HONG KONG MEDICAL FORUM

7th HONG KONG MEDICAL FORUM

6 - 7 July 2002
(Saturday and Sunday)
Phoenix Suite
Hong Kong Convention and Exhibition Centre

PROGRAMME BOOK

Department of Medicine
Faculty of Medicine
The University of Hong Kong
Queen Mary Hospital
Hong Kong

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Welcome Message

The Hong Kong Medical Forum is an annual postgraduate Continuing Medical & Nursing Education (CME/CNE) meeting organized by the Department of Medicine (Faculty of Medicine) of The University of Hong Kong (Queen Mary Hospital, Hong Kong). The Forum is held on a weekend in July each year focussing on topics of current interests and the latest developments in the medical field. Each year distinguished overseas and local speakers are invited to share with us the most recent medical advances in their specialty area. We are confident that participants like yourself, general practitioners, family and specialist physicians, clinicians, basic and higher trainees, nurses and allied health professionals will find the programme of great value in updating your awareness of the new developments and helping with your continuing professional development. We also hope that this will provide an excellent opportunity for our peers to renew friendship and make new ones.

As part of our commitment to providing quality and innovative CME/CNE programmes, we have produced a video compact disc (VCD) to accompany the symposium on "Common Medical Investigative and Treatment Procedures: When, Why & How?". Apart from being distributed to all participants of the 7th HKMF, it will also be used as one of our Department's clinical training tools and distributed to all medical and nursing staff of the University Department of Medicine and medical students. We hope that you will find this useful and we thank you for your support in our CME/CNE activities. Last but not least, we would like to extend our thanks to all the speakers and chairmen for taking the time out of their busy schedule to spend the time with us and for their invaluable participation, contribution and hard work. As well as the medical and nursing staff and patients who have contributed to the content of the VCD. Thank you.

Professor Lam Wah Kit
Head, Department of Medicine

Professor Lau Chak Sang
Chairman, Organizing Committee

Organizing Committee

Chairman : A/Prof Lau Chak Sing

Members :
Prof Lai Kar Neng
Prof Cyrus R Kumana
Prof Kwong Yok Lam
A/Prof Ho Shu Leong
A/Prof Kathryn CB Tan
A/Prof Tse Hung Fat
Ms Selina WK Wong

General Forum Information

Venue
All scientific sessions, including the talks for the dinner and lunch symposium, will take place in Phoenix Suite, Room 301, 3/F (New Wing), Hong Kong Convention & Exhibition Centre (HKCEC), 1 Expo Drive, Wan Chai, HK.

General Forum Information – Cont.

Forum Secretariat
Executive Officer, HKMF, University Department of Medicine, Room 409, Professorial Block, Queen Mary Hospital, Pok Fu Lam, Hong Kong
Tel : +852-2855-4607 Fax : +852-2816-2863 Email : denise@hku.hk http://www.hku.hk/medicine/hkmf/

The Secretariat Room is located in Room 302-303, 3/F, (New Wing) and will be open for the duration of the Forum.

Registration
The Registration Desk is located in the Phoenix Suite foyer, 3/F (New Wing) outside the Secretariat Room. It will be open during the hours of 13:00 to 18:30 on Saturday and 09:00 to 17:00 on Sunday. Participants can register during those times. On-site registrants can make payments in cash or cheque.

Registration is inclusive of :

- Attendance of all scientific sessions
- Admission to industry exhibition
- Coffee/tea breaks on the registered days
- Dinner & luncheon (if applicable – see below Food & Beverages arrangements)
- Name badge
- Programme and abstract book
- Certificate of Attendance
- "Common Medical Investigative and Treatment Procedures: When, Why and How?" VCD
- Forum briefcase/satchel
- Complimentary gift

Food & Beverages
Coffee and tea will be served in the Phoenix Suite foyer, 3/F (New Wing) and nearby the Exhibition Room.

Lunch and dinner will be served in the Egret Suite, Room 601, 6/F (Old Wing). Seating is limited and is subject to a first come first served pre-booking at the time of payment basis. Admission is permitted with dinner and lunch tickets.

Please collect your ticket(s) by the end of the coffee break previous to the dinner / lunch (i.e. 16:40 on Saturday & 11:10 on Sunday) otherwise it will be given away to other participants. Day registrants are only entitled to meals on the day they have registered. Thank you for your co-operations.

The HKCEC also has a number of food & beverage outlets, a list is available at the Registration Desk for your information.

Audio-Visual Preview
PowerPoint/slide previews are available in the Secretariat Room, Room 302-303, 3/F, (New Wing) and will be open for the duration of the Forum. Staff will be on site to offer any assistants.

Admission Name Badges
Participants are requested to wear their name badge (provided at registration) in a clearly visible place as all Forum functions require the Name Badge for admission.

Certificate of Attendance
A Certificate of Attendance will be issue to all pre-registered participants upon registration. On-site registrants can collect theirs at the end of the Forum. No certificate will be issue after the Forum.

Audio-Visual Recordings
Please note that all scientific sessions may be recorded for future publications or production of Forum related material. However participant identification will not be revealed without prior consent.

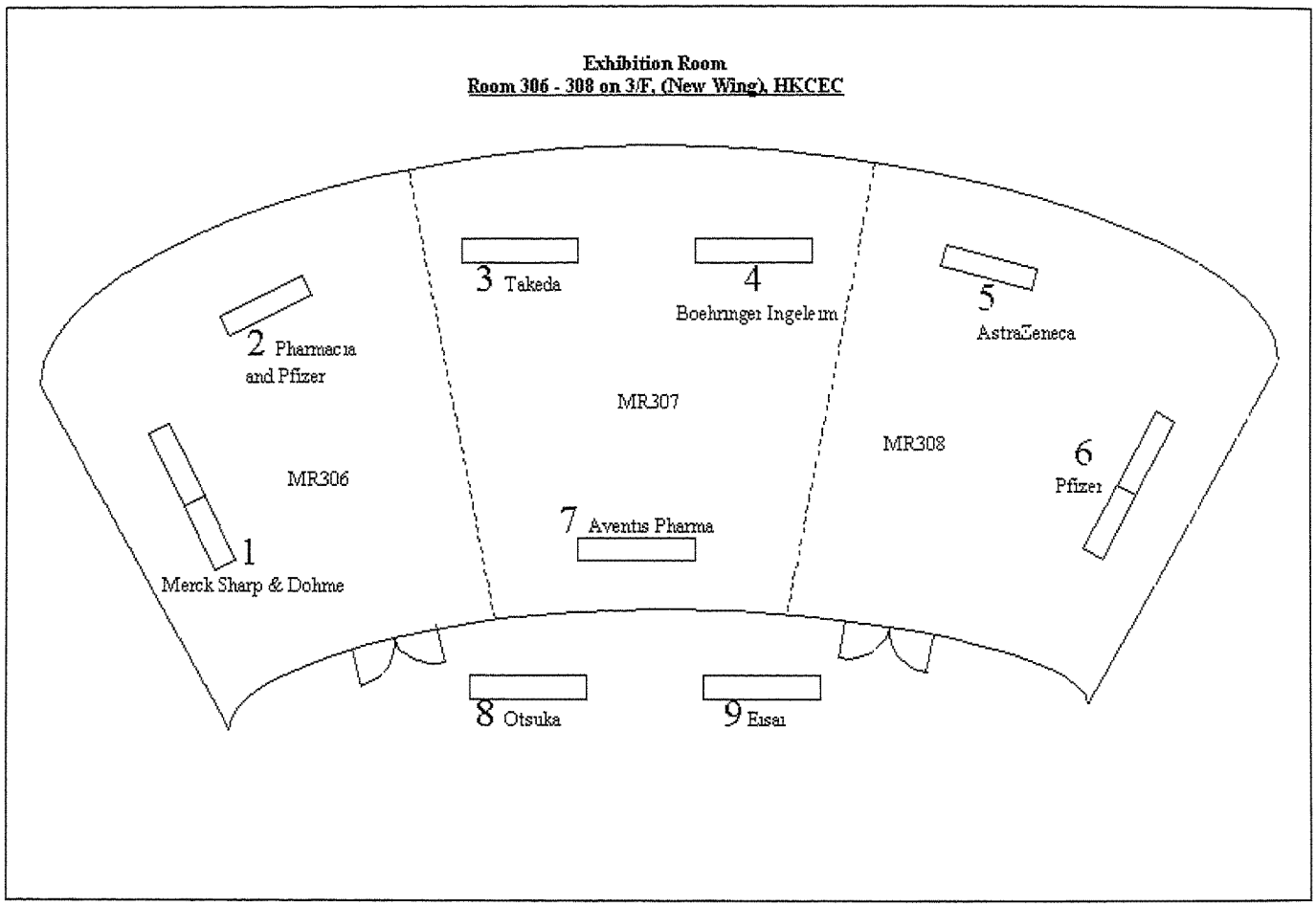
Security
The Hong Kong Medical Forum will not be responsible for lost and damage to the private property of participants. Participants should take care not to leave their belongings unattended at all times.

Language
The official language of the meeting will be English. There will be no simultaneous translation.

Exhibition

An industry exhibition on the latest medical-related products will be held concurrently in Rooms 306-308, 3/F (New Wing) for the duration of the Forum. Please feel free to visit it at any time.

1. Merck Sharp & Dohme (Asia) Ltd
2. Pharmacia and Pfizer
3. Takeda Chemical Industries (Taiwan) Ltd – Hong Kong Branch
4. Boehringer Ingelheim (Hong Kong) Ltd
5. AstraZeneca Hong Kong Limited
6. Pfizer Corporation Hong Kong Ltd
7. Aventis Pharma Limited
8. Otsuka Pharmaceutical (HK) Ltd
9. Eisai



Sponsors

- Otsuka Pharmaceutical (HK) Ltd (Dinner Symposium)
- Eisai (Lunch Symposium)

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- Aventis Pharma Limited
- Boehringer Ingelheim (Hong Kong) Ltd
- Schering HK Ltd
- Rotta Pharmaceuticals

Continuing Medical/Nursing Education (CME/CNE) Accreditations

Fellows/members of each CME/CNE awarding institution should sign the attendance sheets at the Registration Desk. The completed sheets will be returned to each awarding institution at the completion of the Forum. The points listed are for 2 days unless otherwise specified. If it is not stated than the awarding institutions has not specified the points for individual days. Please contact them if you have any queries. Fellows/members should sign the attendance sheets for each individual day or session for which points have been specified..

Continuing Medical Education (CME) Accreditations

- Hong Kong College of Anaesthesiologists - 12 points (non-anaesthetic activities)
- Hong Kong College of Community Medicine - 9 points
- The College of Dental Surgeons of Hong Kong - 6 points (3 points per day)
- Hong Kong College of Emergency Medicine - 6 points
- Hong Kong College of Family Physicians - 7.5 points in Category 6.2
  Saturday – 3.5 points Sunday – 4 points
- Hong Kong College of Obstetricians & Gynaecologists - 5 points (under non O & G category)
- The College of Ophthalmologists of Hong Kong - 4 points
- Hong Kong College of Orthopaedic Surgeons – 2 Category B points
- Hong Kong College of Otorhinolaryngologists – 2 points
- Hong Kong College of Paediatricians - 10 points Category A
  Saturday afternoon (14:00 – 18:30) – 3 points
  Saturday evening (18:30 – 19:30) – 1 point
  Sunday morning (09:00 13:45) – 3 points
  Sunday afternoon (14:30 – 17:40) – 3 points
- Hong Kong College of Pathologists – 11.5 points (1 point per hour)
- Hong Kong College of Physicians -13 points
  Saturday – 5 points Sunday – 8 points
- Hong Kong College of Psychiatrists – 10 points
  Saturday – 4 points Sunday – 6 points
- Hong Kong College of Radiologists –11.5 points
  Saturday – 4 points Sunday – 7.5 points
- The College of Surgeons of Hong Kong - 10.5 points
- Hong Kong Medical Council CME Programme for Non-Specialists - 12 points
  Saturday – 5 points Sunday – 7 points
  Inclusive of the following CME Programme Administrators:
  - Department of Health
  - Hong Kong Academy of Medicine
  - Hong Kong Doctors Union Ltd
  - Hong Kong Medical Association

Continuing Nursing Education (CNE) Accreditations:

- Hong Kong College of Nursing – 7 points
  Saturday – 3 points Sunday – 4 points
- Institute of Advanced Nursing Studies (IANS), Hospital Authority CNE System – 13.5 points

Invited Speakers

Prof Stephen Wing Keung Cheng
Professor, Division of Vascular Surgery, Department of Surgery, University of Hong Kong Medical Centre, Queen Mary Hospital, HK

A/Prof Raymond Tak Fai Cheung
Associate Professor, Division of Neurology, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Miss Frances Hing Chiu
Nurse Specialist, Renal Dialysis Unit, Division of Nephrology, University Department of Medicine, Queen Mary Hospital, HK

Prof Ronnie Fass
Associate Professor & Director of Motility Laboratory, University of Arizona Health Sciences Center, Southern Arizona VA Health Care System, Tucson, Arizona, USA

Dr Wayne Hsing Cheng Hu
Senior Medical Officer, Division of Geriatrics, University Department of Medicine, Queen Mary Hospital, HK

Prof Mary Sau Man Ip
Chair Professor, Division of Respiratory & Critical Care Medicine, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

A/Prof Ip Wing Yuk
Associate Professor, Department of Orthopaedic Surgery, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Prof Cyrus R Kumana
Professor and Chief of Division of Clinical Pharmacology & Therapeutics, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Prof Kwong Yok Lam
Professor, Division of Haematology, Oncology & Bone Marrow Transplantation, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Dr Lam Bing
Medical Officer, Division of Respiratory & Critical Care Medicine, University Department of Medicine, Queen Mary Hospital, HK

Prof Karen Siu Ling Lam
Professor of Medicine & Chief of Division of Endocrinology & Metabolism, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Dr Lam Yui Ming
Medical Officer, Division of Cardiology, University Department of Medicine, Queen Mary Hospital, HK

Invited Speakers —Cont.

Prof Lau Chak Sing
Professor, Division of Rheumatology & Immunology, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Dr Lee Pui Yin
Medical Officer, Division of Cardiology, University Department of Medicine, Queen Mary Hospital, HK

Dr Li Fu Keung
Senior Medical Officer, Division of Nephrology, University Department of Medicine, Queen Mary Hospital, HK

Dr Albert Kwok Wai Lie
Consultant, Division of Haematology, Oncology & Bone Marrow Transplantation, University Department of Medicine, Queen Mary Hospital, HK

Dr Lui Wai Man
Senior Medical Officer, Division of Neurosurgery, Department of Surgery, University of Hong Kong Medical Centre, Queen Mary Hospital, HK

Dr Windsor Mak
Associate Consultant, Division of Neurology, University Department of Medicine, Queen Mary Hospital, HK

Dr Samuel R Money
Clinical Associate Professor of Surgery, Tulane University Medical Center, New Orleans, Louisiana, USA

Miss Josepha Wai Ming Tai
Nurse Specialist, Bone Marrow Transplant Unit, Division of Haematology, Oncology & Bone Marrow Transplantation, University Department of Medicine, Queen Mary Hospital, HK

A/Prof Kenneth Wah Tak Tsang
Associate Professor, Division of Respiratory & Critical Care Medicine, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

A/Prof Tse Hung Fat
Associate Professor, Division of Cardiology, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Dr Nelson Ming Sun Wat,
Senior Medical Officer, Division of Endocrinology & Metabolism, University Department of Medicine, Queen Mary Hospital, HK

A/Prof Benjamin Chun Yu Wong
Associate Professor, Division of Gastroenterology & Hepatology, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Dr Adrian Young Yuen Wu
Assistant Professor, Division of Rheumatology & Clinical Immunology, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Chairmen

Dr Raymond Hon Wah Chan
Senior Medical Officer, Division of Cardiology, University Department of Medicine, Queen Mary Hospital, HK

Prof Stephen Wing Keung Cheng
Professor, Division of Vascular Surgery, Department of Surgery, University of Hong Kong Medical Centre, Queen Mary Hospital, HK

Dr Ignatius Kum Po Cheng
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Miss Tavia Cheng
Ward Manager, University Department of Medicine, Queen Mary Hospital, HK

A/Prof Raymond Tak Fai Cheung
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Dr Ching Chi Kwong
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Dr Yuk Ling Yu
Honorary Clinical Associate Professor, Division of Neurology, Department of Medicine, Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, HK

Ronnie Fass

Ronnie Fass, MD, is an Associate Professor of Medicine at the University of Arizona in Tucson, Director of the Motility Laboratories at the Southern Arizona VA Health Care System and University of Arizona Health Sciences Center. Dr. Fass earned his medical degree at The Faculty of Health Sciences, Ben-Gurion University in Beer-Shiva, Israel and completed an internship and residency in internal medicine at the University of Arizona Health Sciences Center, where he later became a Chief Resident of Internal Medicine. He then completed a fellowship in gastroenterology at the University of California in Los Angeles. Dr. Fass is a member of several professional organizations, including the American College of Physicians, the American College of Gastroenterology and the American Gastrointestinal Association. He is also involved with national committees that include the American Motility Society Accreditation Committee and the Research and Publication Committees of the American College of Gastroenterology.

A frequent lecturer and presenter, Dr. Fass is a journal reviewer and has published abstracts, editorials, letters and articles in journals such as *Gastroenterology, Gut, American Family Physician, American Journal of Gastroenterology* and *Archives of Internal Medicine.*

Programme - 6 July 2002, Saturday
Phoenix Suite, Room 301, 3/F (New Wing)

TIME	ACTIVITY	SPEAKER
13:00 - 18:30	**Registration**	
14:00 - 14:10	**Opening Remarks by Prof Lam Wah Kit, Head of Medicine**	
14:10 – 16:00	**Joint Medical and Surgical Symposium – "Advances in Vascular Diseases" I** Chairmen: Yu Yuk Ling & Stephen Cheng	
	Cardiovascular Risk Factors: Old and New	Karen Lam
	Advances in the Treatment of Acute Ischaemic Stroke	Raymond Cheung
	Recent Advances in Endovascular Neurosurgery	Lui Wai Man
16:00 – 16:40	**Coffee / Tea Break**	
16:40 – 18:30	**Joint Medical and Surgical Symposium – "Advances in Vascular Diseases" II** Chairmen: Kathryn Tan & Raymond Cheung	
	Carotid Endarterectomy for Stroke Prevention	Stephen Cheng
	Medical Therapy for Vascular Disease	Samuel Money
	Distinguishing Benefits from Risk Reduction Conferred by Statins	Cyrus Kumana
18:30 – 21:30	**Dinner Symposium sponsored by Otsuka Pharmaceutical (Hong Kong) Ltd** Chairman: Lau Chak Sing	
	Drug Therapy for Claudication	Samuel Money
	Diabetic Foot Problems	Ip Wing Yuk
19:30	**Buffet Dinner** Egret Suite, Room 601, 6/F (Old Wing) Availability subject to pre-booking with ticket admissions	
21:30	**End**	

N.B. Please note that all scientific sessions may be recorded for future publications or production of Forum related material. However participant identification will not be revealed without prior consent.

Programme - 7 July 2002, Sunday
Phoenix Suite, Room 301, 3/F (New Wing)

TIME	ACTIVITY	SPEAKER
09:00 – 17:00	**Registration**	
09:00 – 10:30	**Common Medical Investigative and Treatment Procedures: When, Why and How? I** Chairmen: Selina Wong & Leonard Li	
	Allergy Skin Testing	Adrian Wu
	Ambulatory Peritoneal Dialysis	Frances Chiu
	Cardiac Catheterisation	Lee Pui Yin
	Care and Maintenance of Hickman's Catheter	Josepha Tai
10:30 – 11:10	**Coffee / Tea Break**	
11:10 – 13:00	**Common Medical Investigative and Treatment Procedures: When, Why and How? II** Chairmen: Ching Chi Kong & Lau Chak Sing	
	Echocardiography	Lam Yui Ming
	Endoscopic Retrograde Cholangio-Pancreatography	Benjamin Wong
	Clinical Neurophysiology	Windsor Mak
	Plasmapheresis	Albert Lie
	Sleep Study	Mary Ip & Lam Bing
13:00 - 14:30	**Lunch Symposium sponsored by Eisai** Chairman: Benjamin Wong	
	Update on Acid Reflux	Ronnie Fass
13:45	**Buffet Luncheon** Egret Suite, Room 601, 6/F (Old Wing) Availability subject to pre-booking with ticket admissions	
14:30 – 15:50	**Managing Common Medical Emergencies I** Chairmen: Ignatius Cheng & George Lau	
	Acute Renal Insufficiency	Li Fu Keung
	Aspiration Pneumonia	Kenneth Tsang & Wayne Hu
	Cardiac Dysrhythmia	Tse Hung Fat
	Gastrointestinal Emergencies	Ronnie Fass
15:50 – 16:20	**Coffee / Tea Break**	
16:20 – 17:40	**Managing Common Medical Emergencies II** Chairmen: Tavia Cheng & Raymond Chan	
	Anaphylaxis	Adrian Wu
	Vein Thrombosis: Diagnosis and Management	Kwong Yok Lam
	Diabetic Emergencies	Nelson Wat
	Emergency Rheumatic Disorders	Lau Chak Sing
17:40	**End**	

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Cardiovascular Risk Factors: Old and New
by
Karen Lam

Cardiovascular diseases are the major causes of mortality in most developed cities including Hong Kong, where stroke and coronary artery disease together constitute the top killer in the local population. Diabetes, smoking, hypertension, high LDL cholesterol and low HDL cholesterol levels, family history of premature coronary artery disease and age have long been recognised as the major cardiovascular risk factors. The contribution of these well-established risk factors has been supported by findings from epidemiological studies, both cross-sectional and prospective. Large multi-centre interventional studies conducted in recent years have provided further evidence for the importance of hypertension, high LDL cholesterol, low HDL cholesterol and diabetes in the development and progression of cardiovascular diseases. The other well-established modifiable risk factors including physical inactivity and obesity, especially central obesity, enhance cardiovascular risk mainly through increased insulin resistance. The importance of insulin resistance and the Metabolic Syndrome as a distinct cardiovascular risk factor is formally acknowledged in the 2001 NCEP ATPIII guidelines. The Metabolic Syndrome is identified by the presence of three of five of the following conditions: abdominal obesity, hypertension, high triglycerides, low HDL cholesterol, and high fasting glucose. In the treatment of such patients, the concept of non-HDL cholesterol and its treatment goals have been introduced. Other cardiovascular risk factors identified in recent years from epidemiological studies include the prothrombotic and atherogenic lipoprotein(a) the level of which is largely genetically determined, the vasculotoxic effect of homocysteinaemia which is determined both genetically and by environmental factors especially the dietary intake of folic acid, and chronic vascular inflammation as suggested by a raised c-reactive protein. Evidence from interventional studies for their involvement in accelerated atherosclerosis is still pending although an improvement in flow-mediated vasodilatation has been reported, following the lowering of homocysteine using folate supplementation, the reduction of c-reactive protein using atorvastatin, and the treatment of patients with coronary artery disease and chlamydia infection using azithromycin. In light of the above coronary risk factors, both old and new, it would appear that in the primary and secondary prevention of cardiovascular diseases, life style modifications are as important as the judicious use of available medications.

Advances in the Treatment of Acute Ischaemic Stroke
by
Raymond Cheung

Stroke is a medical and neurological emergency. The diagnosis of stroke should be accurate, general management is targeted at prevention and treatment of neurological and systemic complications, and specific management depends to the stroke type and the pathogenic mechanisms. Computed tomography of the brain is required to exclude intracranial haemorrhage and brain tumour. Managing stroke patients in an acute stroke unit improves the outcome of stroke. Presence of an unstable ischaemic penumbra permits acute intervention of ischaemic stroke within a short time window. In highly selected patients with ischaemic stroke, intravenous recombinant tissue plasminogen activator or a modified viper venom within 3 hours of onset or intra-arterial pro-urokinase within 6 hours improves functional outcomes. Nevertheless, the treatment time window is narrow, and majority of stroke patients are ineligible. Advanced neuroimaging technologies may reveal the existence of the ischemic penumbra and permit selection of patients for acute therapy. Early use of low-dose aspirin is a cost-effective treatment for patients with ischemic stroke. Early use of anticoagulation prevents recurrent embolism, but the risk of bleeding is increased. Available evidence from clinical trials does not support indiscriminate use of anticoagulation in acute ischemic stroke. Neurosurgical treatment may be useful in selected patients with ischaemic or haemorrhagic strokes. Prevention of recurrence and rehabilitation are the core components in subsequent management.

Recent Advances in Endovascular Neurosurgery
by
Lui Wai Man

Endovascular Neurosurgery deals with minimally invasive endovascular treatment of vascular diseases of the brain, head and neck, and spine. Commonly treated conditions include aneurysms, vascular malformations, tumours, and vaso-occlusive disease. The interventionist navigates from within the vascular system to study and treat these abnormalities. In many cases endovascular neurosurgery is used as an adjunct or alternative to conventional open surgery, to facilitate the treatment of previously untreatable or difficult lesions.

Treatment of Aneurysms

In 1991, Guglielmi detachable coil (GDC) embolization was introduced as an endovascular alternative to surgical clipping of intracranial aneurysms. This procedure involves the controlled transarterial delivery of tiny platinum coils into the aneurysm sac so as to occlude the aneurysm, thus preventing rupture and haemorrhage.

Arteriovenous Malformations

These complicated lesions often require some combination of multi-modality therapy, including radiation therapy, endovascular embolization, and surgical removal. Embolization can reduce the size of, and flow through the lesion so as to facilitate surgical resection or increase the efficacy of radiation.

Tumour Embolization

Many tumours are amenable to preoperative embolization, wherein their blood supply is reduced or eliminated so as to facilitate surgical resection. Embolization results in shorter surgeries, decreased blood loss, and diminished transfusion requirements.

Stroke Treatment and Prevention

Direct intra-arterial thrombolytic infusion into occluded vessels can improve recanalization of proximal cerebral vessels, which has been proven to improve stroke outcome.

Recent advances in stent and balloon technology have expanded revascularization possibilities in the carotid, vertebrobasilar, and intracranial circulations. These procedures can be performed when surgical therapy is difficult, as in previously irradiated necks or redo-endarterectomies.

Medical Therapy for Vascular Disease
by
Samuel Money

Historically, the treatment of peripheral vascular disease has revolved around surgical therapy. It has become obvious that the treatment of the patient with peripheral vascular disease requires more than simple surgical care. The natural history of PVD in the U. S. population has been extensively studied. Of 100 patients with vascular disease, 50% would be asymptomatic, simply have a diminished pulse. Approximately 10% of the patients would have critical limb ischemia and would probably require urgent interventions. The other 40% would present with intermittent claudication. From a medical point of view, approximately 30% of patients will die within five years — most of which will be secondary to cerebrovascular or cardiovascular events. Another 20% will have non-fatal myocardial infarctions or strokes. If one were to look at the outcomes in the extremities in the patients with intermittent claudication, it is quite interesting. Approximately three-quarters will remain stable. Only 4% of the patients in the intermittent claudication group will require major amputation.

These are numerous modifiable risk factors that occur in patients with PAD. It is obvious that diabetes and obesity are two of the most common. However, hypertension, smoking and dyslipidemia are also treatable. In addition to that, there is a whole host of newly diagnosed entities, such as hyperhomocystinemia and factor V Liden that can also be addressed. It is important to state that smoking is the single most important avoidable risk factor for the development of PAD and intermittent claudication. Basic care plan for patients with PAD is necessary. These revolve around patient education, lifestyle changes, risk-factor reduction, exercise and drug therapies.

Distinguishing Benefits from Risk Reduction Conferred by Statins

by
Cyrus Kumana

Whilst it is commonly inferred that coronary heart disease (CHD) risk reduction achieved by long-term intervention with statins equates to benefits, this communication addresses a number of fallacies pertaining to this assumption. To appreciate benefits better, we and others[1,2] have advocated resorting to number needed to treat (NNT) over a finite period of time [calculated as the reciprocal of absolute risk reduction (ARR)], in preference to relative risk reduction (RRR). Using published data from the only large-scale, randomised, double-blind, placebo-controlled clinical trials of CHD prevention involving statins, the table below summarises these parameters with respect to individual trials as well as relevant composites of two or more studies. Thus, in trial No 1 (see table), though taking statin was associated with a clinically and statistically significant RRR in CHD events, all cause mortality was slightly greater. In the latter scenario, arguably averting CHD events and their sequelae was merely at the expense of other adverse outcomes, such that no overall benefit actually accrued. Second, regarding CHD event prevention, RRRs for individual trials and the composite of different studies were all of the same order, whilst respective NNT/year values varied markedly. In the latter context, statins appeared to confer greater CHD benefit (smaller NNT/year values) among patients with prior CHD rather than those with pre-existing hypercholesterolaemia; corresponding differences being statistically significant between 4S and the other trials and between clinical trial combinations incorporating recruits with and without CHD ($P < 0.05$; non overlapping CIs). In terms of effort needed to achieve a single unit of gain in a defined period, NNT/year was therefore a more discriminating measure of benefit than RRR. Third, several authoritative guidelines endorse the view that, irrespective of age, intervention with statins should be undertaken once the absolute risk of CHD (and therefore the scope for ARR) exceeds a predefined threshold.[3-5] However, in the scenario of increasing age, smaller NNTs for CHD events do not necessarily result in greater cumulative benefit. For example, on average a 50-year-old man might expect to survive till age 75 years, but aged 70 years – expected survival may be till 80 years. Therefore preventing a CHD event at age 50 rather than 70 years is liable to yield a much greater cumulative benefit in terms of life years (and quality of life) gained. In conclusion, CHD risk reduction attributable to statins does not always parallel overall benefit.

Analysis of CHD Event Prevention with Statins: Single & Combined Trials

Individual Trials[†] (Combined trials)	Patient/Subject Characteristics	Years[†] of follow-up	CHD Event: mean (95% CIs)		
			% RRR[§]	NNT[§]	NNT/year
1) AFCAPS/TexCAPS	No CHD + 'normal' cholesterol	5.4	37 (21–50)	49 (33 – 99)	256 (170 – 514)
2) WOSCOPS	No CHD + cholesterol	4.9	31 (17 – 43)	44 (29 – 95)	217 (141 – 463)
3) CARE	CHD + 'normal' cholesterol	5.0	24 (9 – 36)	33 (20 – 99)	167 (100 – 496)
4) LIPID	CHD + 'normal' cholesterol [‡]	6.1	24 (12 – 35)	28 (20 – 48)	172 (122 – 294)
5) 4S	CHD + cholesterol	5.2	34 (25 – 41)	12 (9 – 17)	63 (49 – 89)
(1 + 2)	No CHD	5.15	33 (22 – 42)	47 (34 – 74)	241 (177 – 382)
(3 + 4 + 5)	CHD	5.4	26 (20 – 31)	23 (19 – 31)	129 (103 – 172)
(1 + 3 + 4)	'Normal' cholesterol	5.5	25 (18 – 31)	39 (30 – 55)	212 (163 – 302)
(2 + 5)	Cholesterol	5.15	30 (23 – 37)	29 (22 – 43)	151 (114 – 221)

AFCAPS/TexCAPS indicates AirForce/Texas Coronary Atherosclerosis Prevention Study; WOSCOPS, West of Scotland Coronary Prevention Study; CARE, Cholesterol and Recurrent Events; LIPID, Long-term Intervention with Pravastatin in Ischaemic Disease trial; 4S, Scandinavian Simvastatin Survival Study. [‡]Median value but broad range. [†]Published mean or median durations for individual trials, and for combined trials – the average of respective mean or median individual trial follow-up durations. [§] For the analysis of combined trials, a weighted combination of RRRs from the respective trials was calculated; weightings being inversely proportional to the variance of the RRR in each trial. Variance and CI computations for RRR and NNT were based on these weighted estimates.

References: 1) Kumana et al 1999, JAMA. 282: 1899; 2) TH Lam and others 2000, JAMA.; 284: 303; 3) Ramsay et al 1996, Lancet. 348: 387; 4) Jackson 2000 Brit Med J 320: 709; 5) NCEP-ATP III 2001, JAMA; 285: 248

Ilomedin **Composition** 1 ml Ilomedin contains 0.134 mg iloprost trometamol (equivalent to 0.100 mg iloprost) in aequeous solution. **Indications** For the treatment of peripheral occlusive disease especially where there is skin ulceration or where surgery is contraindicated. Also, for the treatment of advanced thromboangiitis obliterans (Buerger's disease) with critical limb ischaemia in cases where revascularisation is not indicated and severe secondary Raynaud's phenomenon unresponsive to other therapies. (Indications vary from country to country. Please refer to the data sheet for country specific indications.) **Contraindications** Pregnancy, lactation, hypersensitivity to iloprost. Conditions where the effects of ilomedin on platelets might increase the risk of haemorrhage (e.g. active peptic ulcers, trauma, intracranial haemorrhage). Severe coronary heart disease or unstable angina; myocardial infarction within the last six months; acute or chronic congestive heart failure (NYHA II–IV); arrhythmias relevant to the prognosis; suspected pulmonary congestion. **Undesirable effects** The most common and frequently observed side effects at the specified dosage are facial flush and headaches. Malaise, nausea, vomiting, cramp-like abdominal pain, diarrhoea, sweating, a sensation of warmth, and weakness are likely to occur during prolonged infusion. Pain in the affected limb, paraesthesia, tiredness, increased temperature, fever, chill, states of confusion, apathy, sedation, agitation, lowering or increase of blood pressure, tachycardias, arrhythmias, extra-systoles, and restlessness have been reported. All the side effects usually disappear quickly with dose reduction. Arthralgia and allergic reactions may occur. Isolated cases of dyspnoea and bronchial asthma have been reported and individual cases of acute pulmonary edema of heart failure have been observed in elderly patients with advanced arteriosclerosis. Reddening and pain may occur at the infusion site. In some cases, cutaneous vasodilation may give rise to streaky erythema above the infusion vein. Iloprost may provoke angina, espedially in patients with coronary heart disease. Hypotensive episodes occuring at low doses of Ilomedin have been reported in individual cases. **Special warnings and special precautions for use** Surgery should not be delayed in patients requiring urgent amputation (e.g. in infected gangrene). Patients should be strongly advised to stop smoking. In patients with low blood pressure care should be taken to avoid further hypotension. The possibility of orthostatic hypotension should be borne in mind in patients getting up from the lying to an upright position after the end of administration. Ilomedin should be used only after dilution. Because of the possibility of interactions, no other drug should be added to the ready-to-use infusion solution. The paravascular infusion of undiluted Ilomedin can lead to local changes at theinjection site. Oral ingestion and contact with mucous membranes must be avoided. On contact with the skin, iloprost may provoke long-lasting but pa inless erythema. Suitable precautions should therefore be taken to avoid iloprost contact with the skin. In the event of such contact, the affected area should be washed immediately with copius amounts of water or saline. Schering Aktiengesellschaft · D-13342 Berlin

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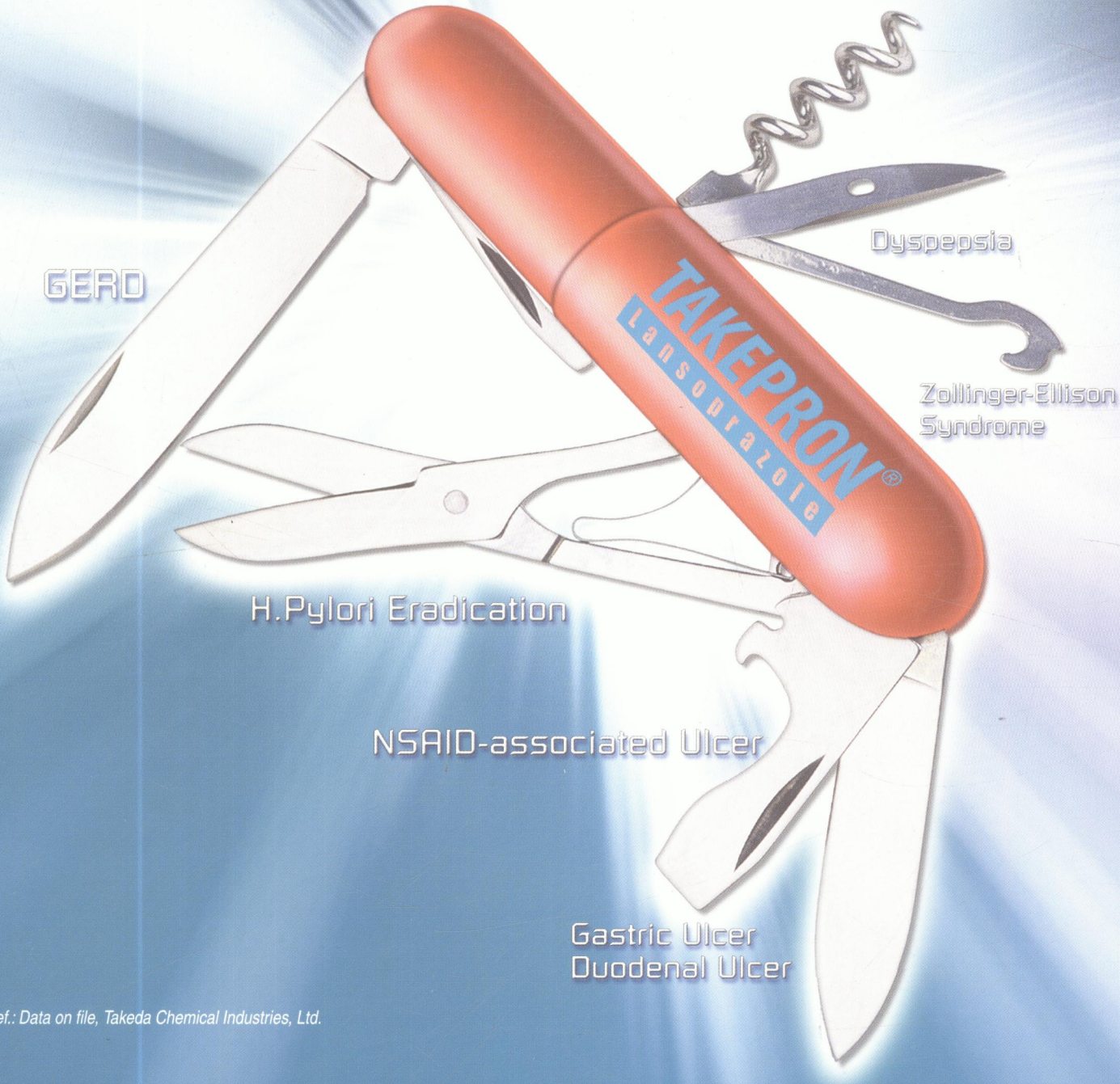

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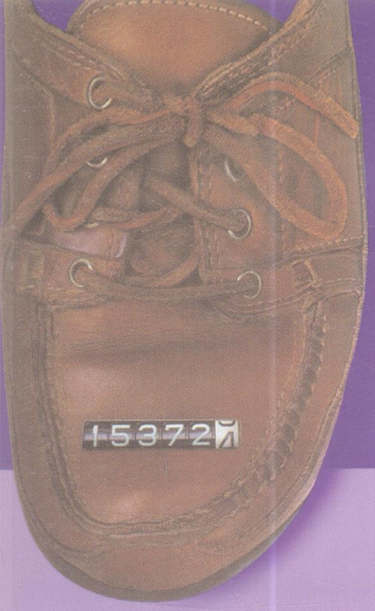

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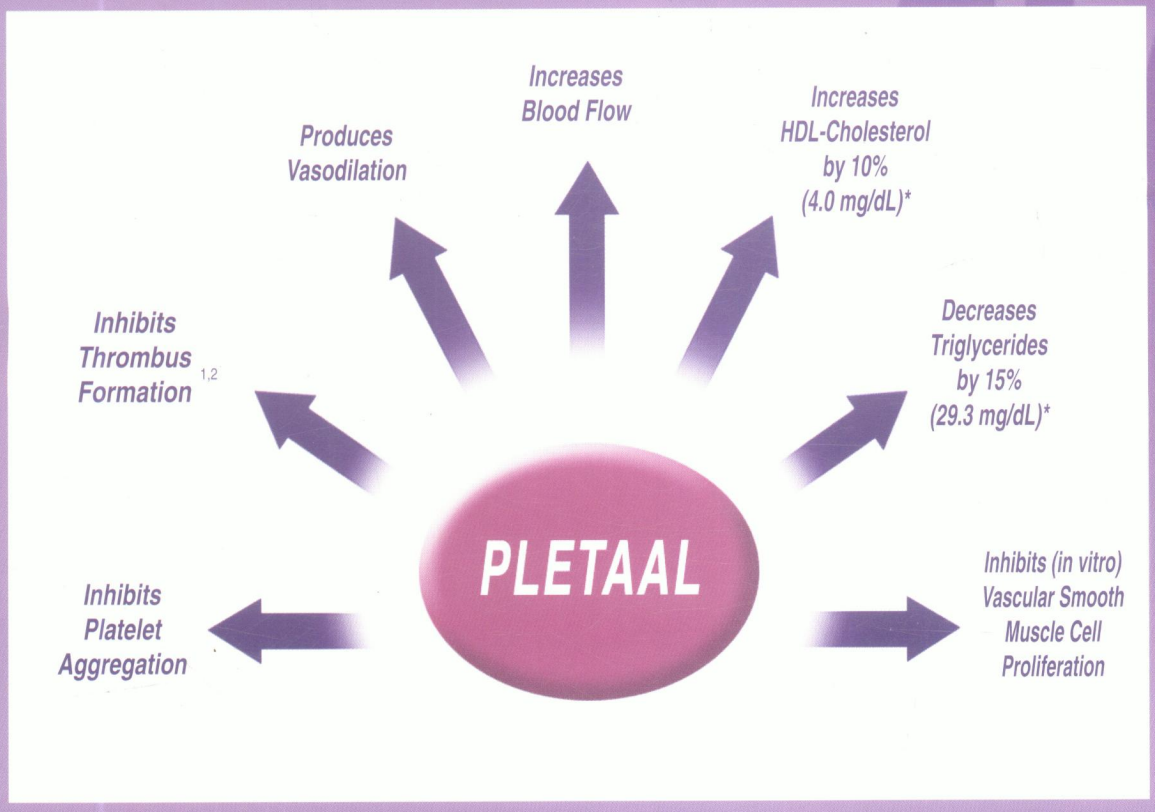

* Data are from a 12-week, multicenter, randomized, double-blind trial comparing PLETAAL 100 mg BID (n=95) with placebo (n=94). PLETAAL is not indicated for the treatment of dyslipidemias.

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Drug Therapy for Claudication
by
Samuel Money

Presently, there are two medications on the market in the United States for the treatment of intermittent claudication. Pentoxifylline (Trental) is a methylxanthine derivative. It was approved in 1984. It is a haemorrheologic agent with some weak anti-platelet activity and has some peripheral vasodilatory actions. This drug has minimal effect in randomised prospective trials. In some of the more recent randomized prospective trials, the effects of the drug parallel that of placebo. Cilostazol is a phosphodiesterase type-3 inhibitor. It was first released commercially in January 1999. This drug is a platelet aggregation inhibitor. In addition to that, it is a vasodilator and has some smooth muscle cell inhibitory effects in vitro. Cilostazol has significant effects improving walking distance in multiple clinical trials. However, these effects are only significant in approximately 40-50% of the patients, so one must realize that not everyone will be helped with medications. Therefore, drug therapy to increase walking distance is helpful but not overwhelmingly. There are other drugs that are presently undergoing clinical testing. Propionyl-l-carnitine is a drug that is probably going to be effective for the treatment of peripheral vascular disease. It increases mitochondrial energy production in ischaemic cells. In addition to that, numerous other agents, including anti-platelet agents and prostaglandin are under study. One of the most intriguing classes of drugs under study is the angiogenic factors. These agents have the potential to actually generate new growth of blood vessels. There are limited published studies on the angiogenic factors.

Allergy Skin Testing
by
Adrian Wu

Allergic diseases are some of the most common chronic illnesses seen in medical practice. Common allergies include allergic rhinitis, allergic conjunctivitis, asthma, food allergies, drug allergies, skin allergies, insect sting allergies and occupational allergies. The most important factor to determine during initial consultation is the cause of the allergy, since successful treatment hinges upon removal of the source of allergen. Detailed analysis of allergic sensitivities is even more important if allergen immunotherapy is considered. A careful history and clinical examination can often give important clues, but diagnostic tests are very valuable in helping to establish a diagnosis. The type of testing to use is dependent on the immunological mechanism involved in the allergic reaction. The most common type of allergic reaction, called immediate type hypersensitivity reaction, is a result of allergen-IgE interaction on the surface of mast cells. Demonstration of allergen-specific IgE is therefore critical in diagnosing this type of allergy. The most sensitive and specific type of test for immediate hypersensitivity is the skin prick test. In this test, a drop of allergen extract is placed on the skin of the forearm or back, and the surface of the skin is lightly punctured by a lancet. The presence of allergen-specific IgE on cutaneous mast cells will result in degranulation and a weal and flare response. The size of the response can be measured with a ruler. This test has very high sensitivity and negative predictive value, but the specificity and the positive predictive value depend on the clinical situation. Intradermal injection of the extract (usually at $1/100^{th}$ of the initial concentration) will increase sensitivity at the expense of reduced specificity. Interpretation of the results requires knowledge of the types of allergens the patient is likely to be exposed to. For example, the types of pollens present in the air at specific times of the year in different geographic areas, the types of allergens present in different home and work environments, the pattern of exposure to food allergens in a cultural context etc. are important aspects to consider. The use of skin prick tests for the diagnosis of food allergies is even more confusing. While some food reactions are IgE mediated, others might be T cell mediated. Furthermore, patients with food-specific IgE and positive skin tests might not necessarily react when exposed, and other external factors such as concomitant medications or exercise might lower the threshold of reactivity. In these situations, skin testing should be followed by challenge testing to ascertain the significance of positive tests.

In conclusion, skin prick tests are technically simple and highly sensitive tests for allergen-specific IgE. However, misinterpretation can lead to over-diagnosis and unnecessary avoidance. A good understanding of the environmental or dietary history is important for correct interpretation. Environmental allergen analysis and challenge testing are underused but valuable adjunctive tests in the diagnosis of allergic diseases.

Ambulatory Peritoneal Dialysis
By
Frances Chiu

At the end of March, 2001, there are 5000 patients receiving replacement therapies in Hong Kong for end stage renal disease. 56% of these patients receives peritoneal dialysis as renal replacement therapies, primarily in the form of continuous ambulatory peritoneal dialysis (CAPD). Peritoneal dialysis is the preferred choice of treatment for its ambulatory nature, that the clients care for themselves at home and return to the renal centre for monitoring of progress, interventions, investigations and necessary education to enhance continuation of self care. Another important issue is patients do not require expensive hospital treatments like intermittent peritoneal dialysis or in-centre haemodialysis.

After the surgical implantation of a peritoneal catheter, sterile dialysis fluid can be introduced into the peritoneal cavity. Uraemic waste products and excessive fluid are removed by diffusion and osmosis. Principles for catheter care include strict asepsis in dialysis procedures; good exit site care, catheter immobilization to prevent unnecessary traction of trauma to the exit site; regular monitoring and early intervention in case of complications.

Education is an important arena of nursing care for peritoneal dialysis patients. Areas to be covered includes principles of aseptic technique, environment and hygiene and skill practice of bag exchange techniques, monitoring of vital signs, exit site care, drugs, diet and fluid regime, handling of minor complications that might occur at home related to the illness or the procedure. Clients will be visited at home after training to assess home conditions and the independent performance of peritoneal dialysis and exit site care at the home setting.

Regular monitoring is achieved through the logistics of client's daily care of the catheter and prompt report of abnormalities. Secondly, through regular clinic visits in 6 week's interval, alternating between nurse and doctor's follow up. During nurse follow up, blood samples and necessary investigations are conducted according to unit's protocol. Clients are assessed for overall condition, fluid and nutritional status, adherence of dialysis and drug regime, laboratory findings and problems encountered. Reinforcement and encouragement are often delivered at the same instance to enhance client's morale. Necessary interventions, education and prompt referrals to relevant health care team members are made if indicated.

Cardiac Catheterisation
by
Lee Pui Yin

Cardiac catheterisation is a combined haemodynamic and angiographic procedure undertaken for diagnostic and therapeutic purposes. It is usually performed to confirm the presence of a clinically suspected cardiac condition, define its anatomic and physiologic severity, and exclude associated conditions when a therapeutic procedure is planned.

Catheterisation of the right and left sides of the heart can be accomplished by the introduction of different catheters via several approaches. During right heart catheterisation, pressures and oxygenation saturations could be measured in the vena cavae, right atrium, right ventricle, pulmonary artery and pulmonary capillary wedge position. The severity of tricuspid and pulmonary lesions, pulmonary hypertension could be evaluated. Intra-cardiac shunt, pulmonary vascular resistance and cardiac output could also be quantified.

With left heart catheterisation, we could assess mitral and aortic valvular function, left ventricular pressures and function, systemic vascular resistance, and coronary anatomy.

Coronary angiography is the most frequently performed cardiac catheterisation procedure, which selectively examines the coronary circulation using contrast radiographic imaging techniques. Despite its limitations, it remains the reference standard for evaluation of patients with coronary artery disease.

Performing cardiac catheterisation in cardiac patients appropriately will greatly enhance the clinical management.

Care and Maintenance of Hickman's Catheter
by
Josepha Tai

Vascular access is both a physical and psychological problem for many individuals as many has described the number of venepunctures endured as the most memorable and unpleasant part of their hospital stay. In order to improve the quality of life of those who required prolonged IV access, central venous catheters such as Hickman's catheters are commonly used. It is situated in the great veins or right atrium, and placed either via the cephalic, or the subclavian, the external / internal jugular veins. Through the catheter lumen(s), blood samples could be withdrawn or blood products, antibiotics, chemotherapy and parenteral nutrition could be administered. However complications such as infection, blockage or breakage do affect the duration of their use.

To prevent the hazards associated with the use of Hickman's catheters, health care professionals should have knowledge of the catheters as related to its contents, insertion procedures and possible complications. In addition, strict catheter maintenance according to standardised procedures (dressing, flushing, blockage clearance, repair) should be adhered to ensure smooth functioning of the catheter. Furthermore good hand-washing techniques should be practised, as this is an important aspect of infection control but one which is frequently overlooked. Moreover, patient / family education on the care of the catheter should not be neglected as they should be empowered to take care of the catheter after discharge.

Despite of their complications, Hickman's-type catheters are still the best option for those with intensive intravenous support. Thus, if stemming from a well-informed and well co-operated team adhering to the strict local guidelines or protocols, certainly success in the care and maintenance of the central venous catheters could be achieved.

Echocardiography
by
Lam Yui Ming

Echocardiography (echo) is the use of ultrasound to examine the heart. It is a powerful and safe technique, which has become widely available for cardiovascular investigation. By using two-dimensional (2D), M-mode, Doppler and colour-flow imaging, echocardiography provides a comprehensive evaluation of the cardiovascular system. The structure, functional status and haemodynamic of the heart can be evaluated in one setting. Assessment of ventricular function and valvular status are the two most common indications for echocardiography. Both the size and systolic function of the left and right ventricle can be evaluated. Regional wall motion abnormalities may suggest the presence of coronary artery disease. Echocardiography can be used to make the diagnosis in patients with valvular heart disease. Information about the aetiology and severity of the lesion can be used to guide the therapy and determine the time for surgical intervention. Other indications for echocardiography include cardiomyopathy, disease of aorta, pericardial disease and congenital heart disease. Echocardiography can also be used to look for source of emboli in patients with arterial embolic event. Patients presented with arrhythmia and syncope may need to have an echo examination to look for structural heart lesion.

Clinical Neurophysiology
by
Windsor Mak

Clinical Neurophysiology or Electrodiagnostic (EDx) Medicine is the objective assessment of neuronal functions by recording action potentials of excitable tissue in the central and peripheral nervous systems and the muscles. Over 3,000 EDx procedures are performed every year at Queen Mary Hospital, consisting mainly of electroencephalogram (EEG), evoked potential (EP) studies, and electromyography (EMG). Most routine EDx tests are non-invasive. EP studies include somatosensory EP to assess dorsal column conduction, visual and brainstem auditory EPs for the visual and auditory pathways, and motor EP for pyramidal functions. The term EMG is often used to include the entire spectrum of peripheral EDx studies; the two main components are nerve conduction studies for large fibre functions, and needle electromyographic examination for bioelectric activity of muscles. Other less commonly performed EMG procedures include late responses for proximal parts of peripheral nerves, cranial nerve reflexes, repetitive nerve stimulation and single-fibre EMG for neuromuscular junction conduction, thermal threshold for small-fibre functions, and sympathetic skin response for autonomic functions. These tests detect physiological malfunctions produced by an underlying lesion rather than directly examine its structural pathology. Abnormal electrophysiological findings are, therefore, not pathognomic of specific diseases. The clinical neurophysiologist has to define and recognize the pattern of involvement to deduce a likely diagnosis or its differentials.

The techniques of most EDx procedures are relatively straightforward. However, appropriate selection and application of tests and the approach to interpretation of findings are the important considerations in EDx consultation. It is essential to integrate clinical and electrophysiological data into a meaningful conclusion that is relevant to the patient s problem. Therefore, EDx examinations should be planned and specifically designed for individual cases. The clinical neurophysiologist should have a good knowledge of neurological and neuromuscular diseases as well as the skills of logistic reasoning and progressive problem solving. He or she must also understand the limitations of EDx procedures and be aware of the possible consequences while formulating a recommendation.

Plasmapheresis
by
Albert Lie

Plasmapheresis is a form of aphaeresis procedure involving the separation of plasma from the cellular components of blood, followed by its collection and removal. The plasma can be collected for donation purpose or, more often, for removal of abnormal or harmful substances in the plasma. It can be performed either by centrifugation using a cell separator machine or by haemofiltration (membrane plasma separation) using a dialysis machine. With centrifugation, whole blood constituents are layered into plasma, platelets, lymphocytes, granulocytes and red cells as according to their increasing specific gravity, and the desired component can then be collected and removed. There are different designs of the centrifugation systems including 1) the spinning bowl, 2) the rotary belt and 3) the two packs systems. With haemofiltration, plasma is separated from blood cells through a highly permeable membrane filter usually of pore size 0.6 μm or less.

Therapeutic plasmapheresis has an accepted role in the management of the following conditions:

- Haematological diseases
  - Hyperviscosity syndrome (multiple myeloma, Waldenstrom s macroglobinemia)
  - Cryoglobulinemia
  - Thrombotic thrombocytopenic purpura
  - Post-transfusion purpura
- Neurological diseases
  - Guillain-Barre syndrome
  - Chronic inflammatory demyelinating polyneuropathy
  - Myasthenia gravis
  - Lambert-Eaton myasthenic syndrome
  - Refsum s disease
- Renal disease
  - Goodpasture s syndrome
- Metabolic disease
  - Familial hypercholesterolaemia

There is also a list of other conditions where the role of plasmapheresis is less clear.

Plasmapheresis is associated with possible problems such as difficult venous access, citrate toxicity, thrombocytopenia, plasma protein reduction, electrolyte & acid-base disturbance, hypotension, hypersensitivity reaction and transmission of infection. Nevertheless, the procedure in general is very safe with an extremely low risk of serious complication.

Sleep Study
by
Mary Ip & Lam Bing

Sleep study, also known as polysomnography (PSG), a multi-channel recording of sleep and breathing, has been widely used in the sleep laboratory to diagnose obstructive sleep apnoea (OSA) and other causes of sleep disruption, such as periodic limb movements (PLMs). Sleep study is usually done at night. At least eight electrodes are needed to determine sleep stages including two central scalp electrodes to record electroencephalogram (EEG), two to record eye movements (elecrooculogram, EOG), two to record a mentalis electromyogram (EMG), and two reference electrodes behind the ears. For assessment of sleep disorders, other sensors are attached to determine airflow, respiratory effort, oximeter to determine oxygen saturation, microphone to detect snoring, body position sensor, electrocardiogram (ECG), surface EMG over the right and left anterior tibialis muscles to detect limb movement. All these parameters are send to a computer and stored. The PSG will be scored and interpreted by experts in sleep disorders in order to identify the problem that causes sleep disruption.

Acute Renal Insufficiency
by
Li Fu Keung

Acute renal failure (ARF) is an important medical emergency that is associated with a high mortality. The diagnosis of renal failure is usually made on an elevated plasma creatinine, electrolyte imbalance or severe metabolic acidosis. The progression of the problem can best be told by comparing the current plasma creatinine concentration or urinalysis with previous results. Other indices include the haemoglobin and serum phosphate level. The causes of renal diseases are traditionally classified by that portion of the renal anatomy most affected by the disorder. Identifying pre-renal and post-renal (obstructive) diseases is particularly important because they are often readily reversible. There are numerous idiopathic and secondary disorders that produce glomerular diseases resulting in renal failure. Acute glomerulonephritis with inflammation on histologic examination produces an active urinary sediment with red cells, white cells, granular and often cellular casts. The renal tubular and interstitial diseases can also produce acute renal failure and the commonest acute tubulointerstitial disorders are acute tubular necrosis, which is often drug-induced. Radiologic studies are used singly or in combination for the detection, diagnosis, and/or the evaluation of renal failure. A renal biopsy is most commonly obtained when non-invasive evaluation has been unable to establish the correct diagnosis. Attention to the fluid, electrolyte and acid-base balance is the mainstay of therapy. Dialysis may be required in patients with severe acute renal failure. Most patients are treated by haemodialysis with the dialysis prescription tailored to the clinical requirement. An alternative approach is the use of continuous renal replacement therapy which is often associated with less haemodynamic instability.

Aspiration Pneumonia
by
Kenneth Tsang & Wayne Hu

Aspiration pneumonia is defined as acute lung injury manifesting as a result of inhalation of regurgitated gastroesophageal or oropharyngeal contents. Aspiration of colonised secretions from the oropharynx is the primary mechanism by which bacteria gain entry to the airways and then lungs. Pulmonary aspiration is an important cause of serious illness and death among residents of nursing homes as well as hospitalised patients, although it is commonly misdiagnosed and poorly treated. Studies indicate that 5-15% of community-acquired pneumonia is due to aspiration. Patients, often elderly, with neurological dysphagia, disruption of the gastroesophageal junction or anatomical abnormalities of the upper aerodigestive tract (e.g. post radiotherapy), are at high risk for developing aspiration. Around 40-70% of stroke patients have increased incidence of swallowing dysfunction and thus are prone to aspiration, particularly silent aspiration. Assessment of the risk of aspiration includes evaluation of cough and gag reflex as preliminary tests, but more formal testing such as videofluoroscopic swallowing study (VFSS) or fiberoptic endoscopic swallowing study with sensory testing (FESST) are essential. Proper assessment will allow institution of appropriate interventions such as selection of the correct food consistency and type, speech therapy training, or even insertion of feeding tubes or percutaneous endoscopic gastrostomy. Reassessment is also essential, as patients could improve after initial illness such as stroke, and could ingest food safely later. Antibiotic treatment should aim to target the bacterial pathogens, including *Streptococcus pneumoniae*, *Staphylococcus auerus*, *Haemophilus influenzae*, and Enterobacteriae in community patients, and predominantly Gram-negative bacilli, such as *Pseudomonas aeruginosa*, among hospital-acquired patients. Other treatment also includes administration of respiratory care, physiotherapy, and measures to control the original illness.

Anaphylaxis
by
Adrian Wu

Anaphylaxis is one of the most urgent medical emergencies that can result in death in a matter of minutes. Anaphylaxis is caused by coordinated degranulation of mast cells resulting in a sudden release of vasoactive mediators such as histamine, serotonin and leukotrienes. As mast cells are commonly found in the skin and the submucosal surfaces of the respiratory and GI tracts, initial symptoms of anaphylaxis commonly present in these organ systems. When the released mediators enter the systemic circulation, systemic symptoms ensue. The pathophysiology of anaphylaxis is the result of vasodilatation and increased vascular permeability. Clinical symptoms include generalized urticaria, angioedema, bronchospasm and hypotension. Common causes of death include asphyxiation due to upper or lower airway constriction, cardiovascular collapse and cerebral or myocardial infarction.

Mast cell degranulation might be the result of IgE-mediated or non-IgE mediated mechanisms. Strictly speaking, the term anaphylaxis should be reserved for IgE-mediated mechanisms, while the term anaphylactoid is used for non-IgE mediated reactions. However, anaphylaxis is often used to cover all clinical situations, since it is sometimes difficult to ascertain the mechanism of the reaction. IgE-mediated causes of anaphylaxis include food, drugs, insect sting and even aeroallergens such as latex and dust mites. Non-IgE mediated causes include osmotic insult (radiocontrast), direct mast cell degranulation (opiates, muscle relaxants, vancomycin, dextran), immune complex reactions (serum sickness) and physical stimuli (heat, cold, exercise). In a small proportion of cases, the cause remains unknown, but might be autoimmune in nature. Drugs such as beta-blockers can lower the threshold for anaphylaxis.

Initial treatment of anaphylaxis aims at correcting capillary leak, bronchospasm and hypotension. The drug of choice is epinephrine (adrenaline). The dose is 0.3mg IM for adults and 0.15mg for children. Studies have shown that survival is inversely proportional to the length of time between initial symptoms and the administration of epinephrine. Systemic steroid is useful in preventing the late phase reaction that might occur in a proportion of the cases, but plays no role in the treatment of the immediate reaction. Anti-histamines might be useful in controlling the cutaneous symptoms of anaphylaxis, but do not affect survival. The usual procedures of maintaining a patent airway and fluid resuscitation also apply.

In patients with recurrent anaphylaxis, the cause should be elucidated. A careful history, food diary and skin prick tests are usually helpful. Such patients must carry self-injectible epinephrine at all times. Patients with recurrent idiopathic anaphylaxis might respond to anti-histamine prophylaxis, but systemic steroid might be needed to prevent attacks.

Diabetic Emergencies
by
Nelson Wat

Blood glucose is kept within a narrow range at all times in normal individuals. Abnormal elevations of blood glucose result in a disease condition known as diabetes mellitus. Diabetes mellitus is classified into type 1 and type 2 according to pathogenesis. Diabetic emergencies occur with extremes of blood glucose level fluctuations, clinically manifested as hypoglycaemia, diabetic ketoacidosis (DKA) and non-ketotic hyperosmolar coma. Recognition of the presence of these conditions is the first step leading to the prevention of serious life-threatening consequences. Early consumption of meals, immediate intake of sweet drinks for conscious patients, while intravenous glucose infusion or intramuscular glucagon injection by relatives for patients who are unconscious, are emergency measures to reverse hypoglycaemia. Presence of ketones in blood / urine samples together with systemic metabolic acidosis establish the diagnosis of DKA. This typically occurs among patients with newly presenting or known diagnosis of type 1 diabetes mellitus. Emergency management includes 1) correction of acid-base and electrolyte disturbances, 2) administration of insulin to lower blood glucose and 3) fluid resuscitation. Sodium bicarbonate infusion to correct metabolic acidosis should only be considered if blood pH falls below 7.0. Pseudohyponatraemia may mask the degree of dehydration gauged by elevation of serum sodium levels. Slow infusion (not in bolus) of insulin through the intravenous route is preferred to either intramuscular or subcutaneous routes. Sliding scale for estimating the amount of insulin required is resorted to when the condition of patients remain unstable.

Emergency Rheumatic Disorders
by
Lau Chak Sing

Although most rheumatological disorders are chronic and progressive, it is not uncommon that patients present to their family physicians with acute complaints that require urgent investigations and treatment.

Acute monoarthritis is probably one of the commonest rheumatological emergencies. Differential diagnoses include trauma related injuries, crystal induced arthropathies and sepsis. It is prudent that infection is excluded in anyone who presents with an acute monoarthritis. Predisposing factors for septic arthritis include diabetes, chronic renal impairment, intravenous drug abuse, underlying joint disease, previous artificial joint replacement, immunosuppressive drug use, adjacent soft tissue infection and recent invasive manipulation of the affected joint. Joint aspiration for total and differential white cell count and microbiological examination is essential before instituting anti-microbial treatment.

Few things are as painful as a severe attack of gout; it often develops overnight and reaches a peak within hours so that, with an affected foot, it is almost impossible to bear weight or even the touch of bedclothes. Acute gouty arthritis should be treated with non-steroidal anti-inflammatory drugs (NSAIDs) which are much more effective than any analgesic. When NSAIDs are contraindicated, colchicines may be useful but diarrhoea is a common side-effect. If all else fails, an intra-articular corticosteroid injection is often effective.

Patients with rheumatoid arthritis (RA) may also present with acute complications such as joint infection, tendon rupture and cervical spine subluxation. Atlantoaxial subluxation or, less commonly, subluxation at lower levels, with subsequent compression of the spinal cord is not uncommon in RA. The earliest and most common symptom is pain radiating up into the occiput. Other symptoms include paraesthesia, sudden deterioration in hand function, sensory loss, abnormal gait, and urinary retention or incontinence. An urgent referral to orthopaedic surgeons for consideration for stabilisation surgery is needed.

Systemic lupus erythematosus (SLE) is a common systemic autoimmune disorder seen in Hong Kong. Certain disease manifestations of SLE may present acutely requiring prompt treatment. Major organ complications such as severe thrombocytopenia, lupus cerebritis, acute thrombosis and the so-called catastrophic anti-phospholipid antibody syndrome are not uncommon. Acute vasculitic digital ischaemia warrants the use of potent vasodilators such as prostacyclin or its analogues and systemic corticosteroids. Probably the most important lupus emergency is sepsis, which is often the result of the patients intrinsic immunocompromised state and / or the use of immunosuppressive agents. Infections should always be suspected in SLE patients who present with a febrile illness.

Acknowledgements

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Last but not least, we would like to extend our thanks to all the speakers, chairmen and participants for their invaluable participation and contribution. Thank you.

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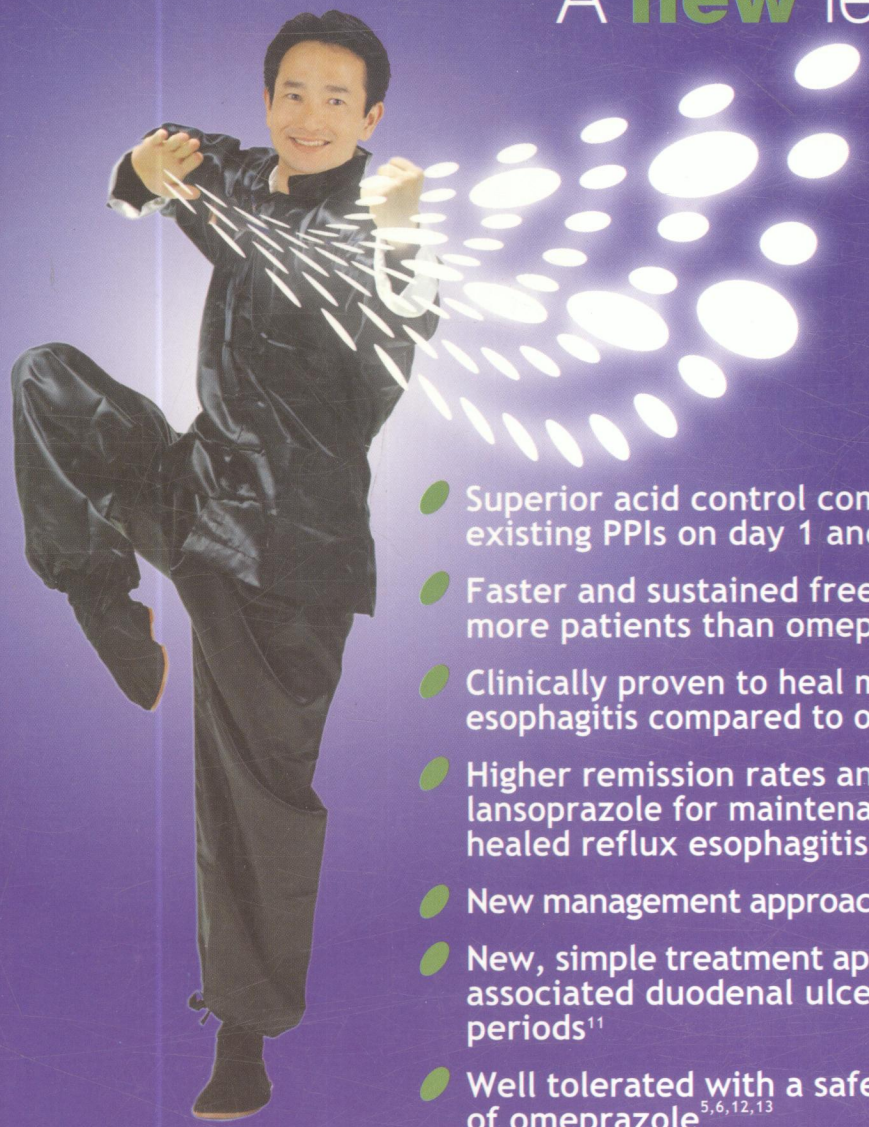

- Superior acid control compared to existing PPIs on day 1 and day 5[1,2,3,4]
- Faster and sustained freedom from GERD symptoms in more patients than omeprazole[5,6] and lansoprazole[7]
- Clinically proven to heal more patients with reflux esophagitis compared to omeprazole[5,6] and lansoprazole[7]
- Higher remission rates and better symptom relief than lansoprazole for maintenance therapy in patients with healed reflux esophagitis[8]
- New management approaches with on-demand therapy[9,10]
- New, simple treatment approach for healing of *H.pylori*-associated duodenal ulcer allowing shorter treatment periods[11]
- Well tolerated with a safety profile comparable to that of omeprazole[5,6,12,13]

ABBREVIATED PRESCRIBING INFORMATION: NEXIUM® (Esomeprazole magnesium). See local prescribing information for full details. **PHARMACODYNAMIC PROPERTIES:** NEXIUM® reduces gastric acid secretion through a highly targeted mechanism of action by being a specific inhibitor of the acid pump in the parietal cell. **INDICATIONS AND DOSAGE: Treatment of erosive reflux esophagitis:** NEXIUM® 40 mg once daily for 4-8 weeks. **Long-term management of patients with healed esophagitis to prevent relapse:** NEXIUM® 20 mg once daily. **Symptomatic treatment of gastro-esophageal reflux disease:** NEXIUM® 20 mg once daily in patients without esophagitis. Once symptoms have resolved, an on-demand regimen of 20 mg once daily can be used when needed, to control subsequent symptoms. ***Helicobacter pylori*-associated peptic ulcer disease:** -healing of *Helicobacter pylori*-associated duodenal ulcer, -prevention of relapse of peptic ulcers in patients with *Helicobacter pylori*-associated ulcer disease. NEXIUM® 20 mg, amoxicillin 1 g and clarithromycin 500 mg, all bid for one week. **CONTRAINDICATIONS:** Known hypersensitivity to esomeprazole, substituted benzimidazoles or any other constituents of the formulation. **PRECAUTIONS:** In the presence of any alarm symptoms (eg significant unintentional weight loss, recurrent vomiting, dysphagia, haematemesis or melena) and when gastric ulcer is suspected or present, the possibility of gastric malignancy should be excluded before treatment is initiated. Patients on long-term treatment should be kept under regular surveillance. **USE IN PREGNANCY AND LACTATION:** Caution should be exercised when prescribing NEXIUM® to pregnant women. NEXIUM® should not be used during breast-feeding. **INTERACTIONS:** Due to the decreased intragastric acidity, the absorption of ketoconazole and itraconazole can decrease during esomeprazole treatment. When NEXIUM® is combined with diazepam, citalopram, imipramine, clomipramine and phenytoin the plasma concentrations of these drugs may be increased and a dose reduction could be needed. Concomitant administration of esomeprazole resulted in a 45% decrease in clearance of diazepam. Concomitant administration of esomeprazole resulted in a 13% increase in trough plasma levels of phenytoin in epileptic patients. The plasma concentrations of phenytoin should be monitored when treatment with esomeprazole is introduced or withdrawn. In healthy volunteers, combined therapy with esomeprazole and cisapride resulted in a 32% increase in AUC and a 31% prolongation of elimination half-life but no significant increase in peak plasma levels of cisapride. **ADVERSE REACTIONS:** In clinical trials with NEXIUM® the following adverse events were reported or suspected to occur in 1% or more of patients: nausea/vomiting, diarrhoea, constipation, abdominal pain, flatulence and headache. In rare cases the following were reported or suspected: dermatitis, pruritus, urticaria, dizziness and dry mouth. **PRESENTATIONS:** NEXIUM® tablets containing esomeprazole magnesium corresponding to 20 mg or 40 mg esomeprazole. Effective date : 5 June 2000. **REFERENCES:** 1. Röhss K, et al. Gut 2000; **47** (Suppl 3): A63. 2. Thomson ABR, et al. Gut 2000; **47** (Suppl 3): A63. 3. Wilder-Smith C, et al. Gastroenterology 2000; **118**: A22. 4. Röhss K, et al. Gut 2001; **49** (Suppl 3): Abs 1956. 5. Richter JE, et al. Am J Gastroenterol 2001; **96**: 656-665. 6. Kahrilas P, et al. Aliment Pharmacol Ther 2000; **14**: 1249-1258. 7. Castell D, et al. Gut 2001; **49**(Suppl 3): Abs 3363. 8. Lauritsen K, et al. J Gastroenterol Hepatol 2002; **17**(Suppl): A1007. 9. Talley NJ, et al. Aliment Pharmacol Ther 2001; **15**: 347-354. 10. Talley NJ, et al. Gastroenterology 2000; **118**: A658. 11. Tulassay Z, et al. Eur J Gastroenterol Hepatol 2001; **13**: 1457-1465. 12. Maton PN, et al . Drug Saf 2001; **24**(8): 625-635. 13. Genta RM, et al. Gastroenterology 2000; **118**: A16.

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