Message from Dr. Yuen Hon, President

Happy New Year to all!

Year 2000 has been a fruitful year for our Society in upkeeping and upgrading our standard of gastroenterology in Hong Kong, thanks to the continuous and unfailing efforts of our members and fellows.

This issue of the Newsletter contains a number of valuable and enlightening articles from Professor Colin McKay, Visiting Professor from Glasglow, Dr. Lee Yuk Tong, Dr. Yuen Siu Tsan and others. I would like to thank all writers for contributing to the Newsletter. I must not forget to thank Dr. Wai-mo Hui and his Publications Committee for their great work in planning and coordination throughout.

The Annual Scientific Meeting was held on September 2, 2000. The Meeting was organized jointly with the Hong Kong Society of Digestive Endoscopy and Hong Kong Society for Coloproctology. The meeting went very well. Over 100 delegates from different specialties including GI Medicine, Surgery, Radiology, Pathology and many others attended. In addition to topical presentations by prominent speakers, both local and overseas, there was a free paper session during which thirteen of our member doctors brightly presented their specialised abstracts. On behalf of the Society, I would like to express my gratitude to all who have participated in and contributed to this important annual event, in particular, the Organizing Committee. I hope our members will continue to give their support to the Third Annual Joint Scientific Meeting planned to be held in the autumn of 2001. Our research project on "Colorectal Cancer Screening" is underway. This has been a hot topic in the past few years and will continue to form an important area for medical research and exploration in the near future.

Last but not least, may I welcome all new members who joined our Society this year and look forward to their enthusiastic support and active participation in our forthcoming events.
In the past decade, many advances have been made in our understanding of the pathophysiology of acute pancreatitis and its complications and several large clinical trials have been carried out which, it was hoped, would lead to specific treatments for this condition. During this period, there has been a steady rise in the incidence of acute pancreatitis and overall mortality has remained unchanged at around 8% (1).

Medical management
Attempts to introduce specific treatments for acute pancreatitis fall into the following categories:

- Inhibition of pancreatic secretion
- Prevention of infection
- Inhibition of the inflammatory response

ERCP_EC

Inhibition of pancreatic secretion
Pharmacological attempts to suppress pancreatic function have included glucagon somatostatin and, more recently, the somatostatin analogue octreotide. There have now been five randomised trials of octreotide in the management of acute pancreatitis reported in the literature. The first of these randomised just 19 patients(2). Next came a study from Israel(3), recruiting 51 patients with predicted severe acute pancreatitis to treatment with octreotide or standard supportive care. The overall complication rate was lower in the octreotide group and hospital stay was shorter. Unfortunately, 25% of the patients were excluded from analysis due to incomplete data and this "preliminary report" was never followed by a report on the completed study. In the West of Scotland study(4), 58 patients with predicted severe acute pancreatitis were recruited. This was the first study to be conducted in a double-blind, placebo-controlled fashion and in contrast to the previous reports, there was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%. In the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in secondary pancreatic infection from 30% to 16%. There was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%. In the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in secondary pancreatic infection from 30% to 16%. There was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%. In the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in secondary pancreatic infection from 30% to 16%. There was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%. In the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in secondary pancreatic infection from 30% to 16%. There was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%. In the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in secondary pancreatic infection from 30% to 16%. There was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%. In the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in secondary pancreatic infection from 30% to 16%. There was no evidence of a beneficial effect of octreotide on outcome. A further non-blinded, small study was reported from Turkey(5) but in the same year, the definitive study was published. A multicentre trial from Germany(6) failed to demonstrate any effect of octreotide and a reduction in extra-pancreatic sepsis from 49% to 15%

Antibiotics
Pederzoli and colleagues(7) reported the results of a multi-centre Italian study which recruited 74 patients with necrotising pancreatitis. The use of imipenem was associated with a reduction in secondary pancreatic infection from 30% to 12% and a reduction in extra-pancreatic sepsis from 49% to 15%. In a single-center study of similar design, Sainio and colleagues(8) randomized 60 patients with pancreatic necrosis to receive cefuroxime, 1.5g three times daily or standard treatment. This study had findings that contrasted sharply with those of the Italian trial(7) in that no difference was observed in the incidence of secondary pancreatic infection but there was an unexplained reduction in mortality associated with cefuroxime prophylaxis. Luiten and colleagues(9) assessed the effect of selective gut decontamination using norfloxacin, colistin and amphotericin on 102 patients recruited from 16 hospitals over...
Introduction
Endoscopic ultrasonography (EUS) combines both endoscopic and ultrasonic capability in one examination and has emerged as a major advance in gastrointestinal endoscopy (1). The examination involves using a high-frequency ultrasound transducer (7.5-20MHz), which is mounted onto the tip of an endoscope and can be directed to the side of interest. Both the luminal and extraluminal structures could be visualized by EUS in great detail. Commonly used EUS equipment include radial and linear echoendoscope and through-the-scope miniature probe. Radial echoendoscope provides 360° cross-sectional images, which is perpendicular to the long axis of the scope. Radial EUS is most commonly used worldwide because the image produced is more familiar to the endoscopist. Linear echoendoscope provides a 180° sector image which is longitudinal to the long axis of the scope, and it allows fine needle aspiration under real-time ultrasound guidance (EUS-FNA) possible. Miniature probe that can pass through the biopsy channel of endoscope is useful in studying small mucosal and submucosal lesions, and in performing intraductal USG (IDUS) examination. EUS, over 20 years of development, has now been recognized as the single best imaging modality in the management of pancreatic diseases.

Acute pancreatitis (AP)
The most common causes of AP are gallstones disease and alcoholic use. ERCP is indicated in patients suspected to have biliary obstruction. However, ERCP carries certain risks including exacerbating pancreatitis and cholangitis. EUS was shown to be highly sensitive and specific in detecting choledocholithiasis. Using EUS to select patients for ERCP may reduce overall complications and may be cost-effective. Previous study has shown that in patients with low to moderate risk of bile duct obstruction (i.e. absent of clinical signs of acute cholangitis or no stone detected on USG), a positive EUS followed by ERCP is more cost-effective than performing ERCP on all patients (2). In patients with signs of cholangitis, direct ERCP should be the treatment choice. For patient not suitable for EUS examination, magnetic resonance cholangiopancreatogram could be an alternative that was also shown to be highly sensitive and specific for choledocholithiasis. EUS based prognostic signs has been found to predict the clinical outcome of patients suffering from AP (3,4). EUS may reliably distinguish edematous from necrotizing AP and the EUS-scores correlate significantly with the duration of hospital stay, number of days of fever, and days spent in the intensive care unit. There is good correlation between EUS scores and CT-prognostic-index (4).

Pancreatic Tumour
EUS, as compared with CT and MRI, is more sensitive in detecting pancreatic tumour, especially for small tumour (<2cm) (8). EUS is also shown to be reliable in predicting resectability of the tumour by detecting local vascular invasion and lymph node metastasis (9). Recently the development of spiral CT has improved the accuracy which is comparable to EUS (93%) (10). With accurate staging, curative resection or palliative treatment could be decided. However, the identification of pancreatic tumour in the background of CP could be difficult. Although signs like vessel invasion, lymphadenopathy and ductal obstruction may help in predicting malignancy, tissue diagnosis is still required for some indeterminate cases. In a recent multicenter study, 164 consecutive patients with pancreatic lesions received EUS-FNA of the lesion or associated lymph node. The overall sensitivity, specificity, diagnostic accuracy, negative and positive predictive values for cancer were 83%, 90%, 85%, 80% and 100%, respectively. The result is significantly better than...
conventional CT (11). However, a negative cytology does not totally exclude an underlying tumour, a careful clinical and EUS follow up is required. Recently positron emission tomography (PET) is also claimed to be accurate in differentiating CP and pancreatic tumour, which may help in difficult cases (12).

EUS is also shown to be highly sensitive in detecting neuroendocrine tumours of the pancreas (13). When combining with somatostatin receptor scintigraphy (SRS), both the sensitivity for insulinoma (89%) and gastrinoma (93%) were improved (14).

Therefore, in patients with clinical suspicion of pancreatic tumour (e.g. biliary or pancreatic ductal stricture), initial spiral CT scan is used to rule out local advanced disease or distant metastases. EUS should be performed when the CT result is inconclusive and EUS-FNA is useful in establishing the diagnosis ERCP should be reserved for drainage and other interventional procedures.

Interventional EUS

With the advantage of real-time monitoring, EUS-guided FNA becomes an important diagnostic and therapeutic tool. In pancreatic pseudocyst, Doppler EUS can detect any intervening vessel between gastric wall and the cyst before puncturing to avoid bleeding. EUS guided puncturing of the cyst is useful especially when there is no luminal bulging observed at the stomach or duodenum. A one-step puncturing followed by stenting of the cyst without the need of changing the endoscope is possible with large-channel echoendoscope equipped with an elevator (15). In patients with inoperable pancreatic cancer, EUS-guided celiac plexus neurolysis by transgastric injection of bupivacaine and steroids or alcohol on either side of the celiac artery could achieve complete pain relief in most patients (16). The treatment may also be useful in patients with chronic abdominal pain related to CP. Most recently, other treatment like EUS-guided fine needle injection of activated T lymphocytes into pain related to CP. (16). Wiersema MJ, Wiersema LM. Endosonography-guided celiac neurolysis by therapeutic ultrasound endoscope. Gastrointest Endosc 1996;44:614-617.

The objective of the Scholarship is to facilitate trainees in gastroenterology (physicians, surgeons, radiologists, pathologists) of the Hospital Authority or the Society to undertake overseas training in gastroenterology.

Award

1. The Scholarship is awarded for a study period of 6 months or more.
2. The award is HK$50,000.
3. The candidate is required to produce evidence of approval to take study leave from his/her serving institution.
4. Upon completion of the training programme, the candidate is required to submit a report to the Society within 2 months, together with original letter of approval on study leave.
5. The award will be paid on satisfactory completion of the training programme.
6. An interview may be required by the Scholarship Selection Committee.
7. The Scholarship Selection Committee has the sole discretion to grant the Scholarship or to refuse any application.

Qualifications

Application is limited to fellow trainees in gastroenterology who are:-
1. staff of Hospital Authority or members of the Society.
2. holder of a post-graduate diploma (MHKCP/MRCP(UK) or equivalent)
3. in the Hong Kong Academy of Medicine Fellowship Training

Application

Interested parties should apply in writing enclosing the curriculum vitae (which should include personal data, professional qualifications, working experience, training experience in gastroenterology, publications and proposed training programme, as well as the reference from at least one supervisor or a referee if the applicant is in private practice). A application should be sent together with supporting documents to the Scholarship Selection Committee, Hong Kong Society of Gastroenterology, c/o MRS Rebeilla Ho, Executive Director, Secretariat: Rm1203 Bank of America Tower, 12 Harcourt Road, Central, Hong Kong (Tel: 2869 5933; Fax: 2869 9533 and E-mail: gastro@netvigator.com)

Deadline of application is February 28, 2001

References

Colorectal Cancer - Hereditary Cancer Syndromes and Significance of Genetic Diagnosis

Dr. Yuen Siu Tsan
Department of Pathology and Hereditary Gastrointedtinal Cancer Registry, Queen Mary Hospital, Hong Kong.

Introduction
Colorectal cancer (CRC) is a common malignancy throughout the world. It is the second most common cause of cancer death in most Western (Parker 1996). Recent epidemiology studies have shown that around 10-15% of CRC have resulted from hereditary factors (Houlston 1992). We have previously reported a rapidly rising incidence of CRC in Hong Kong in recent decades. In addition, there is an excess of patients, by up to four fold, in the younger age groups in Hong Kong as compared with Scotland and other Caucasian data (Yuen 1997). This high incidence in the young Hong Kong (Southern) Chinese was present twenty years ago and has not changed despite the rapid rise in the overall incidence of colorectal cancer in recent years, which is entirely attributable to classical, late-onset (>50 year old) patients. These data encourage search for a genetic basis for increased susceptibility of colorectal cancer in the Hong Kong Chinese population.

The two most well known hereditary colorectal cancer syndromes are familiar adenomatous polyposis (FAP) and hereditary non-polyposis colorectal cancer (HNPCC) syndromes.

Familial Adenomatous Polyposis (FAP) Syndrome
Familial Adenomatous Polyposis is an autosomal dominantly inherited disease. It has a comparable population incidence world wide affecting 1 in 7,000 to 15,000 individuals. The disease is characterised by the development of hundreds to thousands of adenomatous polyps throughout the colon usually in the second and third decades of life. Left untreated, some will progress to invasive carcinomas. Germline mutations in Adenomatous Polyposis Coli (APC) gene had been identified in over 85% of FAP patients (Powell 1993).

Hereditary Non-Polyposis Colorectal Cancer (HNPPC) Syndrome and the Situation in Hong Kong
Hereditary Non-Polypsis Colorectal Cancer (HNPCC) Syndrome was originally termed "Cancer Family Syndrome" highlighting the fact of familial clustering of large bowel cancer occurrence. HNPCC is an autosomal dominantly inherited disease with 80% penetrance. Individuals inherited with the disease show a marked increase in cancer susceptibility, developing especially cancer of the colon and rectum, which differs from sporadic colorectal cancer by that patients have an early age of onset, the tumors are located more in the proximal colon, and synchronous and metachronous colorectal cancer are common. In some families, members can also develop a variety of cancers of the endometrium, urinary tract, stomach, ovary and small bowel. HNPCC are also known as Lynch Syndromes I and II (Lynch 1996). The exact incidence of HNPCC is not known. Based on various data in different countries, the population incidence of HNPCC has been estimated to be about 1 in 500.

HNPCC tumors exhibit genome-wide microsatellite instability resulting from mutations in a group of genes responsible for DNA repair known as mismatch repair (MMR) genes (Aaltonen 1993). Germline mutations in two human MMR genes (hMSH2 and hMLH1) account for the great majority of the HNPCC kindreds (Liu 1996). The diagnosis of HNPCC has been difficult, as there is no known premonitory phenotype stigma like that of FAP. Currently, its diagnosis is based on the careful analysis of detailed family history. Using the Amsterdam criteria, the identification of HNPCC requires (1) three or more relatives with histologically verified CRC, one of whom is a first degree relative of the other two, FAP should be excluded; (2) CRC affecting at least two generations; and (3) one or more CRC cases diagnosed before age 50 (Vasen 1991). This characteristics however, is not invariably. Hong Kong has a high incidence of young colorectal cancer, but analysis of the data in the Hereditary Gastrointestinal Cancer Registry in Hong Kong did not reveal a high proportion of families fitting the Amsterdam criteria.

We recently analysed a series of young colorectal cancer in Hong Kong Chinese.

Our results showed that the incidence of microsatellite instability was very high in the young patients, being observed in more than 60% of those younger than 36 years old. And in many of these young patients, a germline mutation in one of the MTR genes can be found (Chan 1999). Furthermore, patients with Turcot’s syndrome also have germline mutation in the MTR genes (Chan 1999).

These data have important implications for the management of these young patients - total colectomy may be the surgical treatment of choice and subsequent surveillance programme is essential.

Screening, surveillance and genetic diagnosis
Family members of either FAP or HNPCC families are definitely at high risk of inheriting the disease and predisposed to develop colorectal and other cancers. There are enough evidence to support that screening and surveillance of these high-risk individuals can detect premalignant lesions, detect cancer at early stages and overall reduce CRC morbidity and mortality. In the past, all family members are subjected to screening by periodic lower endoscopy (every 1-2 years). As such screening may last up to 50 years; the burden in terms of cost and patient morbidity could be considerable.

It is now possible to improve diagnosis through genetic testing. Those shown to harbour germline mutations in the specific genes should be counselled in a socially, medically and psychologically sound fashion. Vigilant screening program, appropriate prophylactic treatment and possibly chemopreventive agents should be offered.

Family members tested negative for the particular gene mutation will be spared from further surveillance. Moreover, they are also relieved from the psychological burden of the risk of inheriting the disease.

References
a three year period. Once again, there was a reduction in the rate of secondary pancreatic infection in the group random-
ized to selective gut decontamination compared with controls (18% compared with 38%) and this was associated with a significant reduction in late mortality from 23% to 7%.
Early mortality was unchanged. From these data, it is reason-
able to conclude that prophylactic antibiotic therapy with a broad spectrum agent with adequate pancreatic tissue penetration (such as imipenem) will reduce the rate of secondary pancreatic infection in patients with pancreatic necrosis.

Anti-inflammatory therapy - lepipafant
Platelet activating factor is an inflammatory mediator released from many inflammatory cells and is considered to play an important role in the priming and amplification of the inflammatory response. The first clinical trial with the PAF antagonist, lepipafant, was reported by Kingsnorth and col-
leagues(10). This was a randomized comparison of lepipafant, 60mg daily for three days, with placebo in 83 unselected patients with acute pancreatitis. The main findings of this study were a reduction in levels of the pro-inflammatory cytokines, interleukin 6 and interleukin 8, with lepipafant treatment and an associated reduction in organ failure scores at the end of the three day treat-
ment period. Our own group(11) reported the results of a sec-
ond randomized trial, recruiting 50 patients with predicted severe acute pancreatitis, which broadly confirmed the find-
ings of the previous study. These encouraging results led to a multi-centre UK study(12) that recruited 290 patients with predicted severe attacks from 78 hospitals. The primary endpoint was the incidence of complications and secondary endpoints were re-
duction in organ failure scores and plasma markers of the inflam-
matory response. Unfortunately, 75% of patients who had early organ failure had evidence of this prior to study entry and the primary study endpoint was therefore invalidated. There was a significant reduction in organ failure scores at day three in the treatment group but by day seven, at completion of the trial infu-
sion, there was no difference between groups. There followed an international, multicenter study that recruited 1500 patients ran-
domised to receive lepipafant 100mg daily, lepipafant 10mg daily or placebo. This study recruited only those patients with symp-
toms of less than 48h duration and again was restricted to those with predicted severe attacks. Unfortunately, no difference in mortality or complication rate was observed between these groups and further development of lepipafant in acute pancreatitis has been abandoned.

Surgical management
The main indication for surgery in acute pancreatitis is infected pancreatic necrosis. Infection occurs in approximately 30-40% of patients with pancreatic necrosis(7,13,14). In recent years there has been interest in attempting to reduce the morbidity associated with infected necrosis by adopting a number of minimally invasive approaches.

Endoscopic therapy
Successful, endoscopic drainage of infected pancreatic necrosis has been described by Baron and colleagues(15). In their initial report, 11 patients with symptomatic pancreatic collections, associated with pancreatic necrosis, were treated by endoscopic transgastric drainage with two pig-tail stents, augmented in later cases by naso-cavitory lavage. The tech-
nique is widely used for the drainage of simple pancreatic pseudocysts but was previously considered contra-indicated in the presence of significant necrosis.

Percutaneous drainage
Percutaneous drainage has become the standard approach to the management of septic collections in the post-operative period but few would advocate its use in the context of infected pancreatic necrosis, as the presence of solid and semi-solid necrotic tissue within the collection inevitably leads to failure(16). Freney and colleagues(17) have described a modification of percutaneous drainage, where large-bore catheters are placed and com-
binied with repeated vigorous lavage. In their series of 34 patients with infected pancreatic necrosis, 16(49%) had resolution of sepsis without surgery.

Laparoscopic approach
Our group has reported a new technique for the management of infected pancreatic necrosis which we have applied in a sequential group of patients(18). Following placement of a per-
cutaneous drain in the collection, patients were taken to theatre and under general anaesthetic, the track dilated and the collec-
tion explored with a rigid nephroscope. Through the working channel of this instrument, forceps were used to debride the loose necrotic tissue. Continuous lavage with sterile dialysis fluid facilitated the debridement. Post-operative lavage was continued through a dual channel drain. Encouraging results were obtained in a group of ten, unselected patients, eight of whom survived. All had proven infected pancreatic necrosis be-
tween 19 and 187 days (median 24 days) from symptom. An in-
teresting observation was that 5 of these patients were managed without postoperative use of the intensive care unit. The man-
agement of severe acute pancreatitis remains a formidable chal-
lenge and, if anything, current management decisions have be-
come more complex in recent years. Early involvement of a specialist team, including radiologists, endoscopists, intensivists and experienced surgeons, is essential if patient outcome is to be optimised.

References
Major Meetings

International M eating GI Malignancies can be prevented and treated:
from the bench to the bedside Jerusalem and the Dead Sea
Feb 14-17 2001
For further information, please contact:
Nadir Arber, Head GI - Oncology Center
GI Malignancies
P.O. Box 29041
Tel Aviv 61290 Israel
Tel: +972 3 5175 150 Fax: +972 3 5175 155
E-mail: g@targetconf.com

6th Congress of the Asian Society of Hepato-Biliary-Pancreatic
Hong Kong Convention and Exhibition Centre
Feb 6-9 2001
For further information, please contact:
Chinese University of Hong Kong
Department of Surgery, M ichelle Sha
Prince of Wales Hospital, Shatin
Tel: (852) 2632 2951 Fax: (852) 2647 3074
Email: hhp0201@cuhk.edu.hk
URL: www.csu.med.cuhk.hk/sur/sur_proghmt

Hong Kong Society of Gastroenterology
Annual General Meeting & Symposium
March 29 2001
Hong Kong
For further information, please contact:
The Secretariat
Room 1203 Bank of America Tower,
12 Harcourt Road, Central, H. K.
Tel: (852) 2869 5933 Fax: (852) 2869 9533
E-mail: gastro@intivigator.com
URL: www.fhmsk.com.hk/hkg

American College of Physicians & American Society of Internal Medicine, Annual Joint Meeting
Atlanta, Georgia Atlanta Convention Center
March 29 - April 1 2001
For further information, please contact:
American College of Physicians
1901 Independence Mall West
Philadelphia, PA 19106-1572
Tel: (+1 (213) 351 2544 Fax: (+1 (213) 351 2528
E-mail: btume@acponline.org
URL: www.acponline.org

GI Conference
Milwaukee, Wisconsin: Pfister
March 30 - April 3 2001
For further information, please contact:
University of Wisconsin School of Medicine
Continuing Medical Education
2715 M arshall Court
Madison, WI 53705
Tel: (+1 (608) 263 2850 Fax: (+1 (608) 262 8421
E-mail: askelson@facstaff.wisc.edu
URL: www.medsch.wisc.edu/cm/one/conferencehtml

2nd symposium International “Sphincter Saving Treatment in Rectal Cancer”
in Lyon France
April 6 - 7 2001
For further information, please contact:
Univer's Claude Bernard Lyon I
Formation Continue Sante
8 avenue Rockefeller
69373 LYON CEDEX 08 - FRANCE
Tel: (+33 (0)4 78 77 75 52 Fax: (+33 (0)4 78 77 72 61
E-mail: FCCEP@rockefeller.univ-lyon1.fr
URL: focalserv.univ-lyon1.fr/focal/sante/Confs/SPHISTRCreshmt

36th Annual Meeting of the European Association for the Study of the Liver
Prague April 18-22 2001
For further information, please contact:
Liaison Bureau, Hospital Necker
H epatology Unit
149, rue de Sevres
R-75747 Paris Cedex 15 France
Tel: +33 (0)1 44 49 41 26 Fax: +33 (01) 44 49 51 65
E-mail: isabelle.porternd@nck.ap-hop-paris.fr

3rd Congress From Gene to Cure
Vrije Universiteit Amsterdam
April 26-28 2001
For further information, please contact:
De Boeke Elan 1105
NL-1081 HV Amsterdam
Tel: +31 (0)20 444 57 90 Fax: +31 (020) 444 58 25
E-mail: vu_conference@dienst.vu.nl
URL: www.freeinmt.com.my

8th Biennial Congress of the European Council of Coloproctology & 6th Central European Congress of Coloproctology and Viscerosynthesis
Prague April 29 - May 2 2001
For further information, please contact:
Agentur Carolina Ltd.
Jana Fialova, Congress Dept.
Albertov 7-3a P.O. Box 45
CZ-128 01 Prague 2
Tel: +420 (24) 99 08 25 Fax: +420 (24) 91 86 81
E-mail: kongresy2@carolina.cz
URL: www.colon2001.prague.cz

4th International Gastric Cancer New York
April 30 - May 2 2001
For further information, please contact:
Memorial Sloan-Kettering Cancer Center
Dr. Martin S. Karpel
1275 York Avenue
New York NY 10021
Fax: (+1 (212) 794 3184
E-mail: dpbp@dpbp.com.br

Digestive Disease Week, 102th Annual Meeting of GA, The American Gastroenterology Association
Washington, D.C.
May 20-23 2001
For further information, please contact:
American Gastroenterology Association
6900 Grove Road
Thorofare NJ 08086
Tel: +1 (856) 848 1000 Fax: +1 (856) 848 3522
E-mail: scapper@slackinc.com
URL: www.gastro.org/ddw.html

Europa Congress: European Congress of the International Hepato-Pancreato-Biliary Association
Amsterdam: Amsterdam RAI
May 27-30 2001
For further information, please contact:
Nicolaas Tulp Inst.
R. S. E. Weijenhoven
P. O. Box 2213
NL-1100 DS Amsterdam
Tel: +31 (020) 566 48 08 Fax: +31 (020) 696 32 28
E-mail: y.evewijenhoven@amc.uva.nl

International Congress on Pancreatic and Islet T ransplantation - Austria
Innsbruck: Kongresszentrum Innsbruck
June 13-15 2001
For further information, please contact:
Congress Innsbruck - Kongresszentrum Innsbruck
Tel: +43 (0521) 59 36 111
Fax: +43 (0521) 59 36 111
E-mail: m.kostner@congress-innsbruck.at
URL: www.congress-innsbruck.at

Asian Pacific Digestive Week 2001
(incorporating Australian Gastroenterology Week)
Sydney, Australia
September 23-28
For further information, please contact:
Gastroenterological Society of Australia
145 M acquarie Street, NSW 2000 Australia
Tel: 61 (0) 2 9256 5454 Fax: 61 (0) 2 9241 4586
E-mail: gea@acron.edu.au
URL: www.gea.org.au