three infants were delivered vaginally, of which 10 were in poor condition (one-minute Apgar score of five or less and requiring intubation). In all 10 there was usually more than one abnormal feature in the fetal heart record. Specific features were variable decelerations (10/10), baseline tachycardia (7/10), and loss of beat-to-beat variation (7/10). In three cases the cord was found to be prolapsed. A pH estimation was made at the start of the second stage in three cases, the values being 7.31, 7.29, and 7.10 respectively.

In cases of breech delivery where other high-risk factors will not usually apply, compression of the umbilical cord is the most likely cause of asphyxia. There is now considerable evidence demonstrating significant changes in the fetal heart rate when this occurs. Our own data suggest that cord problems are readily detected in breech deliveries by continuous monitoring of the fetal heart and we believe that, because of its continuous nature, this will play a greater role in the management of the breech than fetal blood sampling.—We are, etc.,

TIMOTHY WHEELER
KEITH R. GREENE

King's College Hospital Medical School,
London S.E.5

Assessment of Acid-base Disturbances

SIR,—In general the assessment of acid-base disturbances and the different indices used by various authorities (for example, buffer base, base excess or deficit, standard bicarbonate) have been confusing and difficult for the average clinician to understand. The present need is for a simple, understandable approach, and we were disturbed to find Dr. A. W. Grogono (17 February, p. 381) advocating adding the complexities and apparent authority of a computer, especially when the programme is based on essentially invalid premises. Most of the earlier methods of assessment of acid-base disturbances, including the Siggaard Andersen nomogram adapted by Dr. Grogono, are based on the titration of blood with CO_2 in vitro. It is now generally accepted that the titration of the whole animal with CO_2 produces a titration curve which is significantly different from that obtained in vitro¹ and that an assessment of acid-base disturbances based on the latter can produce a significantly misleading analysis.²

We have proposed a very simple scheme for the assessment of acute acid-base disturbances in man, using only the two readily understood parameters, pH and the PCO_2 .³ The severity of the respiratory acidemia is assessed in the conventional manner as the change in the arterial PCO_2 from the normal range (35–45 mm Hg). The presence of a non-respiratory disturbance is established by plotting the measured values (PCO_2 , pH) on a PCO_2 -pH diagram against a background of CO_2 titration curves in vivo. The displacement of this point from the "normal line" (titration curve in vivo passing through pH 7.40 and PCO_2 40 mm Hg) indicates a non-respiratory disturbance. The pH that would result if the PCO_2 were altered in vivo to 40 mm Hg is then found by following the titration curve through that point and is termed the non-respiratory pH. The deviation of the non-respiratory pH

from 7.40 is used as an index of the severity of the non-respiratory acidemia. A similar approach has now also been adopted by Siggaard Andersen,³ using the term extracellular base excess to describe the shift of the in-vivo titration curve produced by a non-respiratory disturbance. But even he, commenting on his own results, states that the "areas [indicated on his nomogram] should not be interpreted as more than reference areas or indications of the acid-base values to be expected in certain types of acid-base disturbance." There seems little point in continuing the battle when the general has fled the field.

The second important misconception supported by Dr. Grogono is that from a single acid-base analysis one can predict the degree of depletion of bicarbonate stores and therefore the total buffer capacity, and hence calculate the amount of base needed to correct it. Such a conclusion is clearly untenable because all the measurements are related to concentrations, not the total amounts of the extent of the pre-existing bicarbonate stores. The prediction of the degree of depletion must also take into account the time course of the disorder. In addition, our results in dogs suggest that there is a relationship between the rate of infusion of sodium bicarbonate and the amount needed to correct a given acidemia.⁴ We would suggest that the only sensible method of management of an acute non-respiratory acidemia is to infuse sodium bicarbonate, preferably slowly, and to measure the effect by repeated blood gas measurements.

We have now had considerable experience with this approach, both in the clinical field and in the teaching of undergraduates and postgraduates, and are very impressed with the ease by which acid-base disturbances can be assessed and, more important, understood by all concerned.—We are, etc.,

J. B. STOKER
C. T. KAPPAGODA
R. J. LINDEN
H. M. SNOW

Department of Physiology,
The University,
Leeds

- 1 Prys-Roberts, C., Kelman, G. R., and Nunn, J. F., *British Journal of Anaesthesia*, 1966, 38, 500.
- 2 Stoker, J. B., Kappagoda, C. T., Grimshaw, V. A., and Linden, R. J., *Clinical Science*, 1972, 42, 455.
- 3 Siggaard Andersen, O., *Scandinavian Journal of Clinical and Laboratory Investigation*, 1971, 27, 239.
- 4 Kappagoda, C. T., Linden, R. J., and Snow, H. M., *Clinical Science*, 1970, 39, 169.

Poisoning in Children

SIR,—I have recently been analysing the ingestion of poisons in children between the ages of 6 months and 5 years in this hospital in 1970–72. There were 175 such admissions—9.5% of the total of admissions within this age group. Although there were no deaths, this is still a considerable cause of morbidity.

Two points of interest have arisen. The first is that 10 children were admitted after the ingestion of camphorated oil, which appears to be still used extensively, at least in Newcastle, for external use in coughs and colds. This is potentially a serious poison, and 5 ml is said to be fatal, causing cerebral

stimulation and renal damage. There is no reason why it should be kept at all in the home today.

The other point of interest is that of the 65 cases of ingestion of drugs other than aspirin 25 took place in houses other than home, usually in the grandmother's house. Thus even in the unlikely event of every child's home in Newcastle today having locked medicine cabinets, children would still be admitted having ingested dangerous drugs such as digoxin, amitriptyline, and iron, and in fact the only death from ingestion of poison in this hospital in recent years has been that of a child who took several of her grandmother's digoxin tablets. Perhaps child-proof containers will prove the answer to the problem.—I am, etc.,

J. R. SIBERT

General Hospital,
Newcastle upon Tyne

Road Accident Prevention

SIR,—Your interesting leading article, "Road Accidents Epidemic" (17 February, p. 370) seems to make, prima facie, a case for the founding of a national institute of road accident prevention or comparable organization. Such a body, through research, could help to identify predisposing factors both in individuals and in environments, and could bring together various disciplines now working independently.

Those needed for such research would include surgeons, epidemiologists, statisticians, and also psychiatrists to study the behavioural causes of accidents—for example, the presence of severely depressed patients on the road with the risk of urgent suicidal impulses as well as the more obvious conditions of alcoholism and aggression. Those responsible for providing and disseminating health education relating to accident prevention would also be needed, as well as co-ordinators for both medical and non-medical bodies, including representatives from the road vehicle industry and from local safety committees. There would be no lack of co-operation from public health departments, which are in fact conscious of the need for an approach to road accidents as to an epidemic, referred to in your article.

As the matching of numbers of vehicles on the road against space available has presented increasing problems in recent years, so road accidents have been replacing infectious disease as the main killers and disablers of young adults. A pooling of knowledge and endeavour through a joint venture in prevention would do much to alleviate the situation.—I am, etc.,

ELIZABETH VAUGHAN

Public Health Department,
Brentree, Essex

Speech Defects in Children

SIR,—We, two speech therapists and a phonetician, involved both in the training of student speech therapists and in the treatment of children with speech and language disorders, started to read with interest the article by Professor Neville Butler and others (3 February, p. 253). However, we must sadly admit that our disappointment deepened as we read the report, and we feel obliged to