anæsthetist can monitor the pulse, observe respirations and support or elevate the mandible by reaching across the patient’s chest from the front when access to the head is blocked by the seated dentist and his assistant.

DONALD BLATCHLEY
London

REFERENCES

ACID PULMONARY ASPIRATION

Sir,—Dr Gordon Taylor’s report (1975) on “Acid pulmonary aspiration” occurring following the aspiration of gastric contents with a pH of 3.5 is extremely disquieting to those of us who use prophylactic antacids and have, hitherto, considered a pH of 2.5 as critical. Careful scrutiny of the time sequence, however, casts doubt on whether or not aspiration really occurred at the stated pH. The patient’s first dose of antacid was at 2 a.m. and the second dose, 5.5 hr later at 7.30 a.m. Our work shows that an interval of more than 4 hr after ingestion of oral antacid may be followed by acid rebound (Roberts and Shirley, 1974). Hence it is possible that at 7.30 a.m., the pH was lower than it would have been had the first dose been omitted, particularly in view of the large volume involved. Aspiration occurred at 7.50 a.m., 20 min after the second oral dose of antacid. At 8.00 a.m., more senior anaesthetic assistance arrived and the gastric sample was stated to be taken at the time of endotracheal intubation. We must assume that some time passed during which the senior anaesthetist performed endotracheal intubation and brought the situation under control. As such, 8.05 a.m. is probably the earliest time at which the sample could have been taken. Our own work shows that up to 30 min may be required for adequate mixing and buffering of gastric contents by antacids, and if the pH was 3.5 at 35 min after ingestion of antacid, it was not necessarily 3.5 at 20 min after ingestion.

The use of a mixture of aluminium and magnesium hydroxide in 200 cases resulted in the increase of the pH to more than 5 in 67% of elective Caesarean sections, 50% of emergency Caesarean sections and 64% of vaginal deliveries. However, if the antacid was given more than 4 hr before delivery, only 27% and 30% of patients in the latter two groups respectively had a pH higher than 5, while 64% and 44% of patients in the same two groups had a pH of below 2.5.

I believe Dr Taylor’s present contribution in no way diminishes the value of his classical work in introducing the use of prophylactic antacids (Taylor and Pryse-Davies, 1966), but points out again that prophylactic antacids are only one of a number of preventive measures which must be adhered to rigorously in order to prevent the occurrence of the acid pulmonary aspiration syndrome.

R. BRYAN ROBERTS
New York

REFERENCES

Sir,—Thank you for allowing me to respond on this subject. Dr Roberts highlights the time sequence accurately and presents a valid argument. However, there are a number of points which led me to believe that the critical value may be higher than pH 2.5 in man:

(1) The critical value of pH 2.5 in man should be considered as arbitrary and is based solely on animal experiments. There is a species difference of critical pH (rats pH 1.7; rabbits pH 2.1-2.4) (Teabeaut, 1952; Taylor and Pryse-Davies, 1966, 1968). No information is available relating to critical pH in man.

(2) Mixing of magnesium trisilicate and stomach contents with the subsequent elevation of pH occurs very quickly, particularly with patients in labour. This was shown by aspiration of gastric contents of mean volume 150 ml (range 47-348) and pH greater than 4.6 (range 4.6-6.6) from five patients in whom the mixing time was less than 20 min.

(3) In vitro unpublished experiments by the author, mixing antacid and acid, show that equilibration takes place within 5 min and that 70% of the equilibrated pH will be attained within 2 min.

Therefore I believe that the critical pH of 3.5 is reasonably accurate at the time of the aspiration incident in this patient. For those who take the opposite view, I would plead that the details of this case report be retained for use when more evidence becomes available.

GORDON TAYLOR
Stanford, California

REFERENCES

COMPUTING ACID BASE STATE

Sir,—The article by Blackburn, Preston and Strickland (1975), entitled “A simple method for computing acid base state”, is one of the most outrageous and fallacious pieces of work ever published in the anaesthetic literature. To call this “a simple method” and to conclude that their results are sufficiently accurate for clinical purposes shows that the authors must be living in some “plastic tower”.

