ml/min, at fresh-gas inflow of 5 L/min will inhale 1.3% CO_2. However, if his $\dot{V}CO_2$ rises to 250 ml/min during surgery (light anesthesia, loss of muscular relaxation, increased surgical stimulation, change in acid-base status, etc), he will now breathe 3% CO_2 and we *do* consider this excessive.

In answer to the second point, the new equation cited has $\dot{V}CO_2$ in the numerator of two of the fractions, and, therefore, any rise in $\dot{V}CO_2$ will necessarily cause a rise in $PaCO_2$ if the denominators are unaltered (which is the case during mechanical ventilation).

In addition, Conway[3] has shown that during the use of semiclosed rebreathing anesthetic systems:

$$FACO_2 = \frac{(FFO_2 - FAO_2)\ FICO_2}{(FFO_2 - FIO_2)}$$

where $FACO_2$ = alveolar CO_2 concentration, FFO_2 = O_2 concentration in the fresh flow, FAO_2 = alveolar O_2 concentration, $FICO_2$ = mean inspired CO_2 concentration, FIO_2 = mean inspired O_2 concentration. This means that the alveolar CO_2 is inseparably linked to the inspired CO_2. It also implies that when the anesthesiologist is unaware of the magnitude of potential CO_2 rebreathing, any increase in an already high degree of rebreathing will further deteriorate the alveolar gas homeostasis.

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REFERENCES

1. Ramanathan S, Chalon J, Capan L, et al: Rebreathing characteristics of the Bain anesthesia circuit. Anesth Analg 56:822-825, 1977

2. Adriani J: The Chemistry and Physics of Anesthesia. Second edition. Springfield, Illinois, Charles C Thomas, p 168

3. Conway CM: Alveolar gas relationships during the use of semi-closed rebreathing anaesthetic systems. Br J Anaesth 48:865-868, 1976

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STEROIDS AND ANESTHESIA

To the Editor:

The report by Lake[1] concerning the failure of prednisone to restore to normal the action of d-tubocurarine at the myoneural junction in a patient with myasthenia gravis calls to attention the interesting problem of drug and disease interaction. To the discussion of her paper an additional point worth adding is that steroid therapy itself causes a myopathy.[2] The extent of this myopathy and its relation to muscle relaxants given during anesthesia has not been investigated to my knowledge, but it may be there is some potentiation of muscle relaxants. Contrariwise, the actions of pancuronium (which has an aminosteroid structure) may be partially antagonized in patients treated with prednisone.[3] Obviously, the conclusion drawn from these clinical vignettes is that for a variety of reasons patients treated with glucocorticoids may have unusual responses to muscle relaxants given during anesthesia.

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REFERENCES

1. Lake CL: Curare sensitivity in steroid-treated myasthenia gravis: a case report. Anesth Analg 57: 132-134, 1978

2. Afifi AK, Berman RA, Harvey MD: Steroid myopathy clinical, histologic observations. Johns Hopkins Med J 123:158-173, 1968

3. LaFlin MJ: Interaction of pancuronium and corticosteroids. Anesthesiology 47:471-472, 1977

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MORE ON INTERPRETATION of pH DATA

To the Editor:

Allow me to point out a frequently made but nevertheless serious error in the interpretation of data which involve measurements of acidity using pH. This error is repeated in the article by Baraka et al, "Control of Gastric Acidity by Glycopyrrolate Premedication in the Parturient" (Anesth Analg 56:642-645, 1977).

The authors have reported a mean and standard error of pH in the table on page 644, and the editors permitted this computation. pH is defined as the negative logarithm of the hydrogen ion concentration. A quick review of algebra tells us that in a logarithm, the number before the decimal

TABLE*

$$pH = -\log [H+]$$

$$pH = \log \frac{1}{[H+]}$$

<60 minutes		>60 minutes	
pH	[H+]	pH	[H+]
6.6	2.512×10^{-7}	6.0	9.999×10^{-7}
1.8	0.0158	3.0	1.0×10^{-3}
4.4	3.981×10^{-5}	3.4	3.981×10^{-4}
5.3	5.012×10^{-6}	5.8	1.585×10^{-6}
3.7	1.995×10^{-4}	6.7	1.995×10^{-7}
2.1	7.943×10^{-3}	1.8	0.011585
4.2	6.310×10^{-5}	4.6	2.512×10^{-5}
1.7	0.01995	5.2	6.310×10^{-6}
2.0	0.01	2.4	3.981×10^{-3}
1.5	0.03162	$\Sigma x =$	0.0212633144
1.5	0.03162	$x =$	2.3626×10^{-3}
4.2	6.310×10^{-5}	$\frac{1}{x} =$	423.2625
$\Sigma x = 0.1173037732$		$pH = \log \frac{1}{x} = 2.63$	
$x = 9.775 \times 10^{-3}$			
$\frac{1}{x} = 102.2985$			
$pH = \log \frac{1}{x} = 2.01$			

*Adapted from Baraka et al, Anesth Analg 56:644, 1977

is the *characteristic* (tells us where to put the decimal in the real number) and the number behind the decimal is the *mantissa* (gives us the identity of the real number from a table). Logarithms cannot be averaged until they have been converted back to real numbers.

