The following formula gives the quantities for 50 portions of 6 g. each.

Sodium chloride B.P. ...	5.85 g.
Potassium chloride B.P. ...	7.46 g.
Potassium phosphate B.P.C. 1949 ...	4.36 g.
Potassium acid phosphate B.P.C. 1949 ...	3.40 g.
Sodium gluconate ...	21.82 g.
Dextrose ...	257.11 g.

Prepare as granules. Pack in 6 g. quantities. 6 g. of granules dissolved in 100 ml. boiled tap water produces a solution containing:

Sodium ...	4.0 mEq.
Potassium ...	3.5 "
Chloride ...	4.0 "

Phosphate ... 2.0 "
Gluconate ... 2.0 " in 5% dextrose solution.

Although the preparation is designed for babies, it would seem to have some value in certain cases of electrolyte replacement in adults.²—I am, etc.,

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Suppression of Lactation

SIR,—The reports by Daniel and his colleagues¹ and Professor T. N. A. Jeffcoate and others (5 October, p. 19), both suggesting that the suppression of lactation in the puerperium is associated with a higher incidence of thrombosis, prompted us to examine the evidence in this region. The case records of all women delivered in hospital in Aber-

deen require the administration of anticoagulants. In the four years there were 14,754 cases for analysis, of whom 41.2% were lactating.

Table I shows the number and percentage lactating at six days. The cases are divided by mode of delivery into spontaneous vaginal, assisted vaginal, and caesarean section cases. The lowest percentage lactating (5.6%) was in

TABLE I.—Number of Mothers and Percentage Lactating at 6 Days

		Total Nos. and (% Lactating) of Mothers of Parity:			
		0	1 and 2	> 3	All
Delivery	Normal	3,882 (51%)	6,396 (40%)	1,928 (37.6%)	12,206 (42.7%)
	Assisted CS	1,273 (39.4%) [440] (36.3%)	[300] (26%) 375 (28.5%)	[72] (20.8%) [88] (5.6%)	1,645 (36.2%) 903 (30.1%)
Age	< 25	3,104 (50.9%)	2,285 (37.4%)	[186] (26.9%)	5,575 (44.6%)
	> 25	2,491 (42.7%)	4,786 (40%)	1,902 (32.6%)	9,179 (39.2%)
Total		5,595 (47.2%)	7,071 (39.2%)	2,088 (32.1%)	14,754 (41.2%)

Figures in brackets [] are based on fewer than 500 deliveries.

deen over a period of four years, from 1958 to 1961 inclusive, were examined.

Twin deliveries and stillbirths were excluded, as were those patients who were discharged before the sixth day of the puerperium. As in the Cardiff study, "thromboembolism" was defined as any case of pulmonary embolism or deep vein thrombosis which showed signs definite enough to

those of high parity delivered abdominally. The total number of cases of thromboembolism was 78, giving a rate of 5.3 per 1,000 deliveries, a figure comparable to that reported in Cardiff

TABLE III.—Variations of Incidence of Thromboembolism with Age, Parity, and Feeding

		No. of Deep Vein Thromboses and Incidence per 100 Births in Mothers of Parity:			
		0	1 and 2	> 3	All
Age < 25	Lactating	4 (2.5)	2 (2.3)	0 (0)	7 (2.8)
	Not lactating	5 (3.3)	5 (3.5)	0 (0)	9 (2.9)
Total ..		9 (2.9)	7 (3.1)	0 (0)	16 (2.9)
Age > 25	Lactating	4 (3.76)	4 (2.1)	7 (11.3)	14 (3.2)
	Not lactating	9 (6.3)	18 (6.3)	20 (15.6)	48 (8.6)
Total ..		13 (5.2)	22 (4.6)	27 (14.2)	62 (6.7)

TABLE II.—Incidence of Thromboembolism

		Nos. of Deep Vein Thromboses and Incidence per 100 Births in Mothers of Parity:			
		0	1 and 2	> 3	All
Lactating ..		9 (3.4)	6 (2.2)	6 (8.9)	21 (3.4)
	Not lactating	13 (4.4)	23 (5.3)	21 (14.8)	57 (6.6)
Total ..		22 (3.9)	29 (4.1)	27 (12.9)	78 (5.3)

TABLE IV.—Variations of Incidence of Thromboembolism with type of Delivery, Parity, and Feeding

		Nos. of Deep Vein Thromboses and Incidence per 1,000 Births in Mothers of Parity:			
		0	1 and 2	> 3	All
Normal delivery	Lactating	8 (4)	3 (1.1)	4 (6.1)	15 (2.9)
	Not lactating	8 (4.2)	13 (3.4)	13 (10.2)	34 (4.9)
	Total	16 (4.1)	16 (2.5)	17 (8.8)	49 (4)
Assisted delivery	Lactating	0 (0)	[1] (12.8)	[1] (66.6)	2 (3.4)
	Not lactating	2 (2.6)	[1] (4.5)	[0] (0)	3 (2.8)
	Total	2 (1.6)	[2] (6.7)	[1] (1.4)	5 (3)
Caesarean section	Lactating	[1] (6.2)	[2] (18.7)	[1] (200)	[4] (14.7)
	Not lactating	[3] (10.7)	[9] (33.6)	[8] (96.4)	20 (31.7)
	Total	[4] (9.1)	[11] (29.3)	[9] (102.3)	24 (26)

Figures in brackets [] are based on fewer than 500 deliveries.

but greater than the incidence found in Liverpool. Table II shows the incidence of thromboembolism in lactating and non-lactating women by parity.

This Table should be read in conjunction with Table III. It can thus be seen on inspection that the puerperal woman's age is a much more important factor than her parity when the association of thromboembolism is considered, and this is confirmed by statistical analysis.

Applying a χ^2 test to the raw data the difference in incidence of thromboembolism between lactating and non-lactating women is significant ($P < 0.05$). When, however, the figures are standardized for parity and method of delivery, then, although there is still a higher incidence of thromboembolism in non-lactating women than in those lactating, this difference is no longer significant ($\chi^2 = 2.4166$).

Table IV indeed shows the importance of operative delivery in this respect, in that 24 out of 78 cases occurred in women delivered by caesarean section.

These results suggest that operative delivery and age rank high among those factors associated with a high risk of puerperal thromboembolism which were examined, and suppression of lactation is probably much less important. We therefore share, on this evidence, the doubts expressed by Professor Jeffcoate and his colleagues concerning the importance of the administration of oestrogens in the aetiology of puerperal thrombosis. However, the dosage of stilboestrol used may be of some importance, as in this hospital a course consists in all of 80 mg., and this is certainly much lower than the dosage reported by Daniel *et al.* from Cardiff. We are, however, unlike Professor Jeffcoate, in some doubt concerning the effectiveness of oestrogens in the suppression of lactation. In a recent double-blind trial in this hospital Maclean² could show only a slight difference in clinical effect between Mixogen 2 ml. (which contains ethinyloestradiol and methyl testosterone) and placebo in primiparae, and none at all in multiparous patients. However, as Professor Jeffcoate suggests, the prospective studies now being conducted in various centres should throw more light on what is at present a glimmer on the horizon.—We are, etc.,

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Degenerative Tropical Neuropathy and Diet

SIR,—I read with great interest the report of a field survey by Dr. C. K. Osuntokun and colleagues (1 March, p. 547) in which the above relationship to cassava (manioc) consumption was suggested. It is interesting to add¹ that manioc, a corruption of manihot by American and European usage, is of more recent arrival in Africa than the European, and was completely unknown outside the Americas before the voyages of Columbus, not unlike the history of the introduction of tea and tobacco in Great Britain. As Jones further showed in his monograph, it became

disseminated around the world after that great era of discovery and grows mainly in tropical areas, but rarely in the subtropics, in every section of that part of Africa south of the Sahara and north of the Zambezi and other areas within the tropical belt. It is consumed in these areas as either the chief staple or secondary staple diet or as supplementary food. By-products such as starch, adhesives, cosmetics, size for textiles, tapioca, confectionery, and beer and other alcoholic drinks are obtainable from this plant. No soil nutriment or fertilizer is needed for its growth. I have myself seen it flourishing in sandy soil. Except for a virus it is also reported to be free from attack by nearly all plant diseases and locusts. It is therefore of great national importance to the economy of developing tropical countries on account of its lowest cost of production when compared with other cereals and the uses already mentioned.

This report unfortunately did not take the following into consideration in drawing up its conclusions.

(1) The methods of preparation of the diet, which usually result in the reduction of the prussic acid content to safe levels.^{1,2} The methods used are cutting, soaking in several changes of water for some days, followed by shredding, fermenting, grating, and heating, etc. Any remaining unhydrolysed glucoside is further broken down in the gastrointestinal tract.

(2) That manioc (cassava) is not the only food plant that may contain cyanogenetic glucosides; they have also been found in lima beans (*Phaseolus lunatus*), corn (*Zea mays*), sorghum, field beans, kidney beans, sweet potatoes, lettuce, and seeds of various fruits.¹

(3) That the amount of prussic acid that can safely be consumed varies from person to person, and appears to be affected by the variety of other foods eaten.¹

(4) The hydrocyanic acid content is determined not only by the variety but also by age of the plant and local soil and climatic conditions.^{3,4}

In view of the foregoing the postulated relationship in this article has evidently not been fully established. Nevertheless, I feel this report has focused attention on, and again emphasized the possible harmful effects to man of, one of the most important food plants of developing countries in tropical areas. Further co-ordinated scientific and dietetic research would be extremely valuable.—I am, etc.,

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Jakob-Creutzfeldt Disease

SIR,—As Dr. S. Nevin points out (29 March, p. 847) there is at present a good deal of confusion in the use of this term. There is also an unsettled dispute among neurologists and neuropathologists as to whether the term as commonly used covers one disease, or two, or many. Dr. Nevin himself draws a sharp distinction between,

on the one hand, Jakob-Creutzfeldt disease as described by Jakob in 1921 and 1923, and, on the other hand, a condition which he calls subacute spongiform encephalopathy.¹ It is the latter, he believes, in which a transmissible "agent" has been demonstrated.

