The atd angle for the right hand is 102° and for the left hand 88°, the sum of right and left being 190°. This value is very high, coming above the mean of 162° for Down's syndrome,[7] yet not so high as the mean of 216° in Patau's syndrome.[8] Finger prints show an unusually complex pattern of six whorls, three ulnar loops, and one double loop (see Table).

	1	2	3	4	5
Left hand ..	DL	W	W	W	UL
Right hand ..	W	W	UL	W	UL

(W = whorl, UL = ulnar loop, DL = double loop, 1 = thumb)

So far as we are aware, this report is the first to record abnormal dermatoglyphics in a child having the abnormal marker chromosome and typical phenotype of cat-eye syndrome. Previously a child having the marker chromosome was found to have apparently normal dermatoglyphics;[4] while another with a normal karyotype was found to have abnormal hand dermal patterns similar to those in D-trisomy.[9] This contradiction is probably due to the effects of cell selection during embryonic growth of mosaics, resulting in the disappearance of cell lines after they had exerted their normalizing or alternatively their teratogenic effect[10] on embryonic growth.

It is desirable to obtain information on the dermatoglyphics and associated chromosome constitution in a larger number of patients with this syndrome to determine whether the present interpretation is correct. We consider it likely that the chromosome anomaly affects the growth pattern of dermal ridges, except in mosaics where there may not be enough abnormal cells in the hands to affect their growth. Further details of the chromosome studies will be published elsewhere.

We wish to thank Dr. Shelagh Calvert for help with the clinical diagnosis, Dr. C. O. Carter for drawing attention to the abnormal dermatoglyphics, Miss Natalie Anderson for cytogenetical technical assistance, Dr. A. P. Norman, consultant in charge of this case, and Dr. John Quinton, referring consultant, Jenny Lind Hospital, Norwich, Norfolk.—We are, etc.,

CARYL W. DARBY
D. T. HUGHES

Institute of Child Health and the Hospital for Sick Children,
London W.C.1

1 Haab, O., *Albrecht v. Graefes Archiv für Opthalmologie*, 1878, **24**, 257.
2 Schachenmann, G., *et al.*, *Lancet*, 1965, **2**, 290.
3 Schmid, W., *Journal de Génétique Humaine*, 1967, **16**, 89.
4 Weber, F. M., Dooley, R. R., and Sparkes, R. S., *Journal of Pediatrics*, 1970, **76**, 594.
5 Gerald, P. S., Davis, C., Say, B. M., and Wilkins, J. L., *Pediatric Research*, 1968, **2**, 297.
6 Mulvihill, J. J., and Smith, D. W., *Journal of Pediatrics*, 1969, **75**, 579.
7 Penrose, L. S., *Annals of Human Genetics*, 1954, **19**, 10.
8 Penrose, L. S., *Nature*, 1963, **197**, 933.
9 Neu, R. L., Assemany, S. R., and Gardner, L. I., *Lancet*, 1970, **1**, 949.
10 Neu, R. L., Bargman, G. J., and Gardner, L. I., *Pediatrics*, 1969, **43**, 624.

Ampicillin and Mononucleosis

SIR,—It is now well known that the great majority of patients suffering from infectious mononucleosis who are given ampicillin develop a sensitivity rash. Dr. T. Pastor (24 April, p. 222) asks the very pertinent question, is this allergy permanent? The answer would seem to be that it is not. We have been investigating this for some time by giving graduated doses of ampicillin to patients some months after their ampicillin reaction during their attack of infectious mononucleosis. No rash or other evidence of hypersensitivity has developed in the first ten patients who have received a full therapeutic dose. These preliminary results would suggest that such patients can safely be given ampicillin six months after a reaction to this drug during an attack of infectious mononucleosis.

