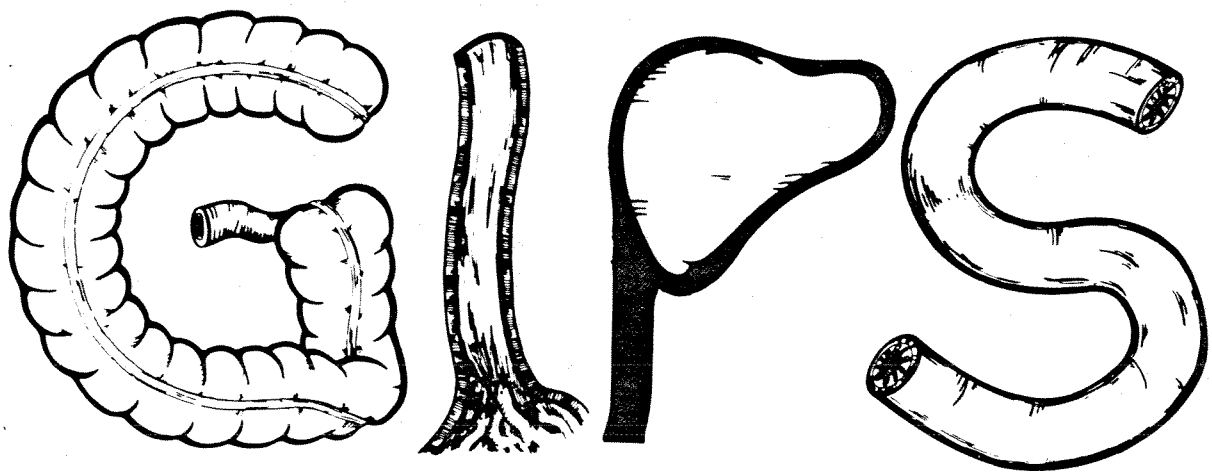

THE GASTROINTESTINAL PATHOLOGY SOCIETY
NEWSLETTER

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GASTROINTESTINAL PATHOLOGY SOCIETY
1988-89 OFFICERS AND COMMITTEE MEMBERS

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Vice-President/President Elect: (1-year term)	
G. Abrams	1989
Secretary-Treasurer: (3-year term)	
S. Hamilton	1990
Education Committee: (3-year term)	
D. Sheahan (Chairman)	1991
R. Pascal	1991
S. Sternberg	1990
J. Wirman	1990
R. Haggitt	1989
F. Mitros	1989
Membership/Nomination Committee: (3-year term)	
A. Qizilbash (Chairman)	1991
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Training Programs Committee: (3-year term)	
K. Barwick	1991
E. Lee	1991
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Publications Committee: (Standing)	
H. Appelman (Chairman)	
R. Riddell	
S. Sternberg (Ex-officio: Editor of Amer J Surg Path)	
R. Rickert (Ex-officio: President of GIPS)	
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Microgrants Committee: (Standing)	
J. Yardley (Chairman)	
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Newsletter Editors: (3-year term)	
D. Keren	1991
W. Dobbins (Associate Editor)	1991
International Liaison: (Standing)	
H. Goldman	

New appointments in **boldface characters**

President's Message

I would first like to thank our outgoing president, Juan Lechago, for a fine year at the helm of the Society. Special thanks must also go to Frank Mitros for his excellent choreography of the Scientific Session in Washington on the Pancreas and Pancreaticobiliary Tree and the session on Gastritis held at the AGA Meeting in New Orleans; to Leonard Kahn for overseeing the application process during the past several years; and to Dave Owen and Juan Lechago, our recently retired Newsletter co-editors, for consistently providing the membership with one of the great literary journals of the English language.

The new line-up of Committee chairpersons and members appears elsewhere in the Newsletter and is testimony to our continued efforts to bring a broad spectrum of the membership into the affairs of the Society. If any of you have interest in serving on GIPS committees in the future, please notify me or our secretary, Stan Hamilton.

A brief look at the horizon for the coming year reveals a number of interesting and important items. For several years we have been concerned about our participation with the AGA during Digestive Disease Week. We believe that the programs given by the GIPS have been of uniformly high quality, and responses from those who have attended seem to support that observation. However, attendance has been variable and publicity about the programs has been generally lacking. We believe that these problems are due mainly to lack of a properly organized communication network between our two organizations. Therefore, we have initiated discussions with AGA in an effort to provide a more systematic approach to the administration of these high quality educational programs.

During the tenure of Juan Lechago we were approached by the American Society of Clinical Pathologists regarding the possibility of having a GIPS educational program held in conjunction with an ASCP meeting. Very preliminary discussions have been initiated to further evaluate the possibility of such a "companion" meeting. I will keep the membership informed as more information becomes available.

Finally, Henry Appelman, our Publications Committee Chairman has been working with the American Journal of Surgical Pathology to resolve the problems concerning the distribution of the supplements. As you are probably aware, the papers which come from the presentations at our Scientific Session are published in a supplement to the American Journal of Surgical Pathology. These have not been available as part of the journal subscription but are marketed separately. Henry has been working with the Journal to resolve this problem.

If any of you have additional items which you feel should be addressed by the Society, please let me, the other officers or committee chairpersons know. Best wishes for a pleasant summer.

Robert R. Rickert, M.D.
President, Gastrointestinal
Pathology Society

EDITORIAL

Where are the letters to the editor?

Editing the Gastrointestinal Pathology Society Newsletter is a rather lonely task. We gather some basic information about the GIPS including its officers and committees, review the abstracts from the previous meeting and wait . . . wait . . . wait . . . for lively and timely topics which our esteemed members may wish to communicate.

