logically and simply, by the entirely different diet in the two groups of negroes. In the first group the carbohydrates in the diet are unrefined, consisting of the coarsest maize, with very little sugar; whereas in the second group the carbohydrates are now those of the white man, consisting mainly of white flour and sugar. The first diet results in two soft stools a day,³ whereas the second diet, through the massive removal of fibre, results in the marked slowing of the passage of the colonic contents so characteristic of the Westernized nations. This colonic stasis incidentally affords a natural explanation of the muscular hypertrophy in the colonic wall to which you refer.

It should be added to the above that the diet in the United States negro once also consisted of unrefined maize (hominy), but the consumption of maize in the United States shows the following decline over the years⁴:

Year	Maize Products Available to Retail Markets (lb. per year per head)		
1889	..	..	117.0
1899	..	..	103.3
1909	..	..	53.0
1924	..	..	32.4
1932	..	..	26.5
1942	..	..	19.8
1950	..	..	11.8
1961	..	..	7.2

There is of course nothing specific about maize: it is just an example of unrefined grain. Meanwhile the consumption of sugar in the U.S.A., as in all the Westernized nations, has shown a very great rise in consumption over this period.

As long as the approach to diverticular disease smacks of the assumption that the body is built wrongly, instead of being used wrongly, so long does the disease appear likely to continue to be the menace that it has become. A very considerable restoration of the carbohydrates to their natural unrefined state alone points to any great success in prevention and arrest.—We are, etc.,

T. L. CLEAVE.

Fareham, Hants.

G. D. CAMPBELL.

King Edward VIII Hospital,
Natal.

REFERENCES

- Cleave, T. L., and Campbell, G. D., *Diabetes, Coronary Thrombosis, and the Saccharine Disease*, 1966. Bristol.
- Kocour, E. J., *Amer. J. Surg.*, 1937, 57, 433.
- Dodd, H., *Lancet*, 1964, 2, 809.
- Antar, M. A., Ohlson, M. A., and Hodges, R. E., *Amer. J. clin. Nutr.*, 1964, 14, 169.

Labelling Drugs

SIR,—I have previously commented¹ on the problem of drug nomenclature in clinical practice, complicated as it is by the tedious nuisance and even danger of there being more than one name for each drug. On the one hand we have the fairly simple two- or three-syllable, not very meaningful, proprietary names, and on the other hand there are the more complex, often polysyllabic approved names, which are not always much more informative.

But, Sir, both the proprietary and approved names do at least specify a particular drug. Unfortunately the same cannot be said for the third category—namely, "The Tablets." Though pharmacologists

and some clinicians may not always agree on the relative merits of "proprietary" versus "approved" names, yet surely they should combine in damning this anachronism which allows potent medicaments to be dispensed in what amounts to virtually unlabelled containers, with resulting confusion, danger, and waste. When will it become required pharmaceutical practice to label containers meaningfully unless the prescribing doctor specifically directs otherwise? Rat poison bought as such must be properly labelled, but not if dispensed into a bottle to be kept in the family medicine chest or on the mantelpiece.—I am, etc.,

C. D. NEEDHAM.

Aberdeen.

REFERENCE

- Needham, C. D., *Brit. med. J.*, 1963, 1, 330.

Osteomalacia in Asian Immigrants during Pregnancy

SIR,—Drs. D. J. C. Felton and W. D. Stones's article (18 June, p. 1521) prompts me to record a different manifestation of calcium deficiency accompanying pregnancy in a woman of similar racial and cultural background.

The patient, a multiparous Pakistani woman, was delivered in St. Mary's (Manchester) district about June of last year. She had had two or three full-term normal deliveries, and was therefore booked under the care of the district midwives. She attended the district clinic regularly, and was seen by either the midwives or one of the resident medical staff.

Pregnancy progressed normally and no complaints were recorded. She went into labour at term and had a normal delivery of a healthy child. As lactation was becoming established on the third and fourth postpartum days she began to complain of tingling, and then cramp-like pains in the hands and feet. A houseman was taken to see her on the fifth day. The pains were much worse, and there was some spasm in the hands and feet, the thumb being held opposed across the palm of the hand. The houseman, to his credit, diagnosed latent tetany due to hypocalcaemia, precipitated by lactation. Chvostek's sign and Trousseau's sign were positive. The serum calcium was below normal levels. She was treated with 10 ml. calcium gluconate intravenously, followed by oral calcium and vitamin D. Lactation was suppressed. The following day she was free from symptoms and remained so.

On inquiring about the diet it was found that she took no milk, little protein, and had not taken her vitamin tablets. For the latter part of the pregnancy she had lived mainly on chappatees, prepared from non-fortified flour and ghee obtained at the local Indian store.

This patient had developed some calcium deficiency in the latter part of pregnancy. It had not persisted long enough to produce osteomalacic symptoms. The sudden demand for calcium, associated with the onset of lactation, had produced the symptoms and signs of latent tetany.—I am, etc.,

Hope Hospital,
Salford 6, Lancs.

