TABLE I—Gall Stones and Ulcer Surgery

	Male	Female	Total
No. of patients with gall stones	127	279	406
No. of gall-stone patients with previous ulcer surgery	16	10	26
Percentage of gall-stone patients with previous ulcer surgery	12.6	3.6	6.4

TABLE II—Summary of 27* Ulcer Operations on 26 Patients Developing Gall Stones Subsequently

Ulcer Operation	No. of Patients	Sex	Years between Ulcer Surgery and Confirmed Gall Stones	
			Mean	Range
Polya†	13	M.	11	2—29
Polya	3	F.	14.5	14—15
Billroth I	3	M.	7	2—11
Billroth I	7	F.	7	1—18
Truncal vagotomy	1	M.	2	

*One patient underwent truncal vagotomy for anastomotic ulcer subsequent to Polya gastrectomy but before gall-stone formation.
†Polya refers to partial gastrectomy with gastrojejunal anastomosis.

women indicates the relatively greater incidence of gastric ulcer (Table II). Conversely, the predominance of Polya operations in men reflects the incidence of duodenal ulcer. It is of interest that only one patient had undergone vagotomy. Wastell (1969) reviewed 192 vagotomies performed at this hospital with a minimum follow-up of 10 years. In only one out of all these cases were gall stones confirmed.

The results indicate that though gall stones are much more common in women than in men very few have had previous ulcer surgery. Male patients predominate in the gastric clinic, and the close follow-up of these patients leads to the detection of gall stones which might escape undiagnosed in a non-attending population, hence the high incidence, 1 : 8, of previous ulcer surgery.

Comment

The development of gall stones in a patient only two years after vagotomy was in keeping with the theory that the altera-

tion of biliary tract dynamics by vagotomy might predispose to gall stones. The object of this review was to see whether, in fact, ulcer surgery might be a significant aetiological factor in cholelithiasis. Johnson and Boyden (1952), Cox *et al.* (1958), and Tinker and Cox (1969) noted alterations of gall-bladder physiology after vagotomy, and Wyatt (1969) noted changes after partial gastrectomy. After vagotomy there is an increased gall-bladder volume with increased emptying rate, the gall bladder becoming supersensitive to cholecystokinin.

Nielsen (1964) quoted four cases of gall stones within one year of vagotomy. Nobles (1966) found that 18% of patients undergoing vagotomy later required cholecystectomy. Majoor and Suren (1947), however, found only 6 cases out of 394 in which ulcer surgery preceded gall stones. Chapa and Engel (1959) found that 7 out of 135 cholecystectomies followed within seven years of gastric resection. Griffiths and Holmes (1964) found 17 out of 238 patients with gall stones who had had Polya gastrectomy.

Peptic ulcer surgery preceded one in eight cholecystectomies in male patients but only 1 in 30 in females. Vagotomy is not apparently of significance. Cholecystography should be performed in all patients presenting with new indigestion some years after ulcer surgery.

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Acute Hypercalcaemia and Renal Failure after Antacid Therapy

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Intensive therapy with antacids, especially with calcium carbonate, is recognized as a cause of hypercalcaemia and also of the milk alkali syndrome. Attention has been drawn by Kirsner (1964) to the occasional occurrence of a hypercalcaemic syndrome after the use of antacids. McMillan and Freeman (1965) pointed out the dangers of oral treatment with any calcium salt, and particularly the possibility of renal damage.

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Case Report

In 1965 a 59-year-old retired farmer had a radiologically proved duodenal ulcer causing only minor symptoms of dyspepsia, which he controlled by self-medication with antacids. In August 1969 he developed severe dyspepsia after drinking a large amount of alcohol. The severity of the symptoms was such that he took large daily doses of antacid. Within three days of the initial attack he developed excessive thirst, polyuria, and anorexia; these symptoms persisted over the next four weeks, during which he continued with the high intake of antacid. He was referred for a urological opinion and further investigations. Clinical examination was negative. Intravenous pyelography showed no dye excretion on either side but cystoscopy and retrograde pyelography showed nothing abnormal. A catheter urine specimen showed red blood cells and 100 mg of albumin per 100 ml, but organisms were not cultured. The blood urea was 93 mg/100 ml, the serum creatinine 7.6 mg/100 ml, and the creatinine clearance 9 ml/minute. The fasting serum calcium on two occasions was 7.1 mEq/litre and the 24-hour urinary calcium excretion was 15.6 mEq. No other serum abnormality was detected.

In view of the history of peptic ulcer, thirst, polyuria, hypercalcaemia, and hypercalciuria a confident clinical diagnosis of hyperparathyroidism was made and the patient was transferred from the surgical to the medical unit. Apart from bilateral Dupuytren's contractures no other positive findings were present on clinical examination. The blood pressure was 130/80 mm Hg and the results of further relevant investigations were as follows: serum

FIG. 1—Serum calcium and phosphate levels after ingestion of BiSoDoL. X—X=Serum calcium. ●—●=Serum phosphate.

FIG. 2—Creatinine clearance and plasma creatinine values over 14-week period. X—X=Plasma creatinine. ●—●=Creatinine clearance.

urea 90 mg/100 ml, serum calcium 4.95 mEq/l, serum creatinine 7.6 mg/100 ml, creatinine clearance 9 ml/minute, and urine S.G. 1005. All other investigations, including skeletal survey and parathyroid scan, were normal.

By the day after admission the patient was symptom-free and the serum and urine calcium levels had returned to normal. These

dramatic changes occurred when the antacid was withdrawn on admission to hospital, and this led us to consider the possibility of drug-induced hypercalcaemia. He had been taking about 15 BiSoDoL tablets a day at home and the calcium intake from these was approximately 3 g/day.

When the patient had been normocalcaemic for 10 days and the serum urea had fallen to 61 mg/100 ml the effect of BiSoDoL ingestion on his serum calcium was studied. A fasting blood specimen having been taken, he swallowed 15 BiSoDoL tablets between 9 a.m. and 1 p.m., and three further blood specimens were taken at 2 p.m., 6 p.m., and 9 a.m. the next day. The results are shown in Fig. 1.

The serum calcium response in five normal individuals to similar amounts of BiSoDoL showed a tendency for the serum calcium to rise in three out of the five subjects.

The patient was discharged home and has remained free of any symptoms other than occasional dyspepsia, which is relieved by magnesium trisilicate. The serum and urine calcium levels have remained normal. The serum urea and creatinine levels decreased towards normal and the creatinine clearance rose from 9 to 56 ml/minute (Fig. 2).

Comment

The initial clinical diagnosis of hyperparathyroidism was not substantiated by either the biochemical findings or the clinical course of this patient's illness. When self-medication with BiSoDoL was stopped the serum calcium level rapidly returned to normal and there was a more gradual improvement in renal function.

We have been unable to find any cause other than BiSoDoL ingestion for the acute reversible hypercalcaemia and renal failure in this patient. BiSoDoL may be found in many homes, and the possibility of drug-induced hypercalcaemia should be borne in mind when a patient presents with symptoms suggestive of hyperparathyroidism. Greater awareness of the potential risk of renal damage should prompt the physician to inquire carefully along these lines.

We wish to thank Dr. J. G. Macleod and Mr. J. E. Newsam for allowing us to study this patient.

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