and by children (Tucker and Gerrish, 1960). Few patients survive after taking sulphuric acid (Gray and Holmes, 1948). Robert's patient lived for nine weeks.

In corrosive damage to the stomach the usual lesion is pyloric stenosis of rapid onset. There is a characteristic histamine-fast achlorhydria. One is particularly likely to make a diagnosis of carcinoma from the radiographs. The possibility of carcinoma may force a laparotomy for a decision. Even at operation the condition may resemble carcinoma to a remarkable degree.

In 80% of lesions of the upper gastrointestinal tract due to acids the oesophagus is spared; 20% have concomitant gastric and oesophageal lesions. Ingestion of alkalis damages the stomach and oesophagus equally or spares the stomach (Skapinker and Crawshaw, 1954). Moynihan's (1926) series all had pyloric lesions. Strode and Dean (1950) described the microscopical lesion, which was similar to that in our case.

Treatment consists of emergency and resuscitative measures and surgery for the results of the damage. The acid should be neutralized with magnesium bicarbonate. Tracheostomy or intubation may be necessary for glottic oedema. Intravenous fluid and electrolyte and calorie intake maintenance will be required for some time.

Surgery is undertaken for the results of the corrosion. Local plastic procedures in the region of the pylorus—for example, pyloroplasty or gastroduodenostomy—are unsatisfactory because of the poor tissues and progressive fibrosis. Gastroenterostomy was the usual treatment before 1948. Gray and Holmes (1948), reviewing 139 cases, could find only three treated by partial gastrectomy. Gastroenterostomy, though often satisfactory, has a number of disadvantages—poor tissues and advancing fibrosis, stomach ulceration later, and gastric mucosal metaplasia. Strode

and Dean (1950) reported two cases treated by partial gastrectomy. Strange *et al.* (1951) and Skapinker and Crawshaw (1954) performed total gastrectomies. Hodgson (1959) advocated partial gastrectomy.

From these reported cases the best management of corrosive gastric injury appears to be partial gastrectomy. It is occasionally necessary to perform a total gastrectomy for the more unusual diffuse lesion.

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Haemolysis and Agranulocytosis Complicating Treatment with Methyldopa

Brit. med. J., 1967, **4**, 94

Methyldopa occasionally causes autoimmune haemolysis (Worledge, Carstairs, and Dacie, 1966). Agranulocytosis has also been attributed to this drug (Hallwright, 1961). A case is presented showing both these complications with demonstrable autoantibodies on the red and white cells.

CASE HISTORY

A man aged 71 suffering from hypertension had been treated with methyldopa (2 g. daily) for 16 months. For the last six of these he had also received chlorpropamide (200 mg. daily) on account of mild diabetes. When admitted to hospital his liver and spleen were found to be enlarged and both drugs were stopped, but no active treatment was given for the haematological disturbance discovered.

The results of the haemoglobin, reticulocyte, and white cell estimations are summarized in the Chart. The bone marrow showed normoblastic hyperplasia, active granulopoiesis to the metamyelocyte stage, and excessive iron storage. The half-life of ^{51}Cr -labelled red cells ($T_{1/2}^{51}\text{Cr}$) was reduced to 20 days. The serum bilirubin was 1.4 mg./100 ml. The red cell osmotic fragility was normal. The direct antiglobulin test was positive and the eluate showed anti-e and nonspecific components.

The mixed antiglobulin reaction (Chalmers, Coombs, Gurner, and Dausset, 1959) for autoantibodies on the patient's white cells was repeatedly strongly positive during the early recovery phase. Both the red and white cells remained weakly sensitized after five months.

Comment.—As the complications found in this patient bear little resemblance to the few blood disorders attributed to chlorpropamide (Grace, 1959; Karlin, 1960; White, 1962), this drug seems unlikely to have been the cause. The immune haemolysis discovered is readily attributable to methyldopa, and the agranulocytosis, associated with an autoantibody, suggests that a similar process affected the white cells.

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