FENTANYL

SIR,—I read with interest the case report by Brockway and colleagues [1] and the article by Noble and colleagues [2] on respiratory depression after extradural fentanyl. In view of the apparent rarity of this complication, I wish to report a similar case which I encountered recently.

A healthy, 30-year-old patient (gravida 1) presented for elective Caesarean section because of uterine abnormality. The current pregnancy had been uneventful. Preoperative examination was unremarkable (weight 56.0 kg, height 1.54 m). She was premedicated with two doses of oral ranitidine 150 mg and sodium citrate 30 ml before surgery.

Before an extradural block was induced, the patient received Ringer's lactate solution 1 litre. Patient monitoring included ECG, non-invasive arterial pressure measurement (Dinamap) and pulse oximetry (Nellcor). With the patient in the left lateral position, the L2–3 extradural space was identified with a 16-gauge Tuohy needle, using loss of resistance to saline. An extradural catheter was passed 4 cm cephalad and 0.5% bupivacaine 3 ml was injected through a Millipore filter as a test dose. After 5 min, and with no evidence of intrathecal or intravascular administration, 0.5% bupivacaine 7 ml was injected in divided doses over 5 min with the patient positioned with the pelvis tilted 15° to the left using a wedge under the right hip. An additional 0.5% bupivacaine 8 ml with fentanyl 100 μg was then injected incrementally over 10 min.

Bilateral sensory block to T6 was obtained after 5 min. Maximum block height was obtained at 10 min: bilateral T2. The motor block was assessed using a Bromage scale: the patient was just able to move both knees and was graded 1. There was a decrease in systolic arterial pressure from 96 to 87 mm Hg and heart rate decreased from 56 to 51 min⁻¹. Ephephrine 8 mg and atropine 0.3 mg were given i.v. Thereafter, arterial pressure and heart rate returned to normal.

Surgery was commenced 30 min after completion of the injection. Before delivery, the mother received oxygen 3 litre min⁻¹ via an intranasal cannula and the pulse oximeter showed 100% saturation. Just before the start of surgery, the patient said she felt drowsy and complained of persistent nausea; she was premedicated with intravenous dexamethasone 5 mg and on command she took deep breaths. After 10 min, when it had become obvious that the patient had not woken, she was instructed to take deep breaths. A fentanyl test dose (60 μg) was administered, enhancing rostral spread via a direct perimedullary vascular channel [3].

It was considered that the respiratory depression in this patient was probably caused by the larger dose of fentanyl being administered, enhancing rostral spread via a direct perimedullary vascular channel [3].

This case further strengthens the importance of continuous monitoring of patients for an adequate period after extradural fentanyl and highlights the need to limit extradural administration of fentanyl to small doses.

C. Y. WANG
Kuala Lumpur

A PUNCTURE TECHNIQUE FOR CONTINUOUS SUBARACHNOID BLOCK

SIR,—Continuous spinal anaesthesia is a well-described technique [1], but the occurrence of postdural puncture headache (PDPH) is the main factor limiting its popularity. Loss of CSF and its relation to intraspinal compliance is one of the causative factors of PDPH [2]. The variability of CSF loss during continuous spinal anaesthesia is a matter of controversy [3, 4]. However, in spite of many of the reports on the technique, there is little information on the technical implementation of the procedure [1, 5].

We have used the technique of continuous spinal anaesthesia since January 1990. Perifix extradural equipment (Braun, Germany) is used with a Tuohy 18-gauge needle and 20-gauge polyamide multiperforated catheter, as part of a technique which detects the subarachnoid space while minimizing initial CSF loss.

Regardless of patient position, the needle bevel is introduced parallel to the ligamentous and dural fibres. The extradural space is located initially by loss of resistance (air) or using an electronic detector (EpiSensor, Palex, Spain) [6]. The detection system is then removed, and a catheter threading aid is fitted, where the catheter tip emerges from the diaphragm end of a transparent blind hub. The total unit is introduced smoothly with the nondominant hand. At the moment when the hub fills with CSF, the needle is rotated with the outlet orifice cephalad; the dominant hand is then used to advance the catheter inside the needle to 3–5 cm within the subarachnoid space.

During conventional dural puncture when the needle stylet is removed, an uncontrolled loss of CSF occurs—particularly when larger needles are used, even when the stylet is replaced quickly by the catheter. When the hub fills with CSF, the needle is rotated with the outlet orifice cephalad; the dominant hand is then used to advance the catheter inside the needle to 3–5 cm within the subarachnoid space.

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**BACTERIAL OVERGROWTH AND GASTRIC DYSFUNCTION**

Sir,—The issue of bacterial overgrowth in patients undergoing mechanical ventilation of the lungs, raised by Dr T. Inglis and colleagues [1], is important because of the serious consequences of nosocomial infections in patients in the intensive care unit (ICU). As the relationship between gastric acidity, bacterial growth and nosocomial infections remains unclear, the authors’ assumption that factors other than acidity are likely to play an important role in gastric bacterial overgrowth is justified.

