will be encountered. In fact, with a tight-fitting mask, a feeling of suffocation would be inevitable and, in practice, the patient would simply augment inspiration through his mouth.

Using time-weighted sampling with gas chromatography, we found ambient levels of nitrous oxide, as measured next to the patient's feet, of 114, 201, 108, and 135 ppm in an exceptionally well-ventilated operating room of our dental clinic. Under the same conditions, we then employed the Brown Scavenging Mask and found the following consecutive levels of nitrous oxide at the patient's feet: 114, 23, 60, 25, 25, 90, 101, 69 and 125 ppm. The first and last high values were measured at times when we also monitored the force of suctioning and found that it was 1 to 2 l/min below the recommended level of 7 l/min. The other high values were, in our opinion, inevitable, if only because of a basic dilemma. The dilemma is that of potentially scavenging the anesthetic before it can reach the patient. Normally, during quiet breathing, the peak velocity of flow attained in the upper airway is of the order of 25 to 30 l/min. For a patient sitting in the dentist's chair, one may safely assume a higher figure. Spillage, and incidentally provision of at least some anesthetic to the patient, are two inevitable results of the use of correspondingly high fresh gas flows. We are trying to find a solution such as McKesson himself provided with his now defunct demand-flow machine, and that should not be beyond the ken of the manufacturers of anesthetic apparatus.

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## Uvular Edema

*To the Editor:*—We were interested in the report by Drs. Ravindran and Priddy[1] of a case of uvular edema following endotracheal intubation. We had a similar case last month. A 50-year-old man was admitted for repair of an epigastric hernia. He had no history of allergy to medications, and physical examination showed him to be in good condition. ECG, electrolyte, hemoglobin and urinalysis values were all within normal limits. He was premedicated with meperidine, 100 mg, diazepam, 5 mg, and glycopyrrolate, 0.2 mg. Anesthesia was induced with thiopental, 300 mg, followed by succinylcholine, 50 mg, and endotracheal intubation was accomplished atraumatically using an 8.5-mm Murphy low-pressure, high-volume cuffed tube. Because the patient had capped front teeth, we elected not to place an oral airway; but, in anticipation of possible airway obstruction after extubation, a no. 30 soft latex rubber nasal airway was placed in the left nostril at the time of intubation. Anesthesia was maintained with nitrous oxide, oxygen, and succinylcholine supplemented with Innovar®, 2 ml, and 0.15 mg fentanyl infusion, and was uneventful. Anesthesia lasted 50 min. The patient was returned to the recovery room awake and breathing normally. The nasal airway was removed at the time of extubation and in fact had not been functionally useful. The following day the patient complained of a feeling that something was "falling down" the back of his throat. On inspection, the tip of the uvula was found to be deep red, swollen and necrotic. There was no respiratory difficulty, and no treatment was needed. Eight days later the tip of the uvula had sloughed off and the area had healed.

Like Drs. Ravindran and Priddy, we could find no report of a similar incidence in our review of the literature until we saw their report. In our case, we felt that the cause was entrapment of the uvula between the nasal airway and the endotracheal tube. With the head turned slightly to the right, as is customary following endotracheal intubation, it would not be surprising if the uvula fell towards that side and were entrapped between the two tubes. This is particularly possible when the nasal tube comes from the left, since its direction in the pharynx would be from left to right towards the midline. Similar entrapment could theoretically occur between an oral airway and the endotracheal tube, and we wonder whether such might have been the cause in the case reported by Drs. Ravindran and Priddy. One might infer that there would be insufficient pressure to occlude blood flow in such a juxtaposition of soft latex rubber with

thermolabile plastic. However, we feel that a "venous tourniquet" effect could occur if slight twisting of the uvula resulted from the airway placement.

In retrospect, we wonder whether such occurrences are not more common than has been supposed but go unrecognized because they are regarded simply to be postintubation sore throat.

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## Ketamine in Acute Intermittent Porphyria—Dangerous or Safe?

