heretofore merely as a useful adjunct to a hospital service, operated more for the benefit of the career structures of its members than for the needs of the community.—I am, etc.,

P. W. HUTTON

New Cross Hospital,
Wolverhampton

SIR,—The article by Drs. H. M. Hodkinson and P. M. Jefferys (2 December, p. 536) must give those of us with less active departments furiously to think. Perhaps Dr. Hodkinson can tell us: (*a*) how he clears the pipeline when it gets blocked by patients waiting for non-existent places in local authority residential homes; and (*b*) what is his arm-twisting technique whereby he has extracted from his regional board not only a senior registrar, a senior house officer, and some G.P. clinical assistants but also a registrar. In this part of the world we are told that we just cannot have registrars. Everyone will agree that results as expressed by turnover are directly related to the number of staff available, and the results reported by Drs. Hodkinson and Jefferys are undoubtedly in great part due to the high doctor : patient ratio of approximately one doctor to 25 patients.

The authors do not state the number of their "initial bonus of some empty beds." My experience in trying to introduce an active geriatric service in this area six years ago, was that a similar bonus of (ex-chest hospital) beds reduced the waiting list in a most gratifying way. Unfortunately, in the course of the next four years there was a slow pile-up of undischargeable patients and a reaccumulation in the waiting list. Perhaps Dr. Hodkinson is a little optimistic in so lightly dismissing his "initial bonus of beds"—and one can only hope that his sustained efforts and those of his whole team will enable him to maintain the highly successful turnover which he describes.—I am, etc.,

A. E. K. SALVI

Department of Geriatric Medicine,
Dorset County Hospital,
Dorchester

Pulmonary Function Laboratories

SIR,—I was most interested to read of the experiences of Drs. D. T. D. Hughes and D. W. Empey (25 November, p. 470), having set up a respiratory function laboratory at about the same time as they must have done.

While I nodded in agreement over most of their comments, I was surprised to see that they consider that "asthma, chronic bronchitis, and emphysema can often be differentiated by simple lung function tests." I have come to the conclusion that all one can be sure of, from the lung function test results alone, is the physiological situation indicated. Similar functional situations can be, and often are, present in different clinical states, and while the tests are of great value in differentiating between diseases with different functional patterns, they are rarely of value in the instances quoted. I am also convinced that in interpreting the lung function test results there is a danger of falling into the trap of arguing from the particular to the general. It is too easy, for example, to say that a reduced FEV_1 means that airway obstruction is present. In fact, it merely represents in numerical terms a peculiarity of the spirometric tracing which reflects expiratory airflow rate and which is liable to be affected by many other factors.

The apparatus in my department at Harefield Hospital is limited at the moment to lung volume and ventilation instruments, home-made diffusing capacity apparatus, and two Astrup blood gas machines. The latter are necessary because of the rapid increase in the heart surgery undertaken in the hospital over the past few years, 12-15 open-heart operations per week now being quite usual. Twenty-four hour coverage for blood gas estimations has been effected partly through the good graces of our anaesthetist friends. The use of the Astrup apparatus is limited to the two members of the staff of the department and the anaesthetists, and as Drs. Hughes and Empey said, this has been found to be the only way of assuring consistent and reliable results.

The number of patients referred to the department for spirometry has remained fairly constant over the years at about 400 per annum. This has been partly because of the limited staff and resources. It is my policy only to accept for testing those patients who require fairly full investigation. Increases in the requirements at ward level, such as Drs. Hughes and Empey describe, have been partly absorbed by entrusting the simpler testing to the junior medical staff, using a Vitalograph and a Wright peak flow meter. While envious of the range and expensiveness of the apparatus available in teaching hospitals, I have been made aware of the scope and value of the tests possible even with our relatively simple apparatus—for example, in the preoperative assessment of the lung function of patients with bronchial carcinoma—I am, etc.,

P. LOCKWOOD

Physiology Department,
Harefield Hospital, Middlesex

Active Management of Labour

SIR,—I was very interested to read your leading article (21 October, p. 126) in which you stated that as a result of the active management of labour in African women in Rhodesia, "the duration of labour was shortened so that only 5% of women had a labour lasting more than 12 hours."