The first value in the authors' table of pH's, 6.6, converts to a real number of 0.0000002512 gram equivalents per litre. The second value, 1.8, converts to 0.0158 gEq/L and so forth. The mean hydrogen ion concentration of the 12 samples in the first column is 0.009775 gEq/L, which corresponds to pH 2.01 and not 3.25 as reported. Furthermore, the pH of the mean hydrogen ion concentration in the second column is 2.60 and not 4.32 as reported.

The issue is of more than academic importance, for if we accept the premise that aspiration of gastric contents of pH 2.5 or less will always result in pulmonary damage, then we see that, using the authors' own data, this critical pH level was not achieved in less than 60 minutes and only barely achieved in more than 60 minutes.

The same mistake has of course been made with blood-gas data; however, with blood gases the *characteristic* is usually 6 or 7 (ie, 1×10^{-6} or 1×10^{-7}), and the magnitude of the error is a factor of 10. By contrast, gastric pH's vary from 1 to 9 and huge errors are possible.

The authors' conclusions and some of the discussion, which is phrased in rather strong language, are weakened by the erroneous interpretation of data. At best, 18% of the authors' patients are still at risk. Rapid acting antacids are available for preinduction use. Milk of magnesia, sodium bicarbonate, and sodium citrate will all mix rapidly with gastric contents and neutralize completely. Aspiration of this neutralized gastric fluid will produce a minimal pneumonitis when compared to the aspiration of acid gastric fluid.[1] The acid aspiration syndrome still

carries a mortality of 30%.[2] Maximum safety could probably be achieved by glycopyrrolate 0.4 mg IM sixty minutes and milk of magnesia 30 ml PO ten to thirty minutes prior to induction of general anesthesia.

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(Editor's Note: see also Statistical analysis of pH data, Anesth Analg 57:143-144, 1978.)

REFERENCES

1. Kuchling A, Joyce T, Cook S: The pulmonary lesion of antacid aspiration. Abstracts of Scientific Papers, ASA Annual Meeting, 1975, pp 281-282

2. Bynum LJ, Pierce AK: Pulmonary aspiration of gastric contents. Am Rev Resp Dis 114:1129-1135, 1976

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HAZARD ASSOCIATED WITH ARMORED ENDOTRACHEAL TUBE

To the Editor:

We would like to call attention to a common, yet manageable complication of armored endotracheal tube use—inability to deflate the cuff.

An 80-year-old white male with a tracheostomy was scheduled for open prostatectomy. After anesthetizing the trachea with 4 cc of 4% lidocaine, a previously tested #38 Rusch armored endotracheal tube lubricated with 2% lidocaine jelly was inserted easily into the tracheostomy, and the cuff then inflated with 6 cc of air to seal the trachea. On inflation of the cuff, a thin-walled bleb was noted extruding just proximal to the pilot balloon, and the cuff tube was clamped proximal to the bleb.

At the end of the procedure the lumen of the endotracheal tube was suctioned, the pilot balloon and bleb deflated completely, and removal of the tube attempted. As this was met with resistance, the pilot balloon was again thoroughly deflated and the tube removed with some difficulty. On removal, the cuff was noted to be fully inflated in spite of a thoroughly deflated pilot balloon. The tube was examined postoperatively, and the cuff tube lumen was found to be sealed shut at the site of clamping.

Numerous reports have appeared regarding inability to deflate cuffed endotracheal tubes, both unreinforced and reinforced. These include separation of the cuff inflation tube from the body of a self-inflatable (Bivona) tube,[1] connector malpositioning such that the cuff tube is interposed between the connector and wire spiral with subsequent compromise of the cuff tube lumen,[2] occlusion of the cuff inflation tube by the inflated cuff,[3] and dissection of the cuff balloon into layers with inability to deflate the innermost balloon.[4]

In order to avoid vocal cord trauma, if extubation meets with resistance in spite of attempted cuff deflation, the cuff tube should be cut proximal to the clamp site prior to tube removal.

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REFERENCES

1. Tavakoli M, Corssen G: An unusual case of difficult extubation. Anesthesiology 45:552-553, 1976

2. Hedden M, Smith RBF, Torpey DJ: A complication of metal spiral-imbedded latex endotracheal tubes. Anesth Analg 51:859-862, 1972

3. Gould AB, Seldon TH: An unusual complication with a cuffed endotracheal tube. Anesth Analg 47:238-240, 1968

4. Davies RM: Faulty construction of a reinforced latex endotracheal tube. Br J Anaesth 35: 128-129, 1963

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CLUNEAL NEUROPATHY

To the Editor:

McKain and Urban (57:138-141, 1978) present an interesting report of cluneal neuropathy from intragluteal injection. Cluneal neuralgias were discussed under the heading of "cluniitis superior" in 1920, with description of traumatic and nontraumatic isolated neuritis of the cluneal nerves.[1] Since these sensory nerves pass over the iliac crest to innervate the skin of the buttocks, the patients complained of pain in the upper, outer part of the gluteal region, and corresponding parts of the iliac crest were tender to pressure. This differs from the pres-