THE GASTROINTESTINAL PATHOLOGY CLUB NEWSLETTER

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SPRING-SUMMER, 1986

CONTENTS

EDITORIAL

"IF IT AIN'T BROKE .... DON'T FIX IT"

This bit of advice derived from popular wisdom would appear to be worth following in the face of the results of the GIPC Questionnaire circulated by Drs. Antonioli and Madara early this year (this issue, page 10). Indeed, we were flattered to learn that 86% of the respondents felt that the format of the GIPC Newsletter was good or excellent, while 89% thought that the contents was good or excellent, with very few dissenting opinions.

However, it would be presumptuous on our part to react to these good news by sitting on our laurels and doing nothing. As a matter of fact, some wag pointed out many years ago that if you are sitting on your laurels probably you are wearing them in the wrong place , anyway. In addition, there were a number of suggestions offered which we feel we should explore. For example, in response to the suggestion that we cut humor of poor taste, we are endeavoring to make this Newsletter as humorless as humanly possible: we are afraid we might be very near achieving such goal. Other well-meaning offerings such as to incorporate work in progress, more critical reviews, and more information including modern techniques are very good but have a catch to them: we need the assistance of our membership. Clearly, your already overworked editors could not possibly come up with such input all by themselves; we have neither the time, nor probably the talent. We felt that contributions such as that of Dr. P. Correa in our last issue offered very valuable insights and a breath of fresh air. May we take this ocassion to invite *all* our readers to join us in making this, your Newsletter, a better publication?

In the meantime, what have we done to nurture the flow of change that will hopefully keep this modest publication vital and interesting? For starters, the availability of better computer hard and software has enabled us to improve, we trust, the format and appearance of the Newsletter. In terms of contents, we have reviewed all GI related contributions at the last IAP Meeting together, whether presented from platform or as posters, so that a more complete picture may be available to the readers. We are also including in this issue a listing of the GI Pathology Training Programs compiled by the Training Programs Committee under the stewardship of Dr. J. Yardley: it does not take much to realize the tremendous effort that putting together such list must have represented.

As for the future, we expect to see further artistic contributions as a counterpoint to Dr. R. Riddell's offering, as well as a growing Poetry Corner now that the potential bards and other creative artists amongst us have been given the green light. Those who, on the other hand, favor prose, please send us your scientific contribution: this Newsletter will thrive and reflect our Club only in the measure that its members care about it and nourish it with their own effort.

Juan Lechago
David A. Owen

GASTROINTESTINAL PATHOLOGY CLUB
MINUTES OF THE EXECUTIVE COMMITTEE MEETING
NEW ORLEANS HILTON - NEW ORLEANS, LOUISIANA
MARCH 9, 1986

The meeting was called to order at 8:30 a.m. by Dr. K. J. Lewin in the Warwick Room, New Orleans Hilton. Members in attendance were: Drs. Antonioli, Appelman, Goldman, L. Kahn, Lechago, Lewin, Madara, D. Owen, Rickert and Yardley.

I. The minutes of the meeting of March 10, 1985 were approved as distributed.

II. Financial Report: presented by Dr. Rickert

A. Financial Summary - see item 3 of this Newsletter

B. Delinquent Dues - Dr. Rickert noted that seven (7) members had not paid 1985-86 dues. They will be reminded of provision in By-laws which calls for dropping from Membership if in arrears for more than one year, failing satisfactory explanation.

III. Committee Reports:

A. Committee Appointments - presented by Dr. Lewin
Please, see minutes of the 1986 meeting, item 3 in Newsletter

B. Education - presented by Dr. Madara

1. Dr. Madara summarized the program for today's GIPC Scientific Session
2. Dr. Madara noted that the program for this year's AGA Session (Digestive Disease Week 86) will be research oriented and 2 hours in length. The speakers will discuss *M cells* (Drs. R. Owen and J. Wolfe) and *Disease Specific Markers in Ulcerative Colitis* (Dr. D. Podowlski). In keeping with our new policy of alternating program formats, next year's program will be clinically oriented.
3. Dr. Madara discussed the results of the questionnaire prepared by him and Dr. Antonioli (attached). The topic receiving the most support and suggested for the 1987 GIPC Scientific Session at the IAP Meeting was *Infectious Aspects of Gastrointestinal Disease*.
4. The Executive Committee members discussed the possibility of developing an award for the best GI pathology presentation (either poster or platform) by a resident, post-graduate trainee, or fellow. Hopefully, an announcement could be made at the time of the Stowell-Orbison Award. Awards of about $ 100 for the winner and $ 50 each for two honorable mention presentations were suggested. The Education Committee will discuss and report back with suggestions for implementation.

C. Membership/Nomination - presented by Dr. Kahn

1. New Members - The committee recommended the following for membership:

Elsa B. Cohen, M.D.	Regular Membership
Kim R. Geisinger, M.D.	Associate Membership
Howard B. Goldstein, M.D.	Regular Membership
Irwin K. Kline, M.D.	Associate Membership
Edward L. Lee, M.D.	Regular Membership
Chan K. Ma, M.D.	Regular Membership
Manuel A. Marcial, M.D.	Regular Membership (formerly Associate)
Jacob J. Steinberg, M.D.	Associate Membership

2. Status of previous Associate Members - The committee affirmed the present policy of a 5 year limit on Associate membership. Responsibility for application to upgrade to Regular membership rests with the member. The Secretary will notify Associate members as they approach the 5 year limit.
3. The Executive Committee recommended that the term of office for the Membership Committee Chairman be 3 years.
4. Nominee for Vice-President - The Committee recommended Dr. Juan Lechago for the next Vice President. Recommendation was enthusiastically endorsed by the Executive Committee.

D. Publications - presented by Dr. Appelman

1. Dr. Appelman updated the Committee on deliberations concerning publication of an Atlas of gastrointestinal pathology by Gower Publishers. The company has decided to identify a specific individual to undertake the project.
2. Dr. Appelman further summarized discussions concerning the relationship between the Gastrointestinal Pathology Club and the American Journal of Surgical Pathology with respect to papers generated from the Scientific Session (see attached Preliminary Report of the Publications Committee; additional communications will follow in the Newsletter).

E. Training Programs - presented by Dr. Yardley

1. Dr. Yardley reported that a listing of training programs will be updated for the next issue of the GIPC Newsletter.
2. Dr. Yardley then presented a recommendation to develop a "microgrant" program to provide small grants as "seed" money to club members to encourage research in gastrointestinal pathology. A limit of $ 2,500 was suggested for disbursement each year with no project requesting more than $ 1,250. An "ad hoc" committee consisting of Drs. Yardley, Antonioli, Haggitt, Lewin, Madara and Rickert was appointed to develop the program. Dr. Yardley will prepare a more formal written proposal (see attached).

F. International Liaison -

Dr. Goldman recommended and the Committee concurred that the Confederation of G.I. Pathology Groups should remain informal and meet periodically at international meetings.

G. Newsletter -

Drs. Lechago and Owen noted that the Newsletter has just been mailed. Comments about the Newsletter made in the recent Questionnaire are attached.

IV. Old Business:

A. Annual Business Meeting - Dr. Rickert reported that eight of the thirteen members responding to our recent questionnaire recommended that the meeting continue to be held immediately following the Scientific Session.

B. Name of Club - Dr. Rickert reported on the results of the recent questionnaire. Twenty-six (26) of the thirty-one (31) responders recommended that the name be changed, and twenty-one (21) of these suggested "Gastrointestinal Pathology Society". The Secretary will send a ballot with the next dues notice. If a majority favor a name change this will be submitted to the Executive Committee as a recommended ammendment to the By-laws. If approved by the Executive Committee, the ammendment needs to be accepted by a two-thirds majority vote of the Regular members present at the 1987 Annual Meeting.

V. New Business:

A. Dr. Lewin referred to correspondence from Dr. W. Weinstein concerning a recommendation for creation of a "nomenclature" group to address problem areas in gastrointestinal pathology. Dr. Weinstein will be asked to further elaborate on his concept of such a group in the Newsletter and additional input from the membership will be solicited.

B. Response to a questionnaire about "direction" of the Club's activities was discussed (see attached).

C. The possibility of developing a social hour with "cash bar" following our Annual Business Meeting was suggested. This suggestion will be further discussed informally and, if interest is great, a recommendation will be made for implementation by the Executive Committee.

VI. There being no further business, the meeting was adjourned by Dr. Lewin at 12:10 p.m.

Respectfully submitted,

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

GASTROINTESTINAL PATHOLOGY CLUB
ANNUAL BUSINESS MEETING
NEW ORLEANS HILTON - NEW ORLEANS, LOUISIANA
MARCH 9, 1986

The meeting was called to order at 5:15 p.m. by Dr. Lewin in the Grand Ballroom D, New Orleans Hilton. Members in attendance were: Drs. Abrams, Antonioli, Appelman, Barwick, Cooper, Correa, Dayal, DeSchryver, Frei, Goldman, Goldstein, Gourley, Hamilton, E. Kahn, L. Kahn, Kelly, Keren, Lechago, R. Lee, S. Lee, Lev, Madara, Marcial, Ming, Mitros, Norris, D. Owen, Owings, Pascal, Qizilbash, Rickert, Riddell, Rotterdam, Sheahan, Smith, Snover, Sobin, Sprinz, Sternberg, Ulich, Wirman, and Yardley.

I. The minutes of the meeting of March 10, 1985 were approved as distributed.

II. Financial Report - presented by Dr. Rickert:

Balance as of February 28, 1985		$ 5,571.87
Receipts 3/1/85 - 2/28/86:	Dues	$ 2,270.00
	Interest	$ 444.84
	Total	$ 8,285.71
Expenses 3/1/85 - 2/28/86:	1985 GIPC Meeting	$ 798.98
	1986 AGA Meeting	$ 150.00
	Newsletter	$ 40.00
	Total	$ 988.98
Balance as of February 28, 1986		$ 7,297.73

III. Committee Reports

A. Dr. Lewin announced the committee assignments for 1986-1987 (please, see item 4 of this Newsletter)

B. Education

1. Dr. Madara noted that the program for this year's AGA Session (Digestive Disease Week '86) will be research oriented and 2 hours in length. The speakers will discuss *M cells* (Drs. R. Owen and J. Wolfe) and *Disease-specific Markers in Ulcerative Colitis* (Dr. D. Podowlski). In keeping with our new policy of alternating program formats, next year's program will be clinically oriented.
2. Dr. Madara discussed the results of the questionnaire prepared by him and Dr. Antonioli (attached). The topic that received most support and was suggested for the 1987 GIPC Scientific Session was *Infectious Aspects of Gastrointestinal Disease*.

C. Membership/Nomination (please, see VI and VII)

D. Publications

1. Dr. Appelman reported that his committee has had preliminary discussions concerning the relationship of the GIPC and the American Journal of Surgical Pathology with respect to papers generated from the Scientific Session. Another meeting will be held on 3/10/86 and a report will be circulated in the next GIPC Newsletter.
2. Dr. Appelman noted in follow-up to last year's meeting that Gower Publishers had decided to select a specific individual to undertake the G.I. Pathology Atlas Project.

E. Training Programs

Dr. Yardley reported that a listing of training programs will be updated for the next issue of the GIPC Newsletter. Additional items were discussed under New Business.

F. International Liaison

Dr. Goldman reported that the Confederation of G.I. Pathology Groups will remain informal and meet periodically at international meetings.

G. Newsletter

Drs. Lechago and Owen noted that the latest issue of the GIPC Newsletter has just been mailed to the membership.

IV. Old Business

A. Annual Business Meeting - Dr. Rickert reported that eight of the thirteen members responding to our recent questionnaire recommended that the meeting continue to be held immediately following the Scientific Session.

B. Name of the Club - Dr. Rickert reported on the results of our recent questionnaire. Twenty-six (26) of the thirty-one (31) responders recommended that the name be changed, and twenty-one (21) of these suggested "Gastrointestinal Pathology Society". The Secretary will send a ballot with the next dues notice. If a majority favor a name change this will be submitted to the Executive Committee as a recommended ammendment to the By-laws. If approved by the Executive Committee, the ammendment needs to be accepted by a two-thirds majority vote of the Regular members present at the 1987 Annual Meeting.

V. New Business

A. It was reported that the Executive Committee discussed a recommendation to develop a "microgrant" program to provide small grants as "seed' money to Club members to encourage research in gastrointestinal pathology. Dr. Yardley will lead an "ad hoc" committee charged to develop further details of this program.

B. Dr. Yardley discussed a proposal for an award to be given for the best G.I. Pathology presentation (either poster or platform). Additional discussions will be summarized in future issues of the Newsletter.

VI. Report of Membership/Nomination Committee

A. It was announced that the chairperson of this Committee will serve for 3 years.

B. Dr. Kahn presented the following new members recommended by the Membership/Nomination Committee and approved by the Executive Committee"

Elsa B. Cohen, M.D.	Regular Membership
Kim R. Geisinger, M.D.	Associate Membership
Howard B. Goldstein, M.D.	Regular Membership
Irwin K, Kline, M.D.	Associate Membership
Edward L. Lee, M.D.	Regular Membership
Chan K. Ma, M.D.	Regular Membership
Manuel Marcial, M.D.	Regular Membership (from Associate)
Jacob J. Steinberg, M.D.	Associate Membership

C. Dr. Kahn announced that Dr. Juan Lechago was nominated as the next Vice President. No additional nominations were needed and Dr. Lechago was elected by acclamation.

VII. Induction of the New President - Dr. Lewin introduced Dr. Donald Antonioli as the new President of the Gastrointestinal Pathology Club. Dr. Antonioli thanked Dr. Lewin on behalf of the membership for the fine job he did as our President.

VIII. There being no further business, the meeting was adjourned at 6:00 p.m.

Respectfully submitted,

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

TABLE OF OFFICERS AND COMMITTEES 1986 - 1987

Executive Committee: 1986-87

Donald A. Antonioli, President
Juan Lechago, Vice President, President Elect
Klaus J. Lewin, Past President
Robert R. Rickert, Secretary Treasurer
Rodger C. Haggit, Chairman, Education Committee
Leonard Kahn, Chairman, Membership/Nomination Committee
John H. Yardley, Chairman, Training Programs Committee
Henry D. Appelman, Chairman, Publications Committee

Education Committee

		Term Ends
*	R. Haggitt (Chairman)	
	W. Weinstein	1989
	H. Rotterdam	1987
	H. Cooper	1987
	R. Owen	1988
*	F. Mitros	1988
		1989

Membership/Nomination Committee

	L. Kahn (Chairman)	
	P. Manley	1987
	B. Dahms	1987
	D. Snover	1988
*	Y. Dayal	1988
*	S. Geller	1989
		1989

Training Programs Committee

	J. Yardley (Chairman)	
	R. Haggitt	1987
	H. Goldman	1987
	J. Frei	1988
	K. Deschryver	1988
*	R. Lee	1989
		1989

Publications Committee

*	H. Appelman (Chairman)
	R. Riddell
*	J. Madara (Ex officio)
	S. Sternberg (Ex officio)

Newsletter Editors

J. Lechago
D. Owen

International Liaison

H. Goldman

* New appointment

SUMMARY OF THE MEETING OF THE PUBLICATION COMMITTEE OF THE GASTROINTESTINAL PATHOLOGY CLUB MARCH 10, 1986, NEW ORLEANS

Members Present: Drs. H. Appelman, R. Riddell, S. Sternberg, J. Madara, K. Lewin, and D. Antonioli.

1. Submission of papers from the GIPC Scientific Session for publication in the American Journal of Surgical Pathology: According to Steve Sternberg, the deadline of symposium papers to be in the hands of the editorial offices of the AJSP is April 1st in order to have a supplement published by the end of the same year. Therefore, ideally, the manuscripts should be submitted to the Chairman of the Education Committee at the time of the Scientific Session.

   It is recommended that if a scientific session derived manuscript is not submitted to the Chairman of the Education Committee in time for inclusion in the symposium supplement of the AJSP, the the author or authors be instructed by that Chairman that the manuscript may be submitted to any journal *with the inclusion of a footnote* stating that the manuscript was presented as part of the Scientific Session of the Gastrointestinal Pathology Club on the specific date and in the specific city.

2. The AJSP will publish abstracts of the presentations at the Scientific Session, but the journal prefers that these abstracts be in the January issue prior to the meeting. This means that they must be submitted in November of the previous year. The AJSP Editorial Board will define an abstract policy regarding length, format, citations, etc. As far as the GIPC is concerned, the purpose of such abstracts is mainly for advertising of Scientific Sessions. Enforcement will become another critical issue in relation to abstracts, since, if all abstracts are not submitted by the November deadline, there is little point in publishing them.

3. The Publication Committee recommends that each member of the Club be polled as to whether he or she wishes to be included on a publisher's assistance list. Such a list will be compiled for publishers who are looking for authors or editors of books or journals in gastrointestinal pathology. The list will be kept in the hands of the Office of the Secretary-Treasurer, and all inquiries to members from publishers may be referred to that office, if the members wish. It must be understood that this list will be for no other purposes and will be given to no one else.

4. The Publications Committee recommends that it should not become a clearing house for books in press, books in preparation, chapters in books, either in press or in preparation or commissioned of for any other literary works the members of the Club are undertaking or plan to undertake.

RESULTS OF THE GIPC QUESTIONNAIRE: MARCH 1, 1986
Submitted by Drs. D. Antonioli and J. Madara

The results presented here are of a total of 37 respondants:

1) Scientific Session at IAP Meeting:

A) Approve 97%

B) Make standard format .....Yes: 65%
No: 8%
Wait and evaluate: 16%

C) Format change suggestions .. Increase basic research: 5%
2 hr specialized/ 1 hr general: 3%
mix research and general: 3%

D) General Topics for Scientific Session

Infection (35%)
Mesenchymal tumors (16%)
Malabsorption/small bowel biopsy (11%)
Anal/perianal tumors (<10%)
Transplantation/GVHD (<10%)
Pathophysiology concepts (<10%)
Genetic/developmental disorders (<10%)
Immunopathology (<10%)
Peptic diseases (<5%)
Endocrine pathology (<5%)
Histochemistry (<5%)
Connective tissue/vascular disorders (<5%)
Metaplasia (<5%)
Gastritis (<5%)
Liver diseases (<5%)

E) Members Interest Subjects

Immunoperoxidase (19%)
DNA probes (16%)
Neoplastic polyps (14%)
Research (10% or less)
Flow cytometry (10% or less)
Pancreas (10% or less)
Cytoprotection (5% or less)
Techniques in surgical pathology (5% or less)
Lectins (5% or less)
Systemic disease (5% or less)
Ulcerative colitis/dysplasia (5% or less)
Neurologic disorders (5% or less)
Cell kinetics (5% or less)

2) Newsletter

A) Format:

Excellent	27%
Good	59%
Fair	5%
Poor	3%

B) Content:

Excellent	27%
Good	62%
Fair	5%
Poor	0%

Suggestions for Improvement:

Include work in progress
Cut humor of poor taste
More information, modern techniques
More critical reviews
List new publications
Bibliography or new important articles
Listing of GI-related meetings

3) Other Activities

A) Development of consultative services: ..... Yes: 70%
No: 22%

B) Other directions:

IAP-related problem slide consultation
Quality control mechanisms
Reception for Club members after meeting (widely acclaimed!)
GI Path fellowship fund
Development of position papers

4) Other Meetings Attended by Membership

AGA	43%
ASCP	16%
ASSLD	16%
CAP	3%
APA	3%

G.I. PAPERS AT THE IAP MEETING IN NEW ORLEANS

As pointed out in our last issue, there has been an upsurge in the quality and quantity of poster presentations. Therefore, in a departure from past issues, we have decided to present our readers with an analysis of all G.I.-related papers at the last IAP Meeting, irrespective of whether they were presented from platform or at poster sessions. We feel that this not only is fairer to authors and readers, but also presents a better integrated picture of the G.I. input at that Meeting. We have divided our analysis into the different areas of interest:

Digestive Tube:

Esophagus.