Last year, while working in West Africa (Benin City, mid-western Nigeria) I performed a survey to establish the basic obstetrical statistics in the area. Taking a random sample of 5,000 out of a total of 7,080 mothers delivered over a period of 18 months, I found that the average length of labour, without the use of uterine stimulants, in the 4,219 mothers for whom this information was recorded was 10 hours 37 minutes and the prolonged labour rate (that is, more than 24 hours) was 4%. It would seem, as previously noted, that the average length of labour is considerably shorter in West Africa than in the United Kingdom and (in regard to Professor Philpott's results) that it is therefore unwise to apply statistics obtained in one part of the world to another without establishing the norms in each area.

In the same survey I found that the average length of labour for 3,186 multiparous patients was 9 hours 23 minutes and for 1,033 primiparous patients 14 hours 28 minutes. The forceps delivery rate was 0·8% and the caesarean section rate 4·4%. The maternal mortality rate was very high, being 46 per 10,000 deliveries, but the causes were similar to those in the United Kingdom, infection, abortion, eclampsia, and haemorrhage accounting for 60%.—I am, etc.,

LOUIS D. COURTNEY

Lisdarn Hospital,
Co. Cavan, Eire

SIR,—Your leading article on the management of labour is timely (21 October p. 126). While fully endorsing the importance of monitoring cervical dilatation in assessing progress in labour, I consider it also important that greater stress be made on the accurate recording of uterine activity.

Many hospitals do not have tocographic facilities and even if available, a number of patients find abdominal tocography uncomfortable. Sometimes frequent repositioning of the tocograph head is required. The information obtained from careful bedside observation is often highly informative. Accurate clinical observation of uterine activity in labour entails the writing down on paper of the time of onset of each contraction and its duration. Observation over a period of 10-15 minutes usually clarifies the picture at the time in question. It is my not infrequent experience that, when labour is becoming prolonged, previously recorded contraction rates of one in two and a half or three minutes become one in four or five minutes during the time of close observation.

I would therefore make a plea that those entrusted to recording observations in labour—usually pupil midwives or medical students—be instructed in this method of accurate clinical recording.—I am, etc.,

P. E. N. SUTER

Whittington Hospital,
London N.19

SIR,—Your leading article on active management of labour (21 October, p. 126) clearly shows the benefit of treating inertia with oxytocin. I doubt, however, whether many gynaecologists are yet aware that every labouring woman is provided by nature with a ready-made "oxytocic drip mechanism" that can be used for "active management." The milk-ejection reflex operates before the breast secretes milk—both in the early puerperium and throughout labour—and its basic mechanism, release of oxytocin in response to suckling, can be utilized by employing artificial suckling during labour.

I am grateful to Dr. D. R. Firth (21 October, p. 175) for pointing out that the above manoeuvre can halve the induction-delivery interval in cases of induction for postmaturity. In 20 years of experiment I have never seen an excessive oxytocic response to artificial suckling, and I believe that—unlike the orthodox oxytocic drip—it can be safely used outside the specialist unit by practitioners or midwives. It is instantly available, requires no apparatus, and costs nothing.—I am, etc.,

K. D. SALZMANN

Reading

Middle Class Syndrome

SIR,—I too have had experience of this syndrome reported by Dr. D. J. R. Morgan

(11 November, p. 366) but in my case I have been on the receiving end of the patients. When a new syndrome is recognized it is not enough to report its existence; one should also try to establish its cause.

The cases I have seen occurred for two reasons. The majority were due to the original doctor failing to explain adequately (or even at all) the nature and effects of the patient's illness or, more usually, the nature of and reasons for investigations and treatment. A small number were due to inadequate investigations or therapy. The supercilious term "middle class syndrome" glosses over these underlying causes; the cases arise because the patients happen to be intelligent and articulate, and by accident have unorthodox access to a member of the medical profession. It would be more honest (an attribute we often lack when examining ourselves and our shortcomings) to call this syndrome the "dissatisfied patient syndrome."