I guess that both of our esteemed members are busy, because the GIPS mailbox is about as empty as a well prepped colon. We encourage you to submit your letters to us on a variety of subjects including great science (GS), bad science (BS?) or heavy political issues (should we change the abbreviation GIPS to the more correct GPS?). If you don't, you will be forced to waste your time reading another editorial like this for which, after all, you are paying!

Please submit your contributions to the address below for consideration by the Gastrointestinal Pathology Society Newsletter.

David F. Keren
Department of Pathology
1500 E. Medical Center Drive
Ann Arbor, MI 48109-0602

Gastrointestinal Pathology Society
USCAP Gastrointestinal Pathology Review

A plethora of gastrointestinal pathology was available at this year's USCAP meeting. In addition to our usual Gastrointestinal Pathology Society meeting and the evening case session, the long course was entirely devoted to gastrointestinal pathology this year. With all of this information, some of you may have overlooked the intriguing papers and poster presentations that were available on GI pathology this year.

Dr. Allaire from the AFIP documented that bile duct adenomas are distinct benign, non-cystic structures which have an immunophenotype of CEA +, EMA +, keratin +, alpha-fetoprotein -, and Leu7 -.

Drs. Bona, et al. from the Cleveland Clinic Foundation provided a detailed description of the inflammation in ileostomy pouches (pouchitis). Key features useful in identifying pouchitis included decreased intracellular mucin, increased epithelial cell regeneration, and patchy intraepithelial and lamina propria acute inflammation. Interestingly, decreased or absent lymphoid follicles in the face of increased chronic inflammation were also seen.

Drs. Burk and Helwig provided a large series demonstrating that gangliocytic paragangliomas of the small intestine have unique immunohistochemical profiles reflecting their differentiation along neural, endocrine and epithelial lines. Greater than 90% of the neoplasms studied stained positively with antibody to neuron-specific enolase, S100 protein and pancreatic polypeptide. Most of the neoplasms also stained with antibodies to chromogranin, somatostatin, and keratin.

An experimental model examining the association of reflux esophagitis with the development of adenocarcinoma of the distal esophagus was described by Dr. Cardesa, et al. from the University of Barcelona, Spain. They found that the presence of surgically induced reflux esophagitis was highly correlated with development of adenocarcinoma in animals given the carcinogen 2-6-dimethylnitrosomorpholine.

Dr. Davessar, et al. from Brown University examined the correlation between the Ming and Lauren classifications of gastric adenocarcinoma. Both calssifications were able to predict survival and, in both, the degree of inflammation was a valuable finding. Interestingly, they found that the majority of cases were example type (Ming) and intestinal type (Lauren). These types can be detected early and have a better survival rate than diffuse or infiltrating types,

Drs. Dayal and Underwood present an immunohistochemical study which determined that endocrine cell hyperplasia is not a requisite precursor for development of duodenal somatostatinomas.

Drs. De Schryver-Kecskemeti and Alpers describe the intracytosolic location of intestinal alkaline phosphatase. They note that intracellular intestinal alkaline phosphatase is associated with fat droplets in all stages of lipid transport through the enterocyte. These findings may explain elevation in serum intestinal alkaline phosphatase following

ingestion of fat. Drs. De Schryver-Kecskemeti, et al. also presented a study on the response of chronic hepatitis B to treatment. By using an objective hepatitis activity index, they were able to predict potential responders to therapy in chronic hepatitis B infection.

Drs. Dudley and Dean from the Baptist Memorial Hospital pointed out that the presence of granulomas in the appendix may be distinct from Crohn's disease. Appendices with granulomatous appendicitis had an average of 13.9 granulomas per tissue section, while those from patients with Crohn's disease had only 0.29 granulomas per tissue section.

The use of plastic-embedded endoscopic biopsies was encouraged by Drs. Farrell and Beckstead from the University of California, San Francisco. By comparing two micron plastic-embedded tissues with their routine paraffin-embedded counterparts, about 25% of the cases rendered more specific diagnoses with the plastic sections than with the paraffin sections.

Drs. Fitzgibbons, et al. from the University of Southern California Medical Center found a high prevalence of C. Pylori-associated gastritis in biopsies from an asymtomatic population of volunteers. Almost half of their subjects had histologic evidence of gastritis and almost all of these individuals had C. Pylori. Inflammation in the duodenum was not accompanied by C. Pylori.

Drs. Foust, et al. from the Baptist Memorial Hospital in Memphis studied the histologic features of 19 patients with dysplasia or carcinoma arising in hemorrhoidal tissue. The vast majority were classified as severe dysplasia/carcinoma in situ and usually contained messenger RNA for human papilloma virus type 16 or type 6. These incidentally discovered dysplasias or carcinomas in situ were found to be biologically nonaggressive which were cured in most cases by hemorrhoidectomy.

Fraire, et al. from Baylor College of Medicine performed an autopsy study in which they examined the relationship between obesity and gallstones. By dividing the subjects into a group of normal weight and mildly obese (editorial note-presumably not enough normal weight people could be found for a group) and a group of moderately to severely obese individuals, no statistically significant difference between the two groups could be found with regard to the frequency of gallstones.

Dr. Gottfried, et al. from Duke University examined the relationship between binding of lectins as markers of intestinal metaplasia and dysplasia in columnar lined esophagus. Both well-differentiated adenocarcinoma arising in columnar lined esophagus and dysplasia showed binding of Eulex europeous at the apex and glycocalyx of tumor cells. There was, however, reduced cytoplasmic staining in all cases studied. Such alterations of mucosal glycoconjugates may be useful markers of intestinal metaplasia and dysplasia in the esophagus.

