tions above the vocal cords are effectively removed thereby. No vocal cord damage has been reported to occur using this technique.

We also personally know of many instances in which the Fome-Cuf was removed without first being deflated, with no untoward effect.

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Local Anesthetic *vs.* Spinal Fluid

To the Editor:—In a recent letter (ANESTHESIOLOGY 44:451, 1976), Dr. Reisner indicates the value of using a urine test strip to distinguish cerebrospinal fluid from local anesthetic solution. I am writing to call attention to another simple method for distinguishing the two solutions. If the fluid dripping from the hub of the needle is allowed to come in contact with a solution of thiopental (*p*H 10), turbidity will occur immediately when the fluid is local anesthetic (*p*H 5) but not when it is cerebrospinal fluid (*p*H 7).

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Cuffs Do Not Seal the Trachea Airtight

To the Editor:—In 31 consecutive patients receiving closed-circuit anesthesia I measured the concentration of nitrous oxide in the pharynx with a Foregger Nitrous Oxide Monitor. In every case I found between 100 and 800 ppm of nitrous oxide in the pharynx in spite of zero concentrations at all other areas such as outside the mouth, around the circle system, and at the ventilator. In all cases, the cuffs were inflated to the point of no audible leak with positive pressure applied to the endotracheal tube.

Mehta found that cuffs do not protect against aspiration of contrast medium, but attributed this to its presence in the ventricle between false and true cords at the time when the cuff is deflated.[1] Egnatinsky believes that dilatation of the trachea normally seen with inspiration allows aspiration around a cuff, and therefore recommends deliberate overinflation of high-volume cuffs.[2] Pavlin *et al.* reported failure of a large-volume cuff to protect against aspiration in a spontaneously breathing patient.[3] Stanley *et al.* recommend a pressure-relief valve to prevent overinflation of cuffs due to disproportionately rapid inward diffusion of nitrous oxide into cuffs, compared with slow outward diffusion of nitrogen.[4] They rightly caution against overinflation in view of possible compression of the wall of the endotracheal tube or its orifices. The membranous posterior wall of the trachea offers the least resistance to overinflation, and when 20 ml of air were injected into an ordinary cuff on a #9 Magill tube a 7 cm-long rupture of the trachea resulted.[5]

It seems timely to emphasize that cuffs do not provide an airtight seal except perhaps prior to utmost stretching of the trachea just preceding its rupture. My measurements show that the usual clinical maneuver of inflating cuffs just to the point of abolishing audible leakage with positive pressure to the airway does not result in an airtight seal. These results are easily verifiable with closed systems, which are without exception associated with leakage in the order of 20 to 100 ml/min. The practicing anesthetist should not place reliance on cuffs to seal the larynx, but should continue to employ additional safeguards against aspiration such as throat packs and avoidance of spontaneous respiratory efforts whenever feasible.

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Anesthesiology
46:310, 1977

Lipolysis by Halothane Questioned

To the Editor:—The article, "Studies of the Dual Effects of Halothane on the Lipolysis of Human Fat Cells," by Drs. J. Bennis and U. Smith (ANESTHESIOLOGY 45:379–384, 1976) falls short of its title and lacks adequate documentation to make the results interpretable for the reader. The data presented to document "the dual effects of halothane on lipolysis" are, at best, questionable, since they were obtained under different experimental conditions.

Fragments of fatty tissue, and not fat cells, were used (although the method for obtaining the latter is available).[1] More importantly, one does not know when halothane "via the gas phase above the medium" was added. Was the experiment performed when equilibrium was achieved? When was it achieved, if known? Was the rate of lipolysis linear during the two-hour incubation? The reader cannot compare the concentrations of halothane added directly to the medium with the concentrations of halothane in the experiments when it was added via the gas phase.

The results listed in Table 2 of the article are even more disturbing. Halothane, 10^{-6} M, produces an increase of lipolysis of 27.7 per cent above control with a standard error of the mean of 34.8 per cent. This result is claimed to be significant as determined by the t test for paired data, but the reader cannot confirm this. The data certainly could not be significantly different when analyzed by unpaired Student's t test. The results showing the effects of propranolol and practolol on lipolysis at different concentrations of halothane are not convincing. In fact, the standard error of the mean in the series with propranolol was as high as ±30.7 per cent of the control, and the maximum change from control in the presence of propranolol and halothane was 12 per cent. It goes without saying that, even if β-adrenergic antagonists would decrease the effect of halothane significantly, one would be very daring in making such a conclusion based on one single dose of the antagonist tested. I find the conclusion, the "lipolytic effect of halothane is exerted via direct β-adrenergic stimulation," not substantiated by the data presented.

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Magnesium Deficiency Causing Persistent Hypokalemia

To the Editor:—I wish to comment on the case of primary aldosteronism with severe total-body depletion of potassium reported by Gangat and colleagues (ANESTHESIOLOGY 45:542–544, 1976). Upon induction of anesthesia the patient had tonic muscular contractions of the upper torso and arms, and possible diagnoses of myotonia dystrophica, malignant hyperpyrexia, electrolyte imbalance, and cerebrovascular accident were considered. Subsequently a marked total-body deficit of potassium was determined to be present; it took nine days of intensive therapy to correct the deficit.

I wish to emphasize that primary aldosteronism may cause profound urinary excretion of magnesium as well as potassium. Once established, hypomagnesemia may make correction of the potassium deficiency more difficult and protracted unless magnesium is also administered.[1] Anesthetists should be aware of the relationship of magnesium to potassium and consider measurement of magnesium levels when they encounter unexplained hypokalemia.

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