antagonism. It is certainly true that the response induced a "false sense of security" within the medical staff as to the aetiology of the sedation and increased the delay before its full investigation.

The message is clear that, when evaluating a patient with unexpected sensory depression or frank CNS symptomatology, consideration must be given to the same diagnoses that would be entertained had the finding occurred independent of a temporal relationship to a recent anaesthetic. Particular care should be exercised in the interpretation of a positive response to naloxone as being diagnostic of narcotic overdosage.

B. C. SELLICK
London

REFERENCE


HYPOACOUSIS FOLLOWING EXTRADURAL INJECTION

Sir,—Three patients have been observed in whom acute loss of auditory acuity (hypoacousis) occurred at the end of the injection of local anaesthetic solution to the lumbar extradural space. A standard technique was used. Puncture was performed at L2-3 using loss of resistance to saline; an extradural catheter was inserted. A test dose of 2% lignocaine 4 ml was injected through the catheter. In no patient were there neurological or cardiovascular symptoms to suggest i.v. injection.

Patient 1 was a 59-yr-old man undergoing metatarsal osteotomies of the right foot. After the injection of the main dose of 2% lignocaine 16 ml with adrenaline, he complained that he felt that someone had placed cotton wool in his ears. The hypoacousis lasted for 10 min before return to normal hearing. Anaesthesia was obtained to T6.

Patient 2 was a 34-yr-old primigravida in labour. After the injection of 0.5% bupivacaine 8 ml she complained that her ears had become blocked. The hypoacousis lasted for 5 min before return to normal. Analgesia was obtained to T9. Subsequent "top-ups" of the same dose were eventful.

Patient 3 was a 25-yr-old primigravida undergoing Caesarean section. An injection of 2% lignocaine 16 ml with adrenaline was given. At the end of this she complained of "fullness" in her head, accompanied by the feeling that her ears had become blocked. The sensation of fullness in the head lasted some 30 s, but the hypoacousis persisted for almost 10 min. Anaesthesia was obtained to T5 and the Caesarean section progressed eventfully.

Injection of fluid to the extradural space gives rise to feelings of fullness in the head as a result of a concomitant increase in intracranial pressure (Burn, Guyer and Langdon, 1973). There is free communication across the cochlear aqueduct between cerebrospinal fluid and the perilymph of the cochlear apparatus (Warwick and Williams, 1973). Changes in CSF pressure are therefore accompanied by changes in cochlear perilymph pressure. The position of the hair cells and basement membrane of the cochlear apparatus is determined by the relative pressures in the perilymph (equals CSF pressure) and the endolymph (active secretion). Post-spinal hypoacousis has been reported in which a decrease in CSF pressure produces a distortion of the structures by pressure imbalance and gives rise to damping of the response to auditory stimuli (Gordon, 1983; Panning, Mehler and Lennhardt, 1983). It is possible, therefore, that the hypoacousis following injection of fluid to the extradural space is a consequence of the increase in perilymph pressure which accompanies the increase in CSF pressure. It is important to differentiate symptoms resulting from the mechanical effect of injection from those caused by intravascular injection of local anaesthetic.

P. A. J. HARDY
Edinburgh

REFERENCES


PROLONGED APNOEA WITH KETAMINE

Sir,—Ketamine is used all over the world, frequently in minor procedures involving children. In the developing countries anaesthetics are usually administered by nurses or auxiliaries and ketamine, as well as ether, is frequently used. However, because of its reputation for general safety, support of the circulation and lack of respiratory depression, relatively little attention is paid to the vital functions during its use (Philips et al., 1970). This is understandable, since medical officers usually operate under primitive circumstances, in undermanned hospitals and with relatively poorly trained personnel (van Wijhe, 1981). Although the use of ketamine presents few problems in the majority of patients, it is a drug with potentially serious adverse effects (Austin and Bevan, 1971).

Since we have treated a child who developed prolonged apnoea despite use of the recommended dose and standard procedures (Gregory, 1983), we advise that adequate means of artificial ventilation be at hand when ketamine is used.

A 4-month old girl was scheduled for an EMG, a cervical myelography and a CT scan of the cervical spinal cord for investigation of a brachial plexus injury obtained at birth. Her weight was 6.3 kg, her birth had been at term, and a general physical examination showed no other abnormalities (notably, no signs of increased intracranial pressure). She was not receiving any drug(s). As the expected duration of the investigations was 1.5-2 h we decided to intubate the trachea so as to be able to assist the respiration when necessary. A premedication of pentobarbitone 25 mg rectally was given 2 h before the induction of anaesthesia, which consisted of ketamine 60 mg i.m. (10 mg/kg body weight), oxygenation by facemask, placement of an i.v. catheter, atropine sulphate 0.12 mg i.v. and suxamethonium 12 mg i.v. Oral intubation with a 3.5-mm diameter tracheal tube (without cuff) was uneventful; auscultation of the lungs confirmed the correct position of the tube. Oxygen was given with the Jackson–Rees non-rebreathing system. Because of some movement 15 min after the i.m. dose, an additional ketamine 10 mg was administered slowly i.v., and the investigator was requested to...