Until this dispute has been settled it would avoid a good deal of misunderstanding if the term "Jakob-Creutzfeldt disease" were dropped entirely. (For one thing, few modern neurologists would accept Creutzfeldt's (1920) case as an instance of the syndrome described by Jakob). The term "subacute presenile polioencephalopathy," though cumbersome, should satisfy those who believe in the unity of the group as well as those who regard the question as unsettled; and I would respectfully suggest to Dr. Nevin that he should use the term "Jakob's encephalopathy," rather than "Jakob-Creutzfeldt disease" for the restricted group originally described by Jakob.—I am, etc.,

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- 1 Nevin, S., *Proceedings of the Royal Society of Medicine*, 1967, 60, 517.

Meralgia Paraesthetica

SIR,—Mr. K. Mukherjee and Dr. I. B. Bassett (5 April, p. 55) were unfortunate in that their case of meralgia paraesthetica had progressed to the stage of neuroma formation. In the three cases I have seen in general practice relief was obtained by correcting the mechanical factors causing pressure on the lateral cutaneous nerve of the thigh at the point where it emerged through the fascia lata. In the two ladies this was the edge of a tight "roll on," which happened to cross the nerve at this point on one side. In the man pressure was caused by prolonged writing in a contorted position at a small desk.

An analogous condition has been seen of severe pain with paraesthesia on the inner aspect of the heel due to the pressure of an unusually high and rigid shoe-upper on the medial calcaneal branch of the posterior tibial nerve at the point where it perforated the flexor retinaculum. The pain and paraesthesia were readily reproduced by pressure at this point and relieved by a change of footwear. This condition could pass under the name of calcaneodynia paraesthetica.—I am, etc.,

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Measles in the Tropics

SIR,—The *B.M.J.* of 8 February has just reached me, and I was very interested in Dr. David Morley's article about severe measles in the tropics (p. 363).

In this rural part of Nigeria our people know measles to be an infectious disease, but, burdened with too many children, there is nothing the harassed mother can do; she often just has to watch in agony the disease strike down two or three of her children in succes-

sion. These mothers in the past were reluctant to come to hospital because they believed an injection—any injection—would worsen the disease and cause the death of the child. The native treatment consists in covering the child's body with white chalk and giving it repeated drinks of palm wine. No matter what treatment the child is being given and no matter the vigilance of the nurses or doctors, the mother must find a chance of smuggling in a keg of palm wine from which the child and herself have generous helpings day and night.

Some 10 to 15 years ago a measles outbreak occurred at the beginning and end of the dry season, but in the last three years we had a very virulent form which occurred in all seasons; this necessitated our keeping a whole ward for measles. Then came last year the World Health campaign in which some three million doses of measles vaccine were given all over Nigeria. To-day our measles ward is closed—a testimony of the efficacy of the vaccine used, and forceful evidence to convince those in charge of health matters in tropical countries like ours, where there is acute shortage of doctors, that emphasis ought to be placed on preventive rather than on curative medicine.—I am, etc.,

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Measles Vaccination

SIR,—Until we know the duration of immunity conferred by measles vaccination there is no strong case for such vaccination. Your leading articles (29 March, p. 794, and 5 April, p. 2) present a few specks of knowledge perched on a mountain of ignorance, and the only rational conclusion is that not enough is known about the dangers of injecting live virus to justify its use for immunization against any but the most deadly diseases.

The grand objective is to eliminate measles altogether, but before this can be attained it may be found necessary to give booster doses of measles vaccine to adults, and these might well be more dangerous than the first doses.

There is one point in the paper by J. H. Connolly and colleagues¹ which has not been sufficiently emphasized. This is that the measles from which their three patients of panencephalitis originally suffered was thought to have been caused by a less virulent strain of virus than usual. This observation would appear to be of some significance in relation to the safety of vaccination with live attenuated measles virus.

Despite Dr. J. Twomey's letter (11 January, p. 116), in which he criticizes my reasoning (7 December, p. 642), I still think there should be a pause in the measles vaccination campaign, and I also think that your leading articles support this view, though you confine yourself to pleas for more adequate surveillance of the effects of all—and particularly new—vaccination procedures.—I am, etc.,

A. W. O. TAYLOR.

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