As has been the trend in the last few years, Barrett's esophagus has been a very popular subject among those interested in esophageal pathology. Dr. Qualman and coworkers from the Children's Hospital in Columbus, Ohio, studied the histopathology of Barrett's esophagus in children. They found that incomplete intestinal metaplasia was present before the age of 7, but complete intestinal metaplasia, as characterized by the presence of goblet cells, was noted after this age and progressed in severity to young adulthood. As might be expected, endocrine and Paneth cells were seen in these patients only after the age of 7. No epithelial atypia was appreciated in pediatric patients with Barrett's esophagus. Dr. Layfield and collaborators from the University of California, Los Angeles and Irvine, examined 66 biopsies from 14 patients with Barrett's esophagus for the presence of endocrine cells containing serotonin, secretin, calcitonin, motilin, ACTH, PP, and somatostatin. Focal immunoreactivity was found for all these hormones, varying from one case to another. Their distribution pattern appeared to be independent from the type of metaplastic epithelium present. Dr. Samowitz and his colleagues from Johns Hopkins University studied the expression of the *ras* gene product *p21* in adenocarcinoma arising from Barrett's esophagus. This gene has previously been shown to have enhanced expression in colonic and breast carcinomas. Using a monoclonal antibody, the authors showed a similar enhancement in Barrett's-derived carcinomas (17/17 cases studied). Focal positivity was also seen in 22/23 dysplastic and 8/8 non-dysplastic Barrett's mucosa. They conclude that the role of enhanced *ras* gene expression in the carcinogenic sequence as well as the diagnostic utility of its demonstration are currently uncertain.

Drs. Di Constanzo and Urmacher from Memorial Hospital in New York presented the findings in seven cases of primary esophageal melanoma. They noted that this tumor is so rare that 34 years were required to collect these cases. The age range was from 30 to 74 years (average 59) and the sex ratio was similar. There appeared to be no specific antecedent features or family history. The tumors tended to be polypoidal and presented with obstruction or hemorrhage. Histologically, both spindle and epithelioid cells were present and many neoplasms also had an in-situ component. Immunocytochemistry was negative for CEA and cytokeratin but positive for S100 protein. Treatment was by esophagectomy. Four patients were dead by 7 months, one died at 55 months, and two are alive at 3.5 and 9 months after operation.

Dr. Jesserun and coworkers from Johns Hopkins University presented a work dealing with the interpretation of esophageal "balloon cells" which have on occasions been misinterpreted as glycogenic acanthosis. They showed that the presence of these cells correlates positively with esophageal injury and particularly with reflux esophagitis. The ballooning is the result of cytoplasmic uptake of plasma proteins causing a "dilution" of the normal eosinophilia in the cytoplasm of the squamous mucosal cells. These balloon cells were noted to be present independently of the classical features of esophageal inflammation and the authors suggest that their presence may be one of the criteria utilized for the diagnosis of esophagitis.

Stomach.

Dr. Geisinger and his colleagues from the Wake Forest University and the Bowman-Grey School of Medicine in North Carolina presented a clinico-pathologic analysis of seven cases of gastric antral vascular ectasia. This previously undescribed condition is manifested as upper G.I. bleeding, and the diagnosis may be strongly suspected at endoscopy. In most cases (5) antrectomy was required to stop the bleeding, although the clinical outcome was uniformly good. The average age of the patients was 55 and 6 were females. Associated conditions included scleroderma (1), pernicious anemia (2), and non-alcoholic cirrhosis without gastric varices (2). Morphologically, the stomachs showed dilated vessels in the submucosa, fibrin thrombi and/or ectasia of mucosal capillaries, and spindle cell proliferation (likely myofibroblastic).

Dr. Paull and coworkers from Johns Hopkins University investigated the presence of campylobacter-like organisms in 246 upper G.I. biopsies from 102 unselected adult patients using Giemsa stained sections. They found a strong correlation between the presence of organisms and the histologic finding of both chronic and active (neutrophils present) gastritis. A correlation between intestinal metaplasia and organisms was also shown but was less clear cut. Organisms were not found in esophageal biopsies, and were seen in only 1/39 duodenal biopsies.

An experimental work dealing with cytoprotection of the gastric mucosa was presented by Dr. Weinstein and coauthors from the Rush Medical College in Chicago and the Weitzmann Institute in Israel. Absolute ethanol was used as the damaging agent and the prostaglandins MPGE1 or PGE2 were used as protective substances. It was found that alcohol changed erythrocytes from biconcave discs to spiculated cells (echynocytes) and also caused the appearance of thrombi. Similar changes in the shape of isolated red blood cells were found in vitro using 1-2% ethanol. The degree of shape change paralleled that of erythrocyte agglutinability. Adding either prostaglandin to the system prevented the changes in agglutination but not those in shape. The authors suggested that prostaglandins act as cytoprotectors by stabilizing the RBC surface properties and maintaining adequate blood flow.

Small Bowel.

Dr. Newman and coworkers from the University of Minnesota reported a detailed study of gastric metaplasia of the duodenum. Simple metaplasia (the presence of gastric foveolar epithelium) and fundic gland

heterotopia (the presence of parietal cells and gastric foveolar epithelium) were recognized. Simple metaplasia was preent in 25% of cases with fundic heterotopia being found in 5% of the samples examined. The corresponding endoscopic appearances were variable but included prominent folds and small nodules. Other histological findings included prominent Brunner's glands, inflammation, GVHD, CMV infection, and ulceration. In one patient who had a bleeding duodenal ulcer, resection was required and the heterotopic gastric mucosa was found to be in continuity with antral gastric mucosa.

Dr. Carey and his coauthors from the University of Michigan in Ann Arbor presented a case study of beef protein allergy occurring in a five year old boy. After a month of abstinence from beef protein, this child was given an oral challenge. Biopsies taken before and two hours after challenge, showed a marked increase in small bowel IgE-secreting plasma cells (presumed evidence of type I hypersensitivity). However, the post challenge biopsy also showed an increase in IgG-containing plasma cells and of IgG in the surface epithelium. This was accompanied by mucosal edema and a neutrophilic infiltrate (presumed evidence of type III hypersensitivity). This child, therefore, appeared to have two possible mechanisms of mucosal damage.

Dr. Barwick and colleagues from Yale University presented a paper dealing with the cytoskeleton of the human small intestinal brush border. The study was prompted by the observation that chicken brush border contains unique proteins and it was felt that a similar situation in man could be exploited for experimental and diagnostic purposes. A protein analogous to the avian counterpart was found in man, and it appeared to be specific for the intestinal brush border. Isolation and further characterization of this protein are in progress.

Large Bowel.

Another paper dealing with the cytoskeleton was presented by Dr. Kahn and coworkers from Toronto. They used immunocytochemistry and gel electrophoresis to examine primary and metastatic breast and colon carcinomas. Immunoreactivity with keratin showed little difference between the two tumor primary sites. However, with actin antibodies, staining concentrated in the apical cytoplasmic margin of 85% of the colonic tumors but only in 10% of breast metastatic tumors. They concluded that this may be useful in practical diagnosis when the primary site of a metastasis is unknown. Dr. Banner and colleagues from Rush Medical College in Chicago and the Montefiore Hospital in Bronx, N.Y., retrospectively looked for DNA aneuploidy in benign colonic polyps by flow cytometry. They found that 14/68 polyps (21%) were aneuploid: this feature was found mostly in villous polyps and those with severe dysplasia or CIS. It was concluded that aneuploidy tends to affect polyps with a known risk of developing cancer and may occur before histologic evidence of malignant change. Dr. Nash and colleagues from Cedars-Sinai Medical Center in Los Angeles reported the evaluation of AIN (anal intraepithelial neoplasia) in homosexual males. They analized all anal tissues received in their laboratory during 1984 (180 specimens from males and 118 from females). It was found that 7% of tissues from males but only 0.8% from females contained foci of atypical epithelium: 72% of males with foci of atypia were definite or probable homosexuals. Three distinct types of atypia were identified: a) AIN

(dysplasia), b) condylomas with atypia and koilocytosis, and c) Bowenoid papulosis. More than one type of atypia could be present in the same specimen. It was postulated that these lesions will behave in the same way as similar atypias in the female genital tract.

A study of experimental verotoxin-producing E. coli infection was presented by Kelly and colleagues from the University of Calgary in Alberta, Canada. Rabbits were innoculated intragastrically with the verotoxin(VT)-producing organisms. Adhesion of the bacteria to the surface of the small bowel mucosa was demonstrated whereas, in the colon, there was transient adhesion but a severe colitis. VT given alone also produced a colitis. Non-VT producing organisms failed to elicit colitis. The authors suggest that both the presence of toxin and the adherence of bacteria are required for the development of colitis. To complement this paper, the same group of workers from Alberta presented the histology of rectal biopsies from fifteen patients with bloody diarrhea and colitis due to the same VT-producing organism. Examination of the rectal biopsies revealed that 7/15 had a neutrophil infiltrate of the lamina propria with crypt abscesses being present in 4 cases, 6/15 had only a focal increase in chronic inflammatory cells within the lamina propria, and 2/15 had normal histology. There was no correlation between biopsy appearance and duration of the symptoms or the serotype of E. coli.

