THE GASTROINTESTINAL PATHOLOGY SOCIETY NEWSLETTER

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CONTENTS

EDITORIAL: CONTROVERSIES IN GASTROINTESTINAL PATHOLOGY

THE MICROSCOPE VERSUS THE ENDOSCOPE

Throughout world history, the pen has proven mightier than the sword! However, to gastroenterophiles, the important question today is whether the microscope is mightier than the endoscope! It is no exaggeration to say that the invention of the flexible endoscope has revolutionized the clinical practice of gastroenterology. Nowadays, for a busy, financially-oriented physician, an endoscope is the next logical major household appliance to be purachased after a VCR. Pathologists are also grateful for this development, as a free flow of G.I. biopsies certainly improves the laboratory cash flow. The question to be addressed is whether the endoscope is in itself a diganostic tool of primary importance or merely an illuminated conduit providing tissue for the more important microscope.

Now, I must thread carefully because I know that some of our Society members are also practicing endoscopists in their spare time. However, it has been drawn to my attention that there are in existence a number of "scientific" journals devoted exclusively to endoscopic topics. A quick perusal of this literature reveals a sequence of dull reddish colored photographs depicting various kinds of blob. As is to be anticipated, the new discipline of endoscopy requires new terminology. Fortunately, endoscopists have elected not to go the route of the dermatologists and introduce a series of descriptive names in Latin or Greek. We are, therefore, spared the prospect of red streaks in the stomach being called "striae rubrae pyloridis multiplex", or hemorrhoids that could become "polyposis ani hemorrhagica dolorosa perstans et vulgaris". However, one nomenclatural nightmare that has not been avoided is the stealing (yes, grand lacerny!) of specific pathologic terms and applying them indiscriminately to gross endoscopic appearances. Thus, anything redder that the endoscopists face is automatically labelled as being inflammed, even though, upon biopsy, neutrophils, lymphocytes, etc. are either absent or present in normal numbers.

For the sake of a little controversy, let us, therefore, examine critically each segment of our beloved hollow tube and boldly inquire into the value of endoscopy versus microscopy.

Esophagus

Some days, I begin to doubt that there is even such a thing as an abnormal esophageal biopsy! Many a minute scrap of maloriented normal-appearaing squamous epithelium passes under the objective, and all with a confident endoscopic diagnosis of "reflux esophag**itis**". Reflux there may be, but rarely do I see inflammatory cells. Barrett's esophagus can be diagnosed endoscopically, if the gastroenterologist can remember to figure

out exactly where in the esophagus he is looking at. However, the endoscopist cannot diagnose dysplasia in Barrett's. Can the pathologist reliably diagnose it? Right ... maybe we should pass on to the next topic.

Stomach

Would you have your stomach removed simply because the endoscopist said the ulcer was malignant? What about a lymphoma? Can the endoscopist perform 1001 brown stains, and interpret them all accurately? To be charitable, however, we can at least say that, with endoscopy, the correct area of the rather vast stomach is biopsied ... usually. The situation with regards to gastritis is so complicated (see Tower of Babel in this issue) that I hesitate even to mention it! Our endoscopists swear that if they look closely they can see campylobacter, or at least something suspiciously spiral in front of their eyes.

Small Bowel

Strike out! Celiac disease or any other form of villous mischief cannot be diagnosed endoscopically. Giardia and cryptosporidia are also too small to be seen without the magical microscope.

Large Bowel

I have to grudgingly admit that endoscopy is highly effective in finding and removing polyps provided, of course, that the field of view is not obscured by copious quantities of the unmentionable. However, as we know, gross appearances are unreliable in determining the type of polyp and whether carcinoma is present. Endoscopy can also accurately diagnose a patchy inflammatory bowel disease. It can, in addition, diagnose aphthoid ulcers and pseudomembranes; however, granulomas, amebae, and collagen bands are the exclusive realm of the microscope!

Lastly, I must draw your attention to a new disease: microscopic colitis. The patients present with diarrhea. Endoscopy and radiology reveal a normal mucosa, but microscopic examination discloses evidence of inflammation. Thanks to the almighty microscope, these unfortunate suffereres now have a bona fide disease to suffer from, and are no longer labelled as neurotics!

At last, the picture becomes clearer! The endoscope is an extension of the naked eye which can look into dark holes and around sinuous corners. It can be effectively used to spot and describe changes, but it cannot, by itself, really diagnose their fundamental nature.

David Owen, M.D.

