Gut, 1960, **1**, 371.

GASTROENTEROLOGICAL SOCIETY OF AUSTRALIA

The second annual general meeting was held at the Royal Melbourne Hospital on May 24, 1960.

Office Bearers:

President	Sir William Morrow
Vice-President	Dr. W. E. King
Hon. Secretary	Dr. S. J. M. Goulston
Hon. Treasurer	Dr. A. Kerr Grant

Other members of Council:
Dr. W. Irwin
Dr. R. Andrew
Dr. R. A. Joske

Honorary membership was conferred upon:
Dr. F. Avery Jones, of Great Britain
Dr. Hugh Butt, of U.S.A.
Dr. N. Henning, of Germany
in recognition of their outstanding contribution to gastroenterology.

After the business meeting, a scientific meeting was held. Summaries of the papers are as follows:—

R. A. JOSKE described the acute cholestatic syndrome in a summary of 13 patients with the acute idiopathic cholestatic syndrome, stressing the diagnosis, pathology, and prognosis of this disease and comparing and contrasting it with the acute cholestatic syndromes due to drugs such as "largactil" and "marsalid".

W. E. KING and S. F. PHILLIPS discussed the treatment of Wilson's disease with penicillamine based on a patient presenting in August, 1956, with a history of three months' anorexia, nausea, and dark urine without manifest jaundice. She had hepatosplenomegaly and supported by liver function tests was diagnosed as viral hepatitis. Over the following three months she improved symptomatically, the liver size decreased although the spleen remained palpable. She suffered amenorrhoea and was thought at this stage to be a case of mild persistent hepatitis.

She returned in September, 1959, with five months' history of personality variation, dysarthia and hypersalivation. Kaiser-Fleischer rings were noted in the corneae and investigation proved her to be a case of hepatolenticular degeneration. Liver function tests, still abnormal, were similar to those of 1956.

She has had treatment with the British antilewisite ("dimercaprol") and penicillamine (dimethyl cysteine) both of which markedly increase her urinary copper output. Penicillamine is preferred for long-term therapy because of its low toxicity and oral administration.

Her urinary copper output varies with the dose of penicillamine. She also has potassium sulphide by mouth to lower absorption of copper from the gut. With these measures a negative copper balance over a long period has been achieved.

Her progress has not been very encouraging. At the best they claimed to have halted what was a quite rapid deterioration. In no way has she improved on this therapy. However, it is known that treatment must be continued for many months before failure is admitted. During all this period seemingly adequate "decoppering" is being achieved.

They stressed that the important point of this case is that persistent hepatitis should not be accepted in a case of juvenile cirrhosis without first excluding Wilson's disease. The diagnosis should be possible clinically and by the use of fairly simple biochemical estimations. If this patient had been treated three years earlier she might now have had no neurological deficit. The second point is that the establishment of a negative copper balance is not the complete answer in therapy, and may in fact indicate that copper toxicity is not the prime factor in the pathogenesis of this condition.

A. P. SKYRING reported four patients with rare heritable diseases who had bled from the gastrointestinal tract as a result of local manifestations of generalized disease. In each case the disease could be diagnosed by examination of the skin. The four conditions were (1) pseudoxanthoma elasticum, (2) neurofibromatosis, (3) Peutz-Jaeger syndrome, and (4) primary amyloidosis.

I. MACKAY discussed the value of the secretin test and surgical pancreatic biopsy in the diagnosis of pancreatic disease, stating that in some cases of pancreatitis, elevated serum and urinary enzyme levels cannot be demonstrated and other evidence of pancreatic dysfunction must be sought. The secretin test and operative pancreatic biopsy were assessed from this viewpoint. The response to the secretin tests in 64 patients was graded as "normal", "equivocal", "abnormal", and "grossly abnormal". In 25 patients with relapsing pancreatitis, results of secretin tests were fairly evenly distributed in these four groups. Six patients with dyspepsia and atropic gastritis had abnormal tests.

The biopsy appearances in 21 cases of relapsing pancreatitis included normal tissue in seven cases, mixed inflammation and fibrosis in three cases, mild to moderate fibrosis in four cases, and dense fibrosis in seven cases. These findings run parallel with results of the secretin test in relapsing pancreatitis. Biopsy established the diagnosis of pancreatic carcinoma in five out of nine subsequently proven cases.