From Henry Ford Hospital, Gottlieb and Maeda report eight cases of primary rectal lymphoma. Interestingly, two of these were from patients with AIDS.

Drs. Gourley and Swedo from the University of Texas report another protozoa which propagates in patients with AIDS. This is Enterocytozoon bieneusi. It was found in both the small and large bowel epithelium in 10% of AIDS patients with chronic diarrhea.

Drs. Ishikura, et al. from Japan presented the intriguing suggestion that mycoplasma-like organisms observed in inflammatory fibroid polyp of the stomach may be an etiologic factor of this disease.

Drs. Jones, et al. from Orlando Regional Medical Center and the University of Alabama review three cases of a new malignant entity-intestinal plexosarcoma. The lesions occur as submucosal mural masses in the small bowel or stomach. Metastases occurred in all seven individuals. The histology is variable within the same lesion and includes large epitheliod cells (scattered or in clusters) and a fascicular spindle cell component, mimicking both endocrine and sarcomatous neoplasms. Fewer than four mitoses per high powered field were seen. These neoplasms usually stain for neuron-specific enolase and neurofilament protein. S100 staining is also occasionally seen.

Drs. Jothy, et al. from McGill University found that carcino-embryonic antigen epitopes identified by the D14 monoclonal antibody are present in all types of colonic neoplasia. However, other epitopes, such as those staining with the B7.8.5 monoclonal antibody vary in relation to the type of colonic polyp, being more prevalent in villous adenomas then in tubular adenomas. Heterogeneity of expression of carcinoembryonic antigen epitopes may be useful histopathologic features of colonic neoplasms.

Drs. Kern, et al. from the Johns Hopkins Medical Institutions demonstrated that ras gene mutations occur in many colorectal tumors and may play a role in neoplastic progression.

From Case Western Reserve, Drs. Kilbridge, et al. report that Campylobacter pylori was almost never seen in the absence of gastritis in children. When compared to adults, children with Campylobacter pylori related gastritis have more chronic and less acute inflammation.

Drs. Kuhajda, et al. from the Johns Hopkins Hospital compared the immunoperoxidase technique using a monoclonal antibody against cyto-megalo-virus early antigen with DNA in situ hybridization using a biotinylated probe from patients with known CMV colitis and 20 patients with ulcerative colitis. Interestingly, immunoperoxidase was found to be more useful than in situ hybridization as the former would stain cells without recognizable nuclear inclusions.

A long term study of liver biopsies after jejunal bypass was described by Drs. Lee, et al. from Washington University Medical School. An average of eight years after the surgery, liver biopsies showed fatty change in 33% of cases with fibrosis and 42%. While progression in pathologic lesions occurred in 33% of patients mainly due to fibrosis, none developed cirrhosis. Drs. Lee, et al. also describe a mucosal thickening adjacent to gastric malignancy and its possible relationship to epidermal growth factor. By calculating the mean width of the mucosa

adjacent to gastric carcinomas, lymphomas, and benign ulcers, they found that overall there was no difference in thickness. However, a subset of carcinomas showed marked thickening. Epidermal growth factor was found by immunohistochemistry especially strongly in this subset. It was also present, however, in all carcinoma cases.

From M.D. Anderson Hospital and Tumor Institute, Drs. Lynch and Cleary report 20 cases of non-ampullary adenocarcinoma of the duodenum. Only two of these patients presented with jaundice. The three patients that survived at least four years had no evidence of metastasis at time of presentation.

From Henry Hospital and William Beaumont Hospital Drs. Ma, et al. report three cases of gastric antral vascular ectasia. Characteristic features include longitudinal hypertrophic mucosal folds in the antrum, villiform hyperplasia of the surface epithelium, fibromuscular hyperplasia of the lamina propria and dilated capillaries in the mucosa which may contain thrombi.

Drs. Manivel, et al. from the University of Minnesota used an immunohistochemical technique to examine the role of isolated cytokeratin positive liver cells in hepatocellular and ductal regeneration. Primary hepatocellular regeneration was characterized by numerous cytokeratin positive single cells in regenerative nodules, ductular regeneration following obstructive disease was characterized mainly by numerous cytokeratin positive cells and ductular proliferation while bile duct destruction was characterized by no or rare cytokeratin positive cells.

Drs. Markin, et al. from the University of Nebraska compared clinical suspicion of rejection with histologic diagnosis of post-transplant liver biopsies from 239 patients. 65% of the clinical diagnoses were confirmed histologically as rejection. When CMV was suspected clinically, it was always identified in the biopsy, however, a few unsuspected cases of CMV were also recovered in the biopsy material. Dr. Markin, et al. also report autopsy findings from eleven patients with liver transplants. Six of the eleven cases had histologic evidence of graft rejection and massive necrosis was present in two cases. Three of the eleven had CMV hepatitis.

Drs. McQuillan and Appelman from the University of Michigan raised the specter of superficial Crohn's disease in a study of eight patients. They found Crohn's disease limited to the mucosa and submucosa. They caution that bowel wall in these patients was thin and pliable rather than thick and rigid. They indicate that Crohn's disease need not always be a transmural process.

