Phenytoin Intoxication

SIR,—It should be noted that delayed onset of phenytoin intoxication of unknown cause (Dr. S. Behrman, 22 November, p. 496) has been described previously.[1–3] Dr. Behrman has mentioned some causes of phenytoin intoxication, but there are many others. Most operate by reducing hepatic parahydroxylation of phenytoin, and many have been described within the last two years.

Over 20 drugs may precipitate phenytoin intoxication. Of particular interest, apart from those mentioned by Dr. Behrman, are other anticonvulsants (phenobarbitone,[4] sulthiame, mephenytoin,[2] trimethadione,[2] and possibly primidone), a wide variety of psychotropic drugs,[5] phenylbutazone, oestrogens,[5] and sulphaphenazole. Children may eliminate phenytoin more rapidly than adults,[3] and, in adults, increasing age makes phenytoin intoxication more likely.[2] I have noted higher blood phenytoin levels and more frequent intoxication in elderly patients. Thus, as the patient grows older, the previously satisfactory phenytoin dosage may prove to be excessive.

Intoxication may be caused by patients exceeding their prescribed intake of phenytoin.[5] They may also take less than their prescribed intake.[5] In this case, on admission to hospital their intake, properly supervised, will increase with possible intoxication. It would be interesting to know if Dr. Behrman's cases had been recently admitted to hospital.

Other possible causes not mentioned by Dr. Behrman include discontinuation of drugs which increase phenytoin metabolism (barbiturates,[4] hydroxyzine, chlorcyclizine, folic acid), acute infection,[3] variation in phenytoin absorption,[5] and folic acid deficiency.

It is likely that other drugs can alter phenytoin metabolism so that a high index of suspicion should be maintained as to the role of concurrent drug therapy in patients treated with phenytoin. It is my experience, however, that the aforementioned factors rarely alter phenytoin metabolism to such a degree that intoxication occurs.

It should be emphasized that intoxication caused by high blood levels of this very safe and often indispensable drug can be easily corrected by reducing the phenytoin dosage.—I am, etc.,

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1 Livingston, S., *Postgraduate Medicine*, 1956, **20**, 584.
2 Roseman, E., *Neurology* (Minneapolis), **1961**, **11**, 912.
3 Svensmark, O., and Buchthal, F., *American Journal of Diseases of Children*, 1964, **108**, 82.
4 Kutt, H., Haynes, J., Verebely, K., and McDowell, F., *Neurology* (Minneapolis), **1969**, **19**, 611.
5 Kutt, H., and McDowell, F., *Journal of the American Medical Association*, 1968, **203**, 969.

Axillary Hyperhidrosis

SIR,—This note describes two severe cases of axillary hyperhidrosis successfully treated with the Hurley–Shelley operation. It appears to be a simple and effective method of dealing with the condition, which is certainly not rare and in its most severe form can be a great social embarrassment to the patient.

Axillary skin excision in the management of axillary hyperhidrosis was first described by Hurley and Shelley[1] in 1963. They explain that they discovered this treatment accidentally in 1957 during an investigation of the histology of axillary sweat glands. A general biopsy specimen taken from the axilla of a patient with intense hyperhidrosis produced a remarkable reduction in the amount of sweat in this axilla. Using Randall's method for assessing the distribution of sweat glands in man with starch–paper–iodine, they observed that 70 to 80% of axillary sweat is produced by glands found in the dome or central portion of the axilla. Having mapped out this region, an ellipse of this central axillary tissue was excised down to and including part of the subcutaneous tela, thus removing a sufficient number of the most active sweat glands in the axilla. The elliptical excision is placed transversely—i.e., across the mid axilla—and not in a longitudinal axis. After haemostasis the skin edges are apposed with vertical mattress sutures. Hurley and Shelley carry out this procedure under local anaesthesia.