I agree with Dr. Morgan's implied deprecation of any new investigations and treatment in this sort of situation not being discussed with the family doctor (although it is ironical that the main cause of the syndrome is failure of communication). However, I cannot agree that these cases will be eliminated by doctors maintaining their ethical code in their relations with one another. The syndrome will disappear (except for the very few patients who actually enjoy multiple consultations) by application of our primary ethic—our responsibility to the patient.—I am, etc.,

D. GWYN WILLIAMS

Ickenham, Middlesex

Detecting Sickle Haemoglobin

SIR,—I fully support Professor J. E. Bowman (9 September, p. 644) in advocating definitive electrophoresis and identification of sickle haemoglobin rather than short-cut visual or automated[1] screening methods, a modification (I doubt for the better) of the solubility test.[2] The use of whole blood in solubility testing[1] may lead to false positives in cases of leukemia, high plasma proteins, etc.

Where there is a high incidence of sickle haemoglobin, as in Negroes, Asians, and Mediterranean peoples, other haemoglobin variants—e.g., C, D, E, G, J, thalassaemia, etc.—occur. Both electrophoretic *and* solubility tests are essential for the correct identification of these and haemoglobins SS, AS, SC, S/thalassaemia, S/high F, etc. There are 34 haemoglobin variants with electrophoretic mobility identical or similar to that of sickle haemoglobin; half these have abnormalities (unstable haemoglobins or haemoglobins with abnormal oxygen affinities) in the heterozygous form and are clearly distinguishable from haemoglobin AS by routine haematological tests, including those for heat stability and Heinz bodies. With a bank of six micro-electrophoresis chambers and double spot application[3] 384 samples can be processed per technician-day.[4] Apart from screening out haemoglobins with electrophoretic mobilities different from that of sickle haemoglobin, one is alerted to α- and β-thalassaemia. Undetected benign sickle-cell anaemia[5] is also immediately apparent.

Apart from sickle haemoglobin, haemoglobins C Harlem (C Georgetown identical), Memphis/S, Barts and H (α-thalassaemia), and I have reduced solubilities when deoxygenated. Using automatic pipettors, 480 visual dithionite (and urea-dithionite) tests can be completed daily at a fraction of the cost of automated methods.[1 6] Further, automatic analysers would have quantitative assays as priority in most parts of the world where sickle haemoglobin is prevalent.

Electrophoresis and solubility testing done pari passu thus delineate the major haemoglobinopathies, sickle haemoglobin and double heterozygotes with other variants, and α- and β-thalassaemias. These are mainly associated with similar ethnic groups and geographical localities.[7] In Indians, Iranians, Negroes, etc. haemoglobin D, a non-sickler with electrophoretic mobility identical to that of sickle haemoglobin, requires to be clearly distinguished. With a heat stability test for unstable haemoglobins, these tests would screen all major haemoglobin variants with serious social and clinical effects.

Nalbandian's programme[8] screens all new pupils entering the school system. The social and psychological trauma at this stage is considerable; it would be far better to perform the screening towards the end of the schooling period or on school-leaving, as advocated for β-thalassaemia screening in London Cypriots.[9] The sickle-trait bearer is not at risk except under conditions of stress; only on marriage between partners with the sickle or thalassaemia trait have the offspring a one-in-four chance of suffering from sickle-cell anaemia or thalassaemia major.

With the rising tide of Asian, West Indian, and Mediterranean immigration into the United Kingdom over the past decade a problem of some magnitude in sickle haemoglobin and thalassaemia is developing. Clearly, screening programmes will avert risks by the use of genetic information and alert patients and clinicians to the medical requirements of patients with sickleaemia.—I am, etc.,

RALPH E. BERNSTEIN

Metabolic Research and Human Biochemical Genetics Unit,
South African Institute for Medical Research,
Johannesburg, South Africa