From Evanston Hospital, Dr. Meiselman reports the occurrence of an invasive Campylobacter pylori in a patient with AIDS. The typical bacteria were seen on a Warthin Starry stain to be in the surface epithelial mucus and passing from the superficial mucosa between epithelial cells into the lamina propria. Although the clinical symptoms were successfully treated with both bismuth salicylate and amoxicillin, Campylobacter still persisted in gastric pits.

From the University of Helsinki, Dr. Miettinen studied 45 benign gastrointestinal stromal tumors using immunohistochemistry. The tumors which resembled typical leiomyomas were consistently positive with antibody to desmin and to muscle actin. In contrast, most of the cellular spindle cell tumors and most round cell tumors were negative for these antibodies. We lack immunohistochemical reagents which will reliably distinguish benign gastrointestinal stromal tumors at the present time.

Drs. Murad, et al. from Northwestern University Medical School looked at the DNA content of adenomatous colonic polyps by flow cytometry. They report a surprising lack of correlation between aneuploidy, proliferative index and the degree of atypia! While their study used paraffin-embedded specimens as the source, this has been a reliable source of tissue for other studies. The size of the polyp alone did not appear to be a determinant factor for DNA aneuploidy.

From the University of Minnesota, Drs. Nakhleh, et al. reported an increased expression of class I major histocompatability antigens on gastric and duodenal epithelium in association with acute graft-versus-host disease. By using immunofluorescence techniques, they graded the intensity of staining. All 16 graft-versus-host disease positive tissues had increased expression of HLA-ABC. They predict that this increased expression of MHC antigens would increase the immunogenicity of these tissues.

Drs. Northway, et al. from the Bowmen Gray School of Medicine presented an animal model of radiation proctitis. These changes progressed from mucosal edema with basal infiltrates of eosinophils to increased numbers of eosinophils with cryptitis and swelling of epithelial cells. Eventually, crypt abscesses evolved into crypt ghosts. Treatment with non-steroidal anti-inflammatory agents had no effect on this process.

Four cases of multiple lymphomatous polyposes were reported by O'Briain, et al. from Trinity College. Multiple polyps involving long segments from the stomach to the rectum were present in each case. All cases were B-cell lymphomas which were positive for B1, Ia, IgMD, with light chain restriction. They suggest that this entity is closely related to mantle zone lymphoma.

From the Mallory Institute and Memorial Sloan-Kettering Cancer Center, O'Brien, et al. report the results of the national polyp study with regard to the relevance of anatomic location to the evolution of colorectal adenomata. Their findings indicate that when the variables of size and villus component are controlled, the occurrence on the lesion on the left side does not increase the frequency of significant dysplasia.

Drs. Ohta, et al. from the University of Ottawa report an animal model to assess changes in cytokeratin expression in griseofulvin-treated mouse liver. The griseofulvin treatment results in hyperplastic liver nodules. Their results indicated that Mallory body containing hepatocytes are phenotypically altered and that the Mallory bodies contain high molecular weight cytokeratins which are not normally found in hepatocytes.

Campylobacter pylori in gastric biopsies were studied by Drs. Pinto, et al. at the Bridgeport Hospital. Of 48 patients with histologically proven antral chronic active gastritis, 34 had Campylobacter detected by the Warthin-Starry stain. Culture was positive in less than half of the patients. They did not detect Campylobacter pylori in histologic sections of normal stomach or in biopsies with carcinoma of the stomach or esophagus.

Drs. Radio, et al. from the University of Nebraska report on recurrence of hepatitis complicating liver transplantation. They used a combination of histologic, immunoperoxidase, and serologic findings to distinguish recurrent hepatitis from viral infections or allograft rejection. Eleven patients were transplanted for chronic liver disease due to infectious hepatitis. Two of five patients transplanted for chronic active hepatitis B experienced recurrent hepatitis as indicated by serum HBsAg, HBcAb, and immunoperoxidase staining. Both died of infectious etiologies with evidence of hepatitis B infection in their graft.

Drs. Ray, et al. from the University of Cincinnati advanced their previous studies showing that vitamin A deficiency in the serum is associated with aggregation of cytokeratin filaments (Mallory body formation) in both hepatocytes and bile ducts. In this study, they find that zinc deficiency in combination with low vitamin A may enhance the aggregation of cytokeratin in bile ducts. The zinc deficiency seems to predominantly affect biliary structures rather than hepatocytes.

Drs. Roberts, et al. from Ohio State University used immunohistochemistry to establish the involvement of endothelial cells by cytomegalovirus in the gastrointestinal tract. Their patients included transplant recipients, AIDS patients, patients with ulcerative colitis and toxic megacolon. They found that cells positive for both CMV and factor 8 (endothelium) ranged from 40-60% of positive cells. There was, in general, clustering of CMV positive cells around vessels. They find CMV infection of the GI tract to be primarily a vasculitis. Clearly, however, the CMV infection is not limited to endothelium.

From Harper Hospital in Detroit, Drs. Sakr and Weaver used an immunohistochemical technique to determine the distribution of basement membrane in colonic neoplasia. While the basement membrane around glands of tubular adenomas was continuous, the distribution in invasive adenocarcinoma was variable. Often, neoplasms infiltrating as single cells had thin patchy basement membrane material. The basement membrane distribution in invasive tumors correlated with the degree of glandular expression.

Drs. Smyrk, et al. from Creighton University reported on the occurrence of adenomas in hereditary non-polyposis colorectal cancer. This autosomal dominant inherited disorder has a predilection for developing early onset right-sided colon cancers. Screening colonoscopy on 34 such patients detected at least one adenoma in twelve patients, nine patients had multiple adenomas. Four patients had adenomas with neoplastic glands concentrated at the luminal surface. These have been termed flat adenomas. The flat adenoma with its right-sided distribution may identify patient's at high risk for colon cancer in this disorder.

