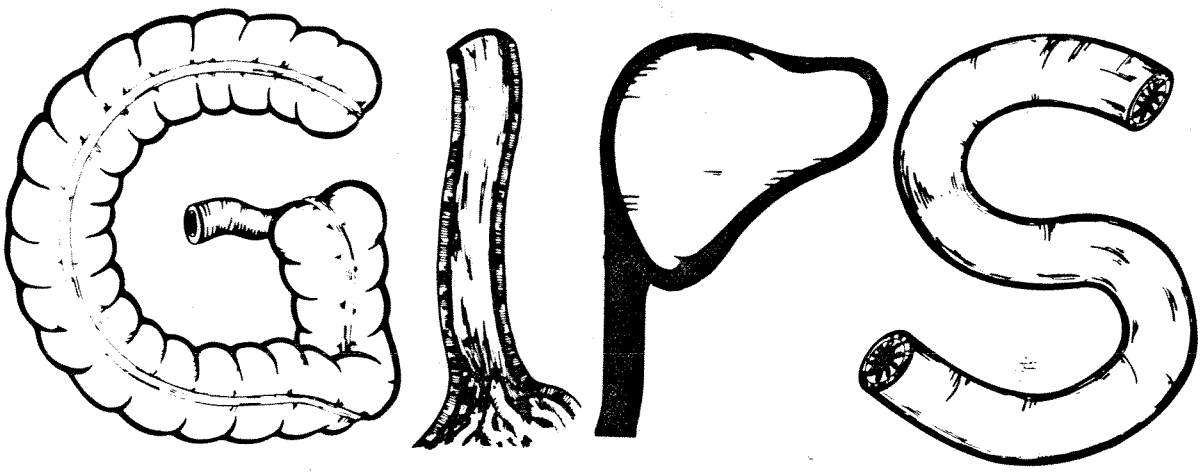

THE GASTROINTESTINAL PATHOLOGY SOCIETY NEWSLETTER

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WINTER 1989

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GASTROINTESTINAL PATHOLOGY SOCIETY
1988-89 OFFICERS AND COMMITTEE MEMBERS

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

New appointments in **boldface characters**

President's Message

In my message to the membership in the Summer 1988 GIPS Newsletter I noted several issues which the Society would be considering during the year. I mention these again since they will constitute important discussion items during our Executive Committee and Annual Business Meetings in San Francisco on March 5, 1989.

First, we will review our participation with the AGA during Digestive Disease Week. Efforts have been made through communication with AGA to improve the dissemination of information about our contribution to their educational program during DDW. Second, we will be considering the possibility of holding a "companion" program in conjunction with the ASCP during their Spring and/or Fall meetings. Finally, we will be discussing the problems associated with publication of the presentations at our Scientific Session in the American Journal of Surgical Pathology. The proceedings now appear in a supplement which is not included as part of the journal subscription; a policy which significantly limits access to these papers. We believe these are all important issues to be addressed by the Society, and we urge your attendance and input.

Elsewhere in the Newsletter you will find the schedule for the GIPS Scientific Session which will be held at 1:30 p.m. on Sunday, March 5, 1989 in San Francisco. Gerry Sheahan, our Education Committee Chairman, has assembled an excellent program on transplantation and the digestive system. We look forward to a large turnout. The Annual Business Meeting will follow immediately. Following the meeting there will be a wine and cheese reception with cash bar (location will be announced at the meeting).

I would like to take this opportunity to thank all of you for your support during my tenure as your president. In San Francisco we will pass the baton to the very capable hands of Gerry Abrams who has worked with the Society since its inception, serving as our first secretary-treasurer. We look forward to continued growth and success as we complete our first decade.

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

Editorial

In this issue of The Gastrointestinal Pathology Society Newsletter, we are pleased to publish a letter to the editor which supports the dialogue suggested in the last issue. The use of this newsletter as a forum for discussion of issues raised at the USCAP meeting, the AGA meeting or in the literature is encouraged by the editor. The informal nature of this communication together with its circulation to an elite group of gastrointestinal pathologists make it an ideal location for your thoughts or concerns on a wide variety of topics. I hope that for the next issue, some of you choose to respond to Dr Makinen's question about microscopic gastritis/colitis and whether these patients require therapy or follow-up.

Also in this issue, we are treated to Dr. Appelman's unique, tongue-in-cheek approach to stromal tumors of the gut. Those of you who have been frustrated in defining some of these lesions will enjoy the extreme views of this extremely well-informed pathologist. No one but Henry Appelman could (or would) have written this article. Remember, you saw it here first!

Lastly, we present the abstracts which will be used at the USCAP meeting of our club in San Francisco in March, 1989. This preview is intended to provide our members with some background information about this rapidly evolving area. Hopefully, it will wet your appetites for learning more about this neglected topic which most of us will be dealing with soon, if we have not been presented with it already.

Submit your contributions for the GIPS Newsletter to the address below.

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LETTERS TO THE EDITOR

To the editor:

We should keep the abbreviation as GIPS - 'GI' being universally recognizable, whereas 'G' can stand for anything such as Godzilla. Also, I really like the bit of esophagus in the logo; it is my favorite letter - possibly because it is the only one in which you can really see pathology - or is the Barrett's esophagus just a figment of my imagination?

Robert H. Riddell, MD, FRCPath, FRCP(C)
Chedoke-McMaster Hospitals
Hamilton, Ontario
Canada

To the editor:

Many thanks for Your Editorial in Vol. 6, No. 2 of the GIPS. This letter is just to let You know, that there is at least somebody in some corner of the world, reading the newsletter You are editing.

As a general pathologist in a district hospital of the city of Lohja (Finland) I really can not write any great science(GS) or even bad science(BS). Anyhow, it would be interesting to hear Your opinion on microscopic gastritis/colitis. Do these patients need any therapy? Do You follow these patients at all?

Being the pathologist of the Finnish group of surgeons dealing with liver transplantation in this country I read the short note on page 8 in the newsletter, concerning the histologic diagnosis of rejection in post-transplant patients. I have difficulties of diagnosing CMV infection in the biopsy material as I almost never see the typical inclusions of this infection in the biopsy specimens. Do You know if Drs. Markin et al. used any specific staining method, immunohistochemistry etc. to make the inclusions visible? As last I would like to encourage You not to stop with the editorials. Even if You were not getting too much post You should be convinced that Your work is not going to land in the wastebasket, but there are always some interested people, wherever they are, who are reading the newsletter with great interest and pleasure.

With friendly greetings to You and the whole editorial office.

Judit Mäkinen, MD, PhD.
Pathologist
Lohja, Finland

Response to Judit Mäkinen:

Dr. Markin, from the Department of Pathology at The University of Nebraska has responded to Dr. Mäkinen's question about the method for detecting the CMV inclusions. At the time of the study, Dr. Markin used in situ hybridization. However, he has since switched to an immunoperoxidase method using an antibody to CMV from DAKO. He observes that greater than 90% of cases with CMV inclusions demonstrated by immunoperoxidase techniques can be detected by the hematoxylin and eosin section. For more details about this study, it has been published in American Journal of Surgical Pathology, 2:362-367, 1988.

Rodney S. Markin, MD, PhD
Department of Pathology and Clinical Chemistry
University of Nebraska Medical Center

John Hart, M.D.
UCLA Medical Center

Disease Recurrence Following Liver Transplantation

Liver transplantation has afforded the chance for cure in many patients with hepatic diseases which previously ran a progressive course culminating in death. In a significant number of patients, however, recurrence of the disease process for which the transplant was performed is a continual source of concern.

Among patients with chronic hepatitis B the incidence of recurrent disease is quite high, dispite immunoprophylaxsis.[1] In fact some authors regard HBsAg and HBeAg positivity as relative contraindications to transplanation.[2] The diagnosis of recurrent hepatitis B can be made quite easily utilizing monoclonal antibodies against HBsAg and HBcAg to identify infected cells in liver biopsies. Serologic tests, although valuable in diagnosing recurrence of infection, may not accurately reflect the extent of hepatocyte damage revealed by biopsy.[1] The diagnosis of recurrent non-A, non-B hepatitis must rely soley on the histologic appearance of a biopsy specimen and the clinical impression. The incidence of recurrent non-A, non-B hepatitis remains undefined at this time.

Early experience with transplantation for hepatic malignancy was disheartening, with the majority of patients dying of recurrent tumor.[3] Efforts to identify and exclude patients with occult metastases or locally advanced disease have been redoubled. The role of perioperative chemotherapy is also being actively explored.[4]

Recurrence of primary sclerosing cholangitis has been the subject of occasional case reports, but substantial experience has not yet accrued. Of much greater concern is the occurence of occult bile duct carcinomas in these patients.[4] These tumors are very difficult to detect preoperatively.

The possibility of recurrence of primary biliary cirrosis (PBC) following transplantation remains controversial. Neuberger, et al[5] reported on three patients in whom the diagnosis was made based on a combination of

clinical findings, laboratory results and histologic features. Histologically the differentiation of recurrent PBC from chronic rejection is difficult, as both apparently involve immunologically mediated events which manifest as damage to bile ductules. Demetris, et al[11] attempted to make the diagnosis of recurrent PBC in their transplant patients utilizing the criteria suggested by Neuberger, et al but were unable to do so. Obviously much further work will be necessary to clarify the natural history of this disease in transplant patients.

Other hepatic diseases that have been reported to recur following transplantation include chronic autoimmune hepatitis and Budd-Chiari syndrome. The complete experience at UCLA with regards to recurrence of hepatits B and non-A, non-B, hepatic malignancies and PBC will be reviewed in detail.

1. Demetris AJ, et al. Recurrent hepatits B in liver allograft recipients: difference between viral hepatitis B and rejection. Am J Pathol 1986; 126: 161-172.

2. Maddrey WC, Van Theil DH. Liver transplantation: an overview. Hepatology 1988; 8: 948-959.

3. Iwatsuki S, et al. Role of liver transplantation in cancer therapy. Ann Surg 1985; 202: 401-407.

4. Colonna II JO, et al. Orthotopic liver transplantation for hepatobiliary malignancy: report of three cases of special interest. Transplantation 1986; 42: 561-62.

5. Neuberger J, et al. Recurrence of primary biliary cirrhosis after liver transplantation. NEJM 1982; 306: 1-4.

6. Demetris AJ, et al. Pathologic analysis of liver transplantation for primary biliary cirrhosis. Hepatology 1988; 8: 939-947.

SMALL BOWEL TRANSPLANTATION

Dr. Barbara Banner

I. INDICATION

A. Vascular insufficiency - thrombosis/embolism; necrotizing enterocolitis

B. Miscellaneous - trauma, Crohn's , radiation, resection; atresias

II. TECHNIQUES

A. Surgery

1. Heterotopic - accessory - recipient S.I. not removed - Thiry-Vella loop
2. Orthotopic - recipient S.I. removed; anastomosis end to end, or ends exteriorized.
3. Ischemia time - 5-6 hours

B. Immunosuppression

1. chemotherapy - Azathioprine, prednisone, Cyclosporine, ALS
2. ablation of lymphoid tissue - radiation, surgery

C. Monitoring

1. Biopsy
2. Absorption tests - maltose, D-xylose, fat, C14 glucose
3. Lab data - serum proteins, triglycerides, stool fat, vitamin levels
4. Clinical - diarrhea, weight gain, nutritional status.

III. SURVIVAL

A. Autografts - Lillehei, Ann. Surg. 150:543 ,1959 - dogs - recovered by 3 weeks. Stools nl. 4-6 weeks. Lymphatics regen. 1-6 weeks. Followed 7 years: motil. and histol normal.

B. Homografts - animal experiments:

Therapy	Graft Survival
1. None	6-9da.
2. Aza. +/- Pred.	m21-42da.
3. CyA +/- Pred.	m24-90da. (some 200 +)
4. Cya + Rad.	m133da.

C. Human trials - 7 reported cases plus two cases with multivisceral tx. : 4 infants, 1 10 yo. and 4 adults. Survivals range from less than 2 days (3 cases) to 7,12, 26 days, to 6 months. The cases with multivisceral tx. lived 3 and 5 months.