Dr. Jessurun and coworkers from Johns Hopkins University presented a clinicopathologic study of 15 cases of collagenous colitis. Fourteen of their patients were women and the mean age was 65.9 years. They all presented with watery diarrhea and biopsies revealed that the subepithelial collagen band had increased to an average of 8 times the normal thickness throughout the colon, the rectum being the least involved segment. All cases showed evidence of either active or inactive inflammatory bowel disease, and responded symptomatically and histologically to antiinflammatory therapy. No changes were observed in the small bowel except for one patient who had Crohn's disease of this gut segment. Drs. Mitros and Johlin from the University of Iowa investigated in a systematic fashion the deposition of hemosiderin within macrophages of the colonic lamina propria. Both H&E and iron stains were used to study 11 cases of diverticular disease, 10 cases of Crohn's disease, 10 cases of ulcerative colitis, and 10 cases of colon cancer using non-neoplastic mucosa adjacent to the tumor. They found that 9/20 cases were positive for iron in inflammatory bowel disease, 4/11 were positive in diverticular disease, and 2/10 were positive in cancer patients. The distribution of the iron pigment correlated with the depth and density of the disease process. The authors concluded that, contrary to widespread belief, hemosiderin deposition is not diagnostic for classical ischemic colitis. Dr. Qilzilbash and colleagues from McMaster University in Hamilton, Ontario, presented an interesting paper about 13 patients who developed an acute self-limiting colitis after colonoscopy. The authors were able to prove in convincing fashion that this non-specific colitis was caused by the glutaraldehyde solution utilized to sterilize the colonoscope.

From Massachusetts General Hospital and Harvard Medical School, Dr. Ross and his coworkers presented a paper on the experimental production of acute colitis and the augmentation of colonic carcinogenesis by formyl-norleucyl phenylalanine (FNLP). In this series of experiments, rats were given injections of dimethyl hudrazine (DMH), a known potent colonic

carcinogen. In addition, some rats received FNLP administered as an intermittent enema. This formylated peptide causes an intense acute inflammatory response and colonic edema. The authors showed that 70% of the rats that received carcinogen and inflammatory agent developed cancer, whereas only 32% of the rats that received carcinogen alone did so. In addition, the rats getting FNLP and DMH were more likely to develop multiple cancers. The authors postulate that inflammation may promote carcinogenesis by the release of oxygen free radicals from activated phagocytic laukocytes.

Chronic inflammatory bowel disease and dysplasia was, as usual, a very popular subject in this meeting. Dr. Cooper and colleagues from the Jefferson Medical College of Philadelphia presented a study on peanut lectin binding sites in the colon of patients with ulcerative colitis. This lectin is known to bind the T blood group antigen, which is a precursor of the MN antigen. In contrast to the normal colon, where the lectin binds to the supranuclear portion of the goblet and columnar cell cytoplasm, in active ulcerative colitis, it binds only to the glycocalyx and apical portion of these cells. In the case of actively regenerating cells, the lectin binds to the total cytoplasm and thus resembles the patterns seen in colonic carcinomas, adenomas, and fetal colon. This pattern was also seen in cases of colonic dysplasia. In inactive colitis, the lectin binding pattern reverts to normal. Drs. Cooper and Steplewski from the Jefferson Medical College and the Wistar Institute in Philadelphia studied ulcerative colitis using immunocytochemical methods with three monocolonal antibodies (19-9, 55-2, 73-3) to intestinal cancer-associated antigen, Y blood group antigen, and a 35 Kd protein. They attempted to use such antisera to separate cases with precancer form those without, but found that much staining occurred in all types of ulcerative colitis making the antisera of little practical value for diagnostic purposes.

Dr. Williams and collaborators from the Bronx VA Hospital and Mt Sinai Hospital in New York analyzed morphometrically nuclear changes in colons with Crohn's disease. They measured nuclear size in non-dysplastic areas, dysplastic areas, and adenocarcinomas arising in long-standing Crohn's disease. Dysplastic nuclei were significantly larger than non-dysplastic ones. It was also seen that in some cancers the dysplastic nuclei were of similar size to the neoplastic nuclei, but that in other cases the neoplasms had much larger nuclei. They concluded that nuclear size measurement may be a viable parameter in defining various types of dysplasia in Crohn's disease. In a less sanguine vein, Dr. Petras and coworkers from the Cleveland Clinic presented the results of a study on the relationship between Crohn's disease and intestinal carcinoma. They reviewed specimens from 3500 cases of Crohn's disease and found 4 small bowel as well as 7 colonic carcinomas. In the patients with small bowel cancer, the average age was 45 and they had suffered from Crohn's disease for an average of 20 years. These cases had adjacent high grade dysplasia which was confined to the mucosa immediately adjacent to the tumor. In the patients with large bowel cancer, the average age was also 45 years and they had suffered from Crohn's disease for an average 19 years. Dysplasia was preent in 6/7 cases, but in two it was noted only immediately next to the tumor. The authors concluded that a screening program for cancer in Crohn's disease is not worthwhile: a) because of its extreme rarity, b) the inaccessibility of dysplasia to biopsy, and c) the limited extent of the dysplastic change.

Drs. Troster and Grignon of the University of Western Ontario in London, Ontario, presented a study on the enteric nerve fibers in Crohn's disease and in ulcerative colitis. They used antisera to NSE and S 100 protein to quantitate nerve fibers and ganglion cells in tissues by immunocytochemistry. They determined that there was a statistically significant increase in the number and size of nerve fibers in Crohn's disease, and increased glial elements in ulcerative colitis. However, ganglion cells did not appear increased in either condition.

Miscellaneous.

The entity of neuroma of the appendix was presented by Dr. Stanley and coworkers from Hennepin County Medical Center, Minneapolis VA Hospital, and the University of Minnesota. Nineteen cases were studied, of which 7 were discovered by prospective examination of 26 consecutive routine specimens (25% incidence), 2 were selected from random cases, and 10 were discovered by retrospective review of cases previously diagnosed as fibrous obliteration. By light microscopy, these lesions were composed of loose spindle cells in a myxoid background often with entrapped fat and fibrous tissue. In only one case was an actual nodule presemt. All neuromas contained S 100 protein and NSE and two contained somatostatin, but stains for VIP, substance P, neurotensin, bombesin, and gastrin were negative. The authors point out that this neural proliferation is rather common in the appendix, and possibly the term fibrous oblitertion should be replaced by appendiceal neuroma.

The somewhat cryptic stromal tumors of the GI tract received a good deal of immunocytochemical attention from two groups of investigators. Dr. Pike and colleagues from the University of Michigan in Ann Arbor stained 31 such tumors for desmin, vimentin, actin, and S 100 protein using the avidin-biotin complex (ABC) technique. All tumors stained with actin and vimentin antibodies. S 100 protein was not immunolocalized, except for some patchy staining found in 13 small bowel tumors. Desmin antibodies immunostained all 5 esophageal and 13 small bowel tumors, but were negative in 12 gastric and 1 rectal neoplasm. Occasional entrapped Schwann cells were seen and no differences were noted in the staining patterns between benign and malignant tumors. Dr. Rast and coworkers from the University of Pennsylvania in Philadelphia examined 76 stromal tumors with antibodies against vimentin, desmin, and S 100 protein using the ABC technique. Like the previous group, they found 100% staining with vimentin antibodies. They failed, however, to stain any tumors with S 100 protein antibodies. In addition, desmin antibodies showed a somewhat different immunoreactivity in different areas: 100% esophageal and colonic, 50% gastric, and 28% small bowel tumors were positive with these antibodies. As a rule, malignant tumors tended to contain less desmin-positive cells than benign tumors; however, there was no difference between epithelioid and non-epithelioid neoplasms. Similarly, no histological differences were observed between the group of gastric and small bowel tumors that were desmin-positive and those that were desmin-negative.

Liver:

Dr. Barwick and coworkers from Yale University studied 112 liver tissue specimens from 19 liver transplant patients. They determined that

the most sensitive indicators of rejection were degenerative injury of bile duct epithelium and endophlebitis of central and portal veins. The most useful indicator of the severity of rejection was the degree of the mixed inflammatory infiltrate, including many eosinophils, which extended from portal areas to the parenchyma in severe cases. In the authors' experience, needle biopsy is a more reliable indicator of rejection than clinical, biochemical, or radiographic assessment. Drs. McLean and colleagues from the University Hospital, London, Ont., Canada also studied the pathology of liver transplantation in 16 adult allograft recipients treated with prednisone and cyclosporine A. They found that clinical rejection was associated with severe bile duct damage, presence of plasma cells, and absence of neutrophils in the portal tracts. On the other hand, features such as lymphocytic infiltration of the portal tracts and other structures as well as endothelialitis (their word, not ours!) were found in both, rejection and non-rejection patients. In another transplant-related paper, Drs. Rutledge and Belknap from the Dallas Childrens Hospital analyzed the possible use of liver transplantation in pediatric patients with fulminant hepatic necrosis. They concluded that it was unlikely that this would prove a valuable treatment method because children with hepatic necrosis tend to have an early onset of serious neurologic complications.

Dr. Ray from the University of Cincinnati looked at the distribution of cytokeratin filaments in alcoholic and non-alcoholic liver disease. Biopsies were examined using monoclonal cytokeratin antibody 50K-Hybritech and the avidin-biotin technique. In the normal liver, only the bile ducts were positive. In all types of alcoholic liver disease, hepatocyte cytoplasmic staining was present (85% compared with 15% positive in controls with viral hepatitis, PBC, etc.). The highest incidence of positives (100%) was registered in alcoholic hepatitis and cirrhosis. The lowest incidence (36%), was seen in fatty change. In alcoholic cirrhosis, the positivity was located mainly in the periphery of the nodules, but in non-cirrhotic alcoholic disease, centrilobular staining was the most common pattern. Dr. Mitros and coworkers from the University of Iowa presented an intriguing paper on liver biopsies in patients with severe rheumatoid arthiritis who had been treated wih standard therapy, including gold. They found that 56% of these patients had lipogranulomas (5% in controls). In addition, 66% had deposition of a golden-yellow to brown-black pigment. In many cases, this was associated with lipogranulomas, but was also found in lipid droplets within portal tracts. By X-ray microanalysis, the pigment was shown to contain gold. It was postulated that this phenomenon is the result of the oily vehicle used in therapeutic gold administration. Fortunately, it appears that these granulomas did not result in structural or functional liver damage. Drs. Camuto and colleagues from NYU Medical Center evaluated the liver morphology in six patients treated with the vitamine A derivative etretinate, for severe psoriasis. They observed a wide range of changes, ranging from minimal damage to chronic active hepatitis and cirrhosis. These changes were similar to those induced by methotrexate and, apparently, could not be predicted from pretreatment liver function tests. Dr. Cho and coworkers at the V.A. and Upstate Medical Centers in Syracuse, N.Y. carried out an attractive scanning and transmission electron microscopic study of rat liver damaged with acetaminophen, and of the protective effects of propylthiouracil. Acetaminophen caused dilatation of the endoplasmic reticulum, endocytic vacuolation, cellular swelling and sinusoidal congestion with impaired blood flow to the centrilobular areas leading to necrosis. Animals

protected with propylthiouracil showed little or none of this hepatotoxic damage.

Drs. Swerdlow and Chowdhury, Michael Reese Hospital, Chicago, examined the liver biopsies of 43 patients with chronic active hepatitis by light and electron microscopy. They determined that bridging necrosis in association with piecemeal necrosis are often associated with definitive progressive disease, while either bridging fibrosis or piecemeal necrosis alone were less significant. These workers contend that finding increased hepatocytic volume, hepatocellular collagen deposits, increased phagosomes in Kupffer cells, and increased frequency of hepatocyte/lymphocyte associations by electron microscopy may be of prognostic value in patients with the less significant findings. Markin and coworkers, from the University of Nebraska Medical Center in Omaha carried out an extensive histopathologic analysis of a pair of somewhat exotic conditions: sporadic fatal infectious mononucleosis and X-linked lymphoproliferative syndrome. Bile ductular cytoplasmic vacuolization, prominent portal fibrosis, massive necrosis, and parenchymal hemorrhage predominate in X-linked lymphoproliferative syndrome, whereas Kupffer cell hyperplasia and sinusoidal inflammation are found almost exclusively in sporadic fatal infectious mononucleosis. Features such as periportal necrosis and portal infiltrates of plasma cells, immunoblasts and atypical lymphoblasts are found in both conditions.

Pediatric conditions were represented by the work of Dr. Witzleben and colleagues from the Philadelphia and Texas Childrens Hospitals. They studied the ultrastructure of the bile canaliculi in arteriohepatic dysplasia and showed a variable canalicular dilatation that was less marked than in other cases of cholestasis of similar severity. They concluded that electron microscopy was not useful as a specific diagnostic method in this condition. Drs. Witzleben and Uri, from Philadelphia Childrens Hospital followed with another pediatric condition. They investigated cases of so-called infantile hemochromatosis and attempted to determine whether this is a specific entity or simply represents a secondary accumulation of iron. They noted that although iron was increased in the liver in many types of chronic liver disease, in infantile hemochromatosis patients there was also an increase of iron in the pancreas, thyroid and heart. They concluded that this provides strong evidence for a primary metabolic defect.

A paper by Dr. Snover and colleagues from the University of Minnesota reported the results of a study of livers with nodular regenerative hyperplasia (NRH) following bone marrow transplantation. Out of 72 biopsies examined, they found 23 cases of NRH: six (22%) of these had hepatomegaly and ascites, and thus clinically mimicked veno-occlusive disease (VOD). Because of the difference in prognosis of these two conditions, it is vital that NRH be considered in the clinical and pathological differential diagnosis of liver disease following bone marrow transplantation. A very interesting and somewhat provocative paper was presented by Dr. Wanless of the University of Toronto. He reviewed 2440 autopsy livers and found a 2.7% incidence of NRH and a 6% incidence of cirrhosis. A detailed morphologic analysis, including measurements of portal tracts, portal veins, and portal arteries was carried out. Many examples of vessel scarring were seen in NRH and the the most severe cases there was an association with portal hypertension. Dr. Wanless postulated

that NRH is a relatively common condition arising as a result of portal scarring. In some cases, this could be secondary to a healed vasculitis (as in rheumatoid arthritis), while in others it may be the result of aging and arteriosclerosis (something analogous to nephrosclerosis).

Oncology of the liver was lavishly represented in this meeting by a variety of papers both in the adult and the pediatric groups. Dr. Sheahan from the University of Pittsburgh compared fibrolamellar hepatocellular carcinomas (FLHCC) with the usual type of hepatocelluair carcinomas, hepatic adenomas, focal nodular hyperplasias, and metastatic carcinomas. Immunocytologic positivity for alpha-fetoprotein (AFP) was noted in 40% of hepatocellular carcinomas and was absent from all FLHCC. On the other hand, alpha-1-antitrypsin was present in 100% of FLHCC and in 93% of the hepatocellular carcinomas, as well as in a number of benign lesions. It is suggested that AFP may be a specific, albeit insensitive, marker in the distinction between hepatocellular carinoma and FLHCC. Drs. Ganjei and colleagues from the University of Miami submitted an abstract, not presented from platform, in which they examined 62 hepatocellular carcinomas and 8 cholangiocarcinomas by immunocytochemistry. Ten gastric and 12 pancreatic adenocarcinomas were used as controls. AFP was present in 15% of hepatocellular carcinomas, 10% of gastric carcinomas and 8% of pancreatic carcinomas. Alpha-1-antitrypsin was found in 37% of hepatocellular carcinomas, 70% of gastric carcinomas, and 50% of pancreatic carcinomas. CEA positivity was seen in 75% of cholangiocarcinomas, 100% of gastric carcinomas, and 90% of pancreatic carcinomas. The authors concluded that these techniques were not particularly useful in the differential diagnosis of these tumors. Dr. Albores-Saavedra headed a team from the University of Miami which examined 9 "intestinal" type adenocarcinomas of the extrahepatic biliary tract (8 gallbladder and 1 major bile duct). Two distinct histologic subtypes were recognized: a type resembling the usual colon cancer, and a type consisting mainly of goblet cells. Both types of tumor had endocrine cells containing somatostatin, cholecystokinin, and pancreatic polypeptide, and occasional Paneth cells.

Dr. Abenoza and coworkers from the University of Minnesota presented an immunologic and ultrastructural study of heptoblastomas, including "mixed" and "pure" types. Of 19 tumors studies, 17 were positive for AFP, 16 for alpha-1-antitrypsin, and 10 for alpha-1-antichymotrypsin. Ductal differentiation was present in 4 cases, two of which were CEA positive. Placental alkaline phosphatase and beta-HCG were not detected.Embryonal hepatocytes and small undifferentiated cells were positive for 54 KD cytokeratin. In 10 cases where there were cells surrounded by osteoid-like material, 9 were positive for epithelial membrane antigen (EMA), and 6 were postiive for AFP and ferritin. Ultrastructural examination of these cells revealed ultrastructural "epithelial" features. The authors concluded that the rare mesenchymal differentiation seen in hepatoblastomas likely arises as a result of metaplasia of epithelial elements. Dr. Schloo and colleagues from the Boston Childrens Hospital and the NIH studied 12 cases of embryonal sarcoma of the liver, 8 of which were subjected to immunocytochemical investigation. Positivity for alpha-1-antichymotrypsin was present in 7/7 of cases, for vimentin in 5/8 of cases, and for desmin, keratin, S100, and F8-A in 1/8 of cases. Negative results were found with myoglobin, GFAP, AFP and HBsAg. The results were considered to add support to the hypothesis favoring a mesenchymal origin for this tumor.

Pancreas:

Dr. Halwani and collaborators from McGill University in Montreal generated a monoclonal antibody by sensitizing mice with an extract of fresh human pancreatic ductal carcinoma. This antibody, named LD-B1, was immunolocalized in pancreatic, gallbladder, and cholangiocarcinomas, as well as in normal ductal and centroacinar pancreatic cells. It was negative, however, in most of gastric, colonic, breast, and lung adenocarcinomas, as well as lung squamous cell carcinomas. The authors believe that detection of antigen LD-B1 may be of potential use in the differential diagnosis of pancreatic tumors. Dr. Skutelsky and coworkers from Tufts University and Angell Memorial Animal Hospital in Boston carried out an experimental work on acinic cell carcinomas of the dog and cat pancreas. They studied these neoplasms for the binding of a large variety of lectins to the malignant cells. They concluded that during differentiation and malignant transformation, there are alterations in the cell surface coat. Furthermore, they also postulated that increased cell surface sialylation correlates with increased malignant potential.