Drs. Soohoo, et al. from the University of California Davis reported that acetylcholinesterase staining of rectal mucosa may be useful in the diagnosis of Hirschsprung's disease. In a series of 41 patients studied, no false negatives were reported. Twelve of fifteen with Hirschsprung's disease were strongly positive, two were weakly positive and a five day old infant had a characteristic staining pattern for his age. They conclude that acetylcholinesterase staining of rectal mucosal biopsies is diagnostic in most cases of Hirschsprung's disease.

From the University of Chicago, Drs. Stephens, et al. looked at the DNA content of squamous cell carcinoma of the esophagus and compared it with the histopathologic and clinical features. Using a computerized image analyzer system, they found that nuclear ploidy did not correlate with transmucosal esophageal penetration nor with survival. However, tumors with nuclear areas greater than 70^2 μ were associated with both.

Drs. Talbert, et al. from Massachusetts General Hospital reported on the prognostic features of malignant stromal tumors of the gastrointestinal tract. Of the 53 malignant stromal tumors studied, mitotic count was the feature with greatest predictive value and was used as the sole determinant for tumor grade. Tumors with less than ten mitosis per 50 high power fields were defined as low grade, greater than 10 mitoses per 50 high power fields were high grade.

The nuclear DNA content of the liver in fibrolamellar carcinomas was reported by Drs. Taylor, et al. from the Children's Hospital of Pittsburgh. They found DNA content abnormalities in 15 of 17 cases. However, no specific relationship existed between the presence of these abnormalities and tumor metastases or fatal outcome of the disease.

Drs. Uri, et al. from Children's Hospital of Philadelphia describe a new subtype of hepatocellular malignancy in children which could be distinguished from hepatoblastoma and hepatocellular carcinoma. These children were younger than one year of age, had an undifferentiated neoplasm by light microscopy with definite but scant epithelial features by ultrastructure (prominent intermediate filaments). Further, the neoplasms have vimentin positive intermediate filaments and lack serum and tumor alphaphetoprotein. They are resistant to chemotherapy and have a poor prognosis.

Another study on DNA content of gastric cancers was performed by Urbanski, et al. from the University of Calgary. They found that all early gastric cancers have close to diploid contents of DNA. Five cases of advanced gastric cancer which were also studied showed no significant differences in DNA profile when compared with early gastric cancer.

Drs. Wange, et al. from Case Western Reserve looked at the DNA content in an experimental rat model of hepatocarcinogenesis. They specifically looked at the enzyme-alterted foci by using Fuelgen-gamma glutamyl transpeptidase stain. No enzyme-altered foci were seen in control animals. Aneuploidy was present in four of twelve enzyme-altered foci from animals treated with a choline deficient phenobarbitol diet. They do not believe that all these foci go on to develop into hepatomas.

Drs. Witzleben, et al. from Children's Hospital and Bryn Mawr Hospital report the findings from five pediatric patients with sclerosing cholangitis. Three of the patients ultimately had biopsy proven inflammatory bowel disease. While all had portal tract fibrosis, bile duct proliferation and inflammation, only one of 14 biopsies had cholestases.

The distribution of Kupffer cells in hepatocellular neoplasms were studied by Drs. Yaldandi and Rao from Northwestern University. Kupffer cells were absent or markedly diminished in hepatocellular carcinoma while the numbers of Kupffer cells in hepatoadenomas were comparable to those in adjacent tissue. These findings are similar to those reported in experimental animals.

Drs. Zhoi, et al. from the Bronx Veterans Administration Medical Center reported an animal model for production of hepatitis B virus. They transfected human hepatoblastoma G2 cells with cloned HBV-DNA. When these cells were injected into nude mice, tumors expressing hepatitis B antigens were found within 2-7 weeks. This model may be useful to study the pathobiology of hepatitis B virus, its relation to oncogene expression and the effects of drugs on the expression of hepatitis B viral markers.

The Society for Pediatric Pathology also had several papers related to the Gastrointestinal Pathology Society. Drs. Ballance, et al. from Case Western Reserve University reported on reparative and chronic changes in acute neonatal necrotizing enterocolitis. All their patients had coagulative or hemorrhagic necrosis and nearly all had inflammation. The vast majority also had reparative or chronic changes. They believe that the high prevalence of reparative and chronic changes in "acute" necrotizing enterocolitis suggests that is it an evolving process which must have been present for several days before clinical symptoms appear.

Drs. Lund and Ruebener reported on the development of the human hepatocytic-ductal plate using immunohistochemistry. By reviewing the livers of nine fetuses ranging in age 7-20 weeks, they present evidence supporting the hypothesis that there is a transformation of periportal hepatocytes into ductular cells.

The rare microvillus inclusion disease was studied by Cutz, et al. from the Hospital for Sick Children in Toronto. Ultrastructural and immunohistochemical findings in rectal mucosa from this child reveal focal disorganization of surface microvilli, intracytoplasmic microvillus inclusions and positive but weak staining for actin and villin in intestinal brush border. Similar defects were seen in kidney tubule microvilli.

Drs. Hoss, et al. from the Children's Cancer Study Group in Pasadena reported on the relationship of cytohistopathology to the outcome of malignant epithelial hepatic tumors in children. Of patients with hepatocellular carcinoma, those with fibrolamellar carcinoma demonstrated superior survival to the typical hepatocelluar carcinoma. In hepatoblastoma, pure fetal histology had a better survival compared to other histologic patterns. Osteoid, chondroid, or squamous epithelial elements were associated with improved prognosis in advanced stage hepatoblastoma.