He considered that the secretin test and pancreatic biopsy should be used to indicate impairment of pancreatic function rather than to establish a specific diagnosis in pancreatic disease.

N. GALLAGHER presented evidence of persistent acute infectious hepatitis being an entity. In these patients

relapses of acute viral hepatitis occurred over long periods, up to three years. Each relapse had the typical clinical and biochemical characteristics of acute hepatitis and serial aspiration liver biopsies confirmed this. All patients recovered without any disturbance of hepatic architecture.

P. PARSONS and I. LYLE discussed the blind loop syndrome in three middle-aged women, all following short-circuit operations for recurrent small bowel obstruction. Two presented with intractable diarrhoea, one with oedema. Results of metabolic and *x*-ray and haematological studies were shown. Operative correction of the blind ileal loop was carried out in all cases.

IAN WOOD praised colour photography of the stomach by the Debray and Housset "gastrophot" gastroscope. This instrument is fitted with an electronic flash of 1/500 second and excellent pictures are obtained. He showed a representative series which included the following:—

(1) Pseudohypertrophic gastritis, the appearance being produced by inadequate inflation of the stomach: it was dispelled by full inflation and gastric biopsy showed atrophic gastritis.

(2) True giant hypertrophic gastritis which had caused gastric haemorrhage and was proven by radiographs and biopsy.

(3) Gastric atrophy which had caused pernicious anaemia and had not regenerated with intensive B12 therapy: the persistence of the atrophy was confirmed by serial gastric biopsies.

(4) Innocent ulcer of the pre-antral region in a pensioner aged 70 not revealed by *x*-ray examination.

(5) Cancer of the antrum, revealed by radiographs and proven at operation.

(6) Anastomotic ulcer with haemorrhage following gastroenterostomy for duodenal ulcer with obstruction: an *x*-ray examination had revealed no ulcer.

(7) "Zonal gastritis" in the region of the stoma following gastroileostomy performed in error for duodenal ulcer and had caused pain, diarrhoea, and severe cachexia. Radiographs had confirmed the error and biopsy showed severe gastritis.

T. KIRKLAND discussed black liver jaundice in a family.

Among six siblings two males have had recurrent jaundice, their liver cells showing a type of pigmentation described in the Dubin-Johnson syndrome.

There appeared to be no significant icterus in other members of the family. Serum bilirubin was measured in the parents and unaffected siblings; it was raised in a third brother who has had no symptoms. In the third generation seven children have not been jaundiced, but their sera have not been examined. Both brothers with symptoms have had abdominal pain, in one case severe. The mechanism of the pain has not yet been explained.

R. ANDREW presented a clinical study of oesophageal hiatus hernia based on his personal experience of 91 cases. Almost all experienced chest or abdominal pain or discomfort, rarely severe and often waking the patient at night. The association of peptic ulcer was high. Oesophagitis was a common feature. About one quarter gave some history of vomiting blood but this was rarely of any severity. Eighty-three patients were managed medically, half doing well and a quarter poorly. Only eight were submitted to surgical repair of the hernia and of these four suffered a recurrence and only two had a good result.

At the annual meeting of the Royal Australasian College of Physicians held concurrently Professor C. R. B. Blackburn presented a case report of a patient who died of liver disease and renal failure. She had cirrhosis of the liver, had a splenorenal anatomosis performed some five years ago, developed high output state and arterial oxygen unsaturation. Full studies revealed the presence of venoarterial shunt of some magnitude and she ultimately died. At necropsy the patency of the anastomosis was demonstrated, and by plastic injection techniques clear-cut pulmonary-arterial venous shunting was demonstrated.

D. W. PIPER gave a paper on the effect of anticholinergic drugs on gastric secretion. A study was made of the effect of anticholinergic drugs on the various components of gastric secretion. With increasing dose, the acid could be inhibited by about 80%. Though pepsin secretion was diminished the output of pepsin was less inhibited than the volume of secretion with a subsequent rise in pepsin concentration. As regards the electrolytes, with increasing anticholinergic suppression of secretion the output of Na and K showed a slight fall, the concentration of Na rose, and the concentration of K showed a slight fall.