The interpretation of the data from this study should, however, take into account the following points. Random, widely-spaced sampling of gastric juice for bacterial count and pH does not allow correlations on the effect of pH on bacterial growth, as almost all bacteria commonly implicated in nosocomial pneumonia in patients undergoing artificial ventilation are killed after 10 min of exposure to gastric juice at a pH < 4, with virtually no regrowth within 24 h [2]. Because brief fluctuations in pH are not reflected in sporadic pH measurements, which would be a mean pH, the results based on such techniques must be interpreted with great caution. This applies especially to evaluation of H₂-receptor antagonist treatment in patients in the ICU, in whom a consistent increase in pH to > 4 is achieved rarely, and in whom recurring episodes of acidity occur [3-5]. Consequently, to examine the effect of pH on bacterial growth, frequent or, preferably, continuous measurements of pH should be performed.

In the study by Inglis and colleagues [1], six patients were given sucralfate, which has a bactericidal action independent of pH [2, 6], and these patients should be excluded from the analysis of the effect of pH on bacterial growth. It would be interesting to have some more information on the bacterial strains cultured, as the susceptibility to acid may vary with individual bacterial properties, such as resistance to antibiotics [1]. Did insertion of the nasogastric tube lead to contamination of the gastric specimens with oral flora in patients who were hypoxic?

The increased duodenogastric reflux documented in these patients indicates post-traumatic and postoperative intestinal motility disorders, which could well predispose to gastric stasis and bacterial regurgitation from the lower intestine. More specifically-designed studies seem to be necessary to elucidate the exact relationship between gastric pH and bacterial growth in ICU patients.

C. H. WILDER-SMITH
Berne, Switzerland


Sir,—The author of this letter made five points which I should like to address.

Work we have performed [unpublished] also has indicated that the pH of gastric specimens fluctuates frequently in some patients undergoing artificial ventilation of the lungs; however, we were unable to sample more frequently on account of the volume of gastric aspirate required. If we had done so, we would not have been able to discard the nasogastric tube and in the sample volume and still have a sample sufficient for analysis. Moreover, Dr Wilder-Smith claims that almost all bacteria in the stomach will be eradicated by very low pH within 10 min. Work in several different centres indicates that small numbers of bacteria exposed to low pH for up to several hours remain viable, and may regrow after a lag phase. As the relationship between bacterial death and exposure to acid is a complex one, the only practical approach to use in such cases is to measure the organisms present and the pH in the same sample, and to try to avoid speculation about fluctuations of each variable during the intervening period.

The author refers to two publications claiming antibiotic activity for sucralfate. Both of these studies used non-standard bacteriological methods. The claim for antibacterial activity for this compound is therefore still controversial. We are investigating this compound currently using conventional antimicrobial analyses, and although our preliminary results support the presence of some effect, this is variable and may depend on different local factors, including: species of bacteria, mucosal binding, local pH and the presence of bile acids. Therefore, it is not possible to anticipate the activity of sucralfate on organisms isolated from gastric aspirate specimens without measurement of these, as yet, unevaluated variables.

The list of bacterial species cultured from samples in this study is shown in table 1.

<table>
<thead>
<tr>
<th>Species</th>
<th>No. of patients colonized</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram-negative bacilli</td>
<td>2</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>2</td>
</tr>
<tr>
<td><em>Pseudomonas fluorescens</em></td>
<td>2</td>
</tr>
<tr>
<td><em>Klebsiella pneumoniae</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Klebsiella oxytoca</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Serratia marcescens</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Serratia liquefaciens</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Proteus mirabilis</em></td>
<td>1</td>
</tr>
<tr>
<td><em>Enterobacter agglomerans</em></td>
<td>1</td>
</tr>
<tr>
<td>Other</td>
<td>10</td>
</tr>
<tr>
<td>Gram-positive cocci</td>
<td>7</td>
</tr>
<tr>
<td><em>Streptococcus spp.</em></td>
<td>7</td>
</tr>
<tr>
<td><em>Staphylococcus spp.</em></td>
<td>3</td>
</tr>
<tr>
<td>Gram-negative cocci</td>
<td>3</td>
</tr>
<tr>
<td>Gram-positive bacilli</td>
<td>1</td>
</tr>
<tr>
<td>Yeast</td>
<td>4</td>
</tr>
</tbody>
</table>

It is true that insertion of a nasogastric tube may introduce bacteria from the bronchial cavity or oropharynx into the stomach, which subsequently colonize the gastric lumen. However, this is an issue of secondary importance, as all these patients had a nasogastric tube, and it does not account for the apparent relationship between gastroduodenal reflux and bacterial overgrowth.

The author states that further studies are required and is, no doubt, keen to conduct one himself! In our publication we have taken great care to present our data as a preliminary report and have sought to draw only tentative conclusions. A follow-up study addressing the relationship between gastroduodenal dysfunction and bacterial colonization of the ventilated airway in a larger number of patients is nearing completion. Although the statistical analysis has yet to be confirmed, preliminary results appear to support the conclusions of our pilot study and have important implications for the prevention of ventilator associated pneumonia.