pH 4.0. The pH of the mixed solution should be close to 6.8 due to the strong buffering action of the phosphate. The pH of a mixed solution can be any value, according to the buffering capacity of each mixing component, and the mathematical average has no relevance to the pH of the mixed solution.

Our view of the thermodynamics of pH and the derived hydrogen ion concentration has already been expressed.² Apparently, his error in the experimental design was caused by confusing acidity expressed by the chemical potential and that expressed by the titration.

From the equations in the Appendix, it follows that a decrease of the ionic strength of an acidic buffer increases the pH, while that of an alkaline buffer decreases it, and an increase of the ionic strength works in the opposite way.

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APPENDIX

The concentration (c) of an ion is related to the activity (a) by the activity coefficient (γ)

$$a = vc$$

and the value of γ varies depending upon, among other factors, the ionic strength of the solution.

The ionic strength (I) of a solution is given by

$$I = \frac{1}{2} \cdot \sum (c_1 z^2)$$

where the subscript i refers to the i-th component and z is the valence of the ion.

An approximate form of the Debye-Hückel equation (see, for instance, a textbook by Perrin and Dempsey³) in *dilute solutions* gives the following relationship. (Theory for a condensed solution is yet to be formulated).

$$-\log \gamma = Kz^2I^{1/2}/(1 + I^{1/2}) - 0.1z^2I$$

where K is a constant that depends only on the temperature.

The approximate generalized equation for a buffer solution is given³ as follows. For an acidic buffer

$$pH = pK_a + \log [H_{n-1}A^{(x+1)-}]/[H_nA^{x-}] - (2x+1)KI^{1/2}/(1+I^{1/2}) + 0.1(2x+1)$$

For an alkaline buffer

$$pH = pK_{a} - \log [H_{n+1}B^{(x+1)+}]/[H_{n}B^{x+}] + (2x+1)KI^{1/2}/(1+I^{1/2}) - 0.1(2x+1)$$

where H_nA^{x-} and H_nB^{x+} are acidic and basic buffer, respectively, and n and x are integer values. The expressions x+ and x- refer to the numbers of positive and negative charges, respectively, carried by the HB and HA ions.

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Pulmonary Aspiration Following Antacid Therapy

To the Editor: —Two articles concerning aspiration pneumonitis appeared in the November 1979 issue of Anesthesiology. 1,2 The first described a study in dogs in which the antacid Kolantyl Gel® was instilled into the mainstem bronchi, demonstrating a more prolonged pulmonary reaction than occurred after instillation of hydrochloric acid. The second was a re-

port of a case of pulmonary aspiration of gastric contents rendered nonacid (pH 6.4) by prior ingestion of the antacid Riopan[®]; the patient had hypoxemia for 72 hours, with pulmonary infiltrates visible on chest x-ray for approximately seven days. Despite disclaimers to the contrary in both articles, one is left with the message that pulmonary

aspiration of antacid-containing liquid gastric contents may produce worse sequelae than aspiration of gastric acid itself.

Many factors in addition to the pH of the aspirate are involved in the genesis of chemical aspiration pneumonitis. Alexander³ has shown that pulmonary edema may develop after aspiration of water or saline solution as a result of distortion of the osmotic gradient across the alveolar-capillary membrane, and that the presence of acid in the fluid merely potentiates the exudative reaction. Intestinal bacteria have been implicated in the pathogenesis of aspiration pneumonitis,4 although bile, digestive enzymes, and gastric bacteria have been found to be of little importance.5 Finally, as pointed out by Gibbs et al.,1 antacid suspensions contain not only the magnesium and aluminum elements, but also preservatives, stabilizers and flavorings. Thus, the antacid preparation used may be of some significance if one of the many additives has the propensity to cause a pulmonary lesion. It may, therefore, be of interest to describe two cases of pulmonary inhalation of the antacid Mylanta® (aluminum hydroxide, 40 mg/ml, magnesium hydroxide 40 mg/ml, simethicone 4 mg/ ml) that resulted in no pulmonary complication.

REPORT OF TWO CASES

The first patient was a healthy primigravida who, about an hour after her last dose of 15 ml of antacid, suffered a grand mal seizure due to inadvertent intravascular injection of lidocaine, 1 per cent, during an attempted pudendal block. She regurgitated and aspirated whitish fluid, the pH of which was above 3. She was treated with pharyngeal and laryngeal suctioning and administration of 100 per cent oxygen via an endotracheal tube. Twelve minutes later she awoke. There was no clinical or laboratory evidence of pulmonary abnormality, and the infant, delivered by outlet forceps immediately after the convulsion, was in good condition.⁶

The second patient, a secundipara in active labor, regurgitated and aspirated white gastric contents during an epileptic convulsion 10 min after her first 15-ml dose of Mylanta. Again, the pH of the aspirate was above 3. The mother, delivered of a healthy infant about three hours later, made a totally uneventful recovery.

These two histories do not prove that the patients involved would have suffered some pulmonary dysfunction had they not previously ingested antacid, or that Mylanta is better than other brands of antacid. We have simply presented another side of the

question in an attempt to spur further investigation of the role of antacids in preventing the acid-aspiration syndrome. Further, when one considers the data obtained by Lewis $et\ al.^7$ in non-obstetric patients, where inhalation of gastric juice of confirmed low pH without preceding oral antacid was followed by hypoxemia of a mean duration of 5.6 days, the 72-hour episode of hypoxemia in the case of Bond $et\ al.^2$ may be seen as an improved outcome due to prior antacid therapy.

Obviously, measures to thwart regurgitation and pulmonary aspiration are the first line in the prevention of the acid-aspiration syndrome, but routine administration of antacids to parturients during labor should be continued until there is solid evidence either that it does no good or that it does more harm than good.

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Avoiding Complications during Jet Ventilation

To the Editor:—Oliverio et al.'s recent report¹ of a ball-valve obstruction and pneumothorax following the use of the Sanders' jet injector during removal

of laryngeal papillomas in a 2½-year-old child deserves comment, for two reasons.

First, in 1974 Smith² pointed out that high intra-