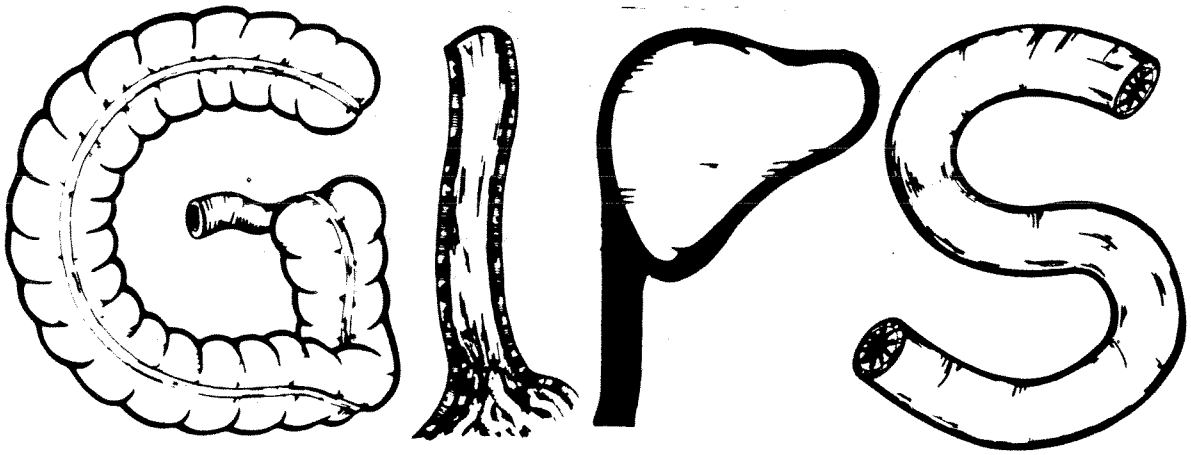
Drs. Hartman and Ray from the University of Cincinnati carried out a light and electron microscopic immunoperoxidase study in an effort to identify the pancreatic islet cell type immunoreactive for alpha-1-proteinase inhibitor (modern English for alpha-1-antitrypsin). The authors conclude that such substance is present free in the cytosol of a subset of A cells as identified by electron microscopy. However, light microscopy immuno-peroxidase had shown that glucagon, the usual peptide produced by A cells in not colocalized inside cells positive for alpha-1-proteinase inhibitor. From the same institution, Dr. Ray presented a related study in which the pancreatic islets of patients with alpha-1-proteinase inhibitor deficiency by light microscopy and by immunoperoxidase for alpha-1-proteinase inhibitor. He found that in patients with this deficiency, there was nesidioblastosis and islet cell atypia, as well as increased numbers of cells immunoreactive for alpha-1-proteinase inhibitor.

Salivary Glands:

Dr. Abenoza and Wick from the University of Minnesota studied 19 Warthin's tumors, 15 mucoepidermoid carcinomas, 2 malignant mixed tumors, and 14 adenocarcinomas NOS of salivary gland origin by immunocytochemistry. PAP and ABC immunoperoxidase were carried out for salivary amylase, S100 protein, epithelial membrane antigen, cytokeratin, CEA and Beta-2-microglobulin. The authors predictably concluded that salivary gland tumors are immunocytochemically heterogeneous, but may be segregated in some cases by the use of appropriate immunostains. In a similar vein, Dr. Zarbo and colleagues from Wayne State University, Detroit, University of Michigan, Ann Arbor, and University of Texas/M.D. Anderson Hospital, Houston studied the immunolocalization of S100 protein in 129 tumors from major and minor salivary glands, including pleomorphic adenomas, adenoid cystic carcinomas, malignant pleomorphic tumors, clear cell carcinomas, mucoepidermoid carcinomas, acinic cell carcinomas, and adenocarcinomas NOS. S100 protein was consistently localized in pleomorphic and monomorphic adenomas and in polymorphous low grade carcinomas. On the other hand, it was invariably absent from the mucoepidermoid carcinomas. Other tumors showed variable immunoreactivity with S100 protein antibodies.

THE (OFF)-CENTERFOLD

A couple of issues ago, we solicited contributions from our membership towards a logo for our Gastrointestinal Pathology Club (or Society, as it may come to be named if so decided by our rank and file). So far, we have received what could be described as a masterpiece from our former president Dr. Robert Riddell. As you will observe, this logo assumes that we will become a Society: Dr. Riddell tells us that should this assumption prove unfounded, he can easily transform the jejunal S at the end into a perfect duodenal C loop. May we present this first, albeit magnificent, effort as a challenge to other "closet" artists among us to send in their own creations?



POETRY CORNER

Dr. Leslie H. Sobin, a member of the Gastrointestinal Pathology Club, has been good enough to share with us a sample of his poetic vein, previously published in the International Journal of Gynecological Pathology (4:274,1985). We are reproducing Dr. Sobin's stanzas in this issue of the GIPC Newsletter with the gracious permission of the original publication. We also take the oportunity to invite other "versifiers" who may be lurking in the GIPC membership to submit their poetic production for this newly created "corner".

SIN, VIN, GIN In - or Out?

That useful term dysplasia
Known from Boston clear to Asia
Is now subjected to some competition:
From a phrase too long unterse
That it scarcely fits in verse
A new way to describe an old condition.

Intraepithelial neoplasia
Is the term for this dyscrasia
Which owing to its length's abbreviated:
In the cervix it's-called CIN
For the vulva make it VIN
In the stomach GIN is what has been created

Further problem with this rival
There's no form that's adjectival
To replace time honored term, i.e. dysplastic:
We could try in cervix CINIC
In the bronchus perhaps BRINIC
VINIC GINIC ESOPHINIC, all sound drastic.

If A calls a rose dysplasia
And B calls it neoplasia
Won't it look as lovely and smell just the same?
Changing terms can be pedantic
And the argument semantic:
Why not stop this game and keep the name the same.

Leslie H. Sobin, M.D.
Washington, D.C.

MESSAGE FROM THE PRESIDENT

As we enter our eight year, the members of the GIPC can look back at an era of productive growth and can anticipate continued development of new Club activities. Our regular sessions at the IAP and AGA meetings this year were, once again, very successful; our association with the *American Journal of Surgical Pathology* is in place; and the Newsletter is thriving under the guidance of Drs. Lechago and Owen.

Before previewing some of next year's activities, I wish to thank all of you who responded to the questionnaire which Jim Madara and I sent out earlier this year. Over 40% of the regular members returned completed forms, a gratifyingly high response to a type of mailing for which a 10 to 15% return rate is considered excellent!

For 1987, the Education Committee, with Rodger Haggitt as Chairperson, is planning our Scientific Session at the next IAP meeting in Chicago based on the results of the questionnaire mentioned above. The subject will be Gastrointestinal Infections and the format will again be a mix of material aimed at a general audience and more specialized items of particular interest to the Club's members. The topics will include the pathogenesis of G.I. infections; intestinal mucosal defenses; the histology and differential diagnosis of infectious enterocolitis; the role of campylobacter species in the genesis of gastric inflammatory disease; and the use of immunocytochemistry and DNA hybridization in the evaluation of enteric infections. This should be a stimulating and informative program.

If you will be in Vienna at the IAP Congress this fall, keep in mind that the International Gastrointestinal Pathology Group Symposium is scheduled for September 3. Harvey Goldman is the moderator and two other Club members, Cecilia Fenoglio-Preiser and Tom Norris, are among the scheduled speakers. The full program was printed in the last issue of this Newsletter.

Finally, two new proposals of direct benefit to the membership are in the works. The first is the establishment of a "micro-grant" program to encourage research in G.I. pathology among Club members by providing small sums of money for such items as technical assistance, purchase of supplies, and travel. This will be a method by which surpluss funds in our treasury can be returned as benefits to the members. Details concerning the application process and deadlines are available eslsewhere in this issue of the Newletter.

The second innovation (to be set up by popular demand) will be a reception for the members after the Business Meeting next year. After a long afternoon of scientific data and business matters, we can look forward to the opportunity to relax and socialize over drinks and snacks.

As you can see, the next year already promises to be another good one for the Gastrointestinal Pathology Club. My best wishes for a very happy Summer.

Don Antonioli
President

GASTROINTESTINAL PATHOLOGY CLUB MICRO-GRANT PROGRAM

The Executive Committee of the GIPC has approved a program of "Micro-Grants" for its members. Its purpose is to provide low-hassle seed money that will encourage research in the area of gastrointestinal pathology, especially that which is interactive between Club members. A total of up to $ 2,500 per year will be made available for the Micro-Grant Program.

Eligibility: Any member (Regular, Associate, or Emeritus) in good staning may apply. The member must be the responsible investigator.

Types of Projects Supported: While stimulation of joint projects between Club members is a major goal of the Micro-Grant Program, other kinds of proposals will be welcomed, especially those that include participation of trainees in GI pathology. Even though funds are very limited, they can be creatively used for such things as obtaining otherwise unavailable help from a technician, purchase of reagents, or travel to meetings between collaborators.

Application Format: Requests for support should be directed to the undersigned. Describe the project in a *maximum* of two pages (single spaced). Include statements which give key aspects of:

a) Rationale and aims
b) Methodology
c) Interpretation of results. Mention previous work, but give only a limited number of references. Attachements (reprints) are *not* encouraged.
d) Identify participants, and state they ways that the project meets Club objectives.

Budget: Give this on a separate page. Salary items should be modest and *limited to support personnel* (technicians, secretaries, etc.). Identify anticipated expenditures for supplies, equipment, travel, etc. The total requested should not exceed $ 1,250.

Identify any other sources which *specifically* support the same work. Indicate to whom a check should be made out (ordinarily, the institution on behalf of the Club member).

Deadlines, Selection Process, etc.: Proposal may be submitted at any time, and awards may be made on an *ad lib* basis if circumstances warrant. However, normal procedure will be to adhere to May 1, and September 1 submission deadlines, with award announcements coming within 6 weeks thereafter. Selections will be made by an *ad hoc* committee comprised of the President, the Secretary-Treasurer, and the Chairpersons of the Education and Training Programs Committees, plus other who may be appointed by the President.

Progress Report: A Micro-Grant is ordinarily awarded for one year. When completed, or on an annual basis if renewed, a brief statement of progress should be provided.

Direct inquiries and applications to:

John H. Yardley, M.D.
Department of Pathology
The Johns Hopkins Hospital
Baltimore, Maryland 21205

GIPC SCIENTIFIC PROGRAM AT THE DDW MEETING IN SAN FRANCISCO

The Gastrointestinal Pathology Club Scientific Session at the 1986 Digestive Disease Week in San Francisco took place in Room 202 of the Moscone Convention Center on Tuesday, May 20, 1986 between 7:30 and 9:30 p.m., under the chairmanship of Dr. James L. Madara from Harvard University and Brigham and Women's Hospital, Boston.

Topics and Speakers

"Structural and functional determinant of Peyer's patch uptake of bacteria, parasites and macromolecules" - Dr. Robert L. Owen, VA Medical Center, San Francisco.

"Viral interaction with the villus and Peyer's patch epithelium" - Dr. Dorsie Bass, Brigham and Women's Hospital, Boston.

"Colonic glycoproteins and inflammatory bowel disease: of man an monkey" - Dr. Daniel K. Podolsky, Massachusetts General Hospital, Boston.

GI PATHOLOGY TRAINING PROGRAMS - 1986

The attached listings of GI Pathology training opportunities are based on questionaires returned by members of the Gastrointestinal Pathology Club in the Spring of 1985 and on updates obtained in the Spring of 1986.

Information is grouped according to whether training is offered in: 1) a residency and/or fellowship program, or 2) Some other type of program. Each reporting institution/department is listed in only one place.

Table 1

GI PATHOLOGY TRAINING PROGRAMS - 1984 VS 1986

Features	**1984 Report**	**1986 Report**
Types of Programs		
Residency	15	33
Residency and Fellowship	6	15
Other Types	2	4
TOTALS	23	52
Fellowship Support		
Via Institution	3	10
Outside or Uncertain	3	5

This is the second compilation of training opportunities in GI pathology, and noteworthy changes have occurred since the initial (1984) listing (Table 1). Total listed programs have increased by 126%, and both residency and fellowship programs have increased proportionally.

The Training Programs Committee believes that the increasing number of fellowship opportunities is especially important. Such opportunities for advanced sub-specialty training are central to increasing the pool of gastrointestinal pathologists. A related development is growing availability of institutionally controlled support for fellowships. These trends in part result from the need for "fifth year" training opportunities in response to regulations of the American Board of Pathology, but we believe they also reflect an expanding interest in GI pathology *per se*. Because of a growing desire for information about fellowships, the

fellowship listing will also be published separately in the American Journal of Surgical Pathology.

The tabulated information about individual training programs gives only a limited impression about their character, and those who want to know more about training opportunities in gastrointestinal pathology are urged to contact individual program directors.

The President of the GIPC has appointed Dr. Katherine DeSchryver to succeed the current Chairman (Dr. Yardley), as of March 1987. In order to provide a smooth transition, the Committee files are being transferred to Dr. DeSchryver with completion of this listing. Feel free to contact her as well as Dr. Yardley and other members of the Committee, about your questions, suggestions, etc.

Training Programs Committee
May 16, 1986

GIPTP.486

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq.	Fellowship Salary	Other Training	Program Director(s) and Address	Comments
- USA (Alphabetical by States) -							
UCLA Center for Health Sciences, Los Angeles, California	REG SEP	1 yr.	MD Pathologist Gastroenterologist	Outside		Klaus J. Lewin, M.D. Wilfred Weinstein, M.D. Department of Pathology Center for Health Sciences 10833 LeConte Avenue Los Angeles, CA 90024	Training in collaboration with GI Div., Dept. Med. (Dr. Weinstein)
Cedars-Sinai Med. Ctr. Los Angeles, California	REG				Contact Dr. Geller	Stephen A. Geller, M.D. Department of Pathology Cedars Sinai Med. Ctr. 8700 Beverly Blvd. Los Angeles, CA 90048	Special training opportunities being developed
Harbor-UCLA Medical Center Torrance, California	REG	1-2yrs.	2 yrs. AP	Inst.	Ad Hoc	Juan Lechago, M.D., Ph.D. Department of Pathology Box 22 Harbor-UCLA Medical Center 1000 W. Carson Torrance, CA 90509	Fellowship combines diagnostic pathol. and research (ca 50%)
Memorial Medical Center Long Beach, California	REG					Joseph Tomasulo, M.D. Department of Pathology Memorial Hospital Med. Center 2801 Atlantic Avenue Long Beach, CA 90801	
Yale University New Haven, Connecticut	REG SEP	1 yr.		Inst.	Ad Hoc.	Kenneth W. Barwick, M.D. Juan Rosai, M.D. Department of Pathology Yale University Hospital New Haven, CT 06510	
Emory University Atlanta, Georgia	REG	See Comments				Victor H. Nassar, M.D. V.A. Medical Center 1860 Clairmont Road Decatur, GA 30033	Fellowships being considered. Contact Dr. Nassar.

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq.	Fellowship Salary	Other Training	Program Director(s) and Address	Comments
Rush-Presbyterian St. Lukes Hospital Chicago, Illinois	REG					Barbara F. Banner, M.D. Department of Pathology Rush Presbyterian Med. Center 1735 West Congress Parkway Chicago, Illinois 60612	3-400 liver bx, 1000 GI bx/yr.
Univ. of Iowa Iowa City, Iowa	REG SEP				Ad Hoc.	Frank A. Mitros, M.D. Department of Pathology College of Medicine University of Iowa Iowa City, IA 52242	
Louisiana State University New Orleans Louisiana	REG	1 yr.	Path. Training Compl.	Contact Dr. Correa		Pelayo Correa, M.D. Department of Pathology LSU Medical Center 1901 Perdido St. New Orleans, LA 70112	Research in GI pathology and epidemiology
Johns Hopkins Baltimore, Maryland	REG SEP	2 yrs.	3 yrs. AP	Inst.	Ad Hoc.	John H. Yardley, M.D. Stanley R. Hamilton,M.D. Department of Pathology Johns Hopkins Hospital Baltimore, MD 21205	Fellowship combines diagnostic and research
Beth Israel Hosp. Children's Hospital Harvard Med. Sch. Boston, Massachusetts	REG	1 yr. (See Comments)	4 yrs. AP or AP/CP	Inst.	Ad Hoc.	Harvey Goldman, M.D. Donald Antonioli, M.D. Department of Pathology Beth Israel Hospital 330 Brookline Avenue Boston, MA 02215	Fellowship includes gen. service responsibility in AP and specific training in GI
Dept. of Pathology Brigham & Women's Hospital, Boston, Massachusetts	REG	2-3 yrs.	AP training	Inst. (NIH T.G.)		James L. Madara, M.D. Department of Pathology Brigham & Women's Hosp. Boston, MA 02115	A basic research fellowship oriented to GI tract.
Massachusetts General Hospital Boston, Massachusetts	REG					G. Richard Dickersin, M.D. Carolyn Compton, M.D. Department of Pathology Massachusetts General Hospital Boston, MA 02114	

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director(s) and Address	Comments
Dept. of Medicine Brigham & Women's Hospital, Boston, Massachusetts		1-2 yrs.	Path. training	Inst. (NIH T.G.)		Jerry S. Trier, M.D. Gastroenterology Div. Brigham & Women's Hosp. 75 Francis Street Boston, MA 02115	Bench research and GI cell biology. Interaction with GI path. group.
Tufts University N.E. Medical Center Boston, Massachusetts	REG				Ad Hoc.	Yogeshwar Dayal, M.D. Department of Pathology New England Medical Center 171 Harrison Avenue Boston, MA 02111	Fellowship being planned. Contact Dr. Dayal.
VA Med Center Boston, Massachusetts	REG					Wilhelm G. Doos, M.D. Laboratory Services V.A. Medical Center 150 S. Huntington Avenue Boston, MA 02130	Much emphasis on GI & Liver in regular Pathol. training program
Univ. of Michigan Ann Arbor, Michigan	REG SEP				Immunology fellowship	Gerald D. Abrams, M.D. Henry D. Appelman, M.D. David F. Keren, M.D. Department of Pathology University Hospital Box 0054 Ann Arbor, MI 48109-0054	Immunol. Fellowship can emphasize mucosal immunity
Univ. Minnesota Minneapolis, Minnesota	REG				Ad Hoc. area of liver transplant	Dale C. Snover, M.D. Dept. Lab. Med. and Pathol. University of Minnesota Box 609 Mayo Building 420 Delaware Street, S.E. Minneapolis, Minnesota 55455	
Washington University Medical School St. Louis, Missouri	REG SEP	1 yr. (initial)	AP/AP-CP	Inst.	Ad Hoc.	Katharine DeSchryver, M.D. Division of Surgical Pathol. Washington University Medical School, Box 8118 St. Louis, MO 63110	Fellowship combines diagnostic and research experience.

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GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director and Address	Comments
VA Medical Center Univ. New Mexico Albuquerque, New Mexico	REG SEP					Cecilia Fenoglio-Preiser,M.D. Department of Pathology Veterans Administration Medical Center 2100 Ridgecrest Drive S/E Albuquerque, NM 87108	Ad hoc for projects
Columbia-Presbyterian Medical Center New York, New York	REG					Karl H. Perzin, M.D. Div. of Surgical Pathology Columbia University College of Physicians & Surgeons 630 West 168th Street New York, New York 10032	
Lenox Hill Hospital New York, New York	REG				Ad Hoc.	Heidi Rotterdam, M.D. Department of Pathology Lenox Hill Hospital 100 East 77th Street New York, New York 10021	30% of SP specimens are GI, many GI conferences.
Long Island Jewish New Hyde Park, New York	REG	1 yr.	4 yrs AP or AP/CP	Inst.	Ad Hoc.	Leonard B. Kahn, M.D. Department of Laboratories Long Island Jewish Medical Center 270-05 76th Avenue New Hyde Park, NY 11042	
North Shore Univ. Hospital, Manhasset, New York	REG	1 yr.	2 yrs. AP	Inst.	Ad Hoc.	Ellen Kahn, M.D. Department of Pathology North Shore Univ. Hospital 300 Community Drive Manhasset, New York 11030	
Univ. of Rochester Rochester, New York	REG SEP				Ad Hoc.	Eric A. Schenk, M.D. Catherine Listinsky, M.D. Department of Pathology University of Rochester Rochester, New York 14642	

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director and Address	Comments
Staten Island Hospital Staten Island, New York	REG				See Comments.	Robert Pascal, M.D. Department of Pathology The Staten Island Hospital 475 Seaview Avenue Staten Island, NY 10305	Residency affiliated with SUNY-Downstate Contact Dr. Pascal about possible fellowship.
Univ. of Cincinnati Cincinnati, Ohio	REG				Ad Hoc.	John A. Wirman, M.D. Department of Pathology University of Cincinnati Med. Ctr. Cincinnati, OH 45267	
Cleveland Clinic Foundation, Cleveland, Ohio	REG SEP					Robert E. Petras, M.D. Cleveland Clinic Foundation 9500 Euclid Avenue Cleveland, Ohio 44106	Research opportunities available.
University Hospital Case-Western Reserve Univ. Cleveland, Ohio	REG					Beverly Dahms, M.D. Geoffrey Mendelsohn, M.D. Case Western Reserve Unit. 2085 Adelbert Road Cleveland, Ohio 44106	
Youngstown Hospital Assoc. Youngstown, Ohio	REG SEP					Ludwig M. Deppisch, M.D. Dept. Pathology & Lab. Med. Youngstown Hospital Assoc. Youngstown, Ohio 44501	
St. Vincent Hospital Portland, Oregon	REG				Ad Hoc. Rotations for clin. residents, fellows.	R. Mark Owings, M.D. Dept. of Pathology St. Vincent Hospital Portland, Oregon 97225	

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director and Address	Comments
Jefferson Med. College Philadelphia, Pennsylvania	REG				Ad Hoc.	Harry S. Cooper, M.D. Department of Pathology Thomas Jefferson University Hospital, 111 South 11th Street Philadelphia, PA 19118	Fellowship planned Contact Dr. Cooper
Temple Medical School Philadelphia, Pennsylvania	REG				Ad Hoc.	Si-Chun Ming, M.D. Department of Pathology Temple University Hosp. Philadelphia, PA 19140	Research opportunities available.
Univ. of Pennsylvania Philadelphia, Pennsylvania	REG					Scott H. Saul, M.D. Surgical Pathology Section Hospital of Univ. of PA 3400 Spruce Street Philadelphia, PA 19140	
Presbyterian-University Hospital Pittsburgh, Pennsylvania	REG				Ad Hoc.	Daniel G. Sheahan, M.D. Department of Pathology Presbyterian-Univ. Hospital Pittsburg, PA 15213	Emphasis on liver transplant available.
Univ. Hospital Univ. Central del Caribe Bayamon, Puerto Rico	REG					Manuel A. Marcial, M.D. Department of Pathology Univ. Hospital Ramon Ruiz Arnau Avenue Laurel Bayamon, PR 00619	
Roger William Gen.Hosp. Brown University Providence, Rhode Island	REG				Rotation in GI Path by GI Fellows	Robert Lev, M.D. M. Peter Lance, M.D. Department of Pathology Roger Williams Gen. Hosp. 825 Chalkstone Ave. Providence, RI 02906	
Univ. of Texas Medical Branch Galveston, Texas	REG					William K. Gourley, M.D. Surgical Pathology Univ. of Texas Medical Branch Galveston, TX 77550	Cooperative research projects-EM, immunostaining, etc. GI infections, liver.

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director and Address	Comments
Univ. of Washington Seattle, Washington	REG SEP	1-2 yrs.	2 yrs. AP or 2 yrs. GI Fellowship	Outside	Ad Hoc.	Rodger C. Haggitt, M.D. Cyrus E. Rubin, M.D. University Hospital Div. of Hospital Pathology BB-220 University Hospital RC-72, Seattle, WA 98195	SEP also avail. to GI Fellows and Path residents. Options incl. myenteric plexus, smooth muscle pathology with Dr. Schuffler.
Univ. of Utah Salt Lake City, Utah	REG					Randall G. Lee, M.D. Thomas V. Colby, M.D. Department of Pathology Univ. of Utah Medical Center Salt Lake City, Utah 84132	
- CANADA (Alphabetical by Province) -							
Univ. of Calgary Foothills Hospital Calgary, Alberta	REG SEP 3 mos.				Ad Hoc. Rotation of GI Subspec. Residents	James K. Kelly, M.D. Department of Histopathology Foothills Hospital 1403 29 Street N/W Calgary, Alberta T2N 2T9 Canada	
Shaughnessy Hospital Vancouver, British Columbia	REG				Graduate Seminars	Dr. W. L. Dunn Department of Pathology Shaughnessy Hospital 4500 Oak Street Vancouver, British Columbia V6H 3N1, Canada	
Vancouver Gen. Hosp. Univ. of Brit. Columbia Vancouver, British Columbia	SEP	1 yr.	4 yrs pathol.	Inst.		David A. Owen, M.D. Department of Pathology Vancouver General Hosp. 855 West 12th Avenue Vancouver, British Columbia V5Z 1M9 Canada	Fellowship includes research. Pediatric GI pathol. avail.

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director and Address	Comments
McMaster University Hamilton, Ontario	REG SEP	1 yr.	2 yrs. AP	Inst.	Ad Hoc.	Ali Qizilbash, M.D. Robert H. Riddell Dept. of Pathology Henderson General Hosp. 711 Concession St. Hamilton, Ontario L8V 1C3, Canada	Prev. exp. required for Ad Hoc.
Queen's University Kingston, Ontario	REG SEP	1-2 yr	AP Compl- eted	Outside	Ad Hoc.	Paul N. Manley, M.D. Department of Pathology General Hospital Kingston, Ontario Canada K7L 2V7	IBD Emphasis
Univ. Western Ontario University Hospital London, Ontario	REG SEP				Ad Hoc. Fellowship (Contact Dr. B. Garcia)	J. V. Frei, M.D. Department of Pathology Health Sciences Center Univ. of Western Ontario London, Ontario N6A 5C1 Canada	1000 GI bx/yr. 2000 Liver bx/y Research: Liver transplant.
Ottawa Civic Hosp. Ottawa, Ontario	REG SEP 4 wks.					J. Robin Barr, M.D. Dept. of Laboratory Medicine Ottawa Civic Center 1053 Carling Avenue Ottawa, Ontario K1Y 4E9 Canada	

-- See End for Notations --

GI PATHOLOGY TRAINING - RESIDENCY & FELLOWSHIP

Institution & Location	Resident Training Types	Fellowship Dur.	Fellowship Prereq	Fellowship Salary	Other Training	Program Director and Address	Comments
UNITED KINGDOM							
Charing Cross Hospital London, England	REG				Ad Hoc.	Sami Shousha, M.D., MRCPath. Department of Histopathology Charing Cross Hospital Fulham Palace Road London W6 8RF, England	

Training in Residency: REG (Regular) - GI path training provided in general context of Regular AP rotations. This usually continues throughout training. SEP (Separate Experience) - Residency includes separate rotation devoted mainly to GI pathology, usually for a specified period of time.

Fellowships: A "Fellowship" is defined as an organized training program lasting 1 or more years. "Inst." refers to salary available via institution. "Outside" salary must come from outside source (by application or otherwise).

Prerequisite (Prereq.): Minimum completed training for participation in Fellowship. Anatomic Pathology (AP) may be specified. Some programs are open to gastroenterologists, internists, and surgeons.

Other Training: "Ad Hoc" means outside persons are welcome for training of varied length. Make arrangements with person(s) named.

GI PATHOLOGY TRAINING - OTHER TYPES

Institution & Location	Type of Training	Program Director(s) and Address	Comments
	-- USA --		
Armed Forces Inst. of Pathology Washington, District of Columbia	Ad hoc. Diagnostic conferences, participation in research projects if qualified. Duration: 2 wks. to several months.	Elson B. Helwig, M.D. Leslie H. Sobin, M.D. Department of Pathology Armed Forces Inst. of Path. Washington, DC 20306	Prereq.: AP 2 yrs., or 6 mos. GI or 1 yr. Med. or Surg.
Dept. of Medicine VA Med. Center Univ. of Michigan Ann Arbor, Michigan	Ad Hoc training with emphasis on intestinal and rectal biopsy. Duration: 4 wks.	William O. Dobbins, M.D. Veterans Admin. Med. Ctr. 2215 Fuller Road Ann Arbor, MI 48104	GI Fellows or Pathology Trainees welcome
GI Division Pacific Med. Center Seattle, Washington	Ad Hoc to learn myenteric plexus and smooth muscle pathology. Duration: Usually 1 wk. to 1 mo.	Michael D. Schuffler, M.D. Pacific Medical Center P.O. Box 3145 Seattle, WA 98114	Can combine with pathology training at Univ. Washington (Drs. Haggitt and Rubin)
	-- UK --		
St. Mark's Hospital London, England	Postgraduate training in pathology. Duration: By arrangement.	Dr. Basil C. Morson Dr. Jeremy R. Jass Pathology Department St. Mark's Hospital City Road London, EC1V 2PS England	Both diagnostic pathology and research experience available.