When a series of drugs were compared as regards their effect on saliva and gastric secretion, he found that those that are the most powerful inhibitors of gastric secretion also cause a fall in saliva flow. Two new drugs were studied, oxyphencyclimine hydrochloride and propionyl atropine methyl nitrate. The former was found to have a longer duration of action than the commonly used preparations; propionyl atropine methyl nitrate was found to have virtually no effect on gastric secretion.

A study on post-gastrectomy malabsorption was presented by KINSELLA, W. B. HENNESSY, and E. P. GEORGE.

Several nutritional problems after gastrectomy are rare, but on the other hand, weight loss of varying degree is common. The reasons for this are probably multiple and include (1) reduced food intake, (2) improper admixture of food with bile and pancreatic secretion, especially after a Billroth II gastrectomy, and (3) too rapid passage of food through the small intestine.

Additional factors also may be important. For example, bacterial flora operating under conditions suitable for their growth can cause gross malabsorption after gastrectomy. Normally the small intestine is sterile and this appears to be due to an antibacterial mechanism which is independent of the secretions of the stomach (Cregan and Hayward, 1953). However in some patients following total or subtotal gastrectomy this mechanism

breaks down and bacterial contamination of the upper small bowel occurs.

Three cases were presented to show that stasis in the afferent limb of a Billroth II anastomosis is a possible cause of bacterial contamination of the upper jejunum. Two of these patients were treated with broad-spectrum antibiotics and responded favourably. Subsequently, bacteriological studies were carried out at operation. It has been suggested (Wirts *et al.*, 1959; Kinsella and Hennessy, 1960) that such cases are examples of the blind loop syndrome. Other possible causes of stasis include obstruction of the efferent loop of a Billroth II anastomosis and vagotomy. Two additional cases were shown to illustrate this complication.

They further stated that post-gastrectomy steatorrhoea may exist without obvious afferent or efferent loop obstruction. Moreover, although more frequent following the Billroth II operation, steatorrhoea may occur after a Billroth I gastrectomy. With the aid of ^{131} labelled triolein, the steatorrhoea in the majority of these patients is found to diminish following the use of chemotherapy and antibiotics. It is assumed that the antibacterial mechanism in the small intestine has broken down in such cases but the real reason for this is uncertain.

THE SEPTEMBER (1960) ISSUE

The September (1960) issue contains the following papers:—

Post-gastrectomy Syndromes: A Review. C. F. W. ILLINGWORTH.

The Use of Cholecystokinin to Test Gall Bladder Function in Man. P. BURTON, A. A. HARPER, HENRY T. HOWAT, J. E. SCOTT, and H. VARLEY.

Gastric Cytodiagnosis: A Review and Appraisal. D. D. GIBBS.

An Assessment of Prednisone, Salazopyrin, and Topical Hydrocortisone Hemisuccinate Used as Out-patient Treatment for Ulcerative Colitis. J. E. LENNARD-JONES, A. J. LONGMORE, A. C. NEWELL, C. W. E. WILSON, and F. AVERY JONES.

A Clinical and Statistical Study of the Effect of Gastrojejunostomy on Human Gastric Secretion. H. I. TANKEL, I. E. GILLESPIE, D. H. CLARK, A. W. KAY, and J. MCARTHUR.

A Comparison Between the Effects of Hexamethonium and Atropine in Combination and of Vagotomy with Gastrojejunostomy on Human Gastric Secretion. J. MCARTHUR, H. I. TANKEL, and A. W. KAY.

Massive Resection of the Small Intestine after Occlusion of the Superior Mesenteric Artery. R. J. HARRISON and C. C. BOOTH.

Factors Influencing the Growth of *Staphylococcus aureus* in the Stomach after Gastric Operations. F. G. SMIDDY and D. PRATT.

Gastric Activity in the South African European and Bantu. I. MACDONALD.

Benign Duodeno-colonic Fistula. G. N. CHANDLER and A. J. LONGMORE.

Surgery in Bleeding Peptic Ulcers. J. N. WARD-MCQUAID, J. C. PEASE, A. MCEWEN SMITH, and R. J. TWORT.

New Methods for Diagnosis and Research

Wireless Telemetering from the Digestive Tract. A. M. CONNELL and E. N. ROWLANDS.

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 17*s.* 6*d.*