GIPS PROGRAM FOR THE 1988 ACADEMY MEETING IN WASHINGTON, D.C.

The GIPS will hold its annual Scientific Session on Sunday, February 28, 1988 at 1:30 p.m. in the International Ballroom West, Washington Hilton Hotel, Washington, D.C. This is the program:

"DISEASES OF THE PANCREAS AND THE PANCREATICOBILIARY TREE"

Moderator: Frank A. Mitros, M.D.
Department of Pathology
University of Iowa
Iowa City, Iowa

1:30 - 2:00 p.m.	**SCLEROSING CHOLANGITIS** JURGEN LUDWIG Mayo Clinic, Rochester Minnesota
2:00 - 2:30 p.m.	**NON-NEOPLASTIC DISEASES OF PANCREAS** JAMES OERTEL A.F.I.P., Washington, D.C.
2:30 - 3:00 p.m.	**EXOCRINE NEOPLASMS OF THE PANCREAS** ERNEST LACK Georgetown University, Washington, D.C.
3:00 - 3:30 p.m.	**RECESS**
3:30 - 4:00 p.m.	**IMMUNOPATHOLOGY AND IMMUNODIAGNOSIS OF PANCREATIC NEOPLASMS** MARGARET TEMPERO University of Nebraska, Omaha, Nebraska
4:00 - 4:30 p.m.	**EXPERIMENTAL PANCREATIC NEOPLASMS** PARVIZ POUR University of Nebraska, Omaha, Nebraska
4:30 - 5:00 p.m.	**EXPERIMENTAL PANCREATITIS** MICHAEL STEER Harvard Medical School, Boston, Massachusetts

AGENDA FOR THE GIPS 1988 ANNUAL BUSINESS MEETING

Sunday, February 28, 1988
Following the Scientific Session
International Ballroom - West
Washington Hilton Hotel, Washington, D.C.

I Approval of Minutes of 1987 Meeting - Dr. Hamilton

II Financial Report - Dr. Hamilton

III Committee Reports

- A. Announcement of new committee assignments - Dr. Lechago
- B. Education - Dr. Mitros
- C. Membership/Nomination - Dr. L. Kahn (after New Business)
- D. Publications - Dr. Appelman
- E. Training Programs - Dr. DeSchryver
- F. Microgrants - Dr. Yardley
- G. International liaison - Dr. Goldman
- H. Newsletter - Dr. D. Owen

IV Old Business

- A. Thanks to membership for response to Questionnaire

V New Business

- A. Vote on continuation of GIPS (Sunset clause of Bylaws)

VI Report of the Membership/Nomination Committee - Dr. L. Kahn

- A. Announcement of New Members
- B. Nomination of Vice President/President Elect

VII Election of Officers

VIII Induction of New President

IX Adjournment

Wine and cheese reception with cash bar will take place in the Caucus Room immediately following the Business Meeting.

MICROSCOPIC COLITIS: NEW CONCEPTS REGARDING PATHOPHYSIOLOGY

Edward L. Lee, M.D.
Department of Pathology
University of Texas Southwestern Medical School

Laboratory Service
Veterans Administration Medical Center
Dallas, Texas

In 1976, Lindstrom described the case of a 48 year old woman with watery diarrhea, accompanied by mucosal inflammation and thickening of the subepithelial collagen table in the colon: he called the condition **collagenous colitis** (10). Read and co-workers reported in 1980 a clinical study on patients with diarrhea of unknown origin which, on microscopic examination of colonic biopsy specimens, was accompanied by non-specific inflammation: they called this presentation **microscopic colitis**, (14). In 1982, Kingham and colleagues described six patients with idiopathic diarrhea who, on microscopic examination of colonic mucosal biopsies, also revealed microscopic colitis (7). Finally, in 1986, a joint report by the G.I. Pathology groups from Johns Hopkins and from Dallas, raised the possibility that the terms **collagenous colitis** and **microscopic colitis** are being applied to the same condition in similar groups of patients (5).

In both conditions, patients typically present with diarrhea of unexplained origin which is resistant to treatment. As a rule, double-contrast barium studies yield normal results, as does proctosigmoidoscopic and/or colonoscopic examination. Microscopic analysis of multiple colonic mucosal biopsies shows cellular injury of the surface epithelium as evidenced by loss of polarity, decreased cell height, decreased mucus production, and focal epithelial detachment. The surface epithelium shows infiltration by variable numbers of lymphocytes, neutrophils and even eosinophils (1,6). The subepithelial collagen layer may be thickened, with an average mean thickness of 15 um, as compared to the normal 3 um seen in control samples. This subepithelial collagen thickening is sometimes patchy and, characteristically, is more severe in the proximal colon (5,6). The lamina propria is expanded by a variable acute and chronic inflammatory infiltrate. Neutrophilic cryptitis is frequently present, but crypt abscesses are rare. The crypt architecture is usually maintained and there is no evidence of ulceration or granuloma formation (1,6).

