined value at some particular shear rate, but a representation of the behaviour of blood at several degrees of shear rate.

Gramstad and Stovner (1979) studied the effects of anaesthetics containing Cremophor EL on plasma viscosity. Orr and colleagues (1982) reported the effects of Althesin on plasma and blood viscosity after i.v. administration; they found small decreases in plasma and blood viscosity as well as in haematocrit, but these were statistically significant effects only at very low shear rates (0.945 s⁻¹). More recently, Gibbs, Oh and Chester (1984) found important increases in plasma viscosity after several days of continuous infusion of Althesin.

We have recently studied the effects of propanidid–Cremophor EL on whole-blood viscosity, haematocrit, leucocytes and platelets, after induction of anaesthesia in eight patients, all submitted to short procedures. Propanidid-Cremophor i.v. reduced whole-blood viscosity after 5 min in vivo from 0.63 ± 0.3 cP (centipoise) (P < 0.001) at 230 s⁻¹ (shear rate). It decreased to 0.8 ± 0.5 cP at 115 s⁻¹ (P < 0.01); to 1.9 ± 1.0 cP (P < 0.01) at 46 s⁻¹; to 1.7 ± 0.8 cP at 23 s⁻¹ (P < 0.001) and to 2.1 ± 0.7 cP at 11.5 s⁻¹ (P < 0.001). Haematocrit decreased by 2.3% (ns), haemoglobin by 0.5 g dl⁻¹ (ns) and platelets by 23000 ± 12000 dl⁻¹ (P < 0.01).