Case 1.—A male, aged 42, employed in a bank, was referred by his general practitioner for consideration of cervicothoracic sympathectomy. Axillary hyperhidrosis commenced a year before his referral. It soon became extremely tiresome and caused extreme embarrassment. Sweating stained his clothes to such an extent that the dry-cleaners were unable to remove these marks and the patient spent more than £100 on new clothes in less than six months. The patient had tried all known antiperspirants and deodorants with no effect. There was no family history of hyperhidrosis. General examination revealed a fit, healthy man and investigations revealed no evidence of hyperthyroidism. Thoracic inlet and chest *x*-rays, electrocardiograph, and urinary steroids were normal.

Case 2.—Male, aged 20, a university student, was an anxious, rather highly strung young man with generalized hyperhidrosis which was much worse in his axillae than other regions of his body. Again, this patient was disturbed by sweat staining his clothes under the axillae.

Treatment.—10% iodine in starch powder was applied to both axillae on a small cotton-wool pad after initially washing and drying the axillae with dry gauze. After two or three minutes sweat from the glands in the central portion of the axilla had turned the starch-iodine-powder dark blue. The stained zone measured 4 by 6 cm. Under general anaesthesia bilateral elliptical incisions were made in the axillae and placed transversely in the line of the skin crease. They were both 7 cm. long and 3 cm. wide at the centre. The skin edges were undercut by 1 cm. on either side, subcutaneous glandular tissue being removed. Warm wet swabs were placed in the wounds and pressure was applied for five minutes. Any remaining bleeding points were treated by diathermy. After haemostasis the skin edges were apposed.

Both patients were very pleased with the results and have remained free of excessive axillary sweating for eight months.—I am, etc.,

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REFERENCE

1 Hurley, H. J., and Shelley, W. B., *Journal of the American Medical Association*, **1963**, **186**, 109.

Treating Preinfected Wounds

SIR,—We write in reply to two points raised in your correspondence columns (15 November, p. 428, and 22 November, p. 493) concerning our paper on topical ampicillin in the appendicectomy wound (25 October, p. 206). The bacteriological findings in the infected wounds were:

	Placebo patients	Ampicillin patients
Coliforms	7	1
Enterococci	1	
Fusiform bacilli	1	
H. influenzae	1	
Staph. coagulase positive	1	
No growth*	1	1
No record†	4	
Total	16	2

*Both these patients had been treated with systemic ampicillin.
†Three of these patients had returned home before the wound discharged. In one patient the wound swab was lost on its way to the laboratory.

From only one patient was an organism cultured which was resistant to ampicillin, and therefore bacteriologically this antibiotic would seem satisfactory.

The use of a placebo powder was considered advisable on the grounds that it might otherwise be argued that any sterile powder used topically might have the same effect as ampicillin. It also eliminated the possibility of observer bias on wound inspection. A careful survey of the literature has failed to convince us that our wound infection rate was any higher than other workers who, like us, observed strict criteria in the diagnosis of wound infection, including, in the infected group, sepsis which only became apparent after discharge of the patient from hospital. In support of this contention are the figures obtained by Drs. J. G. Mountain and P. V. Seal, who have kindly consented to allow us to quote their findings in advance of their paper. They studied wound infection after appendicectomy in a trial along similar lines to our own. Ampicillin used topically was compared with no treatment. The infection rate in the control group was 21% in 75 patients, a figure not significantly different from our own.—We are, etc.

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Brucellosis Still Spreading

SIR,—With reference to Dr. R. W. D. Turner's letter (13 December, p. 685) it should be pointed out that in England and Wales—if not in Scotland—there is legislation to prevent the sale of milk which is known or suspected to be infected with brucella organisms. Under Regulation 20 of the Milk and Dairies (General) Regulations, 1959, if a district medical officer has evidence which satisfies him that a person is suffering from disease caused by the consumption of milk, or that the milk is infected with disease communicable to man, notice may be given stopping the sale of the milk, or alternatively making the sale conditional on the milk being made safe—for example, by pasteurization. If, however, a medical officer has no such evidence, but has reasonable grounds for suspecting that a person is suffering from a disease thus caused, or that milk is infected with such disease, his powers are limited to the second line of action.

In addition to these powers, Section 31 of the Food and Drugs Act, 1955, prohibits the sale for human consumption of milk of any cow which, to the seller's knowledge, is suffering from one of the diseases specified in the Third Schedule to the Act. This list of