We prefer the term **microscopic colitis** to **collagenous colitis** because some recent data suggest that the increase in thickness of the subepithelial collagen layer seen in this disease is not the only, or even the most important, factor in the pathophysiology of the diarrhea experienced by such patients (3,4,8,9,12,14,15). On the other hand, the common denominator linking both entities seems to be the inflammation of the lamina propria. Jessurun and colleagues (6) reported the histologic findings in 15 patients with "typical" collagenous colitis. Whereas 6 of 27 specimens showed borderline or no thickening of the subepithelial collagen layer, all cases demonstrated a significant inflammatory infiltrate within the lamina propria and the surface epithelium. A recent morphometric study by Lee and coworkers showed a statistically significant increase in the number of inflammatory cells of the colonic mucosa in patients with microscopic colitis as compared with a matched group of controls (9). Finally, some reports, using sequential biopsy specimens, have emphasized the relapsing and remitting course of the disease, as well as the development of the subepithelial collagen table thickening with the passage of time (12,15).

A thickened subepithelial collagen layer has been described in other disease processes, e.g. carcinoma of the colon, diverticular disease, and hyperplastic colonic polyps. Gledhill and Cole (4) studied the thickness of the subepithelial collagen layer in colonic biopsies from 457 patients: they found that in 171 cases this layer measured between 3 and 10 um, while in 19 cases it was between 10 and 15 um thick. Seven of these 19 patients experienced diarrhea, but there was no mention of inflammation in the lamina propria or of damage to the surface epithelium. Gardiner and coworkers (3) reported a case of colonic adenocarcinoma associated with an abnormally thick subepithelial collagen table: this varied between 18 and 50 um in thickness, with an average of 35 um. This, however, was not associated with an increase in inflammatory cells of the lamina propria, nor was there a history of watery diarrhea or abdominal pain.

Prostaglandins normally stimulate intestinal motility and transport of water and electrolytes, and it has been suggested that they may play a role in the pathogenesis of collagenous/microscopic colitis. Rask-Madsen and coworkers demonstrated markedly elevated values of PGE2 in the feces of patients with this disease, as compared to healthy controls (13). Lawson and Powell demonstrated a high concentration of PGE2 in the lamina propria of the colonic mucosa, and it is likely that neutrophils are the major source of this substance (8). The possible relationship between autoimmune disease and microscopic colitis is an intriguing one. Erlendsson and colleagues reported a case of collagenous colitis associated with arthritis (2). Mielants and Veys studied 15 patients with HLA-B27 related arthritis who also showed evidence of intestinal inflammation: of seven patients studied by ileocolonoscopy, 3 demonstrated the features of microscopic colitis and 4 showed features of Crohn's disease (11). Finally, Zwillich, Lee and coworkers studied a series of

five patients with HLA-B27 related arthritis treated with sulfasalzine and, on microscopic examination of colonic biopsy, all of them showed histopathological features of microscopic colitis (16).

In conclusion, it appears that collagen band thickening is a somewhat variable parameter, whereas inflammation and surface cell damage, accompanied by reduced water and electrolyte absorption, are constant features in collagenous/microscopic colitis. It seems likely, therefore, that the latter features are of primary importance in the pathogenesis of the nearly intractable diarrhea that afflicts these patients.

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THE POETRY CORNER

The inimitable Leslie H. Sobin strikes again! Our irrepressible resident bard probes once more into the Ampulla of Vater with a zeal and a dead aim worth of an E.R.C.P. virtuoso. The following stanzas are reproduced from Dis. Colon Rectum, 30:990,1988, with their kind permission, and for your enjoyment:

Tales of the Ampulla of Vater: XIII

By the shores of Duodenum
it was once upon a time
That a Villus from far Ileum
did up Papilla climb.

To Ampulla he had journeyed
for advice and consultation
On a lesion most peculiar
that was there astride his nation.

'Twas twelve patches short of Cecum
that a narrow cave they found
It was antimesenteric
murky currents eddied round.

Part was villous like the Ileum
but of a sudden flat
With a pitted surface rugal
just as on the gastric mat.