1 Nalbandian, R. M., *et al.*, *Clinical Chemistry*, 1971, **17**, 1033.
2 Itano, H. A., *Archives of Biochemistry and Biophysics*, 1953, **47**, 148.
3 Fingerhut, B., and Ortez, A., *Clinical Chemistry*, 1971, **17**, 34.
4 Bernstein, R. E., *South African Medical Journal*, 1969, **43**, 1455.
5 Sergeant, G. R. Richards, R., Barbor, P. R. H., and Milner, P. F., *British Medical Journal*, 1968, **3**, 86.
6 Nalbandian, R. M., Nichols, B. M., Heustis, A. E., Prothro, W. B., and Ludwig, F. E., *Journal of the American Medical Association*, 1971, **218**, 1680.
7 Livingstone, F. B., Abnormal Hemoglobins in *Human Populations*, Chicago, Aldine Publishing Co., 1967.
8 Nalbandian, R. M., *Journal of the American Medical Association*, 1972, **221**, 500.
9 Modell, C. B., Benson, A., and Wright, C. R. P., *British Medical Journal*, 1972, **3**, 737.

Gentamicin Dosage

SIR,—Dr. J. H. Darrell (18 November, p. 427) is quite correct in drawing attention to the confusion which reigns over the dose schedule for gentamicin. This antibiotic is one of the most powerful available to the clinician, but there is a tendency to be too cautious in its use, resulting in underdosage. The use of larger doses at eight-hourly intervals which Dr. Darrell advocates would certainly produce bactericidal levels even against *Pseudomonas* species. A further problem arises in a patient with normal renal function, in that the serum levels will be below the desirable therapeutic level for a considerable part of the eight hours.

The application of pharmacokinetic principles to the problem may well be useful. The two variables which the clinician can alter are the dose (D) and the dosing interval (T). Accepting the limitations of a one-compartment model these can be calculated using the formula

$$C_\infty = \frac{F.\,D}{V_d.\,K_e.\,T}$$

where C_∞ is the mean serum level after multiple dosing, V_d the distribution volume, F the fraction of the dose absorbed (= 1 in this case), and K_e the elimination constant for gentamicin. This gives dosing schedules of (*a*) 40 mg intramuscularly 2-hourly, (*b*) 80 mg intramuscularly 4-hourly, or (*c*) 120 mg intramuscularly 8-hourly to achieve a mean serum level of 5 μg/ml.

The elimination constant (K_e) is derived[1] from a knowlegde of the half-life[2] ($t_{\frac{1}{2}}$) of gentamicin:

$$K_e = \log_e 2 \times \frac{1}{t_{\frac{1}{2}}}$$

The volume of distribution for gentamicin[2] is variable but a mean value is 15 l.

Obviously the use of smaller doses more frequently will give rise to less fluctuation in the serum level and avoid the possibility of the peak level coming into the toxic range. In renal failure the serum half-life will lengthen, and to achieve the same mean serum level the dosing interval should be lengthened.—We are, etc.,

RICHARD WISE
D. S. REEVES

Southmead Hospital,
Bristol

1 Janku, I., in *Fundamentals of Biochemical Pharmacology*, ed. Z. M. Bacq. Oxford, Pergamon, 1971.
2 Gyselynck, A-M., Forrey, A., and Cutler, R., *Journal of Infectious Diseases*, 1972, **124**, S.70.

SIR,—The letter by Dr. J. H. Darrell (18 November, p. 427) emphasizing the need for adequate dosage with gentamicin is timely. We would, however, extend this observation to include paediatric practice. We have recently carried out serum assays, using the vertical diffusion technique,[1] on two children aged 5 months and 4 years, both of whom received the standard recommended dose of 0·8 mg/kg body weight three times a day by intramuscular injection. Gentamicin levels at one hour were 3·3 μg/ml and 1 μg/ml respectively. The latter level in particular must be regarded as subtherapeutic.

Evidence has been produced by a number of workers[2-4] that larger doses than those routinely recommended by the manufacturers must be given to neonates and young children if therapeutic levels are to be achieved. For instance, a figure of 2·5 mg/kg 8- or 12-hourly in neonates reducing to adult dosage in older children has been suggested. We understand that evidence is to be submitted to the Medicines Commission by the manufacturers in order to obtain permission to use new dosage recommendations in children. In the meantime we would strongly recommend that the use of gentamicin in children and neonates be