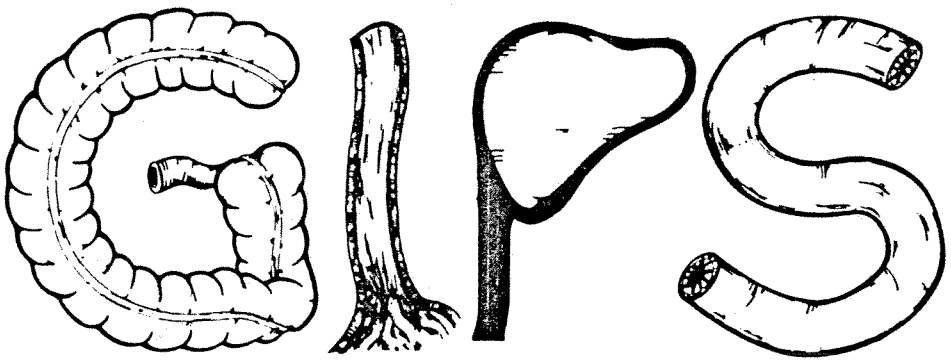

THE GASTROINTESTINAL PATHOLOGY SOCIETY
NEWSLETTER

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CONTENTS

PRESIDENT'S MESSAGE

SHOULD THE G.I.P.S. BE MORE LIVER ORIENTED?

As a result of a recent decision to devote the 1988 scientific session of the Gastrointestinal Pathology Society to pancreas, some people felt that this might have been a good opportunity to have, instead, a program devoted to liver. This has resuscitated a long-standing issue which I think our Club, now Society, has never been able to resolve to everyone's satisfaction. To put it succintly, should the G.I.P.S. be more liver-oriented?

As may be expected, opinions differ. There are those who feel that liver pathology is an integral part of gastrointestinal pathology and, therefore, should be strongly represented both in our membership and in our activities. It has been pointed out that the G.I.P.S. roster includes the presence of several prominent liver pathologists and that we should avail ourselves of their expertise, both in the educational and consultation aspects. Expansion of our activities to encompass strong liver programs would result in a dramatic widening, not only of our membership, but also of the total scope of our activities and, perhaps, enable our Society to carry out more educational programs within, as well as outside, the International Academy of Pathology.

On the other hand, there are those who believe that such expansion into the realm of the liver would be quite undesirable. They point out that our Society has established a solid international reputation as a leading body of pathologists dealing primarily with the gastrointestinal tract. In contrast, and in spite of the several liver experts in our roster, it probably does not have enough such experts to compete successfully with primarily liver-oriented societies. Any educational or other efforts devoted to liver pathology would most likely appear somewhat insignificant compared to those of such societies. Even worse, such efforts could dangerously dilute our capabilities with respect to the digestive tube and result in regression rather than progress.

I agree ... with both viewpoints, inasmuch as both would appear to be valid. It seems to me that we are not faced here with a choice between absolute right and absolute wrong. Either position is tenable and, given the appropriate circumstances, could be implemented, thus rendering this essentially a matter of philosophical position. In other words, should we stay the way we are, placing emphasis on the pathology of the hollow tube and letting our liver-oriented members find their education in liver pathology elsewhere? Or, now that our society has matured for several years and reached a phase of stability, should we seek to renew ourselves by widening our activities and increasing our membership through an agressive, and hopefully competitive, expansion into liver pathology? At the risk of opening a "box of Pandora", I feel that this is a decision that should be made by every member of our Society rather than by an executive body in isolation. Therefore, we solicit the input of all our members to resolve this issue if, indeed, it is perceived as an issue by many of us.

Juan Lechago, M.D., Ph.D.
President

EDITORIAL

STOP PRESS: RECENT ADVANCE IN G.I. PATHOLOGY - ORGANIZATIONAL METAPLASIA

With the solid, but not unanimous, approval of the last general meeting our club is now officially a society. Accordingly, therefore, your editors acknowledge this change with a new revised format for the newsletter. News of this change is regretably slow in spreading and has not yet reached the editorial offices of the American Journal of Surgical Pathology. Check the cover of your latest issue and you will see we are still listed as a sponsoring club.

While a change from a club to a society may mean more prestige for the members, please spare the thought for your over worked co-editors. We are still stuck with the dubious privilege of editing a "newsletter". How much more grand it would be if this name was changed to reflect the increased status of our organization. How about becoming a Journal or Archives or Annals (not Anals!).

On reflection however, such a change to increase prestige would mean that frivolous editorials would be eliminated and replaced by weighty pontifications. In my judgement the newsletter better remain as it is - at least for the time being.

David A. Owen

REPORT OF G.I. PATHOLOGY AND LIVER PATHOLOGY PRESENTATIONS AT THE IAP IN CHICAGO, MARCH 1987

G.I. Platform Presentations

The first presentation was given by Drs. Hamilton and Smith from the Johns Hopkins Hospital and was entitled "Prevalence and extent of Barrett's esophagus in adenocarcinoma of the esophagus and esophagogastric junction". These investigators reviewed 54 consecutive esophageal-gastrectomy specimens with adenocarcinoma and histologically mapped the adjacent mucosa. They found Barrett's epithelium in 65% of cases. The medium length of Barrett's esophagus was 4 cm. with a range of 1 - 12 cm. On average the tumors were centered 2 cm. above the normal esophagogastric junction with a range of 7 cm. to -2 cm. 19 patients (35%) were found not to have identifiable Barrett's mucosa. These tumors were centered at 0.3 cm. above the G.E. junction with a range of +7 to -3 cm. Most interestingly the majority (approximately 80%) of patients with G.E. junction adenocarcinomas were white males. This is compared with a group of 63 Johns Hopkins patients who had adenocarcinoma of the distal stomach where only 29% were white males. The authors conclude that most Barrett carcinomas occur in short segment Barretts which may go undetected by endoscopy.

A 5 year prospective study of Barrett's esophagus was presented by Drs. Chejfec, Sontag, Schnell, Chintam, and O'Connell from the Hines V.A. Hospital, Illinois. They studied biopsies from over 1500 patients with gastro-esophageal reflux. Barrett's was diagnosed when intestinal epithelium was present in the esophagus or when gastric epithelium was identified at least 2 cm. above the lower esophageal sphincter. Their results showed that Barrett's esophagus was present in 13% of cases. In these cases intestinal epithelium was found in 81% either alone or combined with gastric epithelium. High iron diamine positivity (indicating presence of sulphomucin) was present in 57%. Low-grade dysplasia was present in 43% and high grade dysplasia/adenocarcinoma in 2%. However, they did not specifically investigate the relationship between dysplasia and sulphomucin positivity.

From the Cleveland Clinic a presentation on gastric antral vascular ectasia was given by Drs. Suit, Petras, Bauer, and Petrini. They reviewed 9 biopsies from 7 patients with this syndrome which clinically is characterized by acute or chronic upper intestinal blood loss and the endoscopic finding of parallel dilated vessels traversing the gastric antrum. Six of the seven patients were female with an age range from 42 to 81 years. The biopsies were compared with controls taken from individuals with chronic gastritis or peptic ulcer disease. It was found that the vascular ectasia biopsies had vessels with a significantly increased mean cross sectional area and that the percentage of the biopsy area occupied by vessels was also increased. In addition, vascular ectasia biopsies all showed intravascular fibrin thrombi. It was concluded that these features were sufficiently distinctive to permit the recognition of gastric antral vascular ectasia on biopsies.

A presentation entitled "Enterochromaffin Cell Hyperplasia of the Appendix" was given by Drs. Stanley, Cherwitz, and Snover from Minneapolis. In examining routine appendectomy cases they found that approximately 10 to 15% showed no evidence of acute inflammation by routine histology. Using an immunoperoxidase method for serotonin they counted the number of EC cells in these appendixes and compared them with a control group of appendixes removed as incidental appendectomy specimens from asymptomatic patients. In this control group they found an average of 1% EC cells. In the uninflamed appendixes from symptomatic patients they found an average of 1.77% EC cells. This was a significant difference and they postulate the presence of EC cell hyperplasia as a cause of symptoms of acute appendicitis.

From the Presbyterian Hospital, Pittsburgh, the Baptist Memorial Hospital, Memphis and Rush Medical Center, Chicago, Drs. Banner, Dean, and Williams presented the morphology of rejection in long-standing canine small bowel transplants. This study examined graft and host tissues from dogs treated with Cyclosporin surviving longer than two weeks after transplantation. None of these dogs developed systemic graft versus host disease but changes were observed in some of the graft and host bowels. The changes of graft rejection however were a lymphoplasmocytic infiltrate present in the deep bowel wall without the presence of mucosal changes or mural fibrosis. Full thickness biopsies were necessary to detect these lesions.

From McMaster University, Hamilton Ontario Drs Stead, Tomioka, Quinonez, Simon, Felten, and Bienenstock presented a study of intestinal mucosal mast cells in normal and nematode infected rats. These investigators looked at rats infested with Nippostrongylus brasilienis. They used immunohistochemistry, electron microscopy, and quantitative histology. The results showed that in both the normal and infested rats the number of contacts between enteric nerves and mast cells was five times greater than would be expected by chance alone. This is further evidence for a physiological interaction of mast cells with the nervous system.

The next paper presented something we have all long suspected, namely, that subepithelial collagen deposition is not specific for collagenous colitis. This presentation was given by Drs. Owings, and Wang from the Beth Israel Hospital in Boston. For the purposes of this study subepithelial collagen deposition was diagnosed when the basement membrane was greater than 5 microns in thickness. In a 26 month study period seven biopsies with these characteristics were identified. The collagen thickness ranged from 15 to 90 microns. Only 4 of these patients had watery diarrhea; 1 was asymptomatic and 2 were biopsied for reasons other than diarrhea. A control group of 157 consecutive colonic resections were studied. 11 of these had subepithelial collagen deposition exceeding 20 microns in thickness. Only 2 of the 11 had any history of diarrhea; 1 was due to radiation, and the other to diverticular disease. These results suggest that subepithelial collagen deposition is found in a variety of clinical situations and that not all individuals have watery diarrhea.

"Neuron specific enolase and S-100 protein immunoperoxidase stains as an aid to diagnosis in Hirschsprung's disease" was presented by Drs. Robey, Kuhajda, and Yardley from Johns Hopkins Hospital. NSE stains were highly effective in demonstrating ganglion cells in rectal biopsies. One of the H & E slides from each of 53 cases containing ganglion cells was re-stained with NSE and positive results were obtained in 48 cases. In addition, one specimen previously interpreted as aganglionic by H & E was positive with NSE. The S-100 stain was equally effective as an acetylcholinesterase stain in the demonstration of abnormal hypertrophied nerve trunks and increased numbers of nerve fibers.

A group from the Jefferson Medical College and Wistar Institute, Philadelphia (Drs. Cooper, Marshall, Ruggerio, and Steplewski) presented their results of an immunohistochemical study of hyperplastic polyps with monoclonal antibodies against blood group antigens. They compared hyperplastic polyps of the colon with normal epithelium, colonic carcinomas and colonic adenomas. A variety of antibodies to blood group antigens Lewis, A, B, and H were used. In common with normal epithelium all hyperplastic polyps expressed Le^a, Le^x, Le^y, A, B, and H-2. In addition, 25 of 30 extensively expressed sialyated Le^a (gastrointestinal cancer associated antigen). Le^b is usually absent in normal epithelium and was also absent in 26 out of 30 hyperplastic polyps. In the 4 polyps that did express Le^b 3 had synchronous carcinomas. Their conclusion was that inspite of anomalous expession of sialyated Le^a and Le^b hyperplastic polyps are generally similar to normal colorectal epithelium.

From the University of Calgary, Canada, Drs. Urbanski and Tanaka presented a study of C-MYC oncogene protein expression in synchronous colonic carcinomas. These investigators identified 11 cases of synchronous colonic carcinoma studied using an immunoperoxidase method for C-MYC oncogene. In 9 cases identical results were obtained in both of the synchronous tumors. In 2 cases only 1 of the tumors was positive. These results are interpreted to imply a different molecular mechanism in the development of these C-MYC negative tumors.

The hypothesis that inositol hexaphosphate from corn decreased the frequency of colorectal cancer in azoxymethane treated rats was present by Drs. Elsayed, Chakravarthy, and Shamsuddin, from the University of Maryland Medical School. These investigators showed that animals receiving 1% inositol hexaphosphate (phytate) in drinking water developed an average of 3 tumors per animal when injected with carcinogenic doses of azoxymethane. Animals without this dietary supplement developed 4.6 tumors per animal. The reason for this protective effect of phytate is unknown but reinforces the observation in humans that high fiber (also high phytate) diets protect against the development of colorectal carcinoma.

Poster Presentations in G.I. Pathology

In a topic closely related to their platform presentation, Drs. Stead, Tomioka, Riddell, and Bienenstock from McMaster University, Hamilton, Ontario presented a poster entitled "Substance P and/or calcitonin gene related peptides are present in subepithelial enteric nerves opposed to intestinal mucosal mast cells". These investigators examined rat intestines infested with Nippostrongylus brasilensis. Immunostaining of serial sections revealed the presence of both neuropeptides in many of the subepithelial nerves adjacent to mucosal mast cells. This study provided a microanatomical confirmation of the known observation that substance p is an effective mucosal mast cell secretagogue in vitro.

From Johns Hopkins Hospital Drs. Paull, Yardley, Walters, Rawles, and Dick presented their results of a study of Campylobacter pyloridis in patients with Barrett's esophagus. They examined material from 26 patients with biopsy proven Barrett's esophagus and 26 age and sex matched controls. They found organisms in the columnar esophagus in 40% of Barrett's patients with gastric Campylobacter but in 0 out of 16 patients without gastric Campylobacter. Campylobacter were not noted in squamous epithelium. The frequency of gastric Campylobacter was similar in the Barrett's oesophagus group and the controls. There was also an association between active oesophagitis and the finding of esophageal Campylobacter.

Campylobacter in pyloric biopsies was studied by Drs. Qizilbash, Rennie, Thornley, and Castelli from McMaster University, Hamilton, Ontario. They looked at 50 patients who underwent endoscopy and biopsy for upper gastrointestinal symptoms. Organisms were observed histologically in 42% of patients and cultured in 48%. Only 1 patient in whom organisms were found had a normal biopsy. The rest showed chronic gastritis.

A study of mucin histochemistry in Barrett's esophagus was presented by Drs. Jaureugi, Coles, and Hale from Brown University, Providence, Rhode Island. 15 Barrett's esophagus cases were biopsied and examined for the presence of sulphomucins and O-acylated mucins. Sulphomucins were found in 8 patients and O-acylated mucins in 9 cases. 2 caes had an associated carcinoma and both of these showed adjacent sulphomucin positive cells.

From the University of Arizona in Tucson Drs. Spier, Grogan, Rangel, and Richter presented an immunophenotyping study of gastric lymphomas. Endoscopic biopsies from 12 patients with gastric lymphoid infiltrates showed a diverse histologic appearance ranging from overtly malignant to reactive appearing. (9 malignant, 2 atypical and 1 reactive). The majority of overt lymphomas were large cell type. By immunologic analysis all the overt lymphomas and one of the atypical cases typed as monoclonal B cell tumors. The remaining 2 cases were polyclonal and considered as "pseudolymphomas". The authors point out that immunologic analysis can provide valuable assistance in the evaluation of borderline lymphoid proliferations.

An interesting poster on hepatoid adenocarcinoma of the stomach was presented by Drs. Ishikura and Aizawa of Hokkaido University, Japan. They discovered 9 cases of primary gastric adenocarcinomas in which markedly elevated levels of serum AFP were found. Histologically these tumors contained areas with solid sheets of cells that were indistinguishable from hepatocellular carcinoma and with immunostaining were positive for AFP. In one case even bile secretion by the tumor was noted. In addition, cells were positive for alpha I antitrypsin and alpha I antichymotrypsin but not for hCG. In other areas of the tumor there was typical adenocarcinoma mingling with the hepatoid areas and origin from the mucosa could be identified.

From the M.D. Anderson Hospital, Houston, Texas, Drs. Floyd, Ro, Mackay, and Ordonez presented an immunologic and EM study of gastrointestinal stromal tumors. 50 tumors were reviewed, 25 in the stomach, 14 in the small intestine and 11 in the colon. Histologically 43 tumors were malignant. Vimentin was positive in 45 tumors, Desmin positive in 8 tumors, smooth muscle myocin positive in 14 tumors, and S-100 staining positive in 9 tumors. The prognosis in these tumors was generally poor and there was no correlation between prognosis and immunostaining.

From the Armed Forces Institute of Pathology Drs. Federspiel, Sobin, and Helwig presented a poster detailing their analysis of the DNA content of gastrointestinal carcinoid tumors with a view to identifying those that would behave in a malignant fashion. They analyzed 38 carcinoids without metastases and 12 with metastases. They found that all malignant tumors were large and had a DNA value exceeding 5n. This was a valuable discriminatory tool.

Carcinoma of the Ampulla of Vater was presented by Drs. Connolly, and Dawson from the University of Chicago. They studied 26 cases morphologically and with the aid of mucin histochemistry. Histologic review revealed 3 major tumor types. One type (6 cases) appeared to arise from duodenal mucosa and resembled carcinoma of the large bowel containing predominantly non-sulphated mucin. A second group (6) cases arose near the opening of the Ampulla and consisted of tall columnar cells with large glands rich in sulphomucin. The third group (6 cases) arose deep in the Ampulla and consisted of small, well-formed ducts with only small amounts of sulphomucin. The last group had a better prognosis than the others with 60% of patients surviving 5 years.

Drs. Litz and Snover from the University of Minnesota presented an immunohistochemical classification of appendiceal carcinoids. They examined 23 cases of appendiceal carcinoids with antisera to Serotonin, NSE, S-100 and Chromogranin. They found 22 cases stained with NSE. 18 were Chromogranin positive of which 15 were also Serotonin positive. 16 tumors showed an intimate association of S-100 positive cell processes within the tumor nests. There was also polarization of the Chromogranin granules towards the S-100 processes at the periphery of

the nests. The 5 Chromogranin negative tumors showed a trabecular pattern and the majority were associated with fibrous obliteration of the appendiceal lumen. There was no association between S-100 processes and the tumor nests in these cases. The active association of nests of carcinoid cells with nerve twigs raises the possibility that these are forms of endocrine cell hyperplasia rather than true neoplasia.

The relationship between carcinoma of the anus and human papilloma viruses was presented by Drs. Gupta, Taxy, Gupta, and Shah from Johns Hopkins hospital, The Lutheran General Hospital, Parkridge, Illinois and Northwestern University, Chicago. They used in-situ hybridization techniques to search for HPV in anal lesions from 19 patients. Condylomatous changes were noted in or adjacent to 4 of 11 cases with invasive squamous carcinoma, 2 of 5 cases of carcinoma in situ and in 3 patients with warts. HPV-16 was demonstrated in the neoplastic cells of two invasive tumors and 1 case of CIS. HPV-6/11 was found in 4 cases with condylomatous areas but was not present in neoplastic cells. HPV-18 was not found in any case. These results were considered to support an association between HPV-16 and anal cancer.

From the Universities of Calgary, Alberta; Ottawa, Ontario; and the Wellesly Hospital, Toronto, a study of mineral dust in Peyer's patches was presented by Drs. Urbanski, Arsenault, Green, and Haber. They collected 5 cases with patchy terminal ileal pigmentation. This was present within the lamina propria or the submucosa in relation to Peyer's patches. These patches resembled pigment seen in lungs and was not birefringent. Negative staining was obtained for melanin, iron, and lipofuscin. X-ray microanalysis confirmed its identity as dust since it contained predominantly silicates of various kinds. It appears to have a simimlar composition to atmospheric dust.

Using light microscopy, scanning electron microscopy, and transmission electron microscopy Drs. Cho, Hirose, Raheja, and Linscheer of the State University of New York, Syracuse studied the morphology of aging small bowel mucosa in the rat. The study involved measurement of crypt depth relative to villous height before and after 70% enterectomies in young and old rats. The result showed basically no difference between the age groups with both showing an adaptive hyperplasia of crypts with no corresponding change in villous height.

From Trinity College, Dublin, Ireland, and Emory University, Atlanta, Drs. Gaffney, Sweeney, Nolan, Condell, Toner, McDonald and Majmudar, investigated granulomas in human ileocecal tuberculosis. The histopathology of eight cases of ileocecal tuberculosis was studied. All cases had increased numbers of mucosal lymphoglandular complexes and perivascular lymphoid aggregates within the gut wall adjacent to areas of ulceration. Granulomas at various stages of evolution were present within these lesions. The paraffin sections were stained by monoclonal antibodies UCHL 1 (T cell membrane antigen) and LM 1, LM 2, and LM 3 (B cell antigens). Results showed that the central cells of the lymphoid aggregates and lymphoglandular complexes stained like follicular center B lymphocytes and were surrounded by a mantle zone of

T cells and interdigitating reticulum cells. These observations suggest that lymphoglandular complexes are the sites of mycobacterial antigen sampling and potential sites of granuloma formation.

The site of origin of fistulae in Crohn's disease was presented by Drs. Kelly and Preshaw from the University of Calgar, Alberta. In a prospective study these investigators examined 236 consecutive resection specimens from cases of Crohn's disease and made a comparison of the origin of fistulae in first excisions and in re-excisions. 60 fistulae were found (25% of cases). 44 were present in first excisions and 16 in re-excisions. In first excisions 41 fistulae arose in the ileum and 3 in the colon and the majority were enteroenteric or enterovesical. Only 4 fistulae were enterocutaneous. 40 were associated with strictures. 10 of the 16 fistulae from re-excisions arose in the region of the previous anastomosis and 7 of these 10 drained through the skin.

Ultrastructural differentiation between Crohn's disease and other colonic diseases was studied by Drs. Steinhoff, Kodner, and DeSchryver-Kecskmeti of Washington University, St. Louis. In particular, these investigators examined changes in nerves and the presence or absence of adjacent inflammatory changes. Controls included cases of colonic carcinoma, ulcerative colitis and radiation colitis. They found that cases of Crohn's disease were unique in that they showed extensive severe axonal damage unassociated with perineural chronic inflammation.

From the Children's Memorial Hospital and Cook County Hospital, Chicago, (Drs. LLausas-Magana, Hsueh, Arroyave, Torre-Amione, and Gonzalez-Crussi) came a presentation of platelet activating factor and a complement system in the pathogenesis of ischemic bowel necrosis. An experimental model of ischemic bowel necrosis in the rat was presented. This involves injecting rats superior mesenteric artery with platelet activating factor. Three groups of rats were employed, one injected with cobra venom to activate the complement system before an injection of platelet activating factor, a second group injected with venom alone and a third group injected with activating factor alone. They showed that by far the greatest bowel necrosis was produced in rats given the combination of venom and activating factor suggesting that the complement system may have a role in the pathogenesis in ischemic bowel disease.

Drs. Paplanus, Graham, and Bartels of the University of Arizona, Tucson presented an "expert system" for the diagnosis of colonic lesions. This system was developed as part of an overall project involving data aquisition by a high speed laser scanning microscope with scene segmentation analysis and evaluation using multiprocesses. The results they presented show that the expert system is able to distinguish non neoplastic lesions from adenomas, atypical adenomas, and carcinomas.

In an interesting and challenging poster, Dr. Elsayed and Shamsuddin of the University of Maryland School of Medicine claim to have developed a strip test for the detection of colorectal cancer. They obtained

rectal mucus from 38 patients, 11 with cancer and 27 from individuals free of colorectal diseases. The mucus was smeared on a membrane filter and air dried. At a later time another filter impregnated with D-galactose oxidase, was placed under the membrane filter for one hour. The membrane filter was then rinsed with distilled water and developed in Schiff's reagent. All 11 patients with cancer had Schiff positive mucus but only 18.5% of cases without cancer had Schiff positive mucus. The authors postulate that this method may be adaptable for a widespread screening program.

In a closely related study Drs. Elsayed and Shamsuddin studied the glactose oxidase-Schiff sequence to examine biopsies of pre-neoplastic and neoplastic colonic lesions. They claim to have detected the presence of an altered glycocongugate (Beta-D-Gal(1-3)D-GalGalNAc) in neoplastic and pre-neoplastic epithelium.

Altered expressions of the brush border protein villin by colonic neoplasms was investigated by Drs. West, Issac, Morrow, Mooseker and Barwick from Yale University. This study used immunoperoxidase techniques with antibodies raised against chicken intestinal brush border. In normal colons, villin was confined to the brush border and stained more strongly towards the mucosal surface than in the crypts. Villin was also expressed by 5 adenomas and 14 adenocarcinoma (chicken tumors) at the lumen surface of villi or glands. In high grades of tumor the lumenal distribution was sometimes patchy with positivity detected in other areas of the cell.

A study of colonic adenomas using immunohistochemical techniques with blood group antigens was presented by Drs. Ruggerio, Cooper, and Steplewski. They looked at 31 colonic adenomas and stained for normal colonic antigens Le^a, sialated Le^a, Le^x, Le^y, and tumor associated antigens A, B, H2, Le^b. The results showed that 52% of adenomas were positive for both tumor antigens and normal antigens, 10% of adenomas were positive for tumor antigens but negative for normal antigens and 38% were tumor antigen positive but positive for normal antigens.

From Harper Hospital, Detroit, Michigen, Drs. Niebylski, Zarbo, and Crissman presented a study of DNA analysis comparing recovery from fresh and paraffin embedded colon cancer material. They found that fresh tissue preparations contained 100% more DNA intensity than the fixed material. Furthermore, DNA histiograms showed a tendency for aneuploid populations to be decreased in fixed material and in 4 cases (16%) aneuploid propulations were only observed in the fresh specimens.

From the University of Virginia Medical Center, Drs. Frierson and Austin presented their study of colorectal neoplasms in Gardner's syndrome analyzed by flow cytometry. Two cases were studied in depth. In the first case two small adenomas were diploid and two larger adenomas were hyperdiploid. One adenocarcinoma was diploid and two were hyperdiploid. In the second case eight adenomas were hyperdiploid and three were diploid. These results indicate that colorectal neoplasms in Gardner's syndrome whether benign or malignant do not

have identical ploidy profiles.

A study of primary squamous carcinoma of the colon was presented by Drs. Marcus, Lundquest, Thorson, and Massop of Creighton University, Omaha, Nebraska. They performed a literature review and determined that 73% of squamous carcinomas of the colon arose in the right side of the colon and 27% in the left. This should be compared to typical adenocarcinoma of the colon in which 46% are right sided and 54% left sided. They concluded that this highly statistical difference may represent a difference in the pathogenesis of these two types of colon carcinoma.

Proffered Papers in Liver Pathology

A study of the reversibility of methotrexate associated hepatic fibrosis was presented by Drs. Camuto, Neuman, Culubret, Migdal, Auerbach, Tobias, and Feiner from the N.Y.U. Medical Center. They had available for study over 200 patients who had received methotrexate for psoriasis and who had been followed by liver biopsies. Six of these patients had required withdrawal of therapy because of hepatotoxicity and were subsequently rebiopsied to determine their candidacy for resumption of therapy. Fibrosis was graded from 0 to 4+ (cirrhosis). Three patients demonstrated reduction of fibrosis and in one of these cirrhosis reverted to Grade I (mild) fibrosis. One patient remained stable at Grade II and two patients showed mild progression. This data indicates that all grades of methotrexate associated fibrosis are potentially reversible.

A study of vitamin A deficiency and increased intrahepatic expression of cytokeratin filaments in alcoholic liver disease was presented by Drs. Ray, Mendenhall, and Gartside from the University of Cincinnati Medical Center and the Cincinnati V.A. Hospital. These investigators had material from 60 patients with alcoholic liver disease. Cytokeratin filaments were demonstrated in the biopsies by an immunohistochemical technique. They demonstrated an inverse correlation between plasma levels of vitamin A and the presence of cytokeratin filament staining in biopsies without Mallory bodies. There was also a significant positive correlation between the finding of cytokeratin filaments with other features of alcoholic liver disease (fibrosis, cell necrosis, and portal inflammation). There was also a positive correlation between cytokeratin filaments and clinical severity of alcoholic liver disease.

From the Utah Medical Center a study of non alcoholic steatohepatitis was presented by Dr. Lee. He reviewed 543 liver biopsies that had been coded as alcoholic hepatitis and recovered 41 cases which on review were thought to be not related to alcohol. The majority of these cases were middle-aged women who had been found incidentally to have elevated liver enzymes during investigation for other medical problems. Three quarters of the patients were obese and half of them had diabetes mellitus. The histologic findings were indistinguishable from alcoholic hepatitis including the presence of Mallory bodies in 50% of

the biopsies. Clinical follow-up was available for 34 patients and none of these developed further evidence of liver disease. However, histologic follow-up which was available in 12 cases showed that 6 had progression of fibrosis and 2 developed cirrhosis.

Drs. Deschryver-Kecskemeti and Wear from Washington University, St. Louis, and the A.F.I.P. presented their findings in three patients with cat scratch disease of the liver. These individuals had necrotizing granulomas similar to those described in lymph nodes. Around the granulomas was a fibrotic cuff in which thrombosed vessels and areas of vascular proliferation were identified. The diagnosis of cat scratch disease was confirmed by the finding of bacilli on the Brown-Hopps stain.

Veno-occlusive disease of the liver was the topic of a presentation from the University of Washington by Drs. Shulmann, Luk, Deeg, Shuman, and Storb. They treated 64 dogs by various combinations of total body irradiation, localized hepatic radiation and chemotherapy consisting of Busulphan, Phenyalanine mustard, Methotrexate, or Monocrotaline. The major cause of death in these animals was gastrointestinal toxicity. However, as expected, 50% of dogs given Monocrotaline developed VOD. Other regimes were less effective in inducing VOD.

The differential expression of ras oncogene products in liver diseases was reported by Drs. Lee, Gould, Schlom, Banner, and Gould from Rush Medical College, Chicago, NCI, and University of Pittsburgh. This was an immunohistochemical study and showed that ras oncogene expression is most frequent and intense in primary inflammatory liver diseases where there is associated liver cell hyperplasia eg. cirrhosis. The intensity and frequency of expression are lower in benign and malignant hepatic neoplasms. Ras products are not enhanced in normal hepatocytes.

From the University of Nebraska, Drs. Masih, Linder, Shore, Wood, Donovan, White, and Markina presented a comparison of in-situ DNA hybridization, routine light microscopy and viral culture in the diagnosis of CMV hepatitis. These investigators evaluated 44 biopsies from 21 patients who had received organ transplants. Characteristic light microscopic inclusions were found in 13.6% of biopsies, culture was positive in 15.9% of biopsies and in-situ hybridization was positive in 38.6% of biopsies. 20% of biopsies were positive only with in-situ hybridization and this technique detected all cases that were positive by the other methods.

Poster Presentations in Liver Pathology

Immunoperoxidase staining as a diagnostic aid for hepatocellular carcinoma was presented by Drs. Christensen, Kuhajda, and Boitnott from Johns Hopkins Hospital. They reviewed 62 cases of hepatocellular carcinoma and studied them immunologically with antibodies to polyclonal CEA, monoclonal CEA, EMA and keratin. Controls consisted of

a variety of other neoplasms including cholangiocarcinomas, pancreatic and gastric tumors. The most valuable finding was that 66% of hepatocellular carcinomas had polyclonal CEA staining of bile canaliculai. This staining was not observed in other neoplasms. They also noted that negative staining with monoclonal CEA and keratin was suggestive of hepatocellular differentiation.

From the University of Minnesota Drs. Nakhleh and Snover studied the expression of Alpha I antitrypsin in nodular regenerative hyperplasia of the liver. The rationale behind this study was that AAT is considered an acute phase reactant synthesized but not normally stored in the liver. It has been found in other conditions where there is regeneration of damaged hepatocytes. In NRH, AAT was present in 6 out of 11 biopsies compared to 1 out of 18 biopsy controls. The authors suggest that the finding of AAT staining may be helpful in confirming a biopsy diagnosis of NRH.

Liver pathology in morbid obesity was presented by Drs. Silverman, Dabbs, O'Brien, Norris, and Caro from the East Carolina University School of Medicine. These investigators examined liver biopsies from 83 patients who underwent gastric bypass surgery for morbid obesity. They measured the amount of fat and fibrosis within the biopsies and correlated this with the findings on a glucose tolerance test. The results showed a significantly greater amount of fibrosis and fat in the biopsies from individuals who had non-insulin dependent diabetes mellitus or who had an impaired glucose tolerance test.

Drs. Swerdlow, Chowdhury, and Dalton from the Michael Reese Hospital, Chicago studied the effects of prolonged administration of ethanol in rabbits. The alcohol was either added to the drinking water or administered via an intragastric tube. Three rabbits developed hepatic failure at 26, 29, and 31 months. Other rabbits were routinely sacrificed after 6, 12, 24, 30, and 36 months of treatment. After 24 months all the treated rabbits showed extensive fatty change, mitochondral degeneration and mild fibrosis. However, no Mallory's hyaline was found and there as no neutrophilic infiltration. IgA deposition was observed in alcoholic rabbits after 6 months but keratin staining of hepatocytes required 24 months exposure.

From the University of Calgary Alberta, Drs. Kelly, Maung, and Hart demonstrated the effect of intraperitoneal innoculation of C parvum extract. They injected 15 mice with a pyridine extract of C parvum and produced hepatosplenomegaly studied when the experiment was terminated on day 12. All mice showed granulomatous hepatitis. Some had thrombi in intrahepatic portal veins and foci of liver necrosis. Sinusoidal dilatation, acidophil bodies, and increased cell mitosees were noted. Splenomegaly was thought to be the result of increased hemopoiesis stimulated by factors released from activated macrophages within the granulomas.

The histopathology of the liver in AIDS was presented by Drs. Godwin and Felix from Cornell Medical Center. They reviewed the autopsy histopathology of the liver in 100 fatal cases of AIDS. This included 97 males and 3 females. The age range was 22 to 70 years with a mean of 42 years. In 69% of cases the weight of the liver was 1800 grams or greater. There were four cases of cirrhosis. Evidence of CMV infection was noted in 34% and mycobacteria in 15%. Generally, the CMV cells were sparse, isolated and usually periportal. In only 2 cases was CMV prominent in biliary epithelium. Mycobacterial involvement varied from occasional macrophages to discrete granulomas. Occasional cases of Candida, Cryptococcus, Histoplasma, Aspergillus, Herpes, and Cryptsporidium were found. Six cases had Kaposi's sarcoma and two had malignant lymphoma.

An analysis of gene expression in human hepatic tumors was presented by Drs. Sheahan, Rupp, and Locker from the University of Pittsburgh School of Medicine. They studied hepatocellular carcinoma, cholangiocarcinoma, focal nodular hyperplasia, hepatic adenoma and metastatic breast and colon carcinoma. These tumors were analyzed for albumin, AFP, AAT, by hybridization of purified RNA and immunoperoxidase staining. Albumin m RNA expression in normal liver, adenomas and focal nodular hyperplasia was similar. Albumin m RNA was present in low levels in hepatocellular carcinomas as well as breast and colon carcinomas, but was absent from cholangiocarcinoma. AFP m RNA was detected only in some hepatocellular carcinomas. AAT m RNA showed normal or reduced levels in hepatocellular carcinomas and low levels in breast and colon carcinomas. However, most hepatocellular carcinomas showed increased staining for AAT when compared to normal liver cells. This appears not to be due to increased expression of the gene but likely results from altered processing of the protein or up-take from serum.

A demonstration of the diagnosis of metabolic diseases by ultrastructural examination of the liver was presented by Drs. Poucell, Patterson, and Phillips from the University of Toronto. They showed that the ultrastructural appearances can be diagnostic in cases of Farber's disease, Fabry's disease, Pompe's disease, Brancher enzyme deficiency, hereditary fructose intolerance, Gaucher's disease, Metachromatic leukodystrophy, gangliosidosis type I, Alpha 1 antitrypsin deficiency, Dubin Johnson syndrome, erythropoietic protoporphyria, Wilson's disease, Zellweger's syndrome, and infantile Refsum's disease.

From the New York University Medical Center Drs. Camuto, Tillmon, Sidhu, and Feiner presented an ultrastructural evaluation of Ito cells in psoriatic recipients of etretinate and methotrexate. Etretinate is a synthetic vitamin A analog valuable in the treatment of psoriasis. The authors have previously demontrated that 4 of 18 patients treated with this drug developed hepatic fibrosis. In order to investigate this further they morphometrically analyzed the fat content of Ito cells. Controls included biopsies from non-psoriatic individuals and biopsies from psoriatic patients treated with

methotrexate. They found that all cases of psoriasis had an increased percentate of cytoplasmic area occupied by lipid vacuoles. However, there was no difference between the methotrexate and the etretinate treated patients. They conclude that increased lipid content of Ito cells is a nonspecific marker of hepatic damage.

Drs. Johnson, Marnke, Herndier, and Rouse from Stanford University presented an immunohistochemical study of cytokeratin profiles in primary liver tumors. Antikeratin antibodies AE 1 and Cam 5.2 were used to study 21 hepatocellular carcinomas, five cholangiocarcinomas and one mixed tumor. In the normal liver biliary epithelium is positive with AE 1 and biliary epithelium and hepatocytes are stained by Cam 5.2. They found that all cases of cholangiocarcinoma and a single mixed tumor were strongly positive for both antibodies. In contrast, 19 of 21 hepatocellular carcinomas were non reactive for AE 1 but positive with Cam 5.2. One hepatocellular carcinoma was positive with both antibodies and another one was negative with both. The authors conclude that these antibodies may prove valuable in the study of difficult diagnostic cases.

From the University of Ottawa and the Hotel-Dieu in Quebec, Drs. Katsuma, Marceau, and French presented their study of cytokeratin intermediate filaments in rat hepatocytes. This study was performed by transmission electron microscopy and looked at the three dimensional architecture of the cytoskeleton of the normal rat hepatocyte. In addition, cytoskeletal domains were examined by immunofluorescence using antibodies against cytokeratin A (CK-A) and cytokeratin D (CK-D). Four distinct cytoskeletal networks were identified. These included; 1. A nuclear filamentous shell, 2. Cytoplasmic, 3. Cell border, and 4. Pericanalicular. Immunologically CK-A was detected in the cytoplasm and the cell border of hepatocytes but most strikingly in the pericanalicular region. CK-D was primarily located in the cytoplasm and the nuclear rim.

In a related study Drs. Katsuma, Kettery, and French (University of Ottawa and New England Deaconess Hospital) studied Mallory body formation and alteration of cytokeratin filaments. They used monoclonal antibodies AE 1 and AE 3 to study one case of primary biliary cirrhosis and two cases of primary sclerosing cholangitis. They found that AE 1 stained Mallory bodies and bile ducts intensely but did not stain normal hepatocytes. AE 3 stained both Mallory bodies and normal hepatocytes. Although many hepatocytes which contained Mallory bodies did not stain with either of these antibodies, some of them were stained. The authors postulate that Mallory body formation may be linked to the expression of bile duct cytokeratin by altered hepatocytes in the above diseases.

From Washington University, St. Louis, Drs. Bucy, Flye, Hanto, and DeSchryver-Kecskemeti presented the Barnes Hospital experience with liver transplantation. The material consisted of 19 patients who received 21 transplanted livers. The mean follow-up was 129 days with a range of 3 - 376. Eleven of these patients are currently alive and well with properly functioning grafts. 112 per cutaneous biopsies were

available representing protocol biopsies and those done electively. The earliest time of acute rejection was 3 - 4 days. Two patients went on to chronic rejection. The findings in the acute rejection included the presence of activated macrophages and lymphocytes in clusters and necrosis produced by lymphocytes in direct cellular contact with epithelium.

Intraoperative liver biopsies of donor liver from patients undergoing liver transplant were studied by Drs. Markin, Schafer, Zetterman, Wood, and Shaw from the University of Nebraska. They examined pre and post revascularization biopsies in 28 liver transplants. Each biopsy was evaluated for necrosis, loss of cytoplasmic staining, loss of nuclei, pyknotic nuclei, Kupffer cell hyperplasia, steatosis, microabscess formation and bile duct changes. The function or failure of engrafted livers was determined by evaluation of liver function tests. The results showed that two histologic features - zone 3 necrosis and neutrophilic microabscesses were associated with abnormal liver function tests. This correlation was particularly strong in the post revascularization biopsies.

An interesting case of Epstein-Barr virus reactivation after OKT-3 therapy following orthotopic liver transplantation was presented by Drs. Markin, Shaw, Wood, Burnett, Brichacek, and Purtilo from the University of Nebraska. Two biopsy specimens from this patient showed changes consistent with graft rejection, i.e. inflammation, bile duct damage and cholestasis. After OKT-3 therapy the portal areas became markedly expanded by an infiltrate composed of plasma cells and immunoblasts rather than lymphocytes. This infiltrate extended into the parenchyma and produced focal hepatocellular necrosis. Frozen material was stained for EB nuclear associated antigen by indirect immunofluorescence. This was detected within lymphoid cells and adjacent hepatic parenchyma. Viral capsid antigen IgG antibodies increased from 1:40 to 1:1280. Cessation of immunosuppression led to resolution of hepatitis. The authors comment that a missed diagnosis of EBV infection is potentially a dangerous situation in transplanted patients.

Dr. Pinto from the Bridgeport Hospital, Connecticut presented a poster studying CEA in bile in patients with hepatobiliary and pancreatic disease. This author studied 28 specimens of bile obtained from the common bile duct during surgery for a variety of hepatobiliary diseases. Ten specimens were from patients with cholelithiasis and pancreatitis and 18 were from patients with a variety of pancreaticobiliary neoplasms. It was found that in benign disease CEA ranged from 1.4 ng/ml to 12.7 ng/ml (mean 4.5 mg/ml). In malignant disease levels between 2.4 ng/ml to over 600 ng/ml (mean 95.8 ng/ml) were obtained. Using a level of 15 ng/ml as a cut off for benign disease the sensitivity of the test was 78% and the specificity 99%.

THE STATEN ISLAND HOSPITAL

Robert R. Pascal, M.D.
Chief, Surgical Pathology

475 Seaview Avenue
Staten Island, New York 10305
Telephone 718-390-9407

March 23, 1987

Members of the Gastrointestinal
Pathology Society
Department of Pathology; Box 22
Harbor-UCLA Medical Center
1000 W. Carson Street
Torrance, CA 90509

Dear Colleague:

I am asking for your assistance in a project I would like to undertake.

I have been impressed with the variations in histologic patterns among colorectal tumors which have been classified as small cell undifferentiated carcinoma (SCUC), and I would like to review as many of them as possible. As you know, the distinction from poorly differentiated basaloid carcinoma, and, sometimes, from malignant melanoma, may be difficult, and various criteria have been used, including immunohistochemistry, silver stains, and E.M. Some pathologists equate, and possibly correctly, SCUC with "malignant carcinoid". What I plan is to correlate the clinical findings with histologic and immunohistologic data among undifferentiated or poorly differentiated colorectal-anal tumors in which the diagnosis of SCUC has been made or considered. I will also include those discovered in adenomas. As I have only a few of these tumors in my own patient population, I am asking for case contributions from members of the GI Pathology Society.

Should you want to assist me, I will need:

1) One or two representative paraffin blocks with or without an H&E slide.
2) Any available clinical data, including therapy and response.
3) Electron micrographs, when available.
4) Results of any histochemical studies that you have performed.

I will return the blocks and slides promptly. All participants will be contacted with results of studies I perform, and their participation will be acknowledged in any publication resulting from this study. I will welcome any additional ideas and collaboration.

Sincerely yours,

Robert R. Pascal

Robert R. Pascal, M.D.

RRP:cmc

Gastrointestinal Pathology Club
Executive Committee Meeting Minutes
March 8, 1987

The meeting was called to order at 8:30 a.m. by Dr. Antonioli in PDR #11 at the The Palmer House Hotel, Chicago. Members in attendance: Drs. Antonioli, Appelman, DeSchryver, Goldman, Haggitt, Lechago, Lewin, Mitros, D. Owen, Rickert and Yardley.

I. The minutes of the meeting of March 9, 1986 were approved as distributed.

II. Financial Report

A. Dr. Rickert presented the 1986-1987 financial summary which is attached to the minutes of the 1987 Annual Business Meeting.

B. Delinquent Dues - Dr. Rickert reported that ten (10) members had not paid 1986-87 dues and four (4) of these were also in arrears for more than one year. Those in arrears for more than one year will receive a final notice (registered mail) requesting payment within two weeks. If payment is not received, membership will automatically be terminated. Those who did not pay last year's dues will receive a notice explaining our dues policy with the next dues notice (to be sent in April). Two members (Drs. Dunn and Enterline) were recommended for emeritus status.

III. Committee Reports

A. Dr. Antonioli announced the new committee assignments for 1987-88, which are included in the minutes of the 1987 Annual Business Meeting.

B. Education

1. Dr. Haggitt reviewed the schedule for the 1987 GIPC Scientific Session.

2. Dr. Haggitt reviewed the program for the 1987 Digestive Disease Week meeting. The session is scheduled for 7:30 pm on Tuesday, May 12, 1987 and will be a clinically oriented symposium on colorectal biopsy in infammatory disorders of the colon. It was recommended that the 1988 program also be clinically oriented.

3. The committee discussed possible topics for next year's GIPC Scientific Session with Dr. Mitros. The consensus was to have the program devoted to the pancreas.

4. The committee discussed the logistics of selecting and presenting awards for the best presentations on gastrointestinal pathology each year at the Academy meetings. The Education Committee was charged with the responsibility to develop a mechanism for making awards which will fit with Academy policy. Dr. Lechago will contact Dr. Nathan Kaufman for appropriate guidelines.

C. Membership /Nomination

1. Dr. Rickert presented the recommendation of the committee in Dr. Kahn's absence. The following new members were endorsed by the Executive Committee For Regular Membership: Drs. Claude Cuvelier, Patrick Dean, Jeremy Jass and Kenneth Klein. For Change from Associate to Regular Membership: Drs. Kim Geisinger and Robert Mark Owings. For Associate Membership: Drs. Jose Jessurun, Audrey Lazenby, Susan Robey, Wade Samowitz and Daniel Schwartz.

2. Dr. Rickert reported that letters have been sent to those Associate members whose five year membership limit will expire within the next two years as a reminder to apply for Regular member status.

3. Requests for Emeritus status for Drs. William Hawk and Alun Wynn-Williams were approved. In addition, as noted earlier, Drs. W.L. Dunn and H.T. Enterline were also recommended and approved for Emeritus membership.

4. Dr. Rickert reported that Dr. Alan Medline had resigned from the GIPC.

5. Dr. Antonioli presented the recommended Nominees for the following offices: Vice-President - Dr. Robert Rickert; Secretary-Treasurer - Dr. Stanley Hamilton.

D. Publications - The report of the Publications Committee (attached to Annual Business Meeting Minutes) was presented by Dr. Appelman. Major items addressed were the committee membership, Publishers Assistance List and relations between GIPC and the American Journal of Surgical Pathology. With respect to the Publishers Assistance List, it was suggested that each year new members be given the questionnaire to fill out.

E. Training Program - Dr. DeSchryver reported that an updated listing of programs would be published

in the American Journal of Surgical Pathology early each year. The possibility of listing in Gastroenterology and in the AGA Newsletter was discussed.

F. Microgrants - Dr. Yardley announced that the Microgrants Committee did not approve the single project submitted for consideration last year. The committee remains committed to this program and welcomes proposals for consideration.

G. International Liaison - Dr. Goldman reported on a meeting of the International Group of Gastrointestinal Pathologists held on September 4, 1986 in Vienna in conjunction with the International Congress of the IAP. Details of the meeting are attached to the minutes of the 1987 Annual Business Meeting. The group will retain its informal status. The next international program will be in Dublin in 1988 and the topic will be interpretation and nomenclature in gastrointestinal biopsies.

H. Newsletter - Drs. Lechago and D. Owen noted that material for the club publication is difficult to obtain. Suggestions for future editions included more "how to" type articles, book reviews and selective reviews of G.I. papers which appear in some of the journals which may not regularly be read by the memberships. The Newsletter will be routinely sent to international liaison group members.

IV. Old Business

A. Name Change - Dr. Rickert reported that 51 ballots were returned concerning the proposed change in name from Gastrointestinal Pathology Club to Gastrointestinal Pathology Society. Forty two members favored a change to "Society" and nine members voted to maintain the name as "Club". The Executive Committee voted by majority to support the wishes of the membership and change the name to Gastrointestinal Pathology Society. If this recommendation is endorsed by two-thirds of the members voting at the Annual Business Meeting, the By-laws will be ammended to reflect the name change.

B. Consultation Service - The question concerning provision of a consultation service by the GIPC was again discussed. A recommendation was made to include this item in the questionnaire to be distributed to the membership.

C. Nomenclature Group - The Committee suggested that Drs. Fred Weinstein and Klaus Lewin prepare an article for the Newsletter addressing the merits of a standardized nomenclature for gastrointestinal pathology.

V. New Business

A. Liaison with the ASGE - The question of closer liaison with the endoscopists was addressed. In view of the great overlap in membership with AGA and that meetings were held concurrently during DDW, it was suggested that the need for a closer relationship be explored with the president of ASGE by Dr. Fred Weinstein.

B. Recognition of past presidents - It was suggested that certificates be prepared for recognition of past presidents of GIPC. It was further recommended that the name of our organization on any certificate be that in use at the time of a past president's tenure.

C. Questionnaire - The Executive Committee recommended that the secretary prepare a questionnaire for distribution to the membership with the next Dues Notice. Among items recommended for the questionnaire are the following:

1. Which committees of the organization would you be willing to serve on?

2. What topics should be considered for future Scientific Sessions of GIPC and for our presentation during Digestive Disease Week?

3. What types of material should be included in Newsletter?

4. Updating of information concerning GI pathology fellowship/training programs.

5. Would you be willing to serve as a consultant on difficult cases in a specialized area of GI pathology? If so, what area?

D. Dr. Yardley reported that a series of workshops on the pathology of inflammatory bowel disease is being sponsored by the NFIC. Participants will include Drs. Yardley, Goldman, Haggitt and Appelman. The NFIC is interested in local support as well. Members who may wish to organize a workshop at a local or regional level should contact Marjorie Merrick at the NFIC office in New York.

VI. There being no further business, the meeting was adjourned at 11:45 AM.

Respectfully yours,

Robert R. Rickert, M.D.
Secretary-Treasurer

GASTROINTESTINAL PATHOLOGY CLUB
ANNUAL BUSINESS MEETING
March 8, 1987

The meeting was called to order at 5:00 pm by Dr. Antonioli in the Red Lacquer Room, Palmer House Hotel, Chicago, Illinois. Members in attendance: Drs. Antonioli, Appelman, Barwick, Chejfec, Cohen, Cooper, Dahms, Dayal, DeSchryver, Fenoglio-Preiser, Ferrell, Goldman, Goldstein, Gourley, Haggitt, Hamilton, E. Kahn, Kelly, Keren, Lechago, Lee, Lev, Lewin, Madara, Marcial, Ming, Mitros, D. Owen, Owings, Pascal, Petras, Qizilbash, Rickert, Riddell, Rotterdam, Saul, Sheahan, Silverman, Smith, Snover, Sternberg, Tomasulo, Ulich, Wirman and Yardley.

I. The minutes of the 1986 Annual Business Meeting were approved as distributed.

II. Financial Report - presented by Dr. Rickert.

Balance as of February 28, 1986.....	$ 7297.73
Receipts March 1, 1986 - February 28, 1987 (Dues).....	2225.00
Receipts March 1, 1986 - February 28, 1987 (Interest).....	459.36
Receipts March 1, 1986 - February 28, 1987 (Contribution from International Academy of Pathology.....	600.00
	$10582.09

Expenses - March 1, 1986 - February 28, 1987

1986 GIPC Meeting	$408.79
1986 AGA (DDW 86) Meeting	457.28
Newsletter	100.00
	$966.07

Balance as of February 28, 1987..... $ 9616.02

III. Committee Reports

A. Dr. Antonioli announced the following committee assignments for 1987-88:

GIPC COMMITTEES

Education Committee	Term Ends
F. Mitros (Chairman)	1989
R. Haggitt	1989
H. Cooper	1988
R. Owen	1988
* S. Sternberg	1990
* J. Wirman	1990

Membership/Nomination Committee

	L. Kahn (Chairman)	
	B. Dahms	1988
	D. Snover	1988
	Y. Dayal	1989
	S. Geller	1989
*	R. Petras	1990
*	S. Saul	1990

Training Programs Committee

	K. DeSchryver (Chairwoman)	1989
	R. Lee	1989
	H. Goldman	1988
	J. Frei	1988
*	E. Cohen	1990
*	H. Shields	1990

Publications Committee

H. Appelman (Chairman)
R. Riddell
* F. Mitros (Ex-officio)
S. Sternberg (Ex-officio)
President
President-elect

* New appointment

B. Education

1. Dr. Haggitt reported on the program planned for the 1987 Digestive Disease Week meetings which will be held on Tuesday, May 12, 1987.

GASTROINTESTINAL PATHOLOGY CLUB
SYMPOSIUM ON COLORECTAL BIOPSY IN INFLAMMATORY DISORDERS OF THE COLON

Tuesday, May 12, 1987
7:30 P.M.
Rodger C. Haggitt, M.D.
Moderator

What is the Role of Colorectal Biopsy in the Diagnosis and Management of Patients with Idiopathic Inflammatory Bowel Disease?

F. Warren Nugent, M.D.
Lahey Clinic Medical Center

Robert H. Riddell, M.D.
McMaster University

What is the Role of Colorectal Biopsy in the Diagnosis and Management of Patients with Self-Limited Colitis and Colitis due to Specific Infections Agents?

Timothy T. Nostrant, M.D.
University of Michigan

Christina M. Surawicz, M.D.
University of Washington

What is the Role of Colorectal Biopsy in the Diagnosis and Management of Patients with Other Inflammatory Disorders of the Colon?

Douglas S. Levine, M.D.
University of Michigan

Cyrus E. Rubin, M.D.
University of Washington

2. It was reported that the first choice for a topic for the 1988 Scientific Session was pancreas. Dr. Mitros and his committee will develop the program.

3. The Education Committee will develop a mechanism for selection and presentation of awards for the best poster and/or paper in gastrointestinal pathology by a resident at the Academy meeting.

C. Membership/Nomination

1. The following were recommended by the Membership/ Nomination Committee and approved by the Executive Committee:

Regular Membership
- Dr. Claude Cuvelier
- Dr. Patrick Dean
- Dr. Jeremy Jass
- Dr. Kenneth Klein

Regular Membership (change from Associate)
- Dr. Kim Geisinger
- Dr. Robert Mark Owings

Associate Membership
- Dr. Jose Jessurun
- Dr. Audrey Lazenby
- Dr. Susan Robey
- Dr. Wade Samowitz
- Dr. Daniel Schwartz

The new members noted above were approved by acclamation.

D. Publications - The attached report of The Publications Committee was briefly presented by Dr. Appelman.

E. Training Program - Dr. DeSchryver reported that an updated listing of programs would be published early each year in The American Journal of Surgical Pathology.

F. Microgrants - Dr. Yardley reported that the single project submitted for consideration during the past year was not approved by the committee. He noted that the committee remains committed to the program and welcomes proposals for consideration.

G. International Liaison - The attached report of the meeting held in Vienna on September 4, 1986 was presented by Dr. Goldman. The group will continue as an informal liaison body.

H. Newsletter - Drs. D. Owen and Lechago noted that material is difficult to obtain for the Newsletter. A questionnaire to be sent with the next Dues Notice will have an item requesting suggestions for the Newsletter.

IV. Old Business - Dr. Rickert reported on the results of the voting on a proposed name change for the organization. Of 51 ballots returned there were 42 in favor of Gastrointestinal Pathology Society and 9 in favor of Gastrointestinal Pathology Club. The Executive Committee earlier today voted by majority to endorse the wishes of those voting to change the name. This recommendation was put before the membership and by overwhelming majority voted to change the name of this organization to Gastrointestinal Pathology Society. The secretary will make the appropriate change in the By-laws.

V. New Business - Dr. Yardley reported that a series of workshops on the pathology of inflammatory bowel disease is being sponsored by the NFIC. Participants will include Drs. Yardley, Goldman, Haggitt and Appelman. The NFIC is interested in local support as well. Members who may wish to organize a workshop at a local or regional level should contact Marjorie Merrick at the NFIC office in New York.

VI. Nomination and Election of New Offices - Dr. Antonioli reported that the Membership/Nomination recommended and the Executive Committee approved the following nominees:

Vice President - Dr. Robert R. Rickert
Secretary-Treasurer - Dr. Stanley Hamilton

No other nominations were reviewed from the floor and the slate noted above was elected by acclamation.

VII. Induction of New President - Dr. Antonioli introduced Dr. Juan Lechago as the new president of the Gastrointestinal Pathology Club (Society). Dr. Lechago thanked Dr. Antonioli for the excellent job he did as our President and invited the assembled members to a reception immediatley following the meeting.

VIII. There being no further business, the meeting was adjourned at 5:45 pm.

Respectfully submitted,

Robert R. Rickert, M.D.
Secretary-Treasurer

International Group of Gastrointestinal Pathologists

A meeting was held on 4 September 1986 at the Hofburg in Vienna, at the time of the International Congress of the IAP. Present were 32 persons from 14 countries, and a list is appended.

1. Dr. Goldman has spoken with representatives of the various societies and clubs of Australia, Britain, France and USA, and the consensus was that our International Group should remain an informal one. This was reaffirmed at the present meeting, and persons who wish to be a part of this group are encouraged to join one of the existing societies that are listed below. A major goal of the Group would be to promote additional national and regional societies. In the past two years, a new society has formed in Germany, and there are continued efforts in Argentina, Hungary, Italy, and Finland. Dr. Morson will contact persons in China to see whether they would be interested in forming a local society. The extra dues for being a part of the International Group will remain at the reasonable rate of nothing.

2. Existing groups: for those interested in obtaining information and possibly joining an available society, they are listed below.

   Dr. Robin Cooke
   President, Gastroenterological Society, Queensland
   Pathology Department, Royal Brisbane Hospital
   Australia 4029

   Dr. Geraint T. Williams
   Secretary, British Society in Gastroenterology
   Pathologists Group
   Department of Pathology, The Welsh National School of Medicine
   Heath Park, Cardiff CP4 4XN, United Kingdom

   Dr. Claude Degott
   Secretary, Club d'Histologie Digestive
   Departmente of Pathology, Hopital Beaujon
   F 92118, Clichy Cedex, France

   Professor H.F. Otto
   Chairman, Arbeitsgemeinschaft Gastroenterologische Pathologie
   Department of Pathology, University of Heidelberg
   Heidelberg, Federal Republic of Germany

   Dr. Robert R. Rickert
   Secretary-Treasurer, Gastrointestinal Pathology Club
   Department of Pathology, St. Barnabas Medical Center
   Livingston, New Jersey 07039, USA

3. Dr. Goldman will continue to oversee the efforts of the Group, and will be assisted by Drs. Cooke (Australia), Camilleri (France), Heilmann (FRG) and Williams (UK).

4. Activities:

   a. The individual societies and clubs should share newsletters and other announcements. Copy of the Club Newsletter from USA will be sent to representatives of the other societies for distribution. Copy of these minutes and future announcements will be sent to Drs. Sobin and Cotton for possible inclusion in the International Pathology News Bulletin (of the IAP).

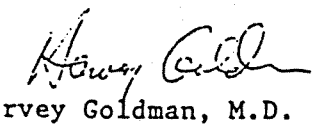
   b. We will attempt to meet on a regular basis at the time of future international sessions. Persons attending the meeting were asked to try to make contact with the organizers of the next European Society of Pathology Congress in Prague (September 1987). We would also hope to participate in the next IAP Congress to be held in Dublin in September 1988 (see below). At these meetings, the International Group will serve to sponsor and provide a symposium, have a business meeting, and possibly add some social function (volunteers needed).

   c. It is further hoped that the Group could serve as a nidus for future collaborative studies.

5. Dublin Meeting:
   We were asked to provide a list of topics that might be included in the Symposium on GI Pathology for that meeting, and this will be transmitted to Dr. Williams who is on the organizing committee. The strongest sentiment appeared to be for the Interpretation and Reporting of Gastrointestinal Mucosal Biopsies. Other topics mentioned included infection, immunology, tumors of the esophagus and stomach (all ~~were~~ or junctional), and neuromuscular disorders.

I thank you all for coming and for supporting the effort, and I look forward to the third round of our Group activity, perhaps in Prague, but definitely in Dublin.

Sincerely,



Harvey Goldman, M.D.
Professor of Pathology
Department of Pathology
Beth Israel Hospital
330 Brookline Avenue
Boston, MA 02215
USA

HG:pkg

REPORT OF PUBLICATIONS COMMITTEE OF THE GASTROINTESTINAL PATHOLOGY CLUB
MARCH 8, 1987

1. The membership of the Publication Committee changes yearly because of changing officers. The membership includes the following:

   a. Chairman - Appelman

   b. Member - Riddell

   c. Exofficio - The Editor of the AJSP, Sternberg

   d. Exofficio - Chairman, Education Committee, Haggitt

   e. Exofficio - President, Antonioli

   f. Exofficio - Vice-president, Lechago

   By six o'clock this evening, f above, will become e, and there will be a new f. The new d will be Frank Mitros.

2. The Publishers Assistance List Questionnaire was distributed to all members in the summer and fall of 1986.

   a. Forty-five of the one-hundred-twelve members polled responded (40%).

   b. Thirty of these forty-five agreed to be on the Publishers Assistance List and noted their preferred topics on the Questionnaire. Thus, these thirty members make up the Publisher's Assistance List.

3. More on the Publishers Assistance List:

   a. The list will probably require periodic up-dating, probably in the form of a modified questionnaire. Within the next year or so, the Publications Committee will look into this issue.

   b. As new members enter the organization, they should be given the questionnaire, and thus given the opportunity to add their names and their chosen topics to the list.

   c. Quite possibly, we may need to define the logistics of the handling of these lists; in other words, we may need to give the Secretary-Treasurer some guidelines as to how to give out the list, to whom to give it, and what credentials are required of the requesting individual in order for that individual to be given the list.

4. Relations between the Club and the American Journal of Surgical Pathology. This is summarized in the report of deliberations of the Publications Committee at their meeting on March 10, 1986 in New Orleans. This report is published in the GIPC Newsletter, Spring-Summer, 1986.

a. The AJSP agreed to publish abstracts of presentations of our Scientific Session if the abstracts are submitted to the AJSP editorial office in November of the year proceeding the scientific session. The abstracts for the 1987 scientific session have been published in the February, 1987 issue of the journal.

b. The full papers will be published as part of a supplement to the AJSP. In order for this supplement to be published by the end of the year, these symposium papers have to be in the hands of the AJSP editor by April 1. The 1986 papers have not yet been published.

5. At the moment, the Publications Committee has no new business to undertake, but the Committee will be delighted to handle any issues as they surface.

Respectfully submitted,
Henry D. Appelman, M.D.
Chairman, Publications Committee

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