Oh no further could they then proceed
to search for cysts and tumors
Because low pH was burning
from the acrid acid humors.

Then Ampulla asked does acid etch
well into the mucosa
Yes quite deeply said the Villus
sometimes to the subserosa.

Does it ever twist upon a stalk
becoming all ischemic
Even purple said the Villus
wet engorged and hyperhemic.

Great Ampulla sat and pondered
 oh how erudite his smile
Then he spoke with resolution
 there beside the River Bile.

Yes, this lesion is antiquity
 a relic pre-enteric
From the days when yolk sac opened
 to the structures mesenteric.

In some two per hundred duct persists
 a diverticulation
Which is in the main quite symptomless
 though some show complication.

It may telescope within the bowel
 a true intussusception
Or by twisting lead to volvulus
 it's death without correction.

Mucoceles and carcinomas
 of course diverticulitis
And yes, some can even simulate
 acute appendicitis.

So then be on guard Ampulla said
 teach this in your curriculum
Beware the complications of
 a Meckel's diverticulum.

Leslie H. Sobin, M.D.
Armed Forces Institue of Pathology
Washington, D.C.

NOTE FROM THE PRESIDENT: A LOOK BACK, AND FORWARD

By the time the 77th Annual Meeting of the U.S. and Canadian Academy of Pathology adjourns, the reins of our Society will have passed into the very capable hands of Bob Rickert. The natural expectation that he will be as good a President as he was a Secretary-Treasurer evokes visions of a great and productive year ahead for our group.

Coincidentally, I will also be bidding adieu as the coeditor of the Newsletter, which for the next three years will be nursed, and no doubt improved, by the ministrations of Dave Keren and Bill Dobbins. Before bowing out, however, I must pay very special tribute to my coeditor, David Owen. There is no question that without his very hard work, often deceptively hidden behind his irreverent and unique wit, there would have been no Newsletter as we have known it for the last three years.

Any time one pauses for a moment on the road, there is a natural tendency to look back, to see how much progress has been made, and then forward, to catch a glimpse of the unfulfilled goals and the new challenges which lie ahead.

A retrospective look reveals that the growth that turned our initial Club into a thriving and respected Society is now slowing down. Certainly, some new blood is still coming in, but some attrition is also taking place. With maturity, there are quite a few achievements at which we can look back with pride. Our activities, including a listing of Gastrointestinal Pathology training programs, are publicized by the American Journal of Surgical Pathology, of which our Society is a sponsor and to which it contributes members of the editorial board. Our Society also organizes an established and very well attended Companion Meeting to the United States and Canadian Academy of Pathology Meetings, as well as a highly successful scientific program to the Digestive Disease Week sponsored by the American Gastroenterological Association. Our membership, in addition, is instrumental in their contributions to the programs of the Academy, such as running the Gastrointestinal Pathology Specialty Conference and, this year, to the organization of the Long Course. Our Microgrant program has awarded the first support for research this year. We have been well represented also at the international level, and will continue to make our presence felt through contributions by our membership in future programs, such as the International Congress to take place in Dublin this year.

Finally, and very importantly, our Society has made a point of incorporating emerging young pathologists to the different committees that run it, thus preventing "inbreeding" and the resulting stagnation. Furthermore, it gives me special pleasure to have brought into these committees a few well-established and prestigious gastrointestinal pathologists who, for various reasons, had not had the opportunity to contribute their wealth of experience to our group.

What is in store for the future? Immediate goals that come to mind include the crystallization of a consultation service offering both, members and non-members, the subspecialized expertise of leading gastrointestinal pathologists. Another project that can be implemented with a little additional work is the establishment of working committees that will tackle aspects such as classification and nomenclature in some problematic areas of gastrointestinal pathology. Other subjects which loom in the not too distant horizon could include the possible expansion of our activities to other societies, some of which are already "courting" our group. Or the creation of a Journal dealing exclusively with Gastrointestinal Pathology. Or, why not?, the establishment of subspecialty boards in Gastrointestinal Pathology, following the steps of other subspecialties such as Neuro and Hematopathology.

Let us keep in mind, however, that these dreams for the future of our Society will remain just that, dreams, unless the whole membership throws its active support behind the efforts of the executive ranks and their committees. No matter how active and imaginative future executives are, or how hard-working our different committees turn out, still the indispensable ingredient must be contributed by a perception by the rank-and-file, that this is a worthwhile adventure in which ALL of us must be personally involved.

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

THE NOMENCLATURE OF CHRONIC GASTRITIS
THE BASEMENT OF THE TOWER OF BABEL

Pelayo Correa, M.D.

Department of Pathology
Louisiana State University Medical Center
New Orleans, Louisiana

Some of my friends took last year's tour of the Tower of Babel (G.I.P.C. Newsletter 4:15-19,1986) and, after their hangover was over, asked me if there was a chance of getting another "high" (or is it "low"?) with more of the same stuff. Since there are so many Towers of Babel, I was tempted to conduct a second tour, although I do it hesitantly because it may induce my colleagues to offer tours of other Towers of Babel they know better than I, in which case we could have an addiction problem.

The basement of our Tower of Babel, the chronic gastritis labyrinth, was closed down for many years, but there have been recent intrusions by nosy pathologists. For centuries, the conventional wisdom was that chronic gastritis was just one more of the nuisances that together bring us to the grave, and we philosophically call them "aging". But then, in 1862, no other than the French master Cruveilhier told the world that chronic gastritis was a real disease, frequently associated with gastric ulcer (1). Shortly after, in 1870, Fenwick agreed that it was a disease, but linked it to a different morbid condition, namely pernicious anemia (2). Thereafter, the people of this planet lived happily without gastritis for 50 years, or at least they did not complain about it in the medical literature. This Arcadia was rocked by one of our great heroes, William Osler, who decided that there was such a thing as chronic gastritis, but it was a clinical syndrome which people had been loosely calling "dyspepsia", a highly temperamental lady (3). Since not even Osler could come up with a good correlation between symptoms and pathology, the medical writings in the ensuing years reflected a "free-for-all" attitude in which it was moore or less concluded that everybody was wrong, but everybody was right.

The heavenly revelation that cleared this mess apparently came first to Motteram, who made a big discovery: there was more than one kind of gastritis. One was "superficial, and the other was "atrophic" (4). We, humans, danced around this heavenly

inspiration and engaged ourselves in a series of circular arguments which have not yet ended, such as: Is it the ulcer that causes gastritis, or is it the other way around? If pernicious anemia is associated with gastritis, why don't we see ulcers in these patients? Is the antrum more important or is the fundus more important? And on, and on, and on. Out of this jazz concert, the words "ulcer", "perniccious anemia", and "topgraphic distribution" of the lesions kept ringing in our ears. Out of desperation, Strickland blew the dust out of the century-old Fenwick paper to remind us that patients with (or candidates for) pernicious anemia had atrophic gastritis of the corpus only and, since they were very important people, their gastritis should be called Type A (5). Everything else was second class, or Type B. This had some dear members of our former Club screaming that they had seen antral gastritis in pernicious anemia patients (6). This fire was put out by our calm Finnish friends who methodically showed that such gastritis was only superficial and disappeared with advancing age (7). Some of us, Latins, didn't like our gastritis being called a second-class citizen and, to show our revolutionary spirit, we decided to split the infamous Type B into subcategories with fancy names such as "environmental" and "hypersecretory", which we later found to be more fanciful than truthful, since Bockus had not yet been told that not all duodenal ulcers were "hypersecretors". The A vs. B battle seemed to upset other minds who could not fit all their cases into two letters and thus came up with a new category: Type AB or "pangastritis" (sounds rather painful, doesn't it?) (8).

By then we were all agreed and found another fancy word for our agreement: "heterogeneity". There was only one little detail to work out among us: what names to give to our "heterogeneous entities"? Some of our friends insisted with the letter games, even if B meant different things to different people. Others wanted to give them numbers: 1, 2, 3, 4 (9). The problem here is that, by previously agreeing that these entities are "heterogeneous", we gave up the idea that such thing as ulcer-associated gastritis and pernicious anemia-associated gastritis might represent part of a continuum.

Our amazing capacity to discover things that everybody had been seeing for years but did not dare to discuss in the open out of fear of being called some unpleasant name, led some of our colleagues to bring a couple of new kids to the block. One of them, liked to intestinal reflux and appropriately baptized "reflux gastritis" (or "chemical gastritis", <u>sotto voce</u>), seems to be blending in, either unnoticed or unopposed (10). The other kid, on the contrary, made quite a splash when we all realized that the ugly monster, baptized "Campylobacter pyloridis", later renamed "pylori" to keep the Latin philologists happy, had been making fun of all of us from its hideout under the gastric mucus (11). Of course, nobody wanted to grant pathogenicity to the new bug or admit that somebody else saw it first. This nihilistic attitude, however, was shattered when two Australian heroes (?), loyal to the tradition of Metchnikoff and Carrion (possibly with the added encouragement of a couple of whiskeys?) drank pure

cultures of the swarming bugs and convinced the world that their stomachs, indeed, became very inflammed (12,13). By then, we were all willing to believe that the little devils may even cause peptic ulcer and many of us rushed to buy stock in bismuth bullet companies.