Not surprisingly, mitotic activity was associated with poorer prognosis in both hepatoblastoma and hepatocellular carcinoma.

Drs. Galvis, et al. from Childrens Hospital of Los Angeles micro-dissected flat-mount preparation of the myenteric plexus of esophagus, small intestine, and colon from eight patients with bronchopulmonary dysplasia, gastroesophageal reflux who had had Nissin fundoplication. While the relative mass of the myenteric plexus was normal for the esophagus it was significantly reduced for the small intestine and colon. It is possible that reduced flow of feeding through the intestines retarded the growth of the myenteric plexus in the small intestine and the colon.

Drs. Kennedy and Triche from the National Cancer Institute report that occult hepatobiliary damage in patients with cystic fibrosis is more severe than is generally appreciated. The biliary fibrosis is progressive with increasing age leading to greater clinical relevance with the prolonged survival that patients with cystic fibrosis can now expect. Older patients had more severe and extensive biliary fibrosis and cirrhosis (50%) then had been reported previously in younger patients.

Drs. Mroczek, et al. from the University of Pittsburgh Childrens Hospital report that there is a humoral component to some cases of early allograft liver loss. While vascular thrombosis was present in 80% of these patients with early liver loss, ABO mismatching was documented in only ten patients (20%). Humoral factors may underline a minority of instances of hepatic vascular thrombosis after transplantation in children.

Tales of the Ampulla of Vater: VIII

By the shores of Duodenum
which had seen its share of trials
Came the emissary foreign
who had travelled many miles.

From the ancient land Cloaca
Pectinate beyond the Treitz
To the crypts of the Papilla
he had journeyed days and nights.

Oh great Vater we beseech you
for our land is racked with pain
Read our signs and hear our symptoms
help prognosticate explain.

The affliction that befell us
is astride the Anal Verge
Where a mass has grown near ulcer
and the two at times converge.

In the submucosa are small cystic
swollen slimy lakes
Near the purple blood-filled caverns
over which mucosa quakes.

The Ampulla contemplative
turned and questioned with great zeal
Is there prolapse ulceration
does it granulate the heal?

Oh exactly the Cloacan said
it's as you have described
Both erosion reparation
just before the Great Divide.

Is it cyclical sporadic
does it wax as well as wane?
Yes precisely said Cloacan
and he added Oh the pain!

The Ampulla with a knowing smile
confirmed he had the answer
Have no fear my distant fellow
it is not a form of cancer.

It is neither adenoma
nor a type of carcinoid
It's the sequel to a prolapse
and mucosa that's destroyed.

Called the solitary ulcer
of the rectum this syndrome
As the prolapse brings ischemia
then mucosa's ulcer prone.

Known as Cystica Profunda
when there is a deep colitis
And the submucosa's filled with
lakes of mucinous detritus.

Pseudotumor on the surface
is a lesion hyperplastic
Called Cloacogenic Polyp
it's inflamed but not dysplastic.

Quickly home I'll speed to Pectinate
with all this vital data
The Cloacan cried departing then
ten thousand thanks Great Vater!

L.H. Sobin, M.D.
Armed Forces Institute of Pathology
Washington, D.C.

Diseases of Colon & Rectum 30:159, 1987.

International GI Scene

Harvey Goldman

At the XVII International Congress of the IAP in Dublin, the International Group of GI Pathologists will co-sponsor a symposium on "Interpretation of Mucosal Biopsies" on Friday, September 9:

Moderator -	B. Morson (UK)
Esophageal biopsy -	H. Goldman (USA)
Gastric biopsy -	V. Bogomoletz (France)
Small intestinal biopsy -	R. Whitehead (Australia)
Colonic biopsy -	A. Price (UK)

There will also be a social gathering of the GI pathologists at the Adelaide Hospital on September 7. For information about the Congress (September 4-9), contact XVII International Congress IAP, 44 Northumberland Road, Dublin 4 Ireland.

The International GI Group continues to expand. There are now eight Clubs (or Societies, if you prefer) in Australia, China, France, Japan, Scandinavia, United Kingdom, United States, Canada, and West Germany.

FINAL NOTE IN GI PATHOLOGY LONG COURSE

Harvey Goldman

For those that wonder what they learned at the US-Canadian Academy of Pathology Long Course on Gastrointestinal Pathology (in Washington, D.C., on 2 March 1988), and for those that inexplicably were missing, we offer the following summary:

1. From Dr. Stanley Hamilton: Persons with excrutiating chest pain may have reflux esophagitis. To confirm, just stand up and jump a bit, preferably with a friend in attendance. If symptom abates, it was reflux disease. If you faint, it is a heart attack.
2. From Dr. John Yardley: It is no longer sufficient to survive by taking megadoses of vitamins and iron. You must add a bismuth tablet, even if you gain weight.
3. From Dr. Donald Antonioli: Gender equality has been finally achieved, at least in cancers of the gastric antrum.
4. From Dr. Juan Lechago: DRGs will permit payment for stains of odd substances such as somatostatin, but not for cheaper silver stains. If you want to blow the whole budget, try a probe and get a black special stain instead of a brown one.
5. From Dr. Henry Appelman: If you are ever confronted with a smooth muscle (oops, stromal) cell tumor of the gut, admit ignorance and rush to his chapter on the subject, preferably in the Course Monograph.
6. From Dr. David Keren: We possess all sorts of immune mechanisms to defend us against dirty food, even if we don't understand them.
7. From Klaus Lewin: The defense mechanisms don't work, once we leave the sanctuary of our private kitchen. On the horizon is a paper, supported by super-probes, demonstrating that chicken soup harbors slow viruses.

