The object of *Gut* is to publish original papers and reviews concerned with practice and research in the field of gastroenterology. The field is that of alimentary, hepatic, or pancreatic disease, and papers may cover the medical, surgical, radiological, or historical aspects. They may also deal with the basic sciences concerned with the alimentary tract, including experimental work. The report of a single case will be accepted only if it is of sufficient interest in relation to a wider field of research.

There will be a section devoted to short papers on laboratory and surgical techniques and methods of investigation where these are not part of a lesser survey.

COMMUNICATIONS Papers should be addressed to the Editor, *Gut*, B.M.A. House, Tavistock Square, London, WC1H 9JR. Papers are accepted only on the understanding that they are not published elsewhere without previous sanction of the Editorial Board. They should be in double-spaced typewriting on one side of the paper only. On the paper the name of the author should appear with initials (or distinguishing Christian name) only, and the name and address of the hospital or laboratory where the work was performed. A definition of the position held by each of the authors in the hospital or laboratory should be stated in a covering letter to the Editor. Communications should be kept short, and illustrations should be included when necessary; coloured illustrations are allowed only if monochrome will not satisfactorily demonstrate the condition. It is not desirable that results should be shown both as tables and graphs.

ILLUSTRATIONS Diagrams should be drawn in indian ink on white paper, Bristol board, or blue-squared paper. The legends for illustrations should be typed on a separate sheet and numbered to conform with the relevant illustrations. Photographs and photomicrographs should be on glossy paper, unmounted. TABLES should not be included in the body of the text, but should be typed on a separate sheet.

ABBREVIATIONS In general, symbols and abbreviations should be those used by British Chemical and Physiological Abstracts. In any paper concerning electrolyte metabolism, it is desirable that data be calculated as m-equiv/l. as well as (or alternatively to) mg/100 ml.

REFERENCES These should be made by inserting the name of the author followed by year of publication in brackets. At the end of the paper, references should be arranged in alphabetical order of authors' names. Such references should give author's name, followed by initials and year of publication in brackets, the *title of the article quoted*, the name of the journal in which the article appeared, the volume number in arabic numerals, followed by the numbers of first and last pages of the article. Abbreviations are according to *World Medical Periodicals* (published by B.M.A. for World Medical Association), thus: Chandler, G. N., Cameron, A. D., Nunn, A. H., and Street, D. F. (1960). Early investigations of haematemesis. *Gut*, **1**, 6-13.

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The July 1970 Issue

THE JULY ISSUE CONTAINS THE FOLLOWING PAPERS

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Notes and activities

Congratulations

We are very happy to offer our congratulations to three contributors to *Gut*, Sir Richard Doll, KB, Professor R. A. Gregory, CBE, and Dr Henry T. Howat, CBE. Sir Richard Doll was for some years on the Editorial Committee of *Gut*, resigning last year when he was appointed to the Regius Chair of Medicine at Oxford. Professor Gregory is a member of the Editorial Committee and has been a frequent contributor to the Journal. Dr Howat was a founder member of the Editorial Committee of *Gut*, and was President of the British Society of Gastroenterology in 1968-69.

72nd Annual Meeting of the American Gastroenterological Association

The 72nd annual meeting of the American Gastroenterological Association, under the presidency of Dr James A. Clifton, was held at the Americana Hotel, Miami, as part of the Annual Digestive Disease Week which is devoted to postgraduate study and research. It began with a two-and-a-half day postgraduate course organized for the A.G.A. by Dr Gordon McHardy and Dr Nicholas C. Hightower on the esophagus. In addition, there were sessions arranged by the Gastroenterology Research Group, by the American Association for the Study of Liver Disease, and the American Society for Gastrointestinal Endoscopy. The week ended with the official meeting of the American Gastroenterological Association. The splendid Americana Hotel was an ideal setting for the week's activities. Situated on Miami Beach, it provided a ready escape to sun and sea, if the heavy pressure of papers threatened to reduce powers of concentration. There were other rival temptations, with deep sea fishing and the Everglades nature reserve. It is to be noted, and might have even provided an excuse for combining work and pleasure, that the Australia antigen has now reached the shark via the biological chain of sea life affected by sea pollution. No such excuse was available for those visiting the Everglades, except perhaps to maintain the evolutionary perspective in gastroenterology. The meeting was exceedingly well attended, and no less than 800 registered and paid $100 for the postgraduate course on the esophagus. Over 400 abstracts were submitted for the various sessions and, in spite of a number of simultaneous sessions, barely a quarter of these papers were on the programme. All the abstracts submitted were reproduced and will be printed in *Gastroenterology*, and they provide an impressive record of the present vigour of American gastroenterological research, and there seemed to be a trend towards more clinically orientated studies.

It will give much pleasure in many countries both to pathologists and gastroenterologists that the Julius Friedenwald medal for 1971 was awarded to Hans Popper and the citation was delivered by Fenton Schaffner, giving a delightful picture of his life and work. The annual memorial lecture, 'The biological aspect of diarrhoea', was given by Dr Norman Kretchmer who looked back on the lactase-deficiency syndrome and put it in a world-wide geographical perspective in relation to dairy farming, and illustrated by his own studies in Africa. The fourth annual Distinguished Achievement Award lecture was given by Dr John S. Fordtran who gave a brilliant analysis of the forces involved in the intestinal transport of ions, water, and sugars.

The following personal selection of papers may give an impression of current American research activity, but the full list of abstracts should be read by all those wishing to keep fully in touch with American work.

THE ALIMENTARY POLYPEPTIDES

The alimentary polypeptide hormones appeared frequently among the papers and abstracts. J. E. McGuigan and J. H. Landor found the half-life of gastrin, with varying rates of infusion, was 5·9 minutes, with a range from 4·4 to 7·0. G. O. Barbezat and M. I. Grossman have demonstrated the effect of glucagon on water and electrolyte movement in the jejunum and ileum of dogs, and have shown that glucagon suppresses absorption and promotes secretion of electrolytes and water by the small intestinal mucosa and that the composition of the secretion is typical of the level of gut (low bicarbonate, high chloride in jejunum and high bicarbonate and low chloride in ileum). This may have a bearing in relation to some cases of the Zollinger-Ellison syndrome. This effect is augmented by pentagastrin and can give rise to diarrhoea. It was suggested that tumours of patients with pancreatic cholera or WDHA (watery diarrhoea, hypocalcaemia, achlorhydria) syndrome might produce both glucagon and gastrin and that there could be a spectrum of tumours with gastrinonoma at one end producing a true Zollinger-Ellison syndrome, and glucagonoma at the other, with a middle group containing both hormones and giving rise to the severe diarrhoea. F. P. Brooks, A. Ertan, W. Hughes, J. Lindenthal, and A. McGuire measured prolonged gastric *p*H recordings and serum gastrin in normal men. The overnight fasting gastric acid tended to remain stable at a *p*H which would suppress gastrin activity. The normal stimulus to acid secretion, ie, a meal, raises gastric *p*H and serum gastrin levels. L. R. Johnson and A. M. Chandler have shown in rat experiments that the chronic injection or release of gastrin in rats can cause hypertrophy of gastric mucosa, and they have found that gastrin exerts a stimulant effect on RNA synthesis, specific to duodenal and gastric mucosa and independent of gastric acid secretion. In a patient with the Zollinger-Ellison syndrome, J. I. Isenberg, J. H. Walsh and M. I. Grossman reported increase in acid secretion after secretin infusion, contrary to expectation. However, it seems that this effect could be related to changes in serum calcium (the abstract is incorrect on this point). Secretin can stimulate parathormone activity, and an increase in calcium leads to release of gastrin. J. Gutierrez, W. Y. Chey, V. Dinoso, and S. H. Lorber studied the effects of intestinal hormones on motor function of the small intestine and sigmoid colon in man. Secretin inhibited jejunal activity, but CCK markedly increased small intestinal transit time, and also stimulated rectosigmoid motility, and this could be inhibited by secretin. It was noted that the CCK effect on the small intestine was present in patients with mucosal diseases but not with smooth muscle disorders, and this observation could be used diagnsotically. W. M. Yau and J. T. Farrar have compared the octapeptide-CCK activity with the naturally occurring CCK and found it to be twelve times more active in its effect on gallbladder muscle activity in guinea

pigs. The relative siting of the sulphated radicle to the tetrapeptide unit was critical in determining this optimum activity.

THE OESOPHAGUS

Much interest has been developed in relation to LES—lower esophageal sphincter (American spelling!), stimulated by the knowledge that gastrin will cause an increase in pressure in the circular smooth muscle at the lower end of the oesophagus where there is a specialized receptor mechanism. It seems likely that this may play a role in the prevention of reflux, and this was the subject of a number of papers during the postgraduate oesophageal course. The action of gastrin in heightening the pressure at the lower oesophageal sphincter may explain the diminished tendency to oesophageal reflux during night time, when measured by periods of *p*H fall, as reported by J. Marshall Garrett. This mechanism may also operate in patients with achalasia, and Bernard R. Cohen and Moises Guelrud have demonstrated this phenomenon in patients who have achalasia, and indeed giving some partial support for the original name, 'cardiospasm'. A movement is clearly building up towards pressure recordings in many centres, and the necessary transducers and recorders, which are expensive, came under detailed discussion. A strong note of warning was sounded by D. A. W. Edwards (London) who felt that the LES mechanism oversimplified the problem of reflux and felt that the normal clinical steps were usually sufficient in determining diagnosis and treatment, but motility tracings still had an important role at research level. This viewpoint gained further support from Mr R. Belsey (Bristol) and other surgeons who realized that successful repair for hiatus hernia with symptoms of reflux needed the establishment of a short segment of intra-abdominal oesophagus exposed to the positive intraabdominal pressure which maintains apposition of the mucosa in this segment and establishes a flutter valve mechanism.

STOMACH

W. R. Trudeau and J. E. McGuigan made comparisons of serum gastrin following vagotomy and pyloroplasty and vagotomy and antrectomy. There was no reduction in the pyloroplasty group, but a greater than 77% in the antrectomy. The results are consistent with the conclusion that reductions in acid secretion after vagotomy and pyloroplasty do not reflect reductions in the level of serum gastrin, but rather may reflect reductions in the sensitivity of the parietal cell mass to gastrin following vasal transection. Bruce M. Smith, John J. Skillman, Bruce G. Edwards, and William Silen, using a new method which distinguishes between H^+ back-diffusion and H^+ secretion or neutralization, demonstrated that acetylsalicyclic acid and ethanol could both break the gastric mucosal barrier in the adult human stomach. The technique which they used with lithium salts came under some criticism in discussion, but nevertheless the results confirm in man the findings previously made in dogs, and furthermore the observations were further supported by studies on transmural potential differences. S. E. Silvis and A. Docherholmen found in 22 patients given histamine for four hours and secretin during the last two hours that there was a change in gastric *p*H from 1·14 to 3·51 and a volume change between 351 to 584 ml per hour between the infusion of histamine and the infusion of histamine and secretin. This stimulation of alkaline secretion seemed to be particularly related to the presence of antral gastritis. G. M. Makhlouf, in an elegant study, provided direct evidence of isosmotic secretion by the stomach and supported the view that osmotic equilibrium of secretions occurs in a restricted space, probably represented by the apical infolding of the secretory cells and their extension into glandular tubules.