W. O. GOLDTHORP.

Rickets in British Children

SIR,—The leading article on rickets (5 March, p. 558) is a good pointer to a need for the clinical reappraisal of nutritional standards in Britain, but it does not indicate the extent of evidence of this disease and of malnutrition generally amongst younger age groups in Britain.

For the past four years I have been examining Army recruits aged 17 to 18 years, with a particular interest in assessing their standard of physique and nutrition, and have been surprised to find much evidence of old rickets, poor physical development, low weight, and other signs of malnutrition. I can confirm that a high proportion of those who are underweight are also anaemic, but I do not agree that large families are a necessary cause of rickets; out of a group of 51 soldiers with signs of old rickets, 23 were the first child.

I have found no evidence to suggest that rickets did disappear during the later post-war years, and much that it continued, particularly in social groups IV–V. Recent examinations of young children, primary and secondary school boys, and of boys joining a junior leaders' unit of the Army indicate that it has been present all the time during the 50's and 60's, but unrecorded. The official assumption that there was no malnutrition in Britain has perhaps discouraged attention being paid to it, and I believe there has been a tendency sometimes to regard rickety deformities as the province of the orthopaedic surgeons only.

Average height and weight records tend to support the official view unless original figures are looked at critically, when it will sometimes, or perhaps often, be found that the "average" represents a mean between two very far separated extremes—the underweight and the overweight—both of whom may be suffering from considerable nutritional defects. Maps giving places of upbringing show that the great cities of the Midlands, the North, London, and the towns to the east of it provide most of the rickety and underweight young men; the smaller county boroughs and country towns provide the better built, but rural Eire provides the best of all.

In addition to the planned investigations advocated in your leading article, which will take some years, as a more immediately effective measure I would suggest that every young person after leaving school should have an assessment of nutriture by doctors who have some skill in this field, at fixed intervals up to 18 years of age. If there appears to be a shortage of such doctors in the existing services, many might be found among those who have come home from H.M. Overseas Civil Service, or from missionary work in various parts of the world.—I am, etc.,

Yeovil, Somerset.

R. Y. TAYLOR.

Amoebiasis—a Diagnostic Problem in Great Britain

SIR,—The physicians and surgeons who allowed Dr. R. Wright (16 April, p. 957) to publish their experience of hepatic amoebiasis are to be commended. But it is per-

haps fair to point out that the significance of certain signs referred to by Dr. Wright—for example, the elevated right diaphragm—has been known for a very long time, and it is difficult to find a better account of the diagnosis and management of hepatic amoebiasis than that of Sir Leonard Rogers and Sir John Megaw.¹

With regard to management, these authors stress the danger of exploratory puncture of the congested liver before a course of emetine (or at least a few grains) has been given. Puncture is, as they say, very rarely a matter of urgency and often unnecessary, and it is remarkable how quickly a liver that has been enlarged, even for many weeks, may subside after a few injections of the drug. For example, an Italian patient had been in hospital for two months because of high fever, loss of weight, and an enlarged liver. When he was seen at the end of that time his liver was palpable four fingerbreadths below the right costal margin, but it subsided long before the course of emetine was completed, and so did his pyrexia. Similar cases could be cited. I have no regrets at having ordered emetine for a patient who had a considerably raised temperature and an enlarged liver but who proved to have metastatic carcinoma.

While agreeing with Dr. Wright that precise diagnosis is desirable, I think those who have experience of hepatic amoebiasis will agree that it is the condition *par excellence* in which very much more is likely to be lost than gained by withholding a specific drug in the hope, often vain, of confirming the diagnosis.—I am, etc.,

Peppermint Grove,
Western Australia.

GERALD C. MOSS.

REFERENCE

- ¹ Rogers, Sir Leonard, and Megaw, Sir John, *Tropical Medicine*, 2nd ed., 1935. Churchill, London.

Diurnal Enuresis

SIR,—I wonder if the following case, which one might report under the heading of "Enuresis diurna," would be of interest to readers of the *B.M.J.*

It concerned an otherwise perfectly normal boy of 9 who had never in his life wetted his bed but was always wet during the day, whether at home or at school. Many remedies, educational and pharmaceutical, had been tried without any effect. The mother, having recently moved into this district, and worried about the boy's standing at school, decided to try yet another doctor. Finding nothing abnormal whatsoever either in the boy's general health or in his psychology or in his environment or family background, I decided to give him 10 mg. of amitriptyline t.d.s.

Almost from the first dose on this boy, who during all the nine years of his life had been wet every day, became dry. The mother, understandably pleased with this result, thought after a "dry" fortnight that all was now well and—without informing the boy—reduced the dose by about one-half. The diurnal enuresis recurred at once but disappeared as soon as the full dose was restored. I should be interested to hear whether colleagues have seen similar cases.—I am, etc.,

Pinner, Middlesex.

