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. Dr. Behrman has mentioned the cases of phenytoin intoxication, but there are many others. Most operate by reducing hepatic parahydoxylation of phenytoin, and many have been described in 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, sulthiamine, mephenytoin, trimethadione, and possibly primidone), a wide variety of psychoactive drugs, phenylbutazone, oestrogens, and sulphamphenazole. Children may eliminate phenytoin more rapidly than adults, and, in adults, increasing age makes phenytoin intoxication more likely. I have noted higher blood phenytoin levels and more frequent intoxication in elderly patients. Thus, as the patient's blood level rises, the previously satisfactory phenytoin dosage may prove to be excessive.

Intoxication may be caused by patients exceeding their prescribed intake of phenytoin. They may also take less than their prescribed single-strength tablet. In such a 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, hydroxyurea, chlorcyclizine, folic acid), acute infection, variance in phenytoin absorption, 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 significantly in the usual course of therapy 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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References
1. Livingston, S., Postgraduate Medicine, 1956, 20, 354.
2. Rosenman, E., Neurology (Minneapolis), 1961, 9, 912.

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 in 1963. They explain that they discovered this treatment accidentally 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 tula, 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 carried 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 cervicotoracicus sympathetic. Axillary hyperhidrosis was evident before his referral. It soon became extremely troublesome and caused extreme embarrassment. Sweating in excess of that which 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 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 the skin and the small cotton-wool pad after initially washing and drying the axillae with dry gauze. After two or three days of application of the dry powder 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 anesthesia 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 5 cm. apart at the bases and 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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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—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 reasonable grounds for suspecting that a person is suffering from disease caused by the consumption of milk, or that the milk is infected with disease communicable to man, he is entitled to stop 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...