SMALL INTESTINE

A. Robert, J. E. Nezamis, and D. F. Stowe produced duodenal ulcers in rats by continuous acid infusion of the stomach, but these were prevented by giving an anticholinergic drug which inhibits pepsin as well as acid. These studies provided renewed evidence that acid together with pepsin may play a direct aetiological role in the development of duodenal ulcer. M. K. Younoszai, E. Urvan, and H. V. Sthedl have studied the mechanism of intestinal calcium transport. They believe a calcium-binding protein situated on the brush border is important for the transport of calcium from the lumen to the mucosa, and they provide further evidence for a calcium pump at the basal lateral part of the mucosal cell which is stimulated by vitamin D. H. P. Sherr, A. Newman, Y. Sasaki, A. G. Banwell, and T. R. Hendrix reported a simple method of detecting bacterial deconjugation of bile salts by a convenient breath analysis technique, and this was further confirmed by Hans Fromm and Alan F. Hofmann. Taurocholate with ^3H inner nucleus (resistant to bacterial attack) and glycocholate with ^{14}C in the glycine moiety (susceptible to bacterial deconjugation and oxidation of the liberated glycine to $^{14}CO_2$) which could be measured by breath sampling. At the same time the labelled taurocholate could demonstrate the excess faecal excretion with steatorrhoea. The studies showed that this procedure could be most helpful in detecting bile salt malabsorption and bacterial deconjugation and the response to therapy. M. Feldman, B. J. Shapiro, and D. Berkowitz showed that medium-chain triglycerides are more potent inhibitors of gastric motility than long-chain triglycerides, and suggest that their effectiveness in malabsorption may be in part due to their ability to delay gastric emptying and consequent slower passage of the small intestine, thus allowing for more complete absorption. M. E. Ament, S. S. Shimoda, D. R. Saunders, and C. E. Rubin studied the pathogenesis of steatorrhoea in the stasis syndrome. Their observations cast doubt on the current belief that the abnormality in jejunal micelle formation is a main factor, and they found extensive absorptive cell damage by electron microscopy in cells which appeared normal by light microscopy. Mucosal invasion by bacteria was noted in two seriously ill patients. F. Goldstein, R. J. Mandle, C. W. Wirts, and G. J. Dammin studied six patients in a 10-year period in Philadelphia who had chronic diarrhoea and in whom excessive bacterial overgrowth, particularly coliform organisms had been found in the upper small intestine in patients who had not been to parts of the world where tropical sprue is endemic. Two such patients in fact had major diagnostic features of tropical sprue, but the others had normal small bowel mucosal biopsies. It was suggested that such bacterial activity in the small intestine, which is a common cause of chronic diarrhoea in many developing tropical countries, can also occur in temperate climates. The absorption of neomycin has been studied by K. J. Breen, R. E. Bryant, J. D. Levinson, and S. Schenker who have shown that gastroduodenal ulceration or regional enteritis does not enhance absorption and does not increase the risk of toxicity.

COLON

There was a clinical panel with Howard M. Spiro as moderator on ischaemic colitis. This can be due to arterial or venous occlusion, or due to a mucosal perfusion defect possibly associated with vasculitis or toxic absorption. It was a diagnosis which had to be kept in mind, particularly in patients who had unexplained abdominal pain, diarrhoea, and a reason for ischaemia. There was support from some members of the panel for immediate arteriography with the idea of urgent arterial embolectomy, which could sometimes avert the need for resection, if done within 30 hours, but it was appreciated that many patients would not require surgery. A plain radiograph could be helpful, and a barium enema could clinch the diagnosis. Some patients had a local mucosal perfusion insufficiency with a vasculitis, with major blood vessels still being patent. A high epidural block to reduce mesenteric activity was one recommendation put forward. Digitalis is to be avoided as it can itself cause mesenteric constriction.

PANCREAS

Eugene D. Jacobson, Stanley J. Konturek, and Leonard R. Johnson believe they have found a link between cigarette smoking and the aggravation of duodenal ulcer and the pancreas. They have shown that nicotine in dogs decreases markedly the hepatic and pancreatic secretion of fluids and bicarbonate into the duodenum. Nicotine acts to depress the pancreatic secretion at two sites, namely, the pacreas itself and the duodenal mucosa, preventing the endogenous release of secretin. Thus, duodenal ulcer formation could be facilitated by the nicotine interfering with the mechanism required for the neutralization of gastric acid within the duodenum. It seems that nicotine is a competitive inhibitor of secretin. A. Ertan, F. P. Brooks, J. D. Ostrow, D. Arvan, C. N. Williams, and J. J. Cerda have studied the release of CCK and secretin from the proximal jejunum and the effect of topical anaesthetic in man. They used a perfusion of essential amino acids in the duodenum to release CCK and secretin, and have shown that this is as effective a stimulant as the CCK. There was also a persistent elevation of the total bile salts during the second hour of the EAA perfusion, but not with the CCK infusion. There was a significant reudction of the response to EAA by jejunal application of topical anaesthetic, which suggested a local neural release mechanism for the endogenous release of CCK. In the discussion, Grossman doubted whether it was necessary to postulate release of secretin to explain the results shown. It certainly seems that EAA may have a role in the regulation of pancreatic and biliary secretions. Moises Guelrud, J. Rudick, and H. D. Janowitz have studied endogenous cyclic AMP and pancreatic enzyme secretion. They suggest that cyclic AMP, which has been implicated as an intracellular mediator of a variety of hormonal actions, was also concerned with pancreatic secretion. They note that theophylline, which increases endogenous cyclic AMP, mimics the action of pancreozymin. There was a sharp and prolonged increase in an enzyme concentration and output lasting more than 60 minutes. The possibility of this being due to glucagon release was raised in discussion.

BILIARY SYSTEM

W. Y. Chey, S. Kosay, H. Siplet, and S. H. Lorber have shown that the dog is a suitable experimental model for demonstrating the effect of alcohol on the liver. They have demonstrated marked hepatocellular changes comparable with those found in man. These include fatty metamorphosis of mild to severe degree, swelling and hydrophic changes of the hepatocytes, necrosis, eosinophilic hyaline bodies, and after 18 months there was severe distortion of the hepatic lobular architecture, proliferation of connective tissue with compression of the space of Disse, Similar studies in primates (baboons) made by C. S. Leiber, E. Rubin, L. M. DeCarli, H. Gang, and G. Walker have shown triglyceride accumulation and alterations in mitochondria and endoplasmic reticulum, strikingly similar to those found in man, and not prevented by a high-protein diet and not potentiated by a 65% decrease in dietary protein. L. Swell, C. C. Bell Jr, and Z. R. Vlahcevic have studied the relationship of bile acid pool size to the formation of lithogenic bile in man. They have found a greatly decreased (1·2 g) bile salt pool and phospholipid excretion which follows as a direct consequence of the reduced bile salt pool, with only slight reduction of the cholesterol excretion. There is a relative decrease in bile salt and phospholipid to cholesterol ratio, and a bile saturated with cholesterol results. The break-point in bile salt pool size in males between lithogenic and non-lithogenic bile was estimated to be between 1·6 and 1·8 g. The incidence of lithogenic bile in siblings of young patients with cholelithiasis was reported by R. G. Danzinger, H. Gordon, L. J. Schoenfield, and J. L. Thistle. The first stage of the pathogenesis of cholesterol gallstones involves the formation of abnormal or lithogenic bile saturated with cholesterol. This state of pre-stone gallstone disease is characterized by low bile acid plus lecithin to cholesterol ratio in bile and has been previously demonstrated in young American Indian women who have a high incidence of gallstone disease. In this study the female siblings of young Caucasian women who had been operated on for cholelithiasis they demonstrated that the older siblings in the third decade of life had a greater proportion of lithogenic bile than the controls and thus constitute a high risk group for cholelithiasis. The bile was obtained by duodenal drainage after gallbladder stimulation by intravenous administration of cholecystokinin. The effect on hepatic bile flow of jejunal perfusion with various micellar fat solutions was reported by William Jabbour, Lidija Trencis, and William Silen who showed that a micellar solution of a long-chain fatty acid (oleic acid) caused a marked increase in the flow of hepatic bile, an effect not observed with its triglyceride or with octanoic acid or trioctanoin. The release of cholecystokinin was postulated, but other factors may be operative. William King, who was awarded the A.G.A. student research prize, gave his paper on rapid biliary secretion of intestinal ^{14}C-Lecithin. The feeding of lecithin increases the phospholipid-cholesterol ratio and cholesterol-holding capacity of human hepatic bile. His data showed that labelled lecithin is secreted into the common bile duct shortly after its intestinal administration and increased steadily over the study period. By eight and a half hours, 70·3% of the radioactivity in the common duct bile was in lecithin. It was suggested that further nutritional studies of lecithin metabolism may provide methods of enhancing the cholesterol-holding capacity of human bile and ameliorating cholesterol cholelithiasis. E. Alpert, P. H. Schur, and K. J. Isselbacher presented their observations on arthritis associated with hepatitis, a study which had started on the basis of Dr Alpert's own personal experience, and subsequently a series of patients with infective hepatitis with similar prodromal syndrome were studied.

These patients all had arthralgias of the distal joints, particularly of the proximal interpharyngeal joints, with morning stiffness, and this was associated with urticarial reactions and fever with jaundice appearing a few days later. All the eight patients studied had a circulating hepatitis antigen (HAA). The serum complement was studied and the total complement at CH_{50} and one or more of its early components, C4, C3, were depressed during the phase, returning to normal later, and this finding was not observed in other patients with infective hepatitis without these symptoms or in association with jaundice from other causes. The syndrome was analogous to a serum sickness immuno-complex disease with an excess of circulating antigen and due to activation of the complement system by circulating immune complexes, presumably the hepatitis virus and homologous antibody. T. B. Reynolds, S. Yamada, and R. L. Peters had previously reported acute hepatitis following the use of a laxative containing oxyphenisatin (*J. Amer. Med. Ass.*, **211**, 86, 1970) and have since seen seven additional cases with more severe and prolonged hepatic damage associated with chronic ingestion of this aperient. Chronic active hepatitis was found without associated HAA, but smooth muscle antibodies were present in four out of six. Two had positive LE cells. All improved markedly after changing the laxative and one patient challenged with oxyphenisatin showed an immediate rise in serum bilirubin and transaminase. A toxic origin is therefore to be considered in all cases of chronic active and lupoid hepatitis. N. D. Grace and M. S. Greenberg reported a controlled trial of phlebotomy in the treatment of iron overload in patients with primary alcoholic cirrhosis with increased deposits of iron, but they failed to demonstrate any benefit in clinical status or liver function.

The session on hepatitis associated antigen (HAA) was introduced by Baruch S. Blumberg and, together with Lewellys F. Barker and Joseph L. Melnick, a full review of the present role of HAA was presented. Confirmation of the finding of small amounts of RNA but not DNA gave support to its identity as a virus, but the possibility was put forward that we are dealing with another type of infective agent, different from true viruses, the atypical immunity reactions giving further support for this possibility. The discussion brought out some interesting medico-legal situations in relation to food handlers and dentists. The clinical session on chronic active hepatitis (moderator Fred Kern, Jr) underlined the distinction between chronic active and persistent hepatitis, with a need to treat the former with prednisolone alone or combined with azathioprine, but azathioprine by itself had proved ineffective. The clinical symposium on chronic liver disease in children, moderated by Fenton Schaffner, dealt with the rare disorders of carbohydrate, lipid, and amino acid metabolism. The gastroenterological research group had arranged a special lecture by John M. Dietschy on the mechanism of bile, acid and fat absorption. In his brilliantly presented, mathematical analysis, Dr Dietschy described the forces operating at the lipoid absorption surface and in relation to the absorption of fatty acids and micelles, and introduced a concept and complications of the 'unstirred water layer of some 200 microns thickness.' However, the final path via micelles into the cell cytoplasm is still very imperfectly understood and nothing further has emerged concerning the passage of fatty acids within the cytosol and its re-acidification and the formation of chylomicrons.

F.A.J.

Notes on books

Immunology of the Liver By Martin Smith and Roger Williams. (Pp. xiv + 314 William Heinemann Medical Books, 1971. £5·30).
This book records the proceedings of an International Meeting held at King's College Hospital Medical School in July 1970. The role of autoimmunity in active chronic hepatitis and primary biliary cirrhosis has become better defined and current experience in both experimental and therapeutic fields is recorded in this admirable publication.

The Small Intestine in Diabetes Mellitus (With special reference to Electrical and Motor Activity, Absorption and Pancreatic Function). By Vagn Mohr Drewes. (190 pages + 9-page Appendix. Munksgaard, Copenhagen. 1971. Dan. Kr. 90.00). This monograph records studies on small intestinal motility and pancreatic function in relation to diabetes with special reference to the problem of diarrhoea in diabetes. Changes in pancreatic exocrine function are demonstrated and there are also motility disturbances in the small intestine.

Human Albumin Metabolism Determined with Radioiodinated Albumin By Niels Rossing. (108 pages, Munksgaard, Copenhagen. Dan. Kr. 60.00). This monograph describes a study on normal and abnormal albumin metabolism in man, with special reference to the mechanisms of hypoalbuminaemia.

Macroglobulins Provoking Haemagglutination in Rheumatoid Arthritis and Other Diseases. By Nanna Svartz. (105 pages, Almqvist and Wiksell, Stockholm, 1971. No price given). Macroglobulins have perhaps only limited associations with alimentary diseases but gastroenterologists may wish to be aware of these research studies by Professor Nanna Svartz so well known for her fundamental work on ulcerative colitis and the development of sulphasalizane.

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