The investigation is continuing and will be reported in detail in due course. It may well be that the altered hypersensitivity which occurs in mononucleosis may disappear much more rapidly than six months. —I am, etc.,

I. J. NAZARETH

St. Ann's General Hospital,
London N.15

Pulmonary Gas Exchange during Dialysis

SIR,—We were interested in the article "Pulmonary Gas Exchange during Peritoneal Dialysis" by Dr. M. J. Goggin and Dr. A. M. Joekes (1 May, p. 247).

We should like to suggest an explanation for the changes in arterial oxygen tension (PaO_2) which would account for the fact that these changes were related to the volume of peritoneal dialysate and also for their rapid reversibility. It has recently been suggested in elderly patients that airways in the dependent parts of the lung may close at a volume ("closing volume") greater than residual volume. "Closing volume" increases with age so that above the age of 44 in supine subjects and 65 in seated ones "closing volume" may exceed functional residual capacity (F.R.C.) and airway closure occurs during ordinary tidal breathing.[1] It has also been shown that on going from the erect to the supine posture "closing volume" is unaltered but F.R.C. falls, and that if it falls below "closing volume" PaO_2 will fall.[2]

We presume that the patients described by Dr. Goggin and Dr. Joekes were recumbent or semi-recumbent during dialysis, and we suggest that the addition of 1-2 litres of fluid to the abdominal contents produced a corresponding fall in F.R.C., which dropped below "closing volume."

In the majority of patients with peritoneal dialysis who are being treated, fluid retention is present at the start of the dialysis. As the "closing volume" can be increased by interstitial oedema in the lungs, this would have accentuated the effects of any fall in F.R.C. Incidentally, the blood gas data in the fourth line of Table IV are impossible, as arterial PO_2 cannot exceed alveolar PO_2.—We are, etc.,

S. FREEDMAN
D. J. MABERLY

Department of Medicine,
Royal Posteraduate Medical School,
London W.12

1 Leblanc, P., Ruff, F., and Milic-Emili, J., *Journal of Applied Physiology*, **28**, 448.
2 Craig, D. B., Wahba, W. M., Don, H. F., Couture, J. G., and Becklake, M. R., *Clinical Research*, 1970, **18**, 742.

**We have shown this letter to Drs. Goggin and Joekes who reply: "We find the comments of Dr. S. Freedman and Dr. D. J. Maberley of considerable interest. The patients, who were recumbent or semi-recumbent during dialysis, had no evidence of pulmonary disease demonstrable clinically or radiologically. None had anything but minor fluid overload and the majority had been under careful metabolic management for enough time to have avoided this.

"As they rightly say, the figures of the derived data in the fourth line of Table IV are impossible and should read 142→137." —ED., *B.M.J.*

Dental Discolouration and Tetracycline

SIR,—The continued widespread practice of prescribing the conventional tetracycline group of drugs to children, despite repeated warnings of its tendency to produce disfiguring discolouration of both primary and secondary dentition, has now become a serious abuse in an age in which there are a host of effective antibiotics for the treatment of specific infections.

I have recently had the misfortune to see a perfectly well child aged 10 years with gross discolouration of her secondary dentition as a result of repeated doses of tetracycline for trivial respiratory infections. There seems no justification for the continued administration of tetracycline under the age of 6 years. — I am, etc.,

L. S. TAITZ

Department of Child Health,
University of Sheffield,
Yorks

Cure of Lung Cancer after Incomplete Surgical Resection

SIR,—In his description of three patients with lung cancer apparently cured following incomplete surgical resection Mr. R. Abbey Smith (5 June, p. 563) suggests that this phenomenon occurs only where the residual cancer remains on the wall of the left atrial chamber of the heart, but not when residual cancer remains on some other intrathoracic organ. That cure is also possible in the latter circumstance is illustrated by the following case.

