FEBRUARY 2015

HONG KONG COLLEGE OF PHYSICIANS

SYNAPSE

RESTRICTED TO MEMBERS ONLY

Photographer

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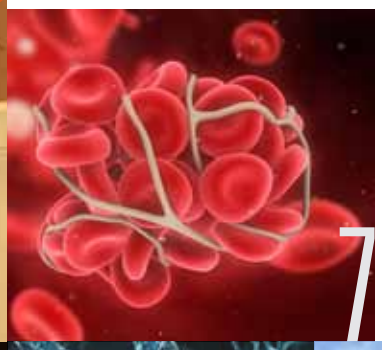
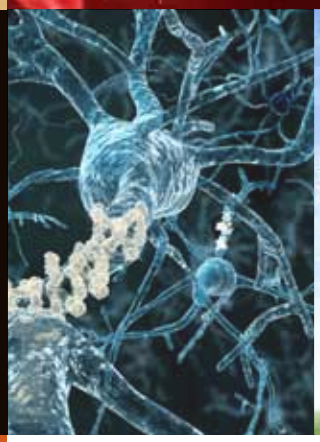




THIS ISSUE

THE PRESIDENT'S ANNUAL REPORT 2014

Patrick CK LI
PRESIDENT, HKCP

The Hong Kong College of Physicians has continued to undertake various initiatives over the past year to uphold the high standard for physician practice in Hong Kong. This has been achieved largely through the effort of our various Standing Committees, our Specialty and Basic Physician Boards, as well as many of our Fellows who have continued to support our training programmes in different capacities.

There is recently heightened public concern over the safety of patients undergoing invasive medical procedures as well as the credentials of the operators. Our College will need to consolidate the framework for delivering the relevant training as well as credentialing of specialists who have attained competence in performing such procedures. The Education and Accreditation Committee and individual Specialty Boards, in particular Cardiology, Respiratory Medicine, and Gastroenterology and Hepatology, are in the process of reviewing the framework for training in high-risk invasive procedures both at trainee and post-Fellowship level. The objectives are to provide assurance of the competence of the specialists who

had completed the required training while ensuring fair training opportunity to the staff of different regional hospitals so that service provision would not be adversely affected. The Specialty Boards are also considering the feasibility of incorporating both skill- and scenario based simulation to enhance their training programme. Issues to consider include availability and accessibility of the simulation modules, the need for a critical pool of trainers who have acquired experience in delivering simulation training, as well as protected time for the trainers and trainees to implement the programme. A Task Force on Simulation Training was set up to facilitate and coordinate its further development within the College.

Within the past year, our College has supported the Department of Health on a number of its initiatives. Written comments were submitted in response to its Consultation on the Draft recommendation of the Working Group on defining high-risk medical procedures/ practices performed in ambulatory setting. The Chairman of our Professional and General Affairs had participated in its advisory group on health effects of use of internet and screen media devices. The Chairman of the Specialty Board in Gastroenterology and Hepatology had joined its task force to oversee the planning, implementation and evaluation of the pilot colorectal cancer screening programme. The Chairman of the Specialty Board in Medical Oncology has joined its Cancer Expert Working Group on Cancer Prevention and Screening. The Specialty Board in Endocrinology, Diabetes and Metabolism has nominated a representative to participate in its preparatory meeting for prevention of iodine deficiency disorders. The Chairman and Secretary of the Specialty Board in Infectious Disease had participated in the publicity events of the Centre for Health Protection in support of World Health Day 2014, with focus on vector-borne diseases, and for promulgation of seasonal influenza vaccination to target groups for the 2014/15 season. Our College has also assisted the Hong Kong Medical Council in updating the lists of nomenclature of procedures and operations on its website.

Our College has also written to the Secretary for Food and Health to reflect our views to the Steering Committee for Review of the Hospital Authority. In particular, we stressed that the Hospital Authority should undertake a comprehensive review of the basis for medical and nursing manpower planning and allocation to all clinical departments, taking into consideration the effect of an ageing population, higher dependency of patients from chronic disabling medical diseases, increase in communicable and non-communicable diseases as well as rising public expectation and heightened requirements for patient safety. We also suggested that the Hospital Authority should review its framework for bed provision and ancillary services within individual hospitals and clusters to cater for the escalating demand on its emergency services, in particular the inevitable and ever-growing seasonal surges. There should also be a mechanism for effective mobilisation of manpower and beds across departments, hospitals and clusters to cater for surges in demand for emergency services. We pointed out that the Hospital Authority should monitor the workload of doctors to ensure that their training and practice are not compromised by excessive clinical responsibilities.

Our College is pleased to note that the projects of some of the recipients of our Young Investigator Research Grant had been published in high-ranking peer-reviewed journals. We hope that more of our younger Fellows can make use of the Grant to launch their career in research.

I wish to extend my sincere appreciation to the Chairpersons and Members of the Committees and Boards for their contributions towards upholding the standard of physician training and supporting the various responsibilities and functions of the College. I would also like to thank the retiring Council Members for their many important contributions to the College over the years in various capacities. I am also very grateful to our Honorary Treasurer for monitoring and maintaining the healthy financial position of the College. I would also like to thank our Council Members, in particular our Immediate Past President and Senior Advisor for their unfailing support and invaluable advice over the past year. Finally, I wish to thank our Honorary Secretary and the hardworking and dedicated secretarial staff in maintaining smooth operation of the College.

THE PRESIDENT'S ADDRESS TO NEW FELLOWS AT THE HONG KONG COLLEGE OF PHYSICIANS CONFERMENT CEREMONY 2014

Patrick CK LI
PRESIDENT, HKCP

Prof. William Wei, Honorary Treasurer of the Hong Kong Academy of Medicine, President of Sister Colleges and their representatives, Honoured guests, Fellows and Members of the College, ladies and gentlemen.

Today is a very special occasion for the newly admitted Fellows and Members. On behalf of the College, I extend to you my congratulations on your well-deserved achievement, which is the result of your dedication and hard work over the past years. You should all appreciate the support from your colleagues, your family and loved ones, without which your success today would not be realised. I am confident that you will all continue to strive for excellence and professionalism in your future career.

One of the important statutory functions of the College is to oversee the standard of internal medicine training in Hong Kong. We should recognise that the standard of physician training is determined by the collective effort of each and every one of our Fellows. The newly admitted Fellows will also make their contributions as they become eligible for appointment as trainers in the respective specialties after completion of their training in advanced internal medicine and an additional two years of work experience in the specialties. Given the vital importance of training in grooming the next generation of physicians and shaping the future of medical care in Hong Kong, I think it would be opportune for us all to reflect on the model for physician training.

Most of us would realise that physician training is much more than didactic teaching of knowledge and demonstration of skills. Medical knowledge is advancing at such a rapid pace that it is imperative for us to undertake continuous medical education to remain current with the accepted standard of practice. Just as some of the contents of textbooks may become out-dated by the time they are published, likewise what we teach our younger colleagues may no longer valid in a few years time. The model of medical education has also changed considerably. Clinicians increasingly make use of web-based tools to directly access original published work as well as reviews articles and practice guidelines. It would be more important for the trainees to acquire the skills to search for and critically review the available information for application in their clinical practice. Nowadays, we also have simulation models to allow trainees to practice interventional skills and management of difficult clinical scenarios. So will the computer take over from us as trainers of the future generations of physicians?

"clinicians..important role to play in training the future generations of physician.. teaching of general principles of good medical practice, serving as role models, and providing mentorship to the trainees"

It is my firm belief that we as clinicians still have an important role to play in training the future generations of physicians. I would consider them under the following three aspects: teaching of general principles of good medical practice, serving as role models, and providing mentorship to the trainees.

Instead of teaching the details of medical knowledge, we should be helping our trainees to develop the capacity for critical and logical thinking so that they can identify the relevant and current resources to assist them in handling difficult clinical scenarios. They should be encouraged to strive for life-long learning and continuous self-improvement. Good communication skills, ethical principles and professionalism should be emphasised. They should also be encouraged to actively engage in discovery of new knowledge, either through scientific or clinical research or quality improvement initiatives to improve patient care.

With the heavy workload facing both the trainees and trainers, any "protected" time for formal training could be compromised. We as clinicians should make full use of any available "contact" time to provide good quality interaction for the benefit of the trainees. This may be through bedside teaching and discussion of problems that they have encountered. An equally important and yet sometimes not realised dimension is our influence to the trainees as role models, either positively or negatively. It will be important for us to personally demonstrate our commitment to life-long learning, strive for excellence, ethical principles and professionalism in our daily practice.

While it may not be so difficult for us to become good trainers, the real challenge lies in providing mentorship to our trainees. This would involve recognising

"personally demonstrate our commitment to life-long learning, strive for excellence, ethical principles and professionalism in our daily practice."

their potential, nurturing and inspiring them to fully realise them, and advocating for their training opportunities and career advancement. Recognising their up-bringing in different social and political climates, we should not impose on them our own value system. We should understand their limitations, frustrations and aspirations and offer them guidance in finding the solution to overcome the hurdles in their career and life.

This year is a special occasion marking the 40th year since the retirement of the late Professor AJS McFadzean from the University of Hong Kong. Professor McFadzean is a legendary figure for internal medicine in Hong Kong. He had laid the foundation for our undergraduate education, specialisation of clinical services as well as medical research. Above all this he was also a great mentor, inspiring the career a whole generation of eminent physicians. I would like to end by sharing with you some of the philosophies of Professor McFadzean. In his views, teaching, research and clinical service must go hand in hand in clinical departments, and in the pursuit of excellence all three aspects should be given adequate attention. As a teacher he always stressed principles rather than details and encouraged clear and logical thinking rather than recall.

For the newly admitted Fellows, I hope that you will be inspired by the example set by Professor McFadzean to join all of us in providing good training and mentorship to your younger colleagues. Only then can the standard of physician training be upheld and our clinical services continue to improve.

"recognising their potential, nurturing and inspiring them to fully realise them, and advocating for their training opportunities and career advancement"

AJS McFadzean Oration 2014

FROM BLOOD CLOTS TO THROMBOSIS AND BEYOND - *A Remarkable Growth of Knowledge in Six Decades*

Hau Cheong KWAAN

Marjorie C. Barnett Professor of Hematology-Oncology

Professor of Medicine

Northwestern University Feinberg School of Medicine, Chicago, IL, U.S.A.

Professor Todd, Dr. Li, Ladies and Gentlemen:

I am deeply honored to be the 19th AJS McFadzean Orator. This honor is especially meaningful to me as this is the occasion of the 40th anniversary of Professor McFadzean's retirement. I am eternally indebted to him for being my mentor and the person who sixty years ago kindled my passion for this fascinating subject of thrombosis. Yes, there are many scientific advances made during these six decades, but, few can match that of the remarkable growth of knowledge in thrombosis from the elucidation of the mechanism of clot formation to that of thrombosis and beyond.

As early as in the days of Hippocrates and Galen, it was known that if blood is taken out of the body, it clots in a matter of minutes. By the 17th century, the Italian anatomist Morgagni found out that clots can also form inside the body. According to Prof. Robb-Smith of Oxford, Galen coined the term "thrombosis", derived from the Greek word "thrombos" meaning a clot. Today, we understand that the term "clot" means fibrin formed after its conversion from fibrinogen, and is cell free; whereas a "thrombus" is a clot formed with a blood vessel, be it in arteries, veins or the microvasculature, and it has cellular components.

The modern concept of thrombosis is credited to Rudolph Virchow, who ascribed the pathogenesis of thrombosis to three abnormalities – that of the blood flow, blood vessels and contents of the blood – often referred to as the Virchow's triad[1] (Figure 1). Today, with advances in biochemistry, biophysics, molecular biology and genetics, many more factors have been added. An example for the thrombogenic factors just in cancer alone is shown here[2] (Figure 2). Note that Virchow's' triad remains the very basic principle of thrombosis.

Today, thrombosis is a world-wide health problem. It is the major cause of death in three leading cardiovascular diseases: ischemic heart disease, stroke and venous thromboembolism. Five days ago, the International Society of Thrombosis and Hemostasis designated October 13 as the first World Thrombosis Day, to raise the much needed awareness of this disorder. October 13 was the day that Virchow was born 193 years ago, in 1821.

Let us look at the magnitude of this health problem (Figure 3). In the United States alone, there were 250,000 new cases of deep vein thrombosis (DVT) a year with 900,000 recurrences accounting for approximately100,000 deaths[3]. Studies have shown that there is a lack of awareness in the public and even among the physicians. Ten years ago, Sam Goldhaber found that in 5400 DVT patients, over 70% did not receive prophylaxis[4]. Today, however, in most countries, there are well defined guidelines for thrombo-prophylaxis especially for patients with high risk such as cancer patients, and patients after surgery.

Rudolph Virchow
(1821-1902)

Pathogenesis of Thrombosis

1. *Aberrant Blood Flow*
2. *Abnormal Vascular Integrity*
3. *Abnormal Contents of Blood*

Figure 1.
Virchow's triad.

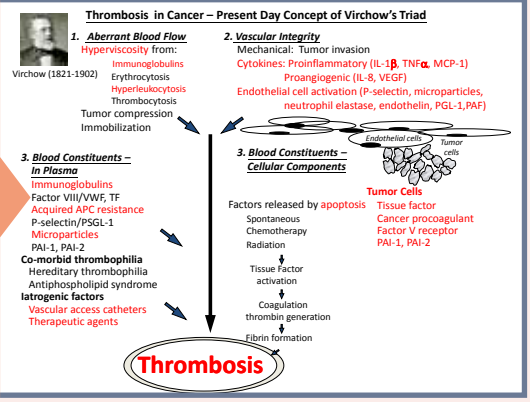


Figure 2.
Present Day Concept of Virchow's Triad applied to thrombosis in cancer.

VENOUS THROMBO-EMBOLISM (VTE)

In United States in 2008

New cases:	249,000 per yr	
Recurrent:	900,000 per yr	*Heit, 2008*
Deaths (estimated):	100,000 per yr	*Surg. Gen., 2008*

Low public awareness: 74% unaware of DVT
95% their physicians did not discuss this.
Amer.Public Health Assoc., 2009

Low physician awareness
5,451 DVT patients: 71% did not receive prophylaxis
Of these, 59% are medical patients
Goldhaber, