to the latter means much. These units could also be used for teaching purposes. At the general-practitioner hospital at which I attend students accompany me regularly. At the outpatients there they see more everyday cases than they would ever see at their teaching hospital, where referred cases tend to be of a highly specialized nature.—I am, etc.,

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London W.1.

enough to go through the night. I usually aim at one ounce per year of age, and at the same time the bad habit of passing urine in the bed must be broken by *waking* the child *before* the bed it wet.

Anyone who wishes to verify these facts can do so by measuring how much an enuretic can hold before training—often less than a sherry glassful.—I am, etc.,

A. R. C. HIGHAM.

St. Paul's Hospital,
London W.C.2.

REFERENCE

- ¹ Higham, A. R. C., *Proceedings of the Royal Society of Medicine*, 1953, **46**, 849.

Causes of Enuresis

SIR,—In your excellent leading article of the causes of enuresis (12 April, p. 63) you include among the things we “know” that do not cause the condition excessively deep sleep.

How do we know this? It is a statement that I have often heard or read, but I have never come across any proof of this opinion. On the other hand, 13 years ago I asked the mothers of 273 enuretic children the simple question, “Is he difficult to wake?” and 68% replied, “Yes.” The same question was put to the mothers of 500 children attending outpatients for other reasons, and only 23% replied in the affirmative.¹

Last year I saw 250 children at the Leicester School Clinic with congenital enuresis and 179 (72%) were said to sleep unusually heavily. Moreover, we have nine enuresis alarms in circulation, which cause a gratifying number of apparent cures in some very difficult cases. The failures are nearly all due to the alarm not waking the patient, although it wakes the parents and all the other non-enuretic members of the family. Lastly, I have this afternoon seen an enuretic girl of 10 who takes 15 mg. of dextro-amphetamine a night—and still sleeps soundly.—I am, etc.,

Leicester.

J. VERNON BRAITHWAITE.

REFERENCE

- ¹ Braithwaite, J. V., *Proceedings of the Royal Society of Medicine*, 1956, **49**, 33.

SIR,—Much as I hate writing letters, I feel I must comment on your leading article (12 April, p. 63) on this subject.

In 1953 I pointed out at a meeting of the urological section of the Royal Society of Medicine¹ that the combination of two factors was almost always associated with what I called then “simple” enuresis, and what is termed “primary” in your article. These two are deep sleep and a bladder which is accustomed to be emptied when it holds only an ounce or two (25–50 ml.) of urine. There simply is not enough capacity to go through the night, and our primitive habit of wetting our nappies (we all do it until we are “pot trained”) is continued into late childhood or longer. Thus far “maturation” is a factor. The low social position of the parents acts, I suggest, because they in many cases could not be trusted to house-train a puppy.

Only the patient can cure himself, and this he can do if he can train his bladder to hold

Abortion Act in Practice

SIR,—As a general practitioner I have become increasingly concerned with the ethics of some members of the profession since the onset of the new abortion laws.

In one case I received a letter from a doctor about a patient of mine whom he had seen without my knowledge. I did not feel the case warranted abortion, knowing the full facts. These I explained to him in my letter, and expressed my opinion that an abortion was not indicated. Nevertheless, he obtained a second recommendation and carried out the abortion himself.

In another case I merely received a letter from a doctor saying he had seen my patient, referred her to a psychiatrist, and arranged termination. Apart from diagnosing this patient's pregnancy, I had no knowledge of any of the proceedings until I received the letter saying the patient had been aborted.

I have had several other patients treated like this. None of the doctors concerned are people I would normally consult and I would certainly not have referred any patient of mine to them. I always choose one of the several teaching hospitals locally. What can be done to stop this cashing in on our patients' predicaments, and raising our blood pressure with utter frustration and annoyance?—I am, etc.,

RICHARD W. PENNY.

London S.E.19.

Severe Self-poisoning

SIR,—I was interested to read Dr. G. R. Burston's account of the management of severe self-poisoning in Sunderland (15 March, p. 679). It is stated that 44% of the sample were discharged directly home to the care of their general practitioners. Does this imply that they did not receive psychiatric screening?

Self-poisoning carries not only an increased risk of further self-poisoning (as the paper shows), but further fatal episodes, regardless of the apparent seriousness of the initial attempt. For this reason the Hill report¹ recommends that all cases of self-poisoning should receive psychiatric attention. In the Bolton group of hospitals, with a case load last year of 260 attempted suicides, 92% of cases were screened. Certainly all cases of

the severity described in the paper were dealt with by either inpatient or outpatient psychiatric services. It has thus been possible to achieve a virtually complete psychiatric service. This may be an advantage of having a psychiatric unit in a general hospital.—I am, etc.,

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Department of Psychiatry,
Bolton District General Hospital,
Bolton, Lancs.

REFERENCE

- Scottish Health Services Council, *Hospital Treatment of Acute Poisoning*, 1968. Edinburgh, H.M.S.O.

Frusemide and Calcium Excretion

SIR,—Drs. J. A. Tambyah and M. K. L. Lim (22 March, p. 751) have shown that in the eight hours after oral frusemide there is an increase in urinary calcium excretion.

We have reported to the Renal Association¹ our preliminary studies on the effect of frusemide on calcium excretion. We have confirmed that both intravenous and oral frusemide causes a marked hypercalciuria in the four hours following administration. However, this is then followed by hypocalciuria so that the overall 24-hour calcium excretion may be little changed. It is probable that if Drs. Tambyah and Lim had studied their subjects for the next 16 hours a period of marked hypocalciuria would have been observed.

Before speculating on any prolonged effect from frusemide therapy it is important to observe the effect of frusemide on calcium excretion and absorption over the 24 hours, and also to consider whether changes persist during prolonged therapy.—We are, etc.,

MARTIN S. KNAPP.

DAVID A. HEATH.

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Bristol 2.

REFERENCE

- ¹ Renal Association, 1968, abstract to be published in *Nephron*, 1969.

Granules of Electrolytes for Infants

SIR,—Prompt oral treatment of infants who are vomiting or have diarrhoea can prevent or lessen the degree of dehydration and make emergency intravenous therapy unnecessary. In these conditions oral dextrose solution is contraindicated, as its use does not replace lost ions; an electrolyte solution is required. In reminding readers of this, you suggested in a leading article (13 January, 1968, p. 70) that it would be helpful to have a solution tablet containing the required replacement electrolytes, which could be used to make into feeds.

This idea was investigated here, but in the end it proved more practicable to prepare granules designed to be dissolved in sterilized distilled or boiled tap-water to produce a solution suitable for use as a “feed.” The formula is based on one given by Talbot and his colleagues,¹ with the addition of dextrose and the minor modifications found necessary for the preparation of granules.

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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University College Hospital,
London W.C.1.

REFERENCES

- ¹ Talbot, N. B., Crawford, J. D., and Butler, A. M., *New England Journal of Medicine*, 1953, 248, 1100.
- ² *British Medical Journal*, 1968, 4, 344

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 ..	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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REFERENCES

- ¹ Daniel, D. G., Campbell, H., and Turnbull, A. C., *Lancet*, 1967, 2, 287.
- ² Maclean, M. W., personal communication.

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