The patient, then aged 50, was first seen on 13 November 1957. He worked as a chimney sweep for one year, previously as a gardener. He had not been exposed to any known industrial hazard, but had regularly smoked 20 cigarettes daily. There was no family history of cancer in primary relatives. There was no previous history of allergy or abnormal endocrine activity. His blood group was AB, Rh. positive. His complaint was of troublesome cough for two months, and chest pains, undue dyspnoea, and slight haemoptysis for two weeks. Chest radiograph showed a mass at the left hilum, with partial collapse of the left upper lobe. Bronchoscopy on 21 November showed the carina normal, but an area of abnormal mucosa at the left upper lobe orifice, which was distorted and bled easily. The bronchial biopsy showed an undifferentiated oat cell carcinoma. He was considered to be operable by left pneumonectomy.

At operation by Mr. Richard Rowlandson on 27 November it was only when the left main bronchus was clamped that it was found to have gone through growth in the bronchial wall. It was then too late to retreat, and the operation was proceeded with, though it was realized that growth was being cut through and that complete removal of the cancer would not be possible. The pneumonectomy specimen showed a growth 3 cm x 2 cm very close to the left main bronchus obstructing the bronchus of the left upper lobe and also invading the bronchus of the left lower lobe. Several large

hilar lymph nodes were involved. The histology of the main tumour showed oat cell carcinoma, while the removed subcarinal lymph node contained deposits of poorly differentiated squamous cell carcinoma. The patient's progress was uneventful, though he was left hoarse following damage to the left recurrent laryngeal nerve. He did not receive radiotherapy or cytotoxic drugs. He has been carefully followed up for 14 years, and despite the bad prognosis initially given, he has remained quite fit, continuing at work, and clinically and radiologically he has shown no signs of recurrence of the lung cancer.

Mr. Abbey Smith lays down the criteria for acceptance of the existence of this phenomenon of cure following incomplete surgical resection of cancer as biopsy confirmation, record of incomplete surgery, and necropsy confirmation of absence of cancer throughout the body. In the case described above, the first two criteria are fulfilled, but the third cannot be met since the patient remains alive after 14 years. It looks therefore as though the policy of Mr. Abbey Smith's unit of removal of the bulk of the tumour where complete removal is not possible, may at times—if but rarely—produce an unexpected successful result.—I am, etc.,

ALEX SAKULA

Redhill General Hospital,
Surrey

Acid-base Balance and Bleeding

SIR,—The criticisms made by Mr. M. H. Irving (29 May, p. 529) of our paper on acid-base balance in acute gastrointestinal bleeding (1 May, p. 242) prompt us to clarify certain points raised in his letter.

We reported that transfusion with stored bank blood, which is known to have a high lactate concentration[1] and a low pH,[2] resulted in a rise in arterial blood lactate concentration in seven patients who were not clinically shocked either before or after blood transfusion. Mr. Irving suggests that this was because the patients remained in a "state of compensated shock", and points out that "a normal arterial blood pressure is no guarantee that hypovolaemia has been corrected". These patients were all transfused to a normal central venous pressure[3] prior to the second blood sample, with the exception of one patient who still had a low central venous pressure at this time. Blood volume measurements were carried out in five of these patients, including the patient with the low central venous pressure, using ^{51}Cr labelled red cells. All five patients had a blood volume well within the normal range,[4] according to weight and sex, at the time of the second blood sample.

We also reported that patients who are clinically shocked may have a severe metabolic acidosis on admission. In one such patient we measured the blood pH again after transfusion of one litre of blood had reversed the state of clinical shock and raised the blood pressure to normal. There had been only a very slight increase in pH from 7·25 to 7·26, but a further increase to 7·43 occurred following infusion of 200 mEq sodium bicarbonate. After this further blood transfusion was continued. Mr. Irving suggests that "the actual volume of the 200 ml of sodium bicarbonate solution was possibly as significant in correcting the acidosis as the buffer itself." It seems unlikely that such a small volume would produce such a marked effect when no significant effect had been obtained with one litre of blood. A further point against this explanation is that the blood lactate concentration remained very high at 128 mg/100 ml following the infusion of bicarbonate, as mentioned in our paper.