IV. BOWEL FUNCTION POST TRANSPLANT

A. Nutritional

1. Acute - transepithelial resistance, Na-glucose absorption and stimulated Cl secretion decrease over first 9 days. (Madara & Kirkman, J. Clin. Inves.75:502,1985)
2. Long term survivors on immunosuppression (usually CyA) :
   a. Clinical: reversible 30% weight loss and diarrhea. Nl. ser. prot./triglyc. (Reznick etal., Can.J. Surg.,25:51,1982; Nordgren etal, Amer.J.

Surg.,147:152,1984; Lee & Schraut, Amer. J. Surg., 151:55,1986; Diliz-Perez etal., Transpl. 37:126,1984)

b. Absorption studies:

(1). D-xylose and C14-glucose - decrease with rejection.(Reznick, Nordgren)

(2). Fat - Absorption of C14 lauric (short chain) and C14 oleic (long chain) fatty acids decrease with onset of rejection(Stamford & Hardy, Surg. 75:496,1974)

(3). Cyclosporine - lipophilic- normal until rejection (Nordgren)

(4). Maltose - absorption decreases before histologic evidence of rejection. Best test for monitoring rejection. (Billiar, J. Surg. Res. 37:75, 1984)

3. Relationship of function to rejection - function maintained until end stage when fibrosis is present.

4. Graft function in humans - ??

B. Motor - spasmotic, uncoordinated electrical activity and vigorous peristalsis on interruption of blood supply; decreased motility during cold ischemia; increased activity and tone on reperfusion. Motor activity maintained until end stage. (Schiller etal. J. Surg. Res. 15:379, ,1973).

V. COMPLICATIONS

A.Technical - vasc. thrombosis (25-50% of early deaths) Perfusion injury.

B. Rejection

1. Early - first 24 hrs. - ischemic injury with congestion, necrosis of villous tips - reversible in 3 days. (Holmes, Gastroent. 61:693, 1971 - classic description)
2. Onset of rejection - 4-6days. See infiltration of mucosa by activated lymphocytes. Untreated, mucosa becomes necrotic. Treated, mucosa returns to normal. (Hardy etal., Ann. Surg.171:51,1970; Rosemurgy & Schraut, Am.J. Surg.151:470,1986; and Holmes)
3. Chronic (postulated) under CyA Rx - lympho-plasmacytic infiltrate around nerves and vessels in bowel wall; mucosa normal; muscle hypertrophy.
4. End stage - fibrosis, obliterative arteriolopathy; stiff bowel

C. Graft vs. host reaction (Deltz etal. Am. J. Surg.151:379,1986; Schraut etal. Transpl.41:286,1986)

D. CyA toxicity ??

E. Lymphoproliferative disorder

VI. GENERAL REFERENCES

1. Deltz E., Thiede A, Hamelmann H. Eds. "Small-Bowel Transplantation. Experimental and Clinical Fundamentals". New York, Springer-Verlag; 1986
2. Schraut W. , Current status of small-bowel transplantation. Gastroent. 94:525,1988
3. Kirkman R.L., Small bowel transplantation. Transplantation 37:429, 1984

Graft-versus-host Disease and the Gastrointestinal Tract

Dale C. Snover, M.D.
University of Minnesota

Graft-versus-host disease (GVHD), that immunological disease caused by attack of immunologically active donor cells against host cells usually following allogeneic bone marrow transplantation, can affect the entire GI tract from lip to anus as well as the liver (1,2). In acute GVHD (generally occurring prior to 100 days post transplant) the stomach, small and large intestine and liver are primary targets whereas in chronic GVHD lip and oral mucosa and liver are the major targets with the remainder of the bowel affected to a lesser degree.

The basic pathology of acute GVHD in the GI tube is necrosis of epithelial cells in the regenerative compartment: the neck region of the stomach and the crypts of the intestine (3,4). The necrosis takes the form of the so-called "exploding crypt cell (ECC)", a vacuole filled with cellular debris. This ECC is characteristic although non-specific. An identical finding is commonly present in neoplastic polyps, carcinomas and bowels afflicted with inflammatory bowel disease, suggesting that this phenomenon is a reflection of cell-mediated killing of epithelium. More pertinent to the differential diagnosis of GVHD, however, is the presence of the ECC secondary to cytoreductive chemotherapy or radiation therapy and in bowels infected with cytomegalovirus (3-5). Because of this, in the first 20 days post transplant GVHD cannot be diagnosed by GI biopsy because of the cytoreductive effect, nor can GVHD be diagnosed (or ruled out) if there are CMV inclusions present in a biopsy. ECC has also been described in patients with acquired immunodeficiency syndrome as "AIDS enteropathy", although the high incidence of CMV in these patients makes it difficult to be certain whether this represents a response to infection or a truly "autoimmune" phenomenon as has been suggested (6).

Although the entire bowel may be affected with GVHD, symptoms will vary with site affected. Small intestinal disease will lead to diarrhea whereas stomach disease often leads to nausea and vomiting (2,5). For this reason, rectal biopsy, although safer and more convenient, will not always be adequate and upper GI biopsy will be needed. If upper endoscopy is performed it is essential to biopsy both duodenum and stomach, even if they appear unremarkable grossly. In our experience in patients with GVHD and simultaneous biopsy of stomach, duodenum and rectum, stomach alone is positive in 21% of cases, duodenum alone in 17% and rectum alone in 8%. We have had essentially no complications from biopsy of the GI tract in these patients.

Acute GVHD is graded on the basis of degree of destruction of crypts, ranging from no crypt loss (grade 1) to total sloughing of mucosa (grade 4) (2,3). Grading has little bearing on prognosis with the exception of Grade 4 disease which is commonly fatal.

Chronic GVHD is a much more generalized disease than acute GVHD, affecting a much wider range of organs (1). From a GI perspective, however, the disease is much more subtle and difficult to diagnose. Oral cavity involvement is common with presentation as lichen planus or sicca syndrome. Lip biopsy is often performed to stage of chronic GVHD. Histological findings in the mucosa are essentially those of lichen planus with basal layer disruption and a submucosal lymphocytic infiltrate whereas in the salivary gland one sees lymphocytic infiltrate with destruction of ducts and acini that may eventually lead to total fibrosis of the gland (1,7). Obviously, these oral manifestations may lead to difficulty eating.

The major manifestations of chronic GVHD of the remainder of the GI tract are those of anorexia, weight loss, malabsorption and sometimes dysmotility. Stricture of the GI tract resembling that found in scleroderma is an uncommon phenomenon. The major histological feature of early chronic GVHD appear to be lymphocytic infiltration and possibly necrosis as in acute disease. However, the epithelium of the GI tract appears to be an uncommon target so that mucosal biopsy is rarely helpful in providing a definitive diagnosis although such biopsy may be useful to rule out concurrent infections of the tract. Fibrosis occurs later in disease and this fibrosis is responsible for the stricturing that is sometimes seen. Recently we have had the opportunity to examine full thickness sections from several patients with chronic GVHD and have found that the major nerve plexuses may be targets of attack. A similar phenomenon has been reported in a dog model of intestinal allograft rejection and it is possible that destruction of nerve may be responsible for the motility problems that are not uncommonly seen (7).

References

1. Sale GE and Shulman HM, eds. The Pathology of Bone Marrow Transplantation. New York: Masson Publishing USA Inc., 1984.
2. McDonald GB, Shulman HM, Sullivan KM, Spencer GD. Intestinal and hepatic complications of human bone marrow transplantation. Gastroenterology 1986;90: 460-477 and 770-784.
3. Sale GE, Shulman HM, McDonald GB, Thomas ED. Gastrointestinal graft-versus-host disease in man. A clinicopathological study of the rectal biopsy. Am J Surg Pathol 1979;3:291-299.
4. Snover DC, Weisdorf SA, Vercellotti GM, et al. The histopathology of gastric and small intestinal graft-versus-host disease following allogeneic bone marrow transplantation. Hum Pathol 1985;16:387-392.
5. Snover DC. Mucosal damage simulating acute graft-versus-host reaction in cytomegalovirus colitis. Transplantation 1985;39:669-670.
6. Kotler DP, Gaetz HP, Lange M, et al. Enteropathy associated with the acquired immunodeficiency syndrome. Ann Int Med 1984;101:421-428.
7. Sale GE, Shulman HM, Schubert MM, et al. Oral and ophthalmic pathology of graft-versus-host disease in man. Predictive value of the lip biopsy. Hum Pathol 1981;12:1022-1030.
8. Banner BF, Dean P, Williams JW. Morphology of rejection in long-surviving canine small bowel transplants. Lab Invest 1987;56:4A.

infection. Occasionally involvement the allograft is present with characteristic histologic features. Adenovirus infection occurs with variable effects. Involvement of the gastrointestinal tract and upper respiratory tract results local symptoms, however, involvement of the allograft usually results in death. Pneumocystis carinii infection occurs occasionally, however, weeks to months after transplant.

The pathologist plays an importand role in the identification of infectious agents or their histologic features. Of particular importance are the correct identification of viral infections since there now specific therapies such as Acyclovir for Herpes virus and Gangcyclovir for CMV. The rapid identification of fungal infection will result in therapy . Evaluation of post-transplant allograft biopsies may yield a diagnosis of cholangitis, abcess or viral infections. Broncho-alveolar lavage(BAL) has become very useful for the rapid identification of viral, fungal and protozoan(PC) infections. The introduction of new technology, such as cDNA hybridization for identification of viral DNA has resulted in rapid therapy for some viral infections. Introduction of polymerase chain reaction(PCR) technology may in the future result in more rapid diagnosis on a greater variety of specimen sources.

- Markin, R.S., Hollins, S., Shaw, B.W., and Wood, R.P. Autopsy Findings in Liver Transplant Patients. Modern Pathology, Submitted.

- Kusne, S., Dummer, J.S., Singh, N., Iwatsuki, S., Makowka, L., Esquivel, C., Tzakis, A.G., Starzl, T.E., and Ho, M. Infections after Liver Transplantation. Medicine, 1988 67(2):132-43.

- Dummer, J.S., Hardy, A., Poorsattar, A., and Ho, M. Transplantation 1983 36:259-67

- Masih, A.S., Linder, J., Shaw, B.W., Wood, R.P., Donovan, J.P., White, R., Markin, R.S. Rapid Identification of Cytomegalovirus in Liver Allograft Biopsies by In Situ Hybridization. Am J. Surg. Path. 1988 12(5):362-7

STROMAL TUMORS OF THE GUT: THE STATE OF THE ARTLESS

Henry D. Appelman, M.D.

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The mystique surrounding gastrointestinal stromal tumors is dependent upon our continuing inability to understand them and to learn anything new about them. As a result, any new, even semi-innovative study of these tumors is eminently publishable, quotable, completely useful for advancing academic careers, and, at the same time, innocuous and intellectually barren, all at the same time. I should know, because I have made my living off of these tumors for the last 20 years, and, to my horror, I find out that my livlihood still depends upon them. As a result, it is important for all of us to recognize that we should never take gastrointestinal stromal tumors seriously, unless we or our significant others have one.

It has come to my attention over the years that almost any kind of stromal tumor can occur anywhere in the gut. The standard lipomas, leiomyomas, glomus tumors, osteogenic sarcomas, chondrosarcomas, and rhabdomyosarcomas, pop up with reportable predictability in various sites, each approximately once per decade except for the lipomas which are fairly common, and the tiny esophageal leiomyomas which everyone has. I have yet to see a typical encapsulated schwannoma attached to a myenteric or submucosal nerve anywhere in the gut, although I suspect somebody else has, perhaps one of the readers of this diatribe. However, I have seen a number of plexiform neurofibromas involving these same nerves in patients with von Recklinghausen's disease. Nevertheless, these are not the most common gut stromal tumors, nor are they the kinds that dominate the world literature. The important gastrointestinal stromal tumors are a group of unusual spindled and epithelioid tumors which don't

appear with great regularity anywhere else except in the gut, although on occasion they pop up in odd places like the retroperitoneum or the myometrium.

This latter group of tumors, indigenous to the gastrointestinal tract, differ in their morphologic expressions, both grossly and microscopically, from one area to another, and their behaviour patterns also differ somewhat with their site of origin. Actually, the whole issue of origin has created a hot and heavy literary debate, inappropriate as it may be. The origin, namely the organ in which the tumor arises, is fairly easy to define. Virtually any of us, highly acclaimed gut pathologists as we are, can tell the stomach from the transverse colon, although I suspect that there are a few in our ranks who find that difficult. On occasion, I have been confused in this area myself. After all, these two structures do lie side by side in the upper abdomen, oriented somewhat in the same direction and with roughly the same external dimensions.

Nevertheless, with these few exceptions, most of us can tell what is the home viscus. What confuses the literature is the fact that people are always going around trying to find the cell of origin. This is ridiculous! Nobody has ever found the cell of origin of a single stromal tumor anywhere in the body, so why should they start trying to find one for the gastrointestinal tumors? Actually, what most people are trying to do is figure out if these tumors have cells which are differentiating into anything in particular. We started out trying to define these differentiating features by light microscopy, and, as a result of that, we call them all smooth muscle tumors of one type or another. Then, since electron microscopes were invented, and since we had to do something with them, we started looking at these tumors under higher magnifications in black and white, and we discovered that it was not so easy to find smooth muscle characteristics in the cells. In fact, most of the cells looked like bits of mesenchymal blob. Nevertheless, a sexy cytoplasmic dense body or two kissing pinocytotic vesicles were enough to throw these tumors into the ultrastructural smooth muscle menagerie.

The introduction of the immunohistochemical detection of cell markers opened the way for a literary explosion. Anyone with a good set of

antibodies could now make a killing in the field. In fact, it wasn't even important to have a good set of antibodies. Someone with a lousy set of antibodies could do just as well, simply by being positive enough to make people think he had a good set of antibodies. As a matter of fact, it was not important if the antibodies were good or bad. Any number of misguided pathologists, thinking their antibodies were good, could use them on these tumors and come up with phenomenally divergent points of view. The important thing was for people with different antibody results to publish their findings in different journals. In this way, certain journals could be marked as favoring certain positions. For instance, there were several groups of hotheads who thought these tumors had smooth muscle markers, who published their results in journal no. 1. Another group of equal hotheads, feeling that their tumors were either undifferentiated or minimally schwannian at best, published their findings in journal no. 2. More recently, there has been a new point of view based upon a whole new set of marker determinants, the plexus group, who we suspect will publish their findings in journal no. 3.

No matter what the point of view, the antibody set, or the journal, the continuing theme in all of the published reports is that the one marker which is present in virtually all of these tumors is vimentin, an antibody of such unimpressive differentiation specificity, that its very universal presence must be offering us some important piece of information, and that is that the cells of the generic forms of gastrointestinal stromal tumors are, in general, undifferentiated. What differentiation they seem to express at any given moment depends upon the antibodies that are available. For instance, in my basement, at this very minute, we are brewing up a concoction of antibodies which we have designated as leiomyin which detects benign smooth muscle tumors, leiomyosarcin which detects malignant smooth muscle tumors, schwannin which detects benign schwann cell tumors, schwannosarcin which detects malignant schwann cell tumors, and the plexus equivalents, plexin and plexosarcin. Using these antibodies in an avidin-biotin system with 14 different chromogens placed on sequentially, these tumors, of different histologic types with different malignant capabilities from different sites, can be divided into 37 different groups, based upon their color characteristics with the 14 chromogens. For instance, there is the blue-grey-purple group from the

lower sixth of the esophagus, compared to the brown-orange-green group from the proximal stomach, the mauve group from the distal duodenum and the heliotrope group from the mid-transverse colon.

It is clear from such studies that the direction of differentiation of these tumors will be worked out in nauseating specificity. What is left, therefore, is to determine their behavior. This has led to a second literary explosion based on how the tumors behave. Do they grow quietly at home or do they metastasize? Where do they metastasize when they do so? Are there some favorite metastatic sites as opposed to some abhorred sites? In order to develop a set of criteria for behavior, it has become necessary to test every parameter we can dream up. We know that the bigger tumors behave worse than the smaller ones, although bigger and smaller differ from one gut site to another. Bigger duodenal tumors, for instance, may be smaller than some of the smaller gastric tumors. Another parameter is the mitotic rate, based upon number of mitoses found in a microscopic field with some unpredictable number of fields accounted. The beauty of this type of measurement is that the size of the microscopic field has not been the same in any two studies, and the number of microscopic fields counted has been equally variable. Furthermore, mitotic counting, being the ultimate in tedium, invariably leads to a slacking off in visual acuity toward the end of the day. Any of us who have counted mitoses recognize clearly that the mitotic counts per high power field decline as the hour grows later. Furthermore, it doesn't matter when you start counting; the same phenomenon will hold. As a result, in the Mitotic Counting Society, a rule was established that counting should be performed for no longer than 22 minutes per day, and only between the hours of 9:00 and 10:30 in the morning. Anyway, this variability has lead to variability in numbers of mitoses considered to be important in predicting good and bad or worse and even worse than that from one study to another.

There are some crazies among us who have decided that cellularity is an important criterion for telling malignant from benign. The cellular tumors tend to be malignant whereas the not-so-cellular tumors tend to be benign. Of course, telling cellular from not-so-cellular can be quite a visual chore, requiring for accuracy a set of templates prominently

displayed over the microscope, to which each microscopic field can be referred. The Cellularity Society has recently published a set of such templates which can actually be placed on the microscopic slide alongside the field to be evaluated, so comparisons can be made effectively. This set of templates sells for $10,000. However, for the stromal tumor devotees, the cost is well worthwhile.

Most recently, DNA analysis in tumor cell nuclei has become the investigational direction of choice. This has become trendy because equipment is prohibitively expensive. However, the machinery is all computerized, so none of us have to think about what we are doing. This has led to a number of critical studies. For instance, we were all surprised to learn that those tumors that were phenomenally aneuploid were likely to be more malignant behaving than those which were not aneuploid at all, especially if they were bigger, more mitotically active, and more cellular. Actually, a study from our institution on a number of gastric stromal tumors, using image analysis coupled with four different interpretive formats, allowed us to take a given stromal tumor and move it in and out of aneuploidy with reckless abandon. In other words, it all depends upon how you look at the data as to what the data means.

Anyway, as you, the readers, can see from the foregoing discussion, gastrointestinal stromal tumors are important. They will continue to supply the newest pathology journals with incomprehensible but publishable manuscripts; they will continue to support the makers of high resolution nuclear DNA analytical equipment, and they will encourage young investigators in gastrointestinal pathology to produce as much nonsense as we, their forebearers, have produced already.

No references have been cited in order to protect both the innocent and the guilty.

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FROM THE INTERNATIONAL SCENE

Howard B. Goldstein, MD
Nyack Hospital, Nyack, NY

A series of unique meetings have occurred in Europe during the past seven years. These have been the gatherings of members of two organizations which are similar to our own Society: the Pathologist's Group of the British Society of Gastroenterology and the french Club D'Histopathologie Digestive. As a french-speaking American anglo/enterophile and a member of both these clubs, I have been privileged to have been present at all five of the reunions which have been held.

Our association began when Dr. Wladimir Bogomoletz of Reims acted as the catalyst to spur Dr. Basil Morson and his Parisian counterpart, Dr. Francois Potet, to agree to try to bridge the language gap, much wider than the Channel, which had long kept pathologists with common interests from knowing each other better. We met in Reims, the capital of Champagne, in 1981. A series of cases were presented in alternating languages and with some mutual difficulty in comprehension. A gala dinner on the evening following the scientific session was held in the cellars of Veuve Cliquot Ponsardin. This meal, based on champagne wines, sauces, regional dishes and cheeses, raised our alcohol and cholesterol levels to a range where we all believed we understood each other thoroughly and decided to meet again, perhaps within two years, in Britain.

Subsequent meetings have been held in London (dinner at the Houses of Parliament), Paris (dinner at la salle capitulaire de Port-Royal), Cardiff, Wales (dinner at Cardiff Castle) and, during October of this year, in Rouen, (dinner at the Palais des Congrès). The parenthetic notations are appropriate as these meetings have evolved into a pleasantly unspoken attempt by each host club to raise the scientific and gastronomic level above that of the previous meeting. These have been truly memorable stimulations of the mind and of the palate. The subsequent gatherings have been marked by improved communication however heavily accented it might be.

In Rouen the meeting was titled the Reunion Franco-Brittanique de Pathologie Digestive. Sixty nine pathologists participated in this meeting and enjoyed an excellent slide seminar. Four cases had been chosen by each of the clubs. Sections were sent east and west and discussers were chosen to analyze and report their interpretation of the challenge cases. The member who had submitted the case would present final comments and discussion of the subject. I list below the cases we shared and note those who participated in each of the rounds as (A) Analyst and (D) Discusser and contributer.

1. Idiopathic nodular regenerative hyperplasia of the liver occurring in a 44 year old woman.
   (A) M. Fabre, Pr. E. Martin (CHU Kremlin Bicetre)
   (D) P.J.Scheuer (Royal Free Hospital, London)

2. Polyclonal plasma cell infiltration of the small intestine in a 29 year old Tunesian man.
(A) K. Henry (Westminster Medical School, London)
(D) J. Nemeth, Pr. A. Galian (Groupe Hospitalier Lariboisiere, Paris)
3. Crohn's disease occurring in a 30 year old man with cystic fibrosis.
(A) F. Berger (Hopital E. Herriot, Lyon)
(D) I.D. Ansell (City Hospital, Nottingham)
4. Ischaemic bowel disease associated with systemic lupus erythematosis.
(A) Pr. N.A. Wright (Hammersmith Hospital, London)
(D) Pr. K. Geboes (UZ St Rafael, Leuven)
5. Hereditary hemorrhagic teleangiectasia of the liver presenting in a 35 year old woman following the birth of her sixth child.
(A) D. Wight (Addenbrooke's Hospital, Cambridge)
(D) M.D. Diebold (Centre Hospitalier Regional, Riems)
6. Gastric antral vascular ectasia "Watermellon stomach".
(A) S. Widgren (Institut de Pathologie, Genève)
(D) M.I.Dixon (University of Leeds Medical School)
7. Wegener's granulomatosis presenting as perianal ulcerations.
(A) H.Thompson (General Hospital,Birmingham)
(D) B.Aymard (CHU, Nancy)
8. Neuroendocrine carcinoma arising in a colonic adenoma.
(A) G.Molas (Hopital Beaujon, Clichy)
(D) P.Anthony (Exeter)

Dr. J.Beurlet of Lyon, currently President of the French division of the I.A.P., presented an expertly composed slide show in which he captured the essence of the previous meetings. It was a moving experience to review the evidence of friendship which has extended beyond these meetings and which has also led to international collaboration in science. Of course this was France and there is the dinner to report:

BARBUE EN LAITUE Au beurre de cidre
AIGUILLETTES DE CANARD AU POMMEAU
Pomme fruit au sucre de canne
Gateau de légumes

SALADE CAUCHOISE
LES FROMAGES NORMANDS
LA SYMPHONIE DE DESSERTS
CAFÉ

COCKTAIL
SAUVIGNON DU HAUT-POITOU
CHATEAU MALHERBES

There is thought being given by the current British secretary, Ashley Price, to try to bring together the American, English and French clubs for a meeting in Québec during 1990 or 1991. This might be followed by visits by their club members to medical centers in the USA. We would probably somehow have to guarantee these friends that we could meet the gastronomic standard. Is anyone else interested?

Book Review: Anorectal Presacral and Sacral Tumors
Localio, Eng & Coppa, W.B. Saunders 1987
Price unknown

This is an interesting book which seems to be aimed somewhat nonspecifically at all having an interest in tumors of this region. It is best suited to those training in surgery. Although it has 18 chapters, the meat of these is in the pathology and pathogenesis of tumors of this region which with the anatomy and physiology of the anus and rectum, take up 10 chapters. There is a huge chapter on surgical treatment. The last 6 chapters are on complications and their management. I looked with particular interest at the anatomy and physiology section; it is done with a series of line diagrams, which in my experience are always much simpler than real life. The anal anatomy is really done quite well, but interestingly the whole concept of the anal transition zone is omitted, as is any of the normal histology, other than for a simple line diagram. The sections on the pathology of the benign and malignant tumors of the anorectum is quite concise and factually pretty well on the money. Because all three major authors are surgeons I tried to find who the pathologist was that supplied much of this factual information. The assistance of Dr. Quentin Valensi, Associate Professor of Clinical Pathology at New York University School of Medicine is acknowledged. In looking through the latest list of GIPS members, he seems not to be on the list. He clearly should be. The histological illustrations are good and are combined with good clinical illustrations in addition. Some of the illustrations are interesting in that the adenoma-carcinoma sequence is illustrated largely using the Spratt-Ackerman diagram in which those authors looked for but failed to find evidence of this sequence. Terms such as focal carcinoma are used that are undefined, as is pseudocarcinoma. In large bowel carcinoma the authors extensively reproduce the (old) TNM classification before going on to state that they do not use it, but these are minor criticisms of a chapter that is otherwise quite well done. The section on "Leiomyosarcoma" of the rectum delves into whether they are really all malignant. The authors choose to use a Broders classification applied to leiomyosarcoma with 4 grades, but fail to relate these to the ultimate prognosis. Nevertheless, the prognosis section puts this in perspective. The chapter on Retrorectal Presacral Tumors is really just a list and literature review but certainly shows the tumors that may be encountered in this region.

I referred the huge chapter on the surgical treatment of these tumors with numerous line diagrams, to one of my surgical colleagues, Dr. John Gately for his opinion. He was really quite impressed with this section of the book and said it goes right along with current practices. John tells me that that the diagrams make it so easy that "even I (meaning me) could do it"! He just comments that he wished it was always as easy in practice as it was in the diagrams. He also thought that the book was good enough to add to his library, which is where it now resides.

The references are a little disappointing in that in some chapters virtually no references are cited after 1980. One therefore wonders whether this book has been "in the works" with a long gestation time. Some references are blatantly incorrect. For instance, Dr. H.G.R. Bussey, is quoted as writing a review article on gastrointestinal polyposis in Gut in 1924, in Vol. 39. Although Dick Bussey officially retired about 10 years ago I think that even he would have been impressed with this, particularly as Gut is currently in Vol. 29.

In summary then, quite a good book for surgeons in training but not one that I would recommend to other members of the Society unless they really wanted to see what their surgical colleagues were up to.

GASTROINTESTINAL PATHOLOGY SOCIETY

Agenda, 1989 Annual Business Meeting

Sunday, March 5, 1989, following Scientific Session

Continental Ballroom 6, San Francisco Hilton Hotel

I. Approval of Minutes of 1988 meeting - Dr. Hamilton

II. Financial report - Dr. Hamilton

III. Committee reports
   a. Announcement of new Committee assignments - Dr. Rickert
   b. Education - Dr. Sheahan
   c. Membership/Nomination - Dr. Qizilbash (to follow new business)
   d. Publications - Dr. Rickert for Dr. Appelman
   e. Training Programs - Dr. DeSchryver
   f. Microgrants - Dr. Yardley
   g. International liaison - Dr. Goldman
   h. Newsletter - Dr. Keren

IV. Old business
   a. Relationship with American Gastroenterological Association
   b. Relationship with American Society for Clinical Pathology

V. New business
   a. Proposed deletion of attendance requirement from By-laws (Article III.E.).

VI. Reports of Membership/Nomination Committee - Dr. Qizilbash
   a. Announcement of new members
   b. Nomination of Vice President/President Elect

VII. Election of officers

VIII. Induction of new President

IX. Adjournment to reception in Cyprus Room, 4th floor, San Francisco Hilton (5:30 PM - 8:00 PM)

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GASTROINTESTINAL PATHOLOGY SOCIETY
ANNUAL REPORT 1988-89

Opening Balance (29 January 1988)		$10,003.00
Credits		
1988 Dues		3,005.00
Interest		400.68
	Total	3,405.68
Debits		
1988 USCAP meeting		
USCAP		1,003.80
Speakers		867.97
1988 AGA meeting		459.45
Microgrant		1,500.00
Secretary-Treasurer expenses		1,077.87
	Total	4,909.09
Closing Balance (31 January 1989)		$ 8,499.59

ANNUALREP.89

GASTROINTESTINAL PATHOLOGY SOCIETY
MINUTES OF ANNUAL BUSINESS MEETING
28 February 1988

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

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

II. Financial Report - Dr. Hamilton.

Balance as of 28 February 1987	9616.02
Credits	
Interest	+ 120.91
Debits	
Secretarial expenses & 1987 meeting reimbursement	- 288.00
Closing balance (Dr. Rickert) and opening balance (Dr. Hamilton) as of 9 June 1987	9448.93
Credits	
1987 dues	+ 2280.00
Interest	+ 284.32
Debits	
1987 USCAP meeting	- 790.75
1987 AGA meeting	- 290.00
Secretary-Treasurer expenses	- 929.50
Balance as of 29 January 1988	$ 10,003.00

The financial report was approved as distributed.

III. Committee Reports

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

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

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

B. Education Committee - Dr. Mitros.

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

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

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

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

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

Regular membership:

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

Associate membership:

Thomas Smyrk

The new members were approved by acclamation.

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

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

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

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

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

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

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

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

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

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

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

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

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BY-LAWS OF THE GASTROINTESTINAL PATHOLOGY SOCIETY

(including Amendments through February 28, 1988)

Article I

Name: Gastrointestinal Pathology Society (GIPS) - Change in name from Gastrointestinal Pathology Club approved by membership on March 8, 1987

Article II

Objectives: The objectives of the GIPS are to disseminate and to increase knowledge about pathology of the gastrointestinal tract and to encourage the development of gastrointestinal pathology as a subspecialty.

Article III

Membership:

A. Categories of Membership -

1. Regular Member. Any individual who has completed post-doctoral training with demonstrated interest and involvement in the field of gastrointestinal pathology as determined by Membership Committee.

2. Associate Member. Any person holding a doctorage (or doctorate-equivalent) degree with an interest in gastrointestinal pathology. This membership is limited to five years and cannot be renewed. Associate members may apply for Regular membership in the usual way at any time if post-doctoral training has been completed.

3. Emeritus Member. A member can become emeritus by requesting this status, in writing, to the Secretary-Treasurer anytime after age 65.

B. Membership Committee -

The President shall appoint five members and one Chairman to the Membership Committee. The term of office will be three years. The initial six appointees shall have their term staggered by lottery, two serving for one year, two serving for two years, and two serving for three years. The Chairman is appointed or reappointed each year by the President from among new and old appointees.

C. Conferring of Membership -

Nomination of an individual to Regular membership can be made by any Regular member. An application form (designed by the Membership Committee) must be countersigned by the Regular member and then sent to the Secretary-Treasurer. The Membership Committee will review the applications and recommend approval or disapproval by the Executive Committee of the GIPS. Once conferred, membership may be retained even though the person discontinues active practice of gastrointestinal pathology.

Nomination for Associate membership can be made by a Regular member. Applicants for Associate membership will not need to provide evidence of their involvement with G.I. pathology in their application. Such decision of involvement will rest with the sponsoring regular members.

All Charter members (persons listed in minutes of the organizational meeting held on March 7, 1979) will automatically become Regular members following approval of the By-laws. However, to retain their Regular membership, Charter members must submit a Regular membership application form within 3 months after receiving the form from the Membership Committee.

Membership can be terminated by written resignation addressed to the President or Secretary-Treasurer.

D. Rights of Members -

Only Regular members will have the right to hold office and to vote. Each Regular member has one vote.

All members have the right to participate in the scientific sessions and in deliberations and discussions at the business meetings.

No member shall use the name, property, or the organization of the GIPS for personal benefit.

Only currently elected officers shall represent the GIPS in official business.

E. Attendance Requirement -

Regular members should attend one annual meeting every three years. Any regular member absent for three successive years will be removed from membership. Reinstatement can take place on written request to the Secretary-Treasurer and approval of the Executive Committee.

Article IV

Governing Body:

A. Elected Offices -

1. President: term of office one year.

2. Vice-President/President-Elect: serves one year as Vice-President and the next year as President.

3. Secretary-Treasurer: term of office three years.

B. Election of Officers -

Any Regular member of GIPS is eligible to hold office. Nominations will be recommended by the Membership Committee and may be offered from the floor by any Regular member. Election oridinarily will be held at the Annual Meeting, or by ballot if deemed necessary by the Executive Committee. A majority vote of the Regular members present, or of all Regular members in case of a mail ballot, is required to elect the officer.

C. Duties of Each Officer -

1. The President shall be the principal executive officer of the GIPS, he shall preside at all meetings, serve as chairman of the Executive Committee, and take responsibility as a representative of the GIPS. The President officially receives donations, bequests, or gifts to the GIPS in behalf of the GIPS. The President shall appoint all standing committees for a term of one year, except, for the Membership Committee, as indicated above. Ad hoc committees are appointed by President as needed.

2. The Vice-President, in absence or incapacity of the President, shall perform the duties of the President. Further, the Vice-President shall serve on the Executive Committee.

3. The Secretary-Treasurer shall keep minutes of the Annual and Executive Committee meetings, distribute notice to members of GIPS, keep custody of documents of GIPS, including mortgages, deeds, and contracts that the Executive Committee has approved, serve on the Executive Committee, receive membership application and dues, keep records of financial documents for governmental agencies, banks or other financial institutions with approval of the President for expenses used solely for the GIPS. All such disbursements will be reported at the Annual Meeting.

D. An officer may not succeed him/herself in office.

Article V

Executive Committee:

A. The Executive Committee shall consit of the currently officers, the past president, and chairman of the standing committees.

B. The Executive Committee shall:

1. Represent the GIPS in official business.

2. Carry out the directives and policies approved by the membership.

3. Organize and coordinate all meetings of the GIPS.

4. Exert leadership in the development and implementation of scientific programs according to the above stated objectives of GIPS.

5. Deal specifically with matters related to the incorporation of GIPS.

Article VI

Meetings

A. Scientific and Business Meeting. This meeting shall be held annually. The time and place will be determined by the Executive Committee. A quorum must be present to conduct business, but the scientific meeting can proceed in absence of a quorum.

B. Special meetings may be called as deemed necessary by the Executive Committee.

C. Quorum* = 1/5 of total Regular members.

Article VII

Standing Committees (to be appointed by President):

A. Education and Specialty Conference. This Committee shall be responsible for design, organization and conduct of scientific meetings and other educational efforts of GIPS.

B. Membership (see Article III).

C. Training Programs. This Committee shall encourage broader availability of organized training opportunities in gastrointestinal pathology and help to upgrade their quality. Committee activities shall include collection and dissemination of information about training programs and identification and development of sources of support for trainees.

D. Publications. This Committee shall review and coordinate arrangements for all publishing activities of the GIPS. Final approval of publishing activities will rest with the Executive Committee.

* Only Regular members are included in quorum.

Article VIII

Amendments:

Amends to the By-laws may be suggested in writing to the Secretary-Treasurer by any Regular member at least two months prior to the Annual Meeting. If approved by the Executive Committee, this amendment needs to be accepted by a two-thirds majority vote of the Regular members at the Annual Meeting.

Article IV

Order of Business at Annual Meeting:

A. Scientific program
B. Business meeting. Determination of quorum.
C. Previous meeting - Secretary-Treasurer.
D. Financial report of Secretary-Treasurer.
E. Reports of committees.
F. Old business.
G. New business.
H. Election of officers.
I. Announcement of new members.
J. Induction of officers.

Article X

Dues and Assessments:

The amount of annual dues shall be determined each year by the Executive Committee. Special assessments may be made by the Executive Committee. New applicants shall be subject to assessments and dues if they participate in GIPS activities while being considered for membership.

After acceptance, new applicants and charter members shall be required to pay an initiation fee not to exceed that of the annual dues.

Any member in arrears of dues for more than one year, failing satisfactory explanation, shall be dropped from membership. Such members may be reinstated on approval of the Executive Committee.

Article XI

Liquidation and Sunset Provision:

A. Liquidation -

Motion for liquidation must be made in writing to the Executive Committee at least 2 months prior to the Annual Meeting. A 2/3 vote of Regular members present at the annual meeting is required.

B. Sunset Provision -

Each three years, the GIPS and its By-laws must be re-approved by a majority of Regular members present at the annual meeting. If this does not occur the GIPS will be dissolved automatically.

C. In the event of liquidation, after payment of obligations, all remaining assets pass to the International Academy of Pathology.

Approved: December 15, 1979
Amended: February 28, 1982
Approved: March 10, 1985
Amended: March 8, 1987
Approved: February 28, 1988