8. From Dr. James Madara: Why the mechanisms don't work. Also, the devastating revelation that one of the last of the good-old traditions, the tight junction, is just another sieve easily manipulated by a bit of sugar.

9. From Dr. Rodger Haggitt: When you develop diarrhea in conjunction with failing to complete the surgicals in advance of the computer print-out, it's not due to nerves but rather to one of a ghastly array of colitides.

10. From Dr. Robert Riddell: Speak kindly to your contributing endoscopist, if you want to keep your head.

BOOK REVIEW

COLOR ATLAS AND TEXTBOOK OF DIAGNOSTIC PARASITOLOGY:

Tsieuh Sun, Igaku-Shoin Medical Publishers, New York, 1987

In light of the frequency of international travel to and from the third world and the common protozoal infections experienced by patients with AIDS, parasitic diseases have more recently attracted global attention. Here is a practical book that consolidates much of the diverse information on the epidemiology, clinical features, and laboratory diagnosis of the common parasitic infections.

The focus of this book is on the clinical and laboratory diagnosis of protozoal and helminthic disease and no information is provided on patient management and treatment. The author has attempted to combine a color atlas with a more standard textbook on parasitology and has been reasonably successful in this task. Each chapter contains a concise summary of epidemiologic, parasitologic, pathologic, clinical and diagnostic features, and combines these with predominantly color illustrations to highlight salient features and life cycles. Where possible the author has attempted to point out distinguising features to aid in the clinical and laboratory diagnosis. Illustrations are generally well reproduced and elevant to the text, although some photomicrographs are somewhat small to clearly define the hitologic features discussed. However as the author points out, the atlas is "not complete" and when encountering certain infections referral to other material may be required. Each chapter is well referenced to facilitate this.

Chapters are also included on somewhat less common but none the less fascinating infections of current interest including anisakiasis and microsporidiosis.

A concise and referenced technical appendix is also included to highlight some important laboratory techniques.

This book will probably be of greatest value to the general pathology or microbiology resident that wants a concise summary of encountered parasites. It should also be of value for the laboratory physician or technologist that wants a synopsis of the pertinent features for the diagnosis of parasitic disease.

Kevin Kain
David Owen
Department of Pathology
University of British Columbia

Bile Pigments and Jaundice.
Molecular, Metabolic and Medical Aspects.

Edited by J. Donald Ostrow, Marcell Dekker, Inc. New York 1986.

This book is Volume 4 in the Monograph Series "liver: Normal Function and Disease", edited by Dr. F.F. Becker. The 35 authors who contribute to this book cover the physiological, biochemical and clinical aspects of bile pigments and their metabolism in health and disease. The 24 chapters in the book permit coverage of fundamental aspects such as the molecular structure and physical chemistry of bile pigments and porphyrins, their hepatic uptake and secretion, the differential diagnosis of jaundice and clinical aspects of the jaundiced patient. Many special topics are dealt with as well, such as fetal bilirubin metabolism and neonatal jaundice, mechanisms of intrahepatic cholestasis, kinetic studies and mathematical modeling. Also included are chapters on pigmented gallstones, comparative bile pigment metabolism in vertebrates and bile pigments in plants. Each chapter is written by the leading experts in the field. The book is very well edited. It provides in a single place a thorough discussion of all aspects of the bile pigments. The book succeeds in providing an up-to-date comprehensive and authoratative survey and bibliography on bile pigments and jaundice. The book is an excellent resource for anyone interested in the biochemistry, physiology and pathophysiology of the bile pigments and to clinicians who manage patients who have jaundice. This book will be of particular value to clinical hepatologists, clinical pathologists and basic scientists.

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GASTROINTESTINAL PATHOLOGY SOCIETY
MINUTES OF ANNUAL BUSINESS MEETING
28 February 1988

The meeting in the International Ballroom West of the Washington Hilton Hotel, Washington, D.C., was called to order at approximately 5:00 PM by Dr. Lechago. Members in attendance were: Abrams, Antonioli, Appelman, Barr, Bostwick, Compton, Cooper, Dayal, Dean, Deschryver, Ferrell, Geller, Goldman, Gourley, Haggitt, Hamilton, E. Kahn, L. Kahn, Kelly, Lechago, Ed Lee, R. Lee, Lewin, Marcial, Mitros, D. Owen, Pascal, Petras, Rickert, Riddell, Sheahan, Sobin, Sternberg.

I. The minutes of the 1987 Annual Business Meeting submitted by Dr. Rickert were approved as distributed.

II. Financial Report - Dr. Hamilton.

Balance as of 28 February 1987		9616.02
Credits		
Interest	+	120.91
Debits		
Secretarial expenses & 1987 meeting reimbursement	-	288.00
Closing balance (Dr. Rickert) and opening balance (Dr. Hamilton) as of 9 June 1987		9448.93
Credits		
1987 dues	+	2280.00
Interest	+	284.32
Debits		
1987 USCAP meeting	-	790.75
1987 AGA meeting	-	290.00
Secretary-Treasurer expenses	-	929.50
Balance as of 29 January 1987	$	10,003.00

The financial report was approved as distributed.

