

years later, as in this case. Other possibilities should therefore be entertained, and particularly a small androgenic tumour of the ovary, such as an arrhenoblastoma or a hilar cell tumour. Such a tumour may be so small as not to be obvious on bimanual examination. Indeed, a hilar cell tumour can pass unrecognized at laparotomy unless the ovary is sectioned. It may be added that these tumours do not always produce all the signs of virilism such as hirsutism and voice changes. Their effects sometimes show only in one target organ, so they may still be present even though the only clinical evidence is hypertrophy of the clitoris. Nor do they usually result in an increased excretion of 17-ketosteroids in the urine. In this respect they differ from an androgenic tumour of the adrenal, the presence of which in this case could be virtually excluded by the finding of a normal level of these excretion products.

Digitalis with Quinidine

Q.—*Can quinidine be given at the same time as digitalis? An elderly lady had a coronary thrombosis a few years ago, and was subsequently digitalized for mild cardiac failure. The digitalis has been continued, but she is now having prolonged attacks of paroxysmal tachycardia.*

A.—Quinidine can certainly be given at the same time as digitalis. Indeed, if it is desired to convert atrial fibrillation to sinus rhythm with quinidine it is advisable for the patient to be digitalized¹ to prevent an increase in ventricular rate due to diminution of atrioventricular block resulting from a reduction in atrial rate. It is, however, inadvisable to give full doses of digitalis and full doses of quinidine at the same time.

In the case quoted it is presumably desired to give quinidine in prophylactic dosage of 6–12 gr. (0.4–0.8 g.) daily to prevent attacks of paroxysmal tachycardia. Provided that the patient is not sensitive to quinidine there is no objection to its administration in this way. However, when paroxysmal tachycardia occurs in a patient who is already having digitalis, the possibility of digitalis intoxication producing the tachycardia must be considered. It is not possible to say whether this might be the cause in the patient quoted without full clinical and cardiographic details.

REFERENCE

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Fish-poisoning in the Pacific

Q.—*Recently there have been two fatal cases of fish-poisoning in Tonga, and the symptoms appeared to correspond with those of organo-phosphorus poisoning. (1) Is the pharmacology of fish-poisoning the same as that of poisoning by the organo-phosphorus compounds? (2) Would the treatment for organo-phosphorus poisoning be suitable for fish-poisoning?*

A.—Poisoning through eating fresh fish in the Pacific was first described in the English literature by Cook.¹ It appears to occur fairly commonly in the tropical part of the Pacific, and most of the islands, with the reputed exceptions of Sikiana and Tonga, have poisonous fish.² It would now seem that these fish extend to Tonga. Very many different species of fish have been found to be poisonous but not all fish of the same species are poisonous, and there can be seasonal variations in their toxicity. The only certain way of diagnosing poisonous fish is by biological assay, the recommended test species being the mongoose.³ The variety of species involved and the frequent relation between the toxicity of the fish and the precise area where they have been caught has led people to think that the toxin is derived from something in the diet of the fish.^{2,4} A possible exception is the puffer fish, whose toxin may be endogenous.

The symptoms of poisoning seem to be much the same wherever they occur. Muscle and joint pains, headache, excessive salivation, vomiting, weakness, and muscular incoordination occur, leading in fatal cases to paralysis and coma. There is loss of the knee and ankle reflexes, and in

non-fatal cases paraesthesiae lasting up to several weeks seem to be a common feature.

The treatment of the condition has been largely symptomatic. Castor-oil has been given at the outset, atropine will relieve the salivation, and promethazine hydrochloride will diminish the paraesthesiae. The postulated resemblance to organo-phosphorus poisoning is only very superficial, and these toxins are not acetylcholinesterase inhibitors.⁵ Their effect on neuromuscular transmission is thought to be due to inhibition of the release of acetylcholine.⁶ Consequently the treatment recommended for organo-phosphorus poisoning would not be suitable for these cases. The extremely high toxicity of these substances has aroused the interest of pharmacologists in many centres, and their mode of action is being strenuously investigated. It is to be hoped that a rational plan of therapy will emerge from these studies.

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Withdrawing C.S.F. from Spitz-Holter Valve

Q.—*Is it possible to withdraw cerebrospinal fluid from the bulbous part of a Spitz-Holter valve, and are there any particular dangers in such a procedure?*

A.—The Spitz-Holter valve used for the treatment of hydrocephalus consists of two cone-shaped valves partly encased in metal tubes and joined to one another by a short silicone rubber tube. It is this central portion which is referred to in the question. Although it is possible to obtain a sample of cerebrospinal fluid by aspiration from this part, there is considerable risk of damage to the apparatus, which is both delicate and costly. The silicone rubber tube splits readily, and after insertion of a needle will leak cerebrospinal fluid through the puncture, even at the low pressure at which the valve operates. Furthermore, there is a chance of damaging the valve mechanism with the point of a needle. These risks are greater than those of obtaining a sample from the lateral ventricle, which can usually be punctured through the coronal suture in these patients, when the lumbar route is not available and the fontanelle is closed. Cisternal puncture is not advised in cases of myelomeningocele.

Correction.—We regret a misprint in Dr. A. M. Foxe's letter on "Tumours in Children." Lord Moynihan's phrase was "the adnormal case," not "abnormal case."

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