state of left ventricular filling or performance. Pulmonary oedema can be, and is effectively, treated by reducing cardiac preload. The benefit of extradural block in this respect is recognized (Moir and Willocks, 1968; Barnes, 1976). Similar gains can be obtained from spinal block and this was exploited in our patient, with success.

From his remarks it is difficult to tell which technique Dr Prince would use: incremental extradural or general anaesthesia. This is not surprising. Management of such complicated cases is always open to criticism. Retrospectively, it is easy to say that other techniques would have been more suitable. However, in an unprepared emergency Caesarean section the time factor is of paramount importance. It seems to me that Dr Prince did not appreciate this urgency. Not only maternal distress was present, but the fetus, who incidently spent 3 weeks in the special baby care unit with meconium aspiration, was facing imminent intra-uterine death. It is my view that neither technique he described would have been suitable for both mother and fetus.

Lunn and Mushin (1982) expressed their opinion that the "quality of the anaesthetist is more important in terms of outcome than the drugs or technique he chooses to use". Their adverse comments in connection with spinal and extradural blocks are related to mismanagement and failure to recognize their disadvantages. The successful outcome in our case is evidence that the technique was fully exploited to the benefit of both mother and infant. Given the knowledge of the disadvantages of other techniques, spinal anaesthesia was not a poor choice; it was, in our view, the only choice in such circumstances.

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REFERENCES


The Problem of Intradural Ketamine

Sir,—I have read with interest the letter of Brock-Utne and his colleagues (1985). In their first publication they had reported that, after extradural ketamine, pain relief was obtained in every patient suffering from cancer pain (Mankowitz et al., 1982).

Other authors (Saissy et al., 1984) also reported on the analgesic effects of extradural ketamine in postoperative pain.

In some of these patients the duration of pain relief was increased when repeated injections, or an infusion, was used. However, in one alcoholic patient the extradural ketamine failed.

Although complications after extradural ketamine in man have not been reported as yet (Mankowitz et al., 1982; Rubin et al., 1983; Saissy et al., 1984), clinical trials should be postponed temporarily, until any possible toxicity has been investigated fully.

In my opinion the differing views as to the toxicity of intrathecal ketamine when used in small or large animals may result from the spinal effects of the different concentrations of the drug used. In rats the volume of the solution injected intrathecally was restricted to 4 to 6 ml (Ahuja, 1983) and the required dose of ketamine had to be injected in a very high concentration. The drug may be diluted by the CSF; however, in small animals this will not be as significant as in larger animals. This high drug concentration in small animals might explain why 9.1% of rats died in Ahuja's experiment. In rats, the concentration of intrathecally administered ketamine was more than 100 times higher than in humans if given extradurally. This might explain the fact that Brock-Utne found nerve degeneration in small monkeys, but not in larger baboons (Brock-Utne, Kallichurum et al., 1982; Brock-Utne, Mankowitz et al., 1982). It seems reasonable, therefore, to devise experiments in which the histotoxic effects of different drug concentrations will be examined at spinal and supraspinal levels both microscopically and electronmicroscopically.

Since, as yet, we do not have a safe, widely used method with which to control pain, it would be very important to examine not only opioids, but also the role of other neurotransmitters in analgesia. As ketamine is acting not only on opioid receptors, but also on the noradrenaline and serotonin systems (Martin, Bouchal and Smith, 1982; Martin and Smith, 1982; Lundy and Jones, 1983), the proposed research may be interesting.

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REFERENCES

Capnograph. However, it is clear that the capnograph, sampling expiratory phase of the respiratory cycle, starts to decrease tracing of concentration of carbon dioxide, measured in the may be a source of error in the measured end-tidal opposed to other measures of the respiratory effects of concentration. In the presence of respiratory depression, the drug for assessment, and their emphasis on tidal volume as comparisons of meptazinol and pethidine in patients anaesthe-
and his colleagues (1985), since we have carried out similar
Sir,—We read with great interest the article of Dr Wilkinson from the strap 149 times out of a possible 180 times that the
which would not have occurred had the mask been lifted off
At laryngoscopy before intubation, a small rectangular metal clip was seen lying on the posterior pharyngeal wall and was removed with Magill forceps.
It was discovered that the 6 mm x 5 mm clip had come from the strap of the Hudson mask, and had become detached when the mask was removed by pulling the elasticated strap through the securing port of the mask. These pliable metal clips are fixed by two short teeth to the ends of the strap and enable it to be threaded easily through the mask.
The presence of a foreign body was not suspected as the patient neither coughed or gagged before induction, and the small clip may have easily been concealed by blood or mucous in the pharynx.
The authors would like to draw attention to this potential hazard with the Hudson disposable "see-thru" oxygen masks, which would not have occurred had the mask been lifted off the patient instead of pulling the strap through the mask.
In a sample of 90 masks, we found that the clip was displaced from the strap 149 times out of a possible 180 times that the strap was drawn through the mask (82%).