*To the Editor:*—Rizk *et al.* report the use of ketamine in a patient with acute intermittent porphyria without complication, and conclude that ketamine appears to be safe in patients with this disease.[1]

Porphyrinogenic activity of drugs may be assessed by examining their effects on the activity of delta-aminolevulinic acid synthetase (ALA-S) in liver homogenates of 17-day-old chicken embryos.

Eight hours after the injection of ketamine (Ketalar®, Parke-Davis), the activity of ALA-S in the liver increased (fig. 1). The increases were highly significant ($P < 0.001$) at doses of 2 mg ketamine/egg or more as compared with the saline control. Ketamine, 0.5, 2, and 5 mg/egg, induced highly significant ($P < 0.001$) increases in the activity of ALA-S four to eight hours after injection (fig. 2).

From our experiments we conclude that ketamine has porphyrinogenic effect and should not be given to patients with acute intermittent porphyria.

The discrepancy between our results and the clinical

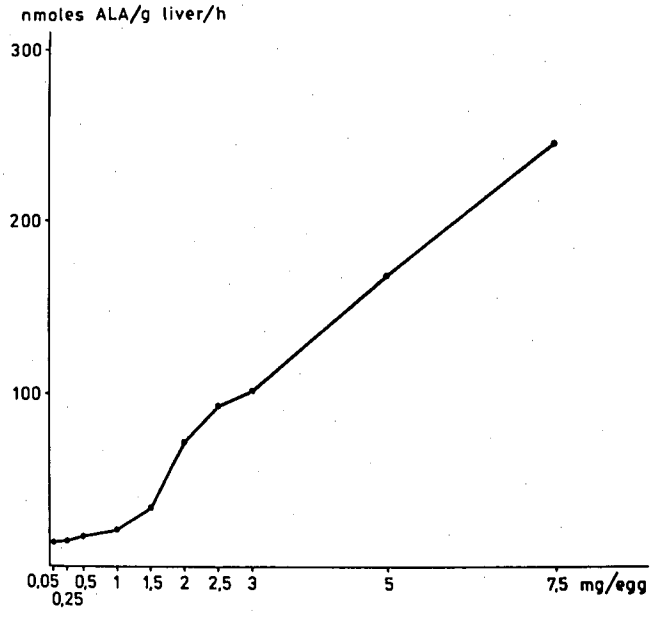


FIG. 1. Activities of ALA-S eight hours after different doses of ketamine. Each point represents the mean from ten examinations.

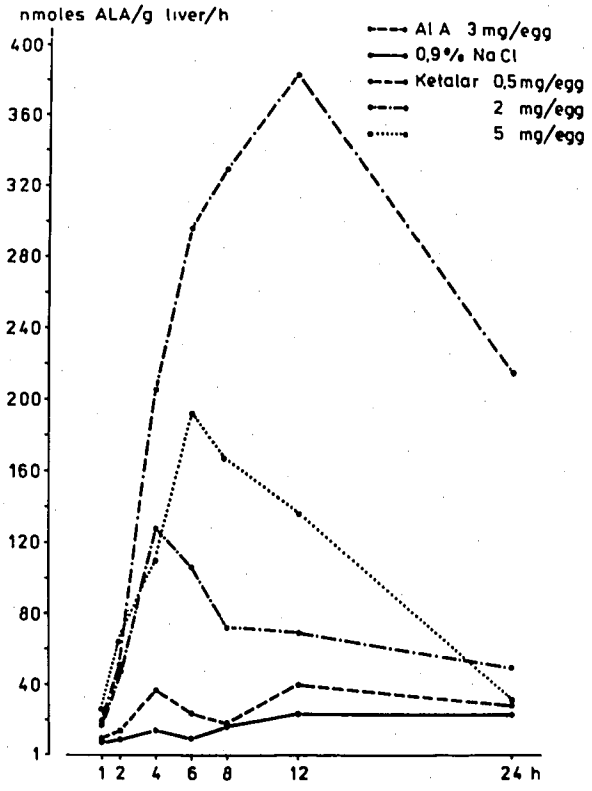


FIG. 2. Activities of ALA-S over a 24-hour period after injection of three doses of ketamine, 0.9 per cent NaCl, and AIA (allylisopropylacetamid)—a strong porphyrinogenic material. Each point represents the mean values from ten examinations.