First, anyone who has read the original article will have to agree that the method is not simple. Using two aliquots
of blood for tonometry and computer solution of everyday acid-base disorders is absolutely unnecessary, time-consuming and expensive as compared with the in vitro titration where the solution can be obtained by mental calculation at the bedside (Rastegar and Thiers, 1972; Levesque, 1975).

I would add that to make one believe that acid-base disorders can be solved using fixed nomograms and fixed equations while discarding patient's history is a step 15 years into the past.

To consider pK as constant and to use the Henderson relation and carbon dioxide solubility coefficient as fixed values can lead to an error of 50%, as shown by Howorth (1974).

Most of all, in vitro titration produces an intrinsic error, Base Excess, which leads often to erroneous conclusions as to the nature of the disturbance. This was shown brilliantly by Schwartz and Relman (1963), Bunker (1965) and Howorth (1974). Artificial concepts like Standard Bicarbonate and Base Excess have no physiological meaning and are the result of a false approach to the solution of a clinical problem.

The British literature has been preeminent in decrying the use of pH to express [H+] and indeed, pH makes calculation more complex and obscures the simple straight-line relationship between [H+] and PCO₂ (Campbell, 1962; Kassirer and Bleich, 1965; Howorth, 1974).

The in vitro titration of the six pure states of acid-base disorders is nearly completed in man (for example: Lemann et al., 1965; DeStrihou, Brasseur and DeConinck, 1966; Arbus et al., 1969; Brackett et al., 1969; Gennari, Goldstein and Schwartz, 1972; Ingram, Miller and Tate, 1973) and to ignore this physiological approach is to demonstrate an addiction to nomograms, equations and artificial complexities.

**Paul R. Levesque**

**Boston, Massachusetts**

**REFERENCES**


Sir.—We are surprised by some of the comments of Dr P. R. Levesque on our paper “A simple method for computing acid base state”, and question their relevance. We do not intend to discuss the well-known differences between the in vivo and in vitro buffer lines which Levesque has expounded enthusiastically, as this topic is not germane to the substance of our paper; although we mentioned briefly criticisms of the in vitro technique, including the contributions of Siggaard-Andersen (1971), Kappagoda, Linden and Snow (1970) and Stoker and colleagues (1972).

Our contribution was, of course, directed at the large number of users of the Siggaard-Andersen interpolation and alignment nomograms, a growing number of whom are considering the use of simple programmable calculators to process their data. Over 19,000 nomograms were sold in Great Britain during the last 6 months. This obviously underestimated the number of determinations as the nomogram can be used repeatedly. We hope that the makers of the “plastic towers” to which Levesque refers have anticipated the demand for this style of accommodation.

A number of other points raised by Levesque appear to require clarification:

(1) Rastegar and Thiers (1972) do not suggest a method for “mental calculation by the bedside”. Both they and Arbus and colleagues (1969) point out the assumptions which must be kept in mind when using “significance bands” to evaluate acid-base disorders. The paper by Levesque (1975) has yet to appear.

(2) Howorth (1974) in his excellent paper did not show that errors of 50% (Levesque does not say in what) arise as a result of taking fixed values for pK and carbon dioxide solubility. Changes in these factors are unlikely to introduce errors in the estimation of acid-base status of more than 10%. Howorth quotes Zimet and colleagues (1970) who analysed cases of severe diabetic acidosis and concluded that the 50% discrepancy between total acid measured and base deficit was related to a number of factors, one of which was the difference between the in vivo and in vitro buffer lines.

(3) We are not aware of any workers who have described Base Excess as an intrinsic error produced by in vitro titration, as maintained by Levesque.

(4) The relative merits of using pH or hydrogen ion activity are well documented and are irrelevant to our paper, since it is a simple matter for both humans and programmable calculators to perform logarithmic transforms of this type. Levesque also maintains that linear relationships are