E. ELKAN.

Treatment of Ulcerative Colitis

SIR,—In view of recent reports in your columns on ulcerative colitis the following case, recently under Dr. S. Locket's care, may be of interest.

A girl aged 19 presented with a history of colicky central abdominal pain and general malaise for one month, diarrhoea (with four soft brown stools a day containing no blood or mucus) for three weeks, pedal oedema, and occasional spotting of fresh blood from the rectum for one week. She had lost 1 st. (6.4 kg.) in weight in the past month. Her previous health had been good. She was anaemic (Hb 58%) and thin (weight 6 st. 1 lb.: 38.6 kg.). She had a clean tongue and a slightly protuberant abdomen with some tenderness in the left iliac fossa. Gross ankle oedema and a raised jugular venous pressure were noted. No rectal bleeding was observed while she was in hospital. A tentative diagnosis of ulcerative colitis was made. Subsequently, electrolytes were repeatedly normal and blood cultures were sterile, but plasma protein values were: albumin 2.0 g./100 ml., globulin 2.6 g./100 ml. Sigmoidoscopy was normal to 20 cm. A barium enema showed a polypoid appearance in the descending colon, suggestive of a polypoid type of ulcerative colitis. Steroid and antibiotic therapy was started and a blood transfusion given. At first she improved symptomatically, but mild diarrhoea continued and her weight fell to 4 st. 10 lb. (29.9 kg.). Six weeks after admission a further blood transfusion became necessary. A swinging pyrexia now developed, and she was transferred to Mr. W. D. Park's care for surgery.

At laparotomy the whole colon from the caecum to the mid-pelvic colon was thickened and inflamed, and the colonic mucosa was extensively ulcerated and showed pseudopolypi; the distal pelvic colon and rectal mucosa were normal. Total colectomy and ileo-rectal anastomosis was performed. The histology of the colon was consistent with ulcerative colitis. Post-operative progress has been reasonably satisfactory.

The case is unusual in that there was only mild diarrhoea of short duration, with the passage of no mucus and very little blood, and yet gross pathological changes were found at operation. In addition the rectum was not involved in the disease process, and it will be interesting to see if this remains so.—I am, etc.,

Oldchurch Hospital,
Romford, Essex.

CARYL W. DARBY.

Disorder of Differential Growth

SIR,—Sir Denis Browne in his letter (21 May, p. 1298) mentions that sterno-mastoid torticollis is a "disorder of differential growth."

It is in fact due to the pressure of the foetal head and neck against the promontory of the sacrum, which flattens the ear and interferes with the growth of the neck muscles. This occurs when the head engages unusually early and deeply. The forehead on the opposite side presses against the symphysis pubis, giving the characteristic flattening.

Babies are frequently born with a slight flattening of the ear on one side and of the forehead on the opposite side, but without

a torticollis—that is, a milder, incomplete picture.—I am, etc.,

Johannesburg.

G. P. CHARLEWOOD.

REFERENCE

- ¹ Charlewood, G. P., *J. Obstet. Gynaec. Brit. Emp.*, 1947, 54, 499.

Syringe-transmitted Hepatitis and Heroin Addicts

SIR,—There has recently been an epidemic of syringe-transmitted serum hepatitis among heroin addicts in this country. This would appear to be spread by the practice of sharing syringes. Most of the cases have appeared in the past eight months. Dr. O. Ben-Arie and I have so far traced 22 such cases but have heard of other cases among addicts from the patients we have seen.

It would be very helpful if anyone who has seen such a case recently could contact either Dr. Ben-Arie or myself at this hospital, or Dr. Vincent Marks at West Park Hospital, as we would like to trace this epidemic more fully.—I am, etc.,

Tooting Bec Hospital,
London S.W.17.

T. H. BEWLEY.

Suture Removal Forceps

SIR,—Standard suture removal forceps seldom prove adequate for grasping very fine sutures, particularly the fine monofilament nylon used in plastic surgery or ophthalmic surgery. The forceps shown in the Figure are produced by Chas. F. Thackray Ltd. and are made on the model of forceps

specially designed by and used by me. They have been in use for some years and have proved ideal for the removal of fine sutures. The blades of the forceps have longitudinal grooves, which allow the finest stitch to be grasped firmly, without fear of it slipping through serrated edges as in the standard type of suture removal forceps.—I am, etc.,

Ballochmyle Hospital,
Mauchline, Ayrshire.

J. C. MUSTARDÉ.

Treatment of Club-foot

SIR,—Over 40 years ago I described¹ a very simple method of treatment with a malleable iron splint.

Provided this was carried out from the moment of birth by the same operator throughout a cure could be expected.—I am, etc.,

London W.1. B. WHITCHURCH HOWELL.

REFERENCE

- ¹ Howell, B. W., *Brit. med. J.*, 1925, 1, 1031.