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RESPIRATORY DEPRESSION—TIDAL VOLUME OR FREQUENCY?

Sir,—We read with great interest the article of Dr Wilkinson and his colleagues (1985), since we have carried out similar comparisons of meptazinol and pethidine in patients anaesthetized with halothane. We believe that both their measurements and their interpretation of the changes in end-tidal carbon dioxide concentrations may be in error. Their use of a 3-min infusion as a method of administering a respiratory depressant: drug for assessment, and their emphasis on tidal volume as opposed to other measures of the respiratory effects of analgesics, would also seem to be inappropriate.
The tracing of carbon dioxide illustrated suggests that there may be an error in the measured end-tidal concentration. In the presence of respiratory depression, the tracing of concentration of carbon dioxide, measured in the expiratory phase of the respiratory cycle, starts to decrease shortly after most of the tidal volume has been exhaled. Accurate estimation of the time relationship of the two events is made difficult because of the delay in response of the capnograph. However, it is clear that the capnograph, sampling gas at 200 ml min⁻¹, is sampling not only gas from the expired tidal volume of the patient, but also from the fresh gas flow. Since, in some circumstances, the expired minute volume of the patient was only 1 or 2 litre min⁻¹ and the fresh gas flow was about 7 litre min⁻¹, the dilution of expired gas by fresh gas could be considerable, and would vary with the degree of respiratory depression and degree of prolongation of the expiratory phase. Consequently, the end-tidal measurements in these circumstances will underestimate the degree of respiratory depression because, the more severe the depression (and hence, the increase in alveolar carbon dioxide concentration), then the more the small volume of gas expired in each breath will be diluted by the fresh gas flow. This effect will also be increased by a prolongation of the duration of expiration since the expiratory flow will become less or even cease before inspiration results in reversal of the flow.

While Dr Wilkinson and his colleagues do come to the conclusion that meptazinol causes significant respiratory depression, administration of a potential respiratory depressant over 3 min in order to avoid the problem of apnoea would seem to be a dangerous precedent to set in an assessment of this type of drug. It also makes interpretation of changes in ventilation caused by the administration of depressant drugs more difficult, as the patient is exposed to other influences that may change at the same time (but not necessarily at the same rate).
The most obvious influence of this type is carbon dioxide itself. Within a few breaths, the respiratory centre will be stimulated by the increase in carbon dioxide tension that has been caused by the reduced ventilation, and this will act to offset the reduction in ventilation that has been caused. In a similar way, a reduction in ventilation will result in a reduction in alveolar and arterial halothane concentrations, and this will, rather more slowly, result in a decrease in the effect of halothane on the ventilation. Depressant drugs, therefore, will tend to be counteracted by the effects they produce. This effect may be evident in the case of meptazinol, with which respiration had increased only 1 min after the end of the injection of the drug. Subsequent measurements are also confused by the respiratory effects of varying surgical stimulation.

In our experience tidal volume is relatively unaffected by pethidine and is decreased by meptazinol. However, this effect is seen only in the first 2 min after rapid bolus administration. After this, tidal volume tends to increase, presumably as a result of increasing respiratory drive from carbon dioxide. The regression data of Wilkinson and colleagues, in which percentage changes in frequency are plotted against percentage changes in minute volume, confirm this effect on tidal volume. Their regression lines, when extrapolated to zero change in frequency, show a 30% decrease in minute volume that could be attributed to a decreased tidal volume after meptazinol. (Small changes in frequency were seen only immediately after the start of administration of the drug.) The slope of the line means that, when decreases in respiratory frequency similar to those observed later in their study are considered, there is a negligible change in tidal volume. This conclusion is supported by their finding that minute volume was increasing, when respiratory frequency was continuing to decrease, 4 min after administration started—indicating an increase in tidal volume although these data were not tabulated. Similar effects are seen after small doses of fentanyl (Drummond, 1983). Because of the different time courses of changes in tidal volume and frequency, it is possibly unsafe to use the pooled data to construct regression relationships, and certainly misleading to extrapolate from them.