It would be clearly impracticable to maintain a central tuberculosis register and a central tuberculosis laboratory for the whole of England, but it is suggested that a centre incorporating these services could be established for the area of a regional hospital board, together with selective mass radiography and intensive vaccination under a unified control. Consideration should be given to annual x-ray examination as a condition of appointment of school staff and other special categories, and to greater use of existing powers under Section 172 of the Public Health Act, 1936, for the isolation of open cases.

My thanks are due to the Danish Government for a scholarship which enabled me to carry out these studies; to the Essex County Council and the Urban District Councils of Chigwell and Waltham Holy Cross, which, as my employing authorities, generously granted me special leave of absence; and to numerous colleagues, here and in Denmark, who kindly gave me much of their valuable time and facilities for study.

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A HAZARD IN THE USE OF THE BEAVER PULMONARY VENTILATOR

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Recently there have been two potentially dangerous incidents connected with the use of the Beaver ventilator. This communication reports the details and suggests how such incidents may be prevented in the future.

Case Reports

Two patients were suffering from respiratory insufficiency and depression of the laryngeal reflexes. In each case tracheostomy was performed and a cuffed tracheostomy tube inserted. This was connected to a Beaver ventilator in the usual way via the inflating inspiratory-expiratory valve (Mushin, Rendell-Baker, and Thompson, 1959) supplied by the manufacturers.

Progress was satisfactory for several days. Then each patient suddenly became cyanosed immediately following a change of the inspiratory-expiratory valve for cleaning purposes. No cause was apparent, but fortunately the inflating valve was changed for still another. The cyanosis at once disappeared.

Examination of the Valves

In both cases the cyanosis followed immediately after changing the inspiratory-expiratory valve, and on each occasion changing the valve again cured the cyanosis. The suspect valves were carefully dismantled and examined. In both cases the edge of the rubber

FIG. 1.—Obsolete valve. Diaphragm with short studs.

FIG. 2.—Current valve. Diaphragm has long studs.

diaphragm was found to be crinkled and turned over upon itself instead of lying flat. In these circumstances, the flow of air from the ventilator passed straight to the atmosphere and the patient's lungs were not inflated.

With the expert assistance of Mr. E. K. Hillard, senior technician to this Department of Anaesthetics, an examination of several other inspiratory-expiratory valves was made. It was found that two types of valve were in use. Although outwardly nearly identical, the valves differ internally in one vital detail. In one type, now obsolete but still in circulation, the diaphragm has two large diameter short studs which fit correspondingly large shallow depressions in the valve casing. In the other, the diaphragm has smaller diameter long studs which fit correspondingly long grooves in the valve casing (Figs. 1 and 2).

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.

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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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