III. Committee Reports

A. Announcement of new committee assignments - Dr. Lechago. The Officers and Committees for 1988-89 are:

POSITION	TERM ENDS
President: (1-year term)	
R. Rickert	1989
Vice-President/President Elect: (1-year term)	
G. Abrams	1989
Secretary-Treasurer: (3-year term)	
S. Hamilton	1990
Education Committee: (3-year term)	
D. Sheahan (Chairman)	1991
R. Pascal	1991
S. Sternberg	1990
J. Wirman	1990
R. Haggitt	1989
F. Mitros	1989
Membership/Nomination Committee: (3-year term)	
A. Qizilbash (Chairman)	1991
P. Correa	1991
R. Petras	1990
S. Saul	1990
Y. Dayal	1989
S. Geller	1989
Training Programs Committee: (3-year term)	
K. DeSchryver (Chairwoman)	1989
K. Barwick	1991
E. Lee	1991
E. Cohen	1990
H. Shields	1990
R. Lee	1989
Publications Committee: (Standing)	
H. Appelman (Chairman)	
R. Riddell	
S. Sternberg (Ex-officio: Editor of Am J Surg Path)	
R. Rickert (Ex-officio: President of GIPS)	
D. Sheahan (Ex-officio: Chairman of Education Committee)	
G. Abrams (Ex-officio: President Elect of GIPS)	
Microgrants Committee: (Standing)	
J. Yardley (Chairman)	
R. Rickert (Ex-officio: President of GIPS)	
S. Hamilton (Ex-officio: Secretary-Treasurer of GIPS)	
K. DeSchryver (Ex-officio: Chairwoman of Training Programs)	
D. Sheahan (Ex-officio: Chairman of Education Committee)	
G. Abrams (Ex-officio: President Elect of GIPS)	
Newsletter Editors: (3-year term)	
D. Keren	1991
W. Dobbins (Associate Editor)	1991
International Liaison: (Standing)	
H. Goldman	

Dr. Lechago thanked the members for agreeing to serve on the various committees.

B. Education Committee - Dr. Mitros.

1. The program planned for 1988 Digestive Disease Week meetings with the American Gastroenterological Association program on gastritis on Tuesday, May 17, 1988, at 5:30 PM was reviewed:

Moderator: Dr. Juan Lechago.
Endoscopic aspects of the gastric mucosa: Dr. Fred Weinstein
Pathobiology of gastritis: Dr. Pelayo Correa.
Campylobacter and the problem it poses: Dr. John Yardley
Metaplasia and dysplasia in the gastric mucosa: Dr. Donald Antonioli.
Neuroendocrine proliferations of the gastric mucosa: Dr. Juan Lechago.

2. The possibility of adding another member to the Committee was raised due to the illness of Dr. Wirman.

C. Membership/Nomination Committee - Dr. L. Kahn.

The following were recommended by the Committee and approved by the Executive Committee:

Regular membership:

Carolyn Compton
Carl A. Illardi
Lawrence D. Jewell
Richard A. Komorowski
A. Scott Mills
Jan Silverman
Herbert van Kruiningen

Associate membership:

Thomas Smyrk

The new members were approved by acclamation.

D. Publications - Dr. Appelman.
Negotiations with the American Journal of Surgical Pathology continue regarding the separate distribution and additional charge for the supplement which contains the papers from the scientific session of the Gastrointestinal Pathology Society.

E. Training Programs - Dr. DeSchryver.
A market for the subspecialty of gastrointestinal pathology seems to be present, as evidenced by numerous requests for information on fellowships. The listing of the programs in Gastroenterology appears to be low-yield, whereas a large number of inquiries come from the listing in the American Journal of Surgical Pathology.

F. Microgrants - Dr. Yardley.
In the past the microgrants have emphasized research. During the past year only two applications were received, one for

laboratory equipment which was not approved, and one for support of a fellow which was approved. The small number of applications led to consideration of broadening the scope of the microgrants. Additional defined purposes which will be considered are visiting scholar, sabbatical project for a member to work in another laboratory, and an interim regional meeting of GIPS members. The amount available will be increased to $1,500. A detailed memorandum describing the changes will be sent with the dues notices.

G. International Liaison - Dr. Goldman.
The international group of GI pathologists will co-sponsor a symposium on "Interpretation of Mucosal Biopsies" on Friday, September 9, 1988, at the 17th International Congress of the International Academy of Pathology in Dublin, Ireland. Eight societies now exist, including Australia, Peoples Republic of China, France, Japan, Scandinavia, United Kingdom, United States - Canada, and West Germany.

H. Newsletter - Dr. D. Owen.
A communication to medical publishers for gastrointestinal pathology books for review resulted in no responses. Members were requested to submit material for the Newsletter.

IV. Old Business - Dr. Lechago thanked the members for their response to the recent questionnaire. The results will be helpful in continuing the activities of the Society.

V. New Business - The vote on continuation of the Gastrointestinal Pathology Society as specified in the sunset clause of the by-laws was unaminous.

VI. Nomination of Vice President - Dr. L. Kahn.
The Membership/Nomination Committee recommended and the Executive Committee approved the nomination of Dr. Gerald Abrams for Vice Present/President Elect. No other nominations were received from the floor and Dr. Abrams was elected by acclamation.

VII. Induction of New President - Dr. Juan Lechago presented Dr. Robert Rickert as the new President. Dr. Rickert thanked Dr. Lechago for the excellent job he did as our President and asked for suggestions regarding future directions of our Society.

IX. There being no further business, the meeting was adjourned to a reception in the Caucus Room.

Respectfully submitted,
Stanley R. Hamilton, M.D.
Secretary-Treasurer

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