Mr. Irving complains that the authors "do not make it clear at what point they believe post-transfusion metabolic acidosis should be corrected. The implication, however, from the interval between their pre- and post-transfusion acid/base measurements is that it may be several hours after the cessation of blood transfusion." The measurements he is referring to here are those obtained in the patients who were not clinically shocked before or after blood transfusion, and we specifically point out that monitoring of acid-base balance "is probably unnecessary in patients who are not clinically shocked." We suggest, however, that it "is advisable in patients with acute gastrointestinal bleeding who are clinically shocked, especially if rapid blood transfusion is contemplated." We intended this to imply that these measurements should be carried out before or during the early stages of blood transfusion, while the patients are still clinically shocked. If a severe acidosis is detected, we would suggest correcting it immediately. We certainly did not imply that it should be corrected "several hours after the cessation of blood transfusion." Thus, in saying that his "main criticism of the paper is the suggestion that a *persistent* metabolic acidosis in shocked patients should be treated by the infusion of sodium bicarbonate" (our italics), Mr. Irving is misinterpreting our suggestion. We accept that eventually "the metabolic acidosis of hypovolaemic shock is self-correcting if adequate volume replacement is provided." Our point is that, in the acute situation of a patient in clinical shock with a severe lactic acidosis, the rapid transfusion of stored bank blood, with a high lactic acid concentration and a low pH, may be dangerous unless the patient's blood pH is monitored.

In support of his opposition to the use of sodium bicarbonate, Mr. Irving quotes Schweizer and Howland's[5] (1962) paper saying that the metabolic acidosis of shocked patients undergoing major surgery responds promptly to treatment of the hypovolaemia with acid bank blood. This paper has been superseded by their more recent publication on this subject,[6] which is the one quoted in our paper. In this, they report an improved mortality rate with simultaneous infusion of alkali, and a similar improvement has been reported under more carefully controlled conditions in experimental animals with haemorragic shock.[2]

Mr. Irving further points out that sodium bicarbonate is "ineffective in treating the shock state." We did not at any stage suggest that it will correct the shock state, only the accompanying acidosis. We share his belief that correction of a low blood pH should not be allowed to divert attention from the need to correct hypovolaemia by blood transfusion.—We are, etc.,

T. C. NORTHFIELD

Guy's Hospital,
London S.E.1

B. J. KIRBY

Royal Infirmary,
Edinburgh

ANNE E. TATTERSFIELD

Hammersmith Hospital,
London W.12

1 Gullbring, B., and Ström, G., *Acta Medica Scandinavica*, 1956, **155**, 413.
2 Nahas, G. G., Manger, W. M., Mittelman, A., and Ultmann, J. E., *Annals of the New York Academy of Sciences*, 1961, **92**, 596.
3 Northfield. T. C., and Smith, T., *Lancet*, 1970, **2**. 584.
4 Birkeland, S. J., *Journal of the Oslo City Hospitals*, 1965, **15**, 43.
5 Schweizer, O., and Howland, W. S., *Surgery, Gynecology and Obstetrics*, 1962, **114**, 90.
6 Howland, W. S., and Schweizer, O., in *Clinical Anaesthesia*, ed. L. R. Orkin, p. 26. Blackwell Scientific, Oxford, 1965.

Myocardial Infarction and the G.P.

SIR,—Dr. L. Adamson (27 February, p. 505) warns against the use of prophylactic procainamide in early myocardial infarction in the absence of E.C.G. control.

In a recent report[1] on experience in a coronary care unit an E.C.G. record was presented which illustrates one of the hazards to which he alludes (Fig.). It demonstrates sinus bradycardia, a run of ventricular premature beats, reversion to sinus bradycardia, and then alteration to nodal bradycardia—all occurring in a period of less than one minute in a patient with fresh infarction. The rapid transition from tachy- to brady-arrhythmia is not uncommon in the early stages of infarction and is impossible to detect clinically.

In this case atropine could have precipitated ventricular fibrillation or tachycardia, and procainamide block-syndrome or asy-