EXTRADURAL MORPHINE AND STOKES-ADAMS ATTACKS

Sir,—There have been several reports of respiratory depression after the extradural injection of morphine, but cardiac depression has been seen only after intrathecal injection and then in combination with respiratory depression (Liolios and Andersen, 1979; Davies, Tolhurst-Cleaver and James, 1980). We describe an extradural injection of morphine complicated by Stokes-Adams attacks without respiratory depression.

A healthy, 40-yr-old woman underwent hysterectomy; general anaesthesia included halothane. One hour before operation the patient received pethidine 50 mg i.m. and morphine chloride 4 mg in isotonic sodium chloride 25 ml through a lumbar extradural catheter. Morphine 4 mg in isotonic sodium chloride 10 ml was injected extradurally 2 5, 3, and 5 h later. The patient suddenly felt nauseated and fainted for 1 min. 4 h after the last injection. The continuously monitored ECG indicated A-V block with a 15-s period of asystole and atropine 0.6 mg was given i.v. with immediate reversal to normal sinus rhythm. There were two similar attacks on the following day, 13 h and 15 h after the second of two doses of morphine 4 mg in sodium chloride 10 ml, which had been given 6 h apart. The patient did not receive further morphine extradurally and morphine diniconnate ester (Vilan) was administered i.m. without complications for the next 2 days. A week later the patient was discharged without any evidence of cardiac disease.

The Stokes-Adams attacks were probably caused by increased parasympathetic activity from stimulation of the dorsal nucleus of the vagus as a result of the rather large dose of morphine. The interval of 4-15 h between the morphine injection and the cardiac depression is in accordance with that reported for respiratory depression following extradurally injected morphine. This indicates the same pharmacodynamic mode of action, but strangely enough, we did not observe respiratory depression.

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POSTERIOR FOSSA SURGERY

Sir,—We were depressed to read the survey of posture and ventilation by Campkin (1981).

The frequency of air embolism during neurosurgery in the sitting position depends on the diagnostic criteria, but has been said to be as high as 93% (Michenfelder, Miller and Gronert, 1972) with a mortality of up to 73% (Ericsson, Gottlieb and Sweet, 1964). Buckland and Manners (1976) studying 36 patients described a frequency of 33%, with two deaths. To this is added the complication of hypotension which may embarrass cerebral blood flow in elderly patients or those whose cerebral circulation is already compromised by the pathology. In contrast the use of the prone or semi-prone position eliminates both these problems, borne out by the fact that there has been no death in this unit from air embolism since the adoption of this position.

It was stated that adopting the sitting position allowed the surgeon to orientate himself as the patient was in the same position as himself. If all surgeons adopted this idea can we expect to see surgeons in the supine or lithotomy positions? In our view the use of the sitting position with all its attendant risks should be limited to a few specific instances when the use of the prone position would necessitate excessive retraction. Surgeons and anaesthetists who favour the routine use of the sitting position and are currently investigating methods of detecting air embolism would be far better studying and practising techniques which use the prone position and thus allow them to eliminate this complication completely.

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