and the sac contained 30 ml. of blood-stained fluid. The heart (460 g.) was commensurate in size with that of the body. The myocardium appeared healthy and there was no evidence of dilatation of either ventricle. All valves were normal. In the coronary arteries there was no macroscopic evidence of atheroma, but a large organizing thrombus was present in the circumflex branch of the left coronary artery (Fig. 3). Both lungs showed fairly marked congestion, with slight oedema. There was no evidence of bronchitis. Pleurae and trachea were healthy. The aorta was elastic and quite free from atheroma. There was marked bruising of the anterior surface of the upper peritoneum, and the cavity contained about 2 litres of blood mixed with bile. In the liver (1,630 g.) there were several extensive ragged lacerations in the under surface of the left lobe. The gall bladder was healthy but was quite empty. There was a small laceration in the common bile duct. The entire alimentary tract, pancreas, suprarenals, spleen (190 g.), and kidneys (120 g. each) were healthy.

Microscopical Examination.—A section of the circumflex branch of the left coronary artery, 0.5 cm. proximal to the proximal end of the thrombus, showed minimal atheroma.

COMMENT

This is a case of primary traumatic coronary thrombosis. It is apparent that the thrombosis occurred from injury to a

previously healthy heart, for the onset of symptoms was contemporaneous with the injury. Further evidence is provided by clinical data at three-quarters of an hour, by electrocardiograms at four hours, and by necropsy after death at 44 hours.

Most cases claimed to be similar are secondary to advanced heart disease or posttraumatic—that is, with an interval between the injury and the thrombosis. It is becoming widely accepted that traumatic heart disease is more common than had been previously recognized. This case, together with that described by Meessen (1940), establishes that primary traumatic coronary thrombosis is an entity.

It is a pleasure to acknowledge my indebtedness to Professor W. A. Mackey for permission to report this case; to Dr. M. M. O'Hare for her report on the electrocardiograms; to Dr. A. Dick for the necropsy report; and to Dr. J. H. Wright for his comments on the manuscript.

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REFERENCE

Meesen, H. (1940). Frankfurt. Z. Path., 54, 307.

Fenfluramine Overdosage

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Fenfluramine hydrochloride (Ponderax), superficially a compound of an amphetamine-type structure, was reported by Munro et al. (1966) to be an effective appetite depressant devoid of stimulant effects on the central nervous system. The only side-effect observed was severe diarrhoea in 2 of the 25 patients who received the drug. This lack of stimulation, together with the lack of hypertensive effect (Le Douarec and Schmitt, 1964; Lambusier, 1965), is in contrast with observed effects with amphetamines. In view of the present concern over the dangers of drug habituation and the widespread use of anorectic drugs this is not without importance.

We report a case of self-poisoning with fenfluramine hydrochloride, together with some laboratory findings.

CASE REPORT

A 13-year-old girl was admitted to New End Hospital half to one hour after having taken approximately 15 tablets of Ponderax (20 mg.)—that is, 300 mg. of fenfluramine hydrochloride. On arrival she was very drowsy and uncooperative, refusing to say what had happened. She had vomited twice, was shivering, and said she felt cold. On examination the extremities were cold, the pulse was of good volume and regular at 88 per minute, and the blood pressure was 110/70 mm. Hg. The pupils were widely dilated and did not react to light until three hours after admission. The cardiorespiratory system and abdomen were normal. The central nervous system was normal apart from sluggish tendon reflexes. This combination of symptoms and signs is most uncommon in other forms of poisoning. A specimen of blood was taken for drug estimation and gastric lavage performed. The patient made an uneventful recovery.

The specimen of blood and a sample of the stomach washout were analysed at Chelsea College of Science and Technology by gas-liquid chromatography. The blood sample was found to contain 0.24 μ g. of fenfluramine and 0.04 μ g. of norfenfluramine, a metabolite of fenfluramine, per ml. The stomach washout contained approximately 3 mg of fenfluramine hydrochloride.

COMMENT

Previous work has been carried out in these laboratories on excretion patterns of this drug after various oral and intra-

venous doses to humans. An oral dose of 180 mg. of fenfluramine hydrochloride has been shown to give blood levels of 0.08 and 0.16 μ g. of fenfluramine per ml. one hour and two hours respectively after taking the dose. Extrapolating from these results, the level of fenfluramine in the blood specimen from the patient indicates that approximately 360 mg. of fenfluramine hydrochloride—that is, 18 tablets—was taken, an estimate in agreement with the report from the patient.

Though the samples were received and analysed two weeks after being taken, storage tests over four weeks show that deterioration is negligible and that no interfering peaks occurred on the chromatogram if the samples were refrigerated. The analysis was carried out on a Perkin-Elmer F.11 gas chromatograph with a flame-ionization detector; the method used was similar to that for the detection and identification of amphetamine (Beckett and Rowland, 1965).

Ten cases of fenfluramine overdosage (unpublished) in France have been reported to the manufacturer, but no fatalities have occurred. Of these 10 patients three took 30 tablets, five took 40, one took 80, and one took 90. For most of these cases full details are not available, but the patient who took 80 tablets presented with symptoms of trismus and transient spasm of other muscles and was semiconscious. Recovery was spontaneous and rapid but there was loss of appetite for one month afterwards.

We are grateful to Dr. Cecil Symons for permission to publish this case; to Dr. L. T. Newman, the patient's family doctor, for preadmission details; and to Selpharm Laboratories Limited for information from sources in France and for supplying fenfluramine hydrochloride for experimental studies.

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REFERENCES

Beckett, A. H., and Rowland, M. (1965). J. Pharm. Pharmacol., 17, 59. Lambusier, P. (1965). Vie méd., 46, 679. Le Douarec, J. C., and Schmitt, H. (1964). Thérapie, 19, 831. Munro, J. F., Seaton, D. A., and Duncan, L. J. P. (1966). Brit. med. J., 2, 624.