**President Message**

**Message from Dr. Benjamin C.Y. Wong, President**

Welcome all of you to the first issue of our newsletter in 2010.

Our Annual General Meeting cum Scientific Meeting was held successfully on 11 March 2010. The new council was formed. I would like to welcome our Vice President Dr. Vincent K.S. Leung and new co-opted council member Dr. Wai-Fan Luk. I would like to thank Dr. Yat-Wah Yeung, our immediate past president, for leading the Society in the past two years. My heartfelt gratitude goes to Professor Joseph Sung for his contributions to the Society and serving the Council in the past 14 years during which he was President from 2002 to 2004.

On behalf of the Society, I wish to express my sincere thanks to all who have contributed to the Society, Prof. Khean-Lee Goh, Prof. Raymond Dubois and Prof. Peter Ferenci for their enlightening lectures, Prof. Justin Wu for organizing the 2010 Annual General Meeting cum Scientific Meeting, Dr. Wai-Mo Hui and Prof. Justin Wu for editing this Newsletter; Prof. John Dent, Prof. Geoffrey Farrell, Prof. Joseph Sung, Dr. James Fung, Dr. David But and Dr. John Chan for contributing to the scientific updates in this Newsletter and last but not least our sponsors from the industry.

I look forward to seeing you all in the Joint Annual Scientific Meeting on 4 September 2010 and other events.

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**Scientific Updates**

**A Symptom-based and Endoscopic Survey of Reflux Disease in China – The SILC Study**

**Professor John Dent**

Department of Gastroenterology, Hepatology and General Medicine  
Royal Adelaide Hospital  
Adelaide, Australia

The Systematic Investigation of reflux in China (SILC) study is the largest study of gastro-oesophageal reflux disease (GORD) in China to date, and the first to apply the Montréal Definition of reflux. The Montréal Definition classifies GORD as heartburn and/or regurgitation that, if mild, occurs at least twice per week; but if moderate or severe, occurs at least once per week.¹

The SILC study took structured population samples from five centres in China: Beijing, Xi’an, Wuhan, Shanghai and Guangzhou. Vigorous efforts by the field researchers resulted in a remarkable final sample of 16,091 completed surveys (89.4% response rate). At each site, urban and rural dwellers were equally sampled.

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¹ The Montréal Definition of reflux is based on the presence of symptoms of heartburn and/or regurgitation, along with endoscopic evidence of reflux. It was developed by the Montréal Group in 2006 and has since become the standard for defining GORD in clinical trials. It classifies GORD as mild if symptoms occur at least twice per week, moderate if at least once per week, and severe if at least daily. This classification is based on the frequency and intensity of symptoms and the presence of oesophagitis on endoscopy.
Professor Dent commented that few reflux patients seek healthcare in China, as 85.3% of these subjects had not been previously diagnosed and had not sought treatment. GORD was also more common amongst rural respondents (3.9%) than urban respondents (2.4%; odds ratio 1.4).²

A sub-sample from Shanghai volunteered for endoscopy (n=1,029). Professor Dent noted that these patients had more symptom-based reflux disease than those who declined endoscopy (n=2,124), were slightly older, had more family history of GI disease and peptic ulcer, and were more from rural areas. With these differences in mind, the results found that 17% had peptic ulcer and 93% of those were H. pylori-positive.³ H. pylori appears to be protective against GORD.⁴ This was supported by the SILC data: Prevalence of reflux oesophagitis in H. pylori-positive subjects was 5.3% compared with 9.3% in H. pylori-negative subjects (odds ratio 0.55).⁵

There was a low prevalence of suspected Barrett's oesophagus, which was seen in only 2.2% of those with symptom-based GORD and 1.8% of those without symptom-based GORD. Measurements of quality of life (QOL) and body mass index (BMI) were carried out on 20% of respondents from each centre (n=3,200). No association was found between BMI and reflux disease. However, subjects with GORD did experience highly significant impaired QOL across all domains of the 36-item medical outcomes study short-form health survey (SF-36), compared with subjects without GORD (p≤0.001).⁶ Professor Dent commented that it was surprising that only 15% of people with GORD sought help, given the impact of the disease on their QOL.

Professor Dent concluded that the prevalence of GORD in China is 3.1%, and that it significantly impairs QOL in affected patients. Prevalence varies between regions and rural Chinese are more affected than their urban counterparts. The rate of healthcare-seeking is low, perhaps indicating a need for education on GORD and available treatments.

References
The latter should eventually lead to rational therapy other than the obvious – it is a good thing, healthwise, not to get fat, and if you are, it is best to exercise, take an optimal (“heart healthy”, “diabetic”) diet and lose some centimetres from your waist circumference! The former (genetic factors) is cold comfort for Asians who have different bodily composition that Europid and African races; a higher proportion of adipose tissue, and a more truncal distribution (VAT versus SAT). Thus, it is possible that Asians (other data indicate south Asians>Chinese>Europids) are at higher risk of diabetes, and may also be at higher risk of NAFD, although this has not yet been established.

It is also becoming clear that accumulation of fat in liver is a failure of bodily lipid homeostasis. Accumulation of VAT could occur either because different factors regulate this site of fatty tissue, and/or because the physiologically more appropriate (or safer) subcutaneous storage sites are no longer expanding in the face of energy excess (the root cause of overweight/obesity). Such “adipose (SAT) restriction” or “adipose failure” is increasingly evident in human studies, and is now well documented in animal models, including our own recent studies with a line of obese mice, the Fat Aussie (faz/foz), attributable to mutation in the murine homolog of the Alström syndrome gene (Alms1). While faz/foz NOD/B10 or C57B16 mice fed a high fat diet develop obesity, profound insulin resistance, diabetes, dyslipidemia and non-alcoholic steatohepatitis (NASH), faz/foz balb-c mice develop similar obesity without diabetes or significant NAFD. The difference is that SAT expansion reaches its maximum in the strains that develop more profound metabolic complications, following which serum adiponectin levels fall profoundly, diabetes is precipitated and NASH occurs. It is clearly now important to establish the factors which determine SAT expansion during states of overnutrition; any critical locus of regulatory control could be a novel therapeutic target.

Somewhat clearer are the implications of “adipose failure”. It appears to occur when insulin resistance is established, and is associated with and could cause an inflammatory state. It results in failure to secrete adiponectin, the master “insulin sensitising” cytokine that is secreted only by differentiated SAT, not VAT or de-differentiated adipocytes.

An Asia-Pacific working party on NAFLD, organised from Hong Kong and convened in this city, emphasised that while the jury was still out on whether NAFLD is as severe among Asian people as in “the west”, it is becoming just as common. The working party also acknowledged practical difficulties in making a confident diagnosis without liver biopsy, an invasive procedure usually not welcomed by the affected person! Pragmatic guidelines on who might have NAFLD were promulgated in JGH (a world first!), but should to be kept under review, particularly as real limitations need to be acknowledged about the sensitivity and specificity of ultrasound and serum ALT level for liver fat. In NASH, the fat has “got out of hand”, or, to use a parlance now familiar to diabetologists and vascular physicians, lipotoxicity of the liver occurs. The manifestations are hepato cellular damage and inflammation that can lead to fibrosis and cirrhosis, placing patients at risk for liver-related mortality from liver failure and hepatocellular carcinoma (HCC).

The natural history of NAFLD/NASH is still being written, and differing views have been issued. We do at least know that a proportion of cases can progress to liver complications (liver failure, bleeding varices, HCC), but progression is slow. Even childhood cases can progress to liver failure, and it recurs with some rapidity following liver transplantation. This chilling scenario may be inevitable, given the metabolic origins of the disease and the nature of immunosuppressive drugs (corticosteroids) to make metabolic settings worse. A clearer picture is emerging of the predisposing factors to “progressive NASH”. These include any fibrosis on original biopsy showing NASH (simple steatosis is usually benign), diabetes, severe obesity, numerous components of metabolic syndrome, and older age. Thus, we have noted that about 10% of our NASH patients have had liver complications over last 3 years; most are aged >65 yrs and virtually all give the history of longstanding diabetes. In light of such a long natural history, it cannot yet be concluded that the outcome will be better in Asians than Europids, particularly as median age in most Asian series is 1-2 decades younger than those from North America, Australia and Europe.

The co-morbidity of obesity and steatosis on other liver diseases is considerable, but varies between diseases. Thus, evidence is accruing that steatosis in those with chronic hepatitis B is usually due to metabolic factors, as in NAFLD. Until recently, there was scant evidence that it could worsen either treatment response or disease outcome (fibrotic stage), but a recent article by Wong and Wong and colleagues disputes this (hep B patients with metabolic syndrome were more likely to have cirrhosis). The exact opposite applies in hepatitis C; obesity, insulin resistance and steatosis worsen disease severity, reduce treatment responsiveness, and may increase risk of HCC in those with cirrhosis. For alcoholic liver disease and hemochromatosis, obesity likely worsens severity of liver injury, but there are few data for rarer diseases like PBC. Obesity does increase risk of methotrexate-induced liver disease.

In closing, some broader perspectives may be apposite for a professional society such as yours. First, obesity increases risk for many gastrointestinal disorders: GERD, gallstones, bowel cancer are just a few. Increased echogenicity on hepatic ultrasonography often comes to light during investigation of abdominal pain, as might abnormal liver function tests or an unexpectedly raised serum ferritin. Before dismissing such findings with the idea “she or he must have a fatty liver, everyone does!”, consider the second point. Undoubtedly the most important health implication of NAFLD is that the liver is the barometer of metabolic health, a window into a waxy interior. Hong Kong colleagues have also taught us that patients often develop diabetes after the discovery of fatty liver disease, and “NAFLD now, heart attack later” is much more than a catchy title for an editorial! Every clinician has a responsibility to consider the wider health ramifications of fat – it is rarely good, perhaps often neutral, but can be VERY BAD for you and your liver!
Selected references
Fan JG, Farrell GC. VAT fat is bad for the liver, SAT fat is not! (Editorial) J Gastroenterol Hepatol 2008;23:829-32.

A meta-analysis of studies of haemoclip or thermocoagulation found that there was no difference between them in terms of successful haemostasis or rebleeding rate. Professor Sung noted the choice will depend on the site of the ulcer, whether the area has already received thermocoagulation treatment, the experience of the doctor and endoscopy assistance available.

The use of high-dose IV proton pump inhibitors (PPIs) to reduce the risk of recurrent peptic ulcer bleeding after endoscopic haemostasis is controversial, as results of studies in this area have been conflicting. While European studies found no overall benefit of PPI treatment, Asian studies reported clinical benefits. As a result, an international study (16 countries) was designed comparing IV esomeprazole and placebo for 72 hours, followed by oral esomeprazole for 27 days. Fewer patients receiving IV PPI had recurrent bleeding within 72 hours than those receiving placebo (5.9% vs. 10.3%; p=0.026). In addition, there was a significant reduction in endoscopic re-treatment, blood transfusions and hospital stays because of recurrent bleeding in the esomeprazole group (p<0.05). While there was a trend toward fewer deaths and less need for surgery in the esomeprazole group, this did not reach statistical significance.

Figure 1. Kaplan-Meier estimate of the cumulative percentage of patients with recurrent bleeding within 30 days

Professor Sung concluded that endoscopy should be offered within 24 hours to patients with bleeding peptic ulcers, and that dual therapy is preferable to monotherapy. There is a role for PPIs in reducing the risk of re-bleeding after endoscopic treatment. Doctors should be alert to non-bleeding complications, as these are more often the cause of death than bleeding complications in ulcer patients.

Recent Advances in the Therapy of Peptic Ulcer Bleeding
Professor Joseph JY Sung
Mok Hiu Yiu Endowed Chair Professor of Medicine
Department of Medicine and Therapeutics
Director, Institute of Digestive Disease; Associate Dean, Faculty of Medicine
The Chinese University of Hong Kong
Hong Kong

Early endoscopy in patients with peptic ulcer bleeding allows for timely diagnosis, location and treatment of the source of bleeding, thus, enables prompt discharge of low-risk patients. The exact timing is debated, but Professor Sung explained that as long as endoscopy is performed within 24 hours, patient outcomes are not compromised.

Professor Sung stressed that it is now clear epinephrine injection should not be used as monotherapy, but should be combined with either thermocoagulation or mechanical treatment. Adding a second procedure after epinephrine injection reduces further bleeding, need for surgery and mortality in patients with bleeding peptic ulcer.1 A meta-analysis found that dual endoscopic therapy is significantly superior to epinephrine injection alone, but has no advantage over thermal or mechanical monotherapy in improving the outcome of patients with high-risk peptic ulcer bleeding.2 Therefore, applying a clip or thermal device to a bleeding ulcer is an effective monotherapy. Adding a sclerosant after injection therapy does not help3 and Professor Sung cautioned it can in fact be harmful.
Hepatitis B (HBV) infection is highly prevalent in Asia, and the natural history of the disease differs between Asians and Caucasians. Asians tend to acquire the disease perinatally or in early childhood, and are more likely to experience eventual progressive disease. In contrast, Caucasians tend to acquire the disease in late childhood or early adulthood, and are more likely to experience eventual quiescent disease after hepatitis B e antigen (HBeAg) seroconversion.

The goal of therapy is to permanently suppress HBV replication.1 In the short-term, this is reflected by HBeAg seroconversion and ALT normalization, to achieve the long-term goals of preventing cirrhosis, hepatic decompensation, and hepatocellular carcinoma (HCC).

Current guidelines recommend stopping therapy approximately 6 months after the patient has undergone HBeAg seroconversion.1-3 Dr Fung noted that while seroconversion was sometimes viewed as the endpoint of therapy, in the Asian context it may only be a milestone as there is a high relapse rate after discontinuation. In one Korean study, 36% of patients had relapsed 1 year after discontinuation of lamivudine (LAM) treatment.4 In another study, 56% of patients relapsed within 6 months of LAM discontinuation.5

Dr Fung and colleagues have investigated the effect of stopping or continuing LAM therapy after HBeAg seroconversion in Asian patients.6 They found that continuing LAM resulted in a much higher proportion of undetectable HBV DNA at last follow-up (78% vs 0%; p<0.001; Figure 1), and a lower number of biochemical flares (16% vs 44%; p<0.001), when compared with those who ceased LAM. Only 10% of patients who continued LAM developed resistance mutations.

References

Figure 1. Undetectable HBV DNA at time of last follow-up in patients continuing or stopping LAM after seroconversion6

HBV, hepatitis B; LAM, lamivudine

Prolonged maintenance of HBV DNA suppression may only be achieved by continuing antiviral therapy after HBeAg seroconversion. For some patients, cost is a limiting factor when considering long-term therapy. A strategy that may reduce cost for patients is to use newer agents such as entecavir (ETV) to achieve early viral suppression, and then switch to LAM for maintenance. Early viral suppression is important in predicting long-term outcome and likelihood of resistance.7

Dr Fung and colleagues are currently conducting a 2-year prospective trial investigating the switch to LAM or continuation with ETV after initial optimal viral suppression with ETV. Preliminary results suggest switching to LAM may be effective, and data from 48 weeks of follow-up will be presented at the Annual Liver Meeting of the American Association for the Study of Liver Diseases in November 2009.
Natural History and Non-invasive Assessment of Primary Biliary Cirrhosis
(Summary of Dissertation May 2009 – Part A)

Dr David Yiu Kuen But
Queen Mary Hospital

Natural History of Primary Biliary Cirrhosis

Introduction
Primary biliary cirrhosis (PBC) is a chronic liver disease characterized by progressive destruction of intrahepatic bile ducts, portal tract inflammation, periporal fibrosis and finally development of liver cirrhosis. In the past, the disease was often diagnosed at late stage because of its non-specific early symptoms of malaise and pruritus. Jaundice often represents advance disease. Nowadays, routine blood tests including liver transaminases and ductal enzymes such as alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT) and even anti-mitochondrial antibodies (AMA) test used in health assessments are becoming more and more common. Therefore, more cases of PBC are diagnosed in the very early phases than in the past. Based on the clinical features and blood tests results, PBC can be divided into 4 phases: pre-clinical phase, asymptomatic phase, symptomatic phase and liver insufficiency phase.

Preclinical Phase
Patients at this phase can be defined as the positivity of AMA without derangement of liver ductal or parenchymal enzymes or development of symptoms of liver disease. The earliest known manifestation of PBC is the appearance of antimitochondrial reactivity in the serum immunoglobulins and on the surface of biliary epithelial cells. Liver histology in these patients are usually diagnostic of or compatible with early PBC. Patients at this phase would eventually develop symptoms typical of PBC and their liver function tests persistently show cholestasis, although the progression of disease is often slow [1]. A longitudinal follow-up study of 29 untreated preclinical patients assessed every year from first-detected AMA for a median of 18 years showed that 24 (83%) developed cholestatic liver tests and 22 (76%) developed fatigue and/or pruritus. But not all patients with anti-mitochondrial reactivity alone will develop clinical manifestations of PBC.

Asymptomatic Phase
The asymptomatic phase is characterized by persistently abnormal liver function test without any symptoms of PBC such as pruritus, fatigue and jaundice. A number of prospective and retrospective studies have shown that a significant proportion of patients in this phase will develop symptoms or have progression of liver disease with jaundice and portal hypertension over time [2]. Those remain asymptomatic seem to have similar survival compared with the general population while the symptomatic patients will have shortened survival. However, clinical, biochemical and histologic variables could not identify any prognostic significance in the prediction of who would develop symptoms or who would remain symptom-free in the study [2]. Patients at this phase may already have advanced histologic lesions [3].

Symptomatic Phase
Symptoms in PBC can be divided into systemic symptoms such as fatigue, pruritus and abdominal pain, and portal hypertensive symptoms such as ascites and bleeding varices. Typically, the systemic symptoms of fatigue and pruritus begin years before evidence of portal hypertension appears, although this rule certainly has exceptions. Fatigue is the most common symptom experience by patients with PBC. It has been estimated that 40% to 80% of PBC patient experience various degree of fatigue and with more than half of those who have fatigue considering it the worst symptom of the disease [4]. The presence and the severity of fatigue and/or pruritus have not found to be associated with disease progression. However, once features of portal hypertension are noted, the disease outlook is abridged. The mean time from clinical diagnosis of ascites or peripheral edema to death in 143 patients with PBC was 3.1 years in a study [5].

Liver Insufficiency Phase
This is a preterminal phase characterized by worsening jaundice and encephalopathy. The mean survival once the bilirubin is 2.0 mg/dL is 4 years. When it reaches 6.0 mg/dL, the mean survival is only 2 years [6]. Liver transplantation is only treatment option that improves survival at this phase. Paradoxically, pruritus, ALP elevation, and hypercholesterolemia may all improve in this preterminal stage.

Mayo Risk Model
The development of the Mayo risk model was based on 312 patients who had been carefully diagnosed and enrolled in clinical trials for D-penicillamine [7]. As this medication was found ineffective and the study protocol stipulated that patients should not take any other medications which may potentially influence the clinical course of the disease, the progression of disease in this group of patients was deemed appropriate to represent the natural history of PBC. The survival models were based on Cox’s proportional hazards assumptions. Out of the 312 patients, 125 died and 19 underwent liver transplantation after a median follow-up of 66 months. Out of an array of demographic, clinical, biochemical and histologic variables in these patients, age, level of serum bilirubin, albumin, prothrombin time (PT) and the severity of edema were identified as statistically and clinically significant predictor of survival. Based on these variables, a summary score, or risk score R, was obtained and patient survival estimated. Later, in a study looking at the longitudinal follow-up of risk scores in a group of patients with PBC for a minimum of 2 years, it was found that the original Mayo model tended to overestimate the survival time of patients with poor short-term survival. In a group of PBC patients who died within 2 years, the estimated survival was more than 6 months of the actual survival in 67% of the patients. Subsequently, an updated Mayo model risk for prediction of short-term survival was re-developed based on the extended follow-up of the original patient cohort in the natural history study [8].
The Mayo risk score for PBC is the most commonly used model for prediction of patient survival because it has been extensively validated in external populations and does not require histological examination. Its calculation is simple and involves mainly objective parameters except the detection and the severity assessment of edema. It is also applicable to patients on ursodeoxycholic acid (UDCA) treatment.

Biochemical Response to Ursodeoxycholic Acid
In recent years large studies [9,10,11] examining the effects of UDCA on transplant-free survivals in patient with PBC show that the biochemical improvements, namely ALP, bilirubin and albumin levels, from the treatment is associated with better survivals. In a Spanish study [9], 192 patients with PBC treated with UDCA for 1.5 to 14 years were assessed for the disease course and survival. The response to treatment was defined by an ALP decrease greater than 40% of baseline values or normal levels after 1 year of treatment (Barcelona Criteria). The result was compared with the predicted survival obtained by the Mayo model and the standardized matched Spanish population. The observed survival of the treated patients was higher than that predicted by the Mayo model but lower than the control population. But for the responders to treatment, the survival was similar to that of the control population (p=0.15). A French study [10] used several combinations of serum bilirubin, ALP, and aspartate aminotransferase (AST) threshold values to predict outcome after 1 year of UDCA treatment in 292 patients with PBC. Patients showing ALP < 3 upper limit of normal (ULN), AST <2 ULN, and bilirubin ≤ 1 mg/dL after 1 year of UDCA had a 10-year transplant-free survival rate of 90%, compared to 15% for those who did not. In a Dutch study [11], 375 patients were followed-up for 9.7 years prospectively. The patients were divided into early, moderately advanced or advanced groups according to their baseline bilirubin and albumin levels. The definition of biochemical response was the normalization of abnormal bilirubin and/or albumin levels. Similarly to the Spanish and French studies, responders had a better prognosis. The prognosis of early PBC was comparable for responders and non-responders; but the prognosis of those with (moderately) advanced disease was significantly better for the responders. These study results show that treatment-induced changes may help to fine-tune predictions regarding the course of the disease, allowing the identification of patients in whom further alternative or additional therapy is needed and a more frequent monitoring of their disease progression is warranted. With the different biochemical response criteria set in these studies, the response rates range from 61% to 76%. Given the very slow and progressive nature of the disease, the longer-term outcomes of the responders are awaited.

Liver-to-Spleen Volume Ratio
In Japan, the investigators examined liver-to-spleen volume (LV/SV) ratios in PBC patients using computed tomography (CT) in 77 patients [12]. Using the median values of the ratios in their cohort, the patients were divided into low and high LV/SV ratio groups. A low LV/SV ratio was found to be a poor prognostic factor. The frequency of esophageal varices was significantly higher. Twelve of the 39 patients having low LV/SV ratio died or underwent liver transplantation compared with one of the 38 patients with a high ratio (p=0.001). Moreover, a low LV/SV ratio predicted the progression from asymptomatic stage to symptomatic stage. The LV/SV ratio provides an easy and objective method of PBC prognostication.

Aspartate Aminotransferase-to-Alanine Aminotransferase Ratio
Aspartate aminotransferase (AST) to alanine aminotransferase (ALT) ratio (AAR) has been applied in the evaluation of liver fibrosis in patients with PBC, although less extensively than in patients with chronic hepatitis C infection. In two studies [13,14], a high AAR was associated with advanced fibrosis (histologic stage 3 & 4 according to Scheuer’s criteria) Using a cut-off value of 1.0, AAR was found to have a better sensitivity (82-91%) than specificity (77-79%); with a good negative predictive value of 93 to 96.4%. The reason for increased AAR in liver fibrosis is unknown. There are suggestions that the sinusoidal clearance of AST decreases in cirrhotic patients. But its use in heavy alcohol users is limited because of the invariably raised AST.

(To be continued)
Computed tomography colonography (CTC) is a minimally invasive examination of the colon via computed tomography (CT) instead of optical colonoscopy. After full bowel preparation, patients undergo insufflation of the colon with room air or carbon dioxide and CT scans are taken. The images are reviewed at an off-line workstation (Figure 1) by combination of two- and three-dimensional images and findings reported.

Dr Chan stressed that proper bowel preparation was essential to obtaining useful results. Tagging of residual fluid and stool, using various oral contrast preparations or a commercial product such as Tagitol V®, can help distinguish these items from polyps. Adequate insufflation is also vital. Multi-detector CT allows for shorter scan times and better imaging. Dr Chan noted that the average effective radiation dose from CTC is 10 mSv, which is comparable to barium enema,¹ but that low-dose techniques can reduce this to 2 mSv.

Contrast-enhanced CTC may be used in cases where the colon is poorly prepared. It increases the specificity for polyp detection as polyps are enhanced when contrast is given. If malignant polyps or stenotic tumours are detected, contrast will also be given.

Visualization software allows for several different views of the colon: automatic segmentation and centerline for endoluminal view, endoscopic view and cut-open view. The endoscopic view mimics the view seen by an optical endoscope. The cut-open view allows the whole colon to be reviewed rapidly. The software can also electronically subtract tagged fluid and stool from the image. Computer-aided diagnosis tools are also available.

CTC is indicated for patients who cannot undergo sedation or the manipulation required for barium enema; for elderly, infirm or anticoagulated patients; or for patients where regular colonoscopy failed to reach the caecum or where an obstructing tumour was found. The use of CTC as a screening tool for colorectal cancer is more controversial. The 2008 guidelines from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology now accept CTC as a screening option (every 5 years),² as does the American College of Gastroenterology.³ However, the Asia Pacific Working Group on Colorectal Cancer states that CTC is “not preferred” as a screening modality.⁴

References
Coming Soon

12th JOINT ANNUAL SCIENTIFIC MEETING
4 September 2010 (Saturday), 1:00 – 6:10 p.m.
Level 7, Langham Place Hotel
555 Shanghai Street, Mongkok
Kowloon, Hong Kong

Organizing Chairperson: Dr. Annie O.O. Chan
Co-Sponsors: AstraZeneca & GlaxoSmithKline

Co-organizers:
The Hong Kong Society of Gastroenterology
Hong Kong Society of Digestive Endoscopy
Hong Kong Society for Coloproctology
The Hong Kong Association for the Study of Liver Diseases
The Hong Kong Society of Gastrointestinal Motility

<table>
<thead>
<tr>
<th>Speakers</th>
<th>Topics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prof. Philip Quirke (UK)</td>
<td>Impact of margins in rectal cancer</td>
</tr>
<tr>
<td>Dr. Garrett C.L. Ho (HKSH)</td>
<td>PET/CT in detection of peritoneal carcinomatosis from colonic malignancy</td>
</tr>
<tr>
<td>Prof. Philip Quirke (UK)</td>
<td>Recent development in GI pathology</td>
</tr>
<tr>
<td>Prof. Hans L. Tillmann (USA)</td>
<td>Understanding HBV resistance to prevent treatment failure</td>
</tr>
<tr>
<td>Dr. Grace L.H. Wong (PWH)</td>
<td>Contrast-Enhanced Ultrasonography (CEUS) of the Liver</td>
</tr>
<tr>
<td>Prof. Hans L. Tillmann (USA)</td>
<td>HBsAg quantification; from a research tool to a standard diagnostic</td>
</tr>
<tr>
<td>Dr. William C.S. Meng (OLMH)</td>
<td>Multi-discipline approach to treatment of Chronic Constipation</td>
</tr>
<tr>
<td>Dr. In-Son Leong (PYNEH)</td>
<td>Achalasia in Hong Kong</td>
</tr>
<tr>
<td>Dr. Larry H. Lai (CUHK)</td>
<td>New Advances in Small Bowel Enteroscopy</td>
</tr>
</tbody>
</table>

More information is available from www.hksge.org/event

Highlights

Annual General Meeting and Scientific Meeting
11 March 2010, Langham Place Mongkok, Hong Kong

Organizing Chairman: Professor Justin C.Y. Wu

The scientific meeting was successful attended by 101 specialists and medical practitioners. Professor Khean Lee Goh from the University of Malaya Medical Center, Kuala Lumpur, Malaysia and Professor Raymond DuBois from The University of Texas M.D. Anderson Cancer Center, Houston, USA were the distinguished guests awarded Honorary Fellowship of the Society. In commemoration of the award, both of them were presented a plaque of the Society by Dr. Yat-Wah Yeung, President of the Society.

Professor Goh delivered a talk on “Asia At The Crossroad-Changing Epidemiology of Gastrointestinal Diseases in Asia” and Professor DuBois’ was on “Inflammation and Cancer: Molecular Targets and Opportunities.” Both talks being captivating and informative were well received and aroused a number of questions from the floor.

The subsequent EGM/AGM was attended by 36 fellows and members during which proposed amendments to the Memorandum & Articles of Association were passed and the Society’s annual report and financial statements for the year of 2009 were presented by the Chairman and Hon. Treasurer respectively. Seven Council members were re-elected to the Council for the term 2010-2012.

A Certificate of Appreciation was presented to each of the ten sponsors in appreciation of their support and contributions towards the meeting and they were AstraZeneca, Bristol-Myers Squibb, Eisai, Ferring, Given Imaging, GlaxoSmithKline, Novartis, Pfizer, Roche and Takeda.

Most attendants stayed for dinner and continued sharing their views.
This symposium, jointly organized by The Hong Kong Society of Gastroenterology and The Hong Kong Association for the Study of the Liver Diseases, was a successful one attended by 56 gastroenterologists, hepatologists and medical practitioners. Professor Peter Ferenci from the Medical University of Vienna, Austria delivered a talk on "The role of Silymarin in the management of liver diseases: latest clinical updates" which was enlightening and informative. The role of IV infusion in treating toxic mushroom ingestion is well established in Europe and its role in treatment of resistant Hepatitis C is encouraging. After the symposium, most attendants stayed and enjoyed the dinner.