With all this old and new material, my "classification mania" led me to place all the proposed names in a hat and, when I threw them on the floor, they rearranged themselves into the Table shown below, which I rushed to our friend Whitehead to be included in his upcoming Bible, "Gastrointestinal Pathology" (Note: all "plugs" in this assay are gratis unless the beneficiary parties wish to make a voluntary contribution to the Gastrointestinal Pathology Society). Before your read the table (which I intend to post as a map at the entrance of our Tower of Babel's basement labyrinth) and start shooting, I will seek haven in the Colombian Andes and try to find a place in the map for the reflux gastritis, just in case any part of the Table is still standing when I come back.

CLASSIFICATION OF CHRONIC GASTRITIS

NAME		SYNONYMS
MORPHOLOGIC	MECHANISTIC	
Non-atrophic		
Superficial	Initial stage of other entities	Simple
Diffuse antral	Hypersecretory? Campylobacter?	Antral Type B Follicular Diffuse Interstitial
*		
Atrophic		
Diffuse corporal	Autoimmune	Type A
Multifocal	Environmental	Dietary Type B Pangastritis Type AB

* Reflux gastritis to be added somewhere

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INTERNATIONAL G.I. PATHOLOGY SCENE

Harvey Goldman, M.D.

The XVII International Congress of the I.A.P. will be held in Dublin, Ireland, on September 4-9, 1988. Information and forms can be obtained from: XVII Congress IAP, 44 Northumberland Road, Dublin 4, IRELAND. Aside from the daily poster and platform presentations, the G.I.-related activities will include: slide seminars on Pathology of the Liver, Pancreas and Gallbladder (September 7), and Gastrointestinal Pathology (September 9); Short Symposium on Reporting of Gastrointestinal Biopsies (September 9); and Long Symposium of Pathology of the Liver in Childhood (September 9). In addition, there will be a social gathering for the pathologists interested in G.I. Pathology on Wednesday evening, September 7 at the Adelaide Hospital. As of this time, formal groups in G.I. Pathology have been organized in U.S.A., U.K., France, Australia, West Germany, Scandinavia, Japan, and China. There is hope that our meeting will help promote the formation of additional groups.

For those among us who are part of the "jet set", there will be the meeting of the International Congresses of Gastroenterology and Digestive Endoscopy in Rome, Italy, from September 4 to the 10, followed by an auxiliary meeting in Bologna, Italy, on September 10-11, 1988.

Finally, for those that must plan their schedules well in advance, the next International Congress of the I.A.P. will be in Buenos Aires, Argentina, in 1990. We can expect at that time that Dr. Lechago will host an appropriate wine-tasting ceremony.

PRELIMINARY PROGRAM OF THE G.I.P.S. SESSION IN THE A.G.A. MEETING IN NEW ORLEANS

As has been done in years past, the Gastrointestinal Pathology Society will present a scientific session at the Digestive Disease Week held by the American Gastroenterological Association in New Orleans. This session will take place on Tuesday, May 17, 1988 from 5:30 to 7:30 p.m. The preliminary program is as follows:

"RECENT ADVANCES IN GASTRITIS"

- **ENDOSCOPIC ASPECTS OF THE GASTRIC MUCOSA**
  WILFRED M. WEINSTEIN, Department of Medicine
  UCLA School of Medicine, Los Angeles, California

- **THE PATHOBIOLOGY OF GASTRITIS**
  PELAYO CORREA, Department of Pathology
  Louisiana State University, New Orleans, Louisiana

- **CAMPYLOBACTER, AND THE PROBLEM IT POSES**
  JONH H. YARDLEY, Department of Pathology
  Johns Hopkins University, Baltimore, Maryland

- **METAPLASIA AND DYSPLASIA IN THE GASTRIC MUCOSA**
  DONALD A. ANTONIOLI, Department of Pathology
  Beth Israel Hospital, Harvard Univ., Boston, Massachusetts

- **NEUROENDOCRINE PROLIFERATIONS OF THE GASTRIC MUCOSA**
  JUAN LECHAGO, Department of Pathology
  U.T. Southwestern Medical School, V.A.M.C., Dallas, Texas