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,
Brintree, 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

comment on certain aspects of the study.

The absence of a speech therapist from the assessment team at once raised doubts in our minds. Although the speech therapy profession recognizes the need to re-evaluate techniques and methodology in many ways, assessment procedures are perhaps its forte, if methods of treatment leave much to be desired. The "clinical speech test" on which the assessment of speech was based involved a rating of the child's ability to repeat six sentences covering "most of the common vowels, single consonants, and clusters of consonants used in everyday English speech." It is, of course, very easy to criticize tests used in such situations, but the sentence test devised as a "clinical test" seems to have many shortcomings and cannot be regarded as "potentially valuable as a means of screening." One must conclude that such factors as vocabulary, frequency of usage, and, more important, sentence length and sentence structure were not taken into account in any systematic way. While one cannot deny the diagnostic value of a sentence repetition test in certain specific cases of language disorder, we feel it can hardly be regarded as a satisfactory general screening test for speech. Furthermore, while allowing that non-expert examiners are hardly likely to notice any "defect" other than "mispronunciations" the emphasis on accurate repetition of speech sounds is surely misplaced in the light of recent widely available findings of linguists and speech pathologists, where stress is laid on the importance of investigating language structure, a point taken up favourably by the recent Government report on speech therapy services.¹

One might also wonder how such a procedure would detect children who are echolalic, who may have repeated either the last word in the sentence or indeed the whole sentence in the complete absence of comprehension. How also can a child repeat six sentences of up to seven words without the involvement of auditory discrimination and memory? Recording of a stammer during this test must also be questioned; some child stammerers would perform normally at such a task, but this performance would not necessarily relate to the child's level of fluency at a communicative level.

The factor of high percentage of mispronunciations in the north-west region is of note and it seems possible that dialectal factors might have been significant here. Although the teachers' evaluation of language use and comprehension helps to restore the balance in the right direction, with reference to the low rating of children from manual social class background, we would refer the authors to the studies of Bernstein² and others^{3,4} whose findings demonstrate not so much "language deprivation" but rather the use of language for different purposes, teachers' "language" being that of school and the 7-year-old child still operating with the "home code."

Other more vital points were eventually taken into account, such as visuo-spatial problems and the obvious fact that "poor speech could itself occur as a result of mental subnormality." Yet in the opening summary the authors declare that "the methods used for screening provided a reliable guide in the selection of children who require further investigation."

Those with experience in the field of

speech and language problems will surely agree that the age of seven is too late for screening and it is the younger child whom we should be concerned about. It is to the authors' credit that they recognize this in their conclusion, but it is regrettable that they then seem to recommend school entry age as the ideal time. This is already too late: if a child comes to school with speech and language problems, he will be doubly handicapped since remediation will require his absence from the classroom and his disability will hamper both his educational and social development. Speech and language disorders and delay can be detected and dealt with very early on in the development of language. Let us make the expertise of the speech therapists and others in the assessment and remediation team available to those children with no speech, instead of waiting until the problem is more defined and more severe. Indeed, the whole study serves as an indictment of the services that are available for all children; why are children not receiving advice and help earlier, if indeed they are even being referred for it? Medical practitioners will be asking themselves this question; speech therapists, teachers, psychologists must ask if they are giving the right advice and management. One must hope that this study will motivate all concerned to evaluate carefully the kind of services they themselves are providing.—We are, etc.,

J. STENGELHOFEN
Head of School of Speech Therapy

C. L. DAVIES
Lecturer in Speech Pathology

P. GRUNWELL
Lecturer in Phonetics and Linguistics

City of Birmingham Polytechnic,
Birmingham

¹ Department of Education and Science, *Speech Therapy Services*. London, H.M.S.O., 1972.

² Bernstein, B., in *Language and Social Context*, ed. P. P. Giglioli, ch. 8. Harmondsworth, Penguin, 1972.

³ Labov, W., in *Language and Social Context*, ed. P. P. Giglioli, ch. 9. Harmondsworth, Penguin, 1972.

⁴ Honeyford, R., *British Journal of Disorders of Communication*, 1972, 7, 206.

Abuse of Diuretics by Jockeys

SIR,—I have recently been approached by two jockeys racing at Cheltenham for what they call "pee pills"; I did not, of course, prescribe them. On further questioning it appeared that there is a substantial black market in diuretics among jockeys, the most popular brand being Lasix (frusemide). As a rule they take these a few hours before the weigh-in, and the usual dose is from two to three tablets (up to 120 mg) though this may be exceeded. They then have a violent diuresis and achieve a loss of weight of up to about 6 lb (2.7 kg). They then take no further fluids until after the race and the further weighing.

The prospect of the side effects that may result from a high dose of frusemide—hypotension, dehydration, and many others—in someone taking part in a very athletic sport, I find alarming. In this particular sport they are not only endangering their own lives, but the lives of fellow jockeys, horses, and possibly the general public.

Is this situation to be allowed to continue, or should there be some sort of inquiry similar

to that which was applied to athletes and amphetamines?—I am, etc.,

DAVID PRICE

Cheltenham

Erysipelothrix Septicaemia

SIR,—We were most interested in the report by Dr. R. H. Townshend and others (24 February, p. 464), of a case of *Erysipelothrix rhusiopathiae* septicaemia, as we recently had a similar case, though with endocarditis. This patient recovered with penicillin but died a few months later from carcinoma of the bronchus. The presence of carcinomatosis in our patient and of chronic alcoholism in four and hypogammaglobulinaemia in one of the previously reported cases raised the possibility that erysipelo-thrix septicaemia is essentially an opportunistic infection, particularly since it is so rare although the organism widespread in nature and the local skin infection, erysiploid, not uncommon.

We wondered if Dr. Townshend's patient had been receiving steroids for her psoriasis and might therefore have had some degree of immunosuppression. We are not sure that he should so dogmatically state that there was no evidence of endocarditis. The patient had multiple pulmonary infarcts and normal lower limb phlebography; these infarcts might well have been due to septic emboli from a focus of endocarditis and, despite the absence of a heart murmur, this surely must have been the most likely source of emboli, since it is such a characteristic lesion in erysipelo-thrix septicaemia.—We are, etc.,

WILLIAM D. ALEXANDER
C. STEWART GOODWIN

Royal Infirmary,
Bristol

Special Risks for Hospital Doctors

SIR,—The recent concern over the lack of proper insurance for doctors at risk in chronic renal dialysis units and in motorway accident teams was discussed at length at a recent meeting of the South-western Regional Committee for Hospital Medical Services. At the request of the committee I would like to express our whole-hearted support for the recent initiative taken by Dr. C. E. Astley, Chairman of the Central Committee for Hospital Medical Services, in making public the concern widely held throughout the hospital service at the intransigence of the Department of Health and Social Security in the matter.¹

We believe that it is grossly unjust, particularly to doctors in the training grades, that their lives should be put at risk in the course of their daily duties without adequate insurance cover being supplied. The cost to the D.H.S.S. would not be great and this would ensure that where death ensued, doctors' families would have adequate pensions.

Under the present arrangements, should death ensue in the course of their work, their future earning capacity is not reflected in the pension their families would get, and recent awards by the courts reflect that future earning capacity should now be considered. We therefore strongly urge Dr. Astley and his committee to continue their efforts on the profession's behalf and wish