Re-examination of the suspect valves showed that a diaphragm with long studs had been placed in a valve-case designed for a diaphragm with short studs. This was an accidental occurrence which probably happened when several valves had been cleaned simultaneously.

Prevention

As long as the two types of valve are in circulation the accident can happen again. It is suggested that the only satisfactory way of preventing this accident is to take out of use all valves having diaphragms with short studs. These are, in any case, considered to be obsolete by the manufacturers. They have not been made for some years and were only supplied with early models of the Beaver ventilator.

It is understood that the manufacturers are anxious to exchange, free of charge, all such valves for the new type.

Summary

Two cases are described in which dangerous cyanosis occurred as a result of an obsolete type of diaphragm being fitted to the inspiratory-expiratory valve of a Beaver Ventilator.

It is recommended that the obsolete valves should be taken out of use at once.

REFERENCE

Mushin, W. W., Rendell-Baker, L., and Thompson, P. W., (1959) *Automatic Ventilation of the Lungs*, Blackwell Scientific Publications, Oxford, p. 305.

Jaundice during Prochlorperazine Therapy

Prochlorperazine ("stemetil") (1-[3-(3-chloro-10-phenothiazinyl) propyl]-4-methylpiperazine dimaleate) is a phenothiazine derivative related to chlorpromazine (see Formulae). It has been used in the treatment of psycho-

Chemical structure of prochlorperazine and chlorpromazine.

neurotic patients (Vischer, 1957), and is an effective anti-emetic agent (Smithy and Homburger, 1957). Side-effects that may result from its use are drowsiness, dryness of the mucous membranes, interference with accommodation, Parkinsonism, bizarre neuromuscular disorders, and photosensitive dermatitis (report from May and Baker, Dagenham). There has been no mention in the literature of jaundice following the use of prochlorperazine. We report a case in which jaundice appeared during administration of this drug.

CASE REPORT

A 69-year-old man was admitted to hospital on April 3, 1958, with jaundice. He complained of weakness and lethargy for two weeks, during which time he had observed that his urine was dark and his motions were clay-coloured.

There was no anorexia, nausea, vomiting, abdominal pain, fever, or weight loss. He had suffered from an anxiety psychoneurosis for many years and had begun treatment with prochlorperazine in November, 1957. He continued taking the drug, approximately 10 mg. daily, until admission to hospital. The total quantity administered was 1,500 mg.

Physical examination revealed a well-nourished jaundiced man. The liver was tender, and was enlarged to 4 cm. below the right costal margin. The liver edge was smooth and regular. The stools were grey and the urine was dark. Examination was otherwise negative. There was no fever.

Laboratory studies disclosed the presence of bile in the urine without increase in urobilinogen. Serum bilirubin was 5 mg. per 100 ml., serum alkaline phosphatase 39 King-Armstrong units, thymol turbidity 0.2 unit, zinc sulphate turbidity 1 unit, serum proteins 8.1 g. per 100 ml. (albumin 5.1 g., globulin 3 g.). Cholecystography and choledochography revealed normal function of the biliary tree without evidence of obstruction to the major bile passages. Examination by barium meal and enema revealed no abnormality. A full blood count showed a haemoglobin value of 13.5 g. per 100 ml., white-cell count 9,200 per c.mm. (neutrophils 5,520, eosinophils 844, lymphocytes 1,548, monocytes 1,196, and band forms 92). A liver biopsy showed no evidence of hepatocellular damage. There was occasional bile plugging of canaliculi.

Prochlorperazine was suspended on admission to hospital, and during the subsequent two weeks the patient's jaundice subsided. At this time serum bilirubin was 1 mg. per 100 ml. and the serum alkaline phosphatase 23 King-Armstrong units; thymol turbidity 0.2 unit, zinc sulphate turbidity 1.1 units, serum proteins 7.1 g. per 100 ml. (albumin 4.5 g., globulin 2.6 g.). The eosinophil count had returned to normal. Follow-up liver-function tests and full blood counts performed three months later were within normal limits.

COMMENT

This patient received prochlorperazine for four months as an ataractic agent, the total dose being 1,500 mg. There was no history of exposure to other recognizable hepatotoxic agents or drugs. He developed an obstructive type of jaundice, with a raised serum alkaline phosphatase level; hepatocellular function tests were normal. There was also a transient eosinophilia. Liver biopsy revealed no evidence of parenchymal injury. On withdrawal of the drug the jaundice subsided. Jaundice is known occasionally to follow the administration of chlorpromazine (Lemire and Mitchell, 1954; Zatuchni and Miller, 1954; Hartnett, 1955), and Hubble (1941) has reported the hepatotoxic action of phenothiazine.

Prochlorperazine is chemically related to chlorpromazine and promazine, and shares a common, phenothiazine group. Prochlorperazine would also appear to share with these drugs the propensity to induce jaundice.

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REFERENCES

- Hartnett, B. S. (1955). *Brit. med. J.*, **1**, 1458.
Hubble, D. (1941). *Lancet*, **2**, 606.
Lemire, R. E., and Mitchell, R. A. (1954). *J. Lab. clin. Med.*, **44**, 825.
Smithy, G., and Homburger, F. (1957). *New Engl. J. Med.*, **256**, 27.
Vischer, T. J. (1957). *Ibid.*, **256**, 26.
Zatuchni, J., and Miller, G. (1954). *Ibid.*, **251**, 1003.