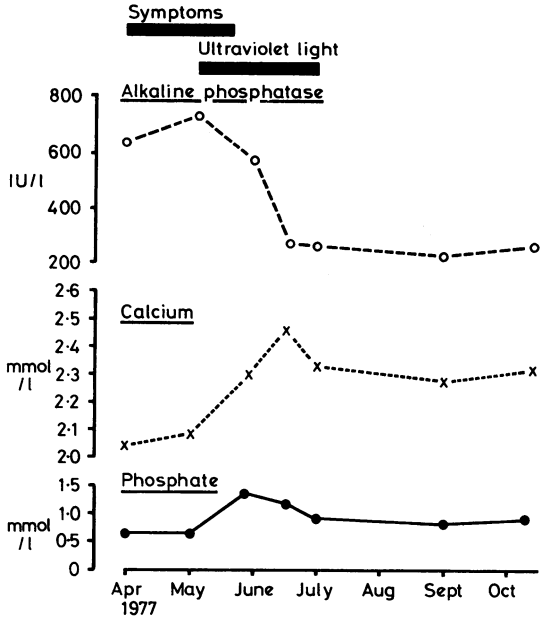
Before vitamin D was discovered nutritional rickets was treated by exposure to sunlight.[3] We report the successful treatment of osteomalacia by ultraviolet light in a patient with malabsorption and draw attention to the value of this long-established, safe, and unduly neglected form of treatment.

Case report

A 42-year-old mentally defective man presented in February 1976 with a one-year history of diarrhoea and loss of weight. He was found to have steatorrhoea and a jejunal biopsy specimen showed partial villous atrophy. Although the steatorrhoea improved on a gluten-free diet (fat excretion falling from 83 mmol/24 h to 27 mmol/24 h; normal < 18 mmol/24 h), complete recovery was never achieved, partly (at least) because of dietary indiscretions. In May 1977 the patient complained of soreness over the ribs and both tibiae for two months. The serum concentration of calcium was 2·08 mmol/l (8·32 mg/100 ml), phosphate 0·67 mmol/l (2·07 mg/100 ml), and alkaline phosphatase 720 IU/l (normal < 92 IU/l). A bone biopsy specimen showed wide osteoid seams and a defective calcification front and the plasma 25-OHD concentration was low at 3·9 ng/ml.

Whole-body treatment with ultraviolet light was begun in May 1977 using four Westinghouse lamps FS40. The patient was treated three times per week, with increasing exposure times. Within one week of starting treatment (total exposure 20 min) his bone pains had been relieved and after two weeks' treatment (50 min) the plasma 25-OHD concentration had risen to 18·3 μg/l. Serum concentrations of calcium had risen to 2·31 mmol/l (9·24 mg/100 ml), phosphate to 1·33 mmol/l (4·12 mg/100 ml), while alkaline phosphatase had fallen to 590 IU/l (figure).



Effects of treatment with ultraviolet light.
Normal concentrations: calcium 2-2·6 mmol/l (8-10 mg/100 ml), phosphate 0·8-1·4 mmol/l (2·5-4·3 mg/100 ml; and alkaline phosphatase 30-92 IU/l.

Conversion: SI to traditional units—Serum calcium: 1 mmol/l ≈ 2 mg/100 ml. Serum phosphate: 1 mmol/l ≈ 3·1 mg/100 ml.

After four weeks' treatment (113 min) the serum calcium concentration was 2·45 mmol/l (9·80 mg/100 ml) and alkaline phosphatase 288 IU/l. Treatment was discontinued after seven weeks (212 min). Eighteen weeks after beginning treatment the plasma 25-OHD concentration was 21·5 μg/l and a bone biopsy specimen showed healing of the osteomalacia.

Comment

Our data show that a short exposure to ultraviolet light (20 minutes) results in a rapid rise in the plasma 25-OHD concentration and quick resolution of symptoms. A similar resolution of symptoms and rapid rise in the plasma 25-OHD concentration has been found in a patient with postgastrectomy osteomalacia after only five minutes' (50 joules) exposure to ultraviolet light.[4] This suggests that a shorter course of ultraviolet light might have been equally as effective in our patient. An advantage of ultraviolet light treatment is that hypercalcaemia, which may occur with the more polar metabolites of vitamin D, is not a problem. To prevent further relapses patients should be instructed to go out in the sunlight, but a yearly course of treatment (in winter) may be needed if malabsorption persists.

We thank Miss Lane for giving the ultraviolet light and Dr D E M Lawson for estimating plasma 25-OHD.

Correspondence to: Dr R T Jung, Addenbrooke's Hospital, Trumpington Street, Cambridge.

[1] Stamp, T C B, *Lancet*, 1974, **2**, 121.
[2] Compston, J E, Morton, L W L, and Tighe, J R, *British Medical Journal*, 1977, **2**, 612.
[3] Huldschinsky, K, *Deutsche medizinische Wochenschrift*, 1919, **45**, 712.
[4] Pittet, P G, Davie, M, and Lawson, D E M, *Nutrition and Metabolism*, in press.

(*Accepted 2 March 1978*)

Addenbrooke's Hospital, Cambridge
R T JUNG, MB, MRCP, honorary senior registrar and MRC clinical scientific staff, Dunn Nutrition Unit
M DAVIE, MB, MRCP, honorary senior registrar
J O HUNTER, MD, MRCP, consultant physician
T M CHALMERS, MD, FRCP, consultant physician

Relative chloroquine resistance of P falciparum in Zambia

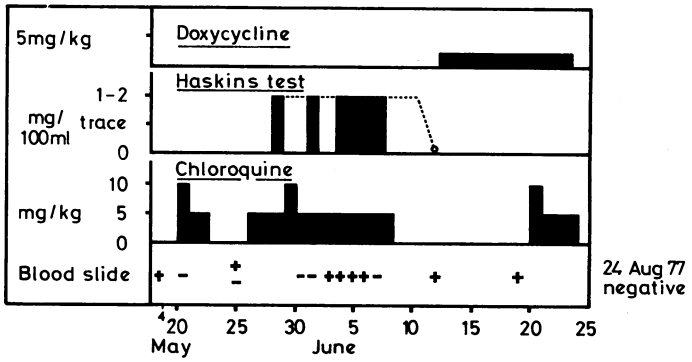
We have suspected that some strains of *Plasmodium falciparum* in Zambia show resistance to chloroquine, but because the patients are ill we have been unable to prove this according to the WHO criteria.[1] We report a case that apparently fulfils these criteria.

Case report

A 2-year-old child was admitted on 4 May 1977 for treatment of a lower respiratory tract infection. The child came from Lusaka and had not been outside that area. A diagnosis of tuberculosis was entertained but subsequently discarded in favour of that of bronchiectasis. While in the ward she had several episodes of fever, some with convulsions, and malaria parasites were seen in the blood film. On 20 June she was given a course of chloroquine but the blood film remained positive for parasites so testing for resistance was instituted. Between 25 June and 7 July she received 1·3 g of chloroquine (130 mg/kg)—that is, an oral dose of between 5 and 10 mg/kg/day (figure). During the course the Haskins's test in the urine was repeatedly positive, proving that she was absorbing the drug. Despite this parasites were still present in the blood film on 12 July. Subsequently the infestation cleared with a 10-day course of doxycycline, 5 mg/kg/day—that is, 50 mg daily by mouth—to which chloroquine was added. On 24 August parasites were no longer present in the film.

Comment

The dosage of chloroquine between 28 June and 7 July is more than adequate by the WHO criteria and the degree of resistance would



Summary of treatment and findings.

appear to be of the RII category. Though we have seen several other patients with apparent chloroquine resistance, either they have been too ill to delay alternative treatment or they have had several courses of chloroquine before testing for resistance and have finally responded to the test course. It is now our practice to use chloroquine by mouth in a dose of 10 mg/kg, repeated after six hours, then daily for three days. This is the general dosage by WHO recommended standards.[2]

We believe that chloroquine resistance is appearing in Zambia and that clinicians must watch for this. In such cases we have had success with a combination of doxycycline and quinine or chloroquine.

[1] World Health Organisation, *Resistance of Malaria Parasites to Drugs*. WHO Technical Report No 296, 1965.
[2] World Health Organisation, *Chemotherapy of Malaria*. WHO Technical Series No 375, 1967.

(*Accepted 2 March 1978*)

Department of Paediatrics and Child Health, University Teaching Hospital, PO Box R W 110, Lusaka, Zambia
A A KHAN, MB, FRCP, professor
M J MAGUIRE, MB, MRCP, honorary lecturer

Predicting plasma concentrations of common biochemical values from residual peritoneal fluid in patients on peritoneal dialysis

Peritoneal dialysis is now widely used in the management of renal failure. Since frequent blood sampling is needed to assess the adequacy of dialysis, we decided to investigate whether equilibrated peritoneal fluid could be used instead of blood samples to monitor biochemical values in patients being treated with peritoneal dialysis.

Patients, methods, and results

Fifteen patients being treated with maintenance peritoneal dialysis were studied. Dialysis was for an average of 40 hours a week by an indwelling soft catheter. Each morning before starting dialysis, blood and peritoneal fluid samples were taken simultaneously, the latter directly from the peritoneal cavity after discarding the first 2 ml from the catheter. Both samples were analysed for concentrations of urea, creatinine, sodium, potassium, calcium, phosphate, total protein, and albumin using standard techniques.

The table shows the highly significant correlations between plasma and peritoneal fluid concentrations of creatinine, urea, sodium, potassium, calcium, and phosphate. Plasma calcium concentrations can be closely approximated by adding 0·5 mmol/l (2 mg/100 ml) to peritoneal fluid calcium concentrations. Nevertheless, the margin of error for plasma calcium lay between ±7·8%. Proteins also tended to equilibrate across the peritoneal membrane. There was no appreciable difference between plasma and peritoneal fluid albumin to globulin ratios.

Correlation between peritoneal fluid and plasma concentrations of some common biochemical parameters

	No of tests	Plasma (mean ± SD)	Peritoneal fluid (mean ± SD)	Significance of difference (P)*	Correlation coefficient (r)†
Urea (mmol/l)	35	31·26 ± 8·48	31·43 ± 8·50	NS	0·99
Creatinine (μmol/l)	35	1335 ± 666	1338 ± 691	NS	0·99
Na^+ (mmol/l)	35	136·62 ± 3·31	136·34 ± 4·91	NS	0·81
K^+ (mmol/l)	35	4·48 ± 0·89	4·46 ± 0·97	NS	0·96
Calcium (mmol/l)	29	2·23 ± 0·25	1·73 ± 0·25	<0·001	0·93
Calcium (mmol/l)	29	2·23 ± 0·25	2·23 ± 0·25	NS	0·93
Phosphate (mmol/l)	29	1·84 ± 0·87	1·85 ± 0·91‡	NS	0·99
Protein (g/l)	19	59·97 ± 7·39	25·15 ± 7·85	<0·001	0·83
Albumin (g/l)	19	26·76 ± 4·00	11·81 ± 4·59	<0·001	0·75
A:G ratio	19	0·809 ± 0·10	0·899 ± 0·30	NS	

*Calculated by means of paired *t* test.
†Between plasma and peritoneal fluid concentrations.
‡Calculated mean based on regression equation $y = x + 0·5$, where *y* represents plasma and *x* peritoneal fluid calcium concentrations.
NS = Not significant.
Conversion: SI to traditional units—Urea: 1 mmol/l ≈ 6·02 mg/100 ml. Creatinine: 1 μmol/l ≈ 0·011 mg/100 ml. Na, K: 1 mmol/l = 1 mEq/l. Calcium: 1 mmol/l ≈ 4 mg/100 ml. Phosphate: 1 mmol/l ≈ 3·1 mg/100 ml.

Comment

Several points clearly emerged. Peritoneal fluid concentrations of urea, creatinine, sodium, potassium, and phosphate approximated to their plasma concentrations after at least an overnight equilibration. For practical purposes the two concentrations may be regarded as identical. While complete equilibration for calcium cannot be achieved after overnight equilibration, calcium concentrations in plasma and peritoneal fluid correlated closely enough to validate estimations of plasma calcium from peritoneal fluid. In fact complete equilibration of ionised calcium between plasma and peritoneal fluid is probably achieved after overnight equilibration, as peritoneal clearance of ionised calcium is similar to that of urea.[1]

These findings show that routine blood sampling can be abandoned in patients on peritoneal dialysis in favour of peritoneal fluid analysis, and furthermore, sampling can easily be done by the patients with instruction. Sampling peritoneal fluid rather than blood eliminates an obvious cause of blood loss and prevents damage to sites of blood access. Samples may be mailed without fear of distortion of potassium and phosphate concentrations by leakage from damaged red cells.

An intriguing aspect of this study is the fact that the peritoneal membrane appears to be highly and non-selectively permeable to large protein molecules. The finding of similar albumin to globulin ratios in plasma and peritoneal fluid agrees with those of other studies on immunoglobulin loss in peritoneal dialysis.[2] Protein loss is a familiar hazard of peritoneal dialysis, and our results suggest that it is caused by equilibration across the peritoneum rather than by some ill-understood irritation of the membrane. The good correlations between plasma and peritoneal fluid concentrations of total protein and albumin after overnight equilibration help to explain the increased protein loss in the first few cycles of each dialysis.[3] It is impracticable to try to derive an equation to predict plasma protein concentrations from those in peritoneal fluid, but will be of interest to measure hormones such as thyroxine in the peritoneal fluid after overnight equilibration to see if losses by such a route contribute to the increased incidence of subclinical hypothyroidism in patients treated with peritoneal dialysis.[4]

Requests for reprints to Dr M K Chan.

[1] Stoltz, M L, Nolph, K D, and Maher, J F, *Journal of Laboratory and Clinical Medicine*, 1971, **78**, 389.
[2] McKelvey, E M, *et al*, *Archives of Internal Medicine*, 1974, **134**, 266.
[3] Bianchi, R, *et al*, *European Journal of Clinical Investigations*, 1975, **5**, 409.
[4] Boss, A M B, *et al*, *European Dialysis and Transplant Association, XIII Congress*, abstract, p 299.

(*Accepted 23 February 1978*)

Department of Nephrology and Transplantation, Royal Free Hospital, London NW3 2QG
M K CHAN, MB, MRCP, registrar
Y C HUANG, MB, BS, senior house officer
Z VARGHESE, MSC, chief biochemist
R A BAILLOD, MB, BS, first assistant
J F MOORHEAD, MB, FRCP, director

Correction

Creamatocrit

In the article by Dr A Lucas and others (22 April, p 1018) the formula for the relation between creamatocrit reading and energy content of human milk should have read: kcal = 290 + (66·8 × creamatocrit (%)).

Serum lipid concentrations in obesity

In the paper by Dr R G Wilcox (10 June, p 1513) the key in the legend to fig 2 was printed incorrectly. It should have read: × — — — × = Weight. ● — — — ● = Cholesterol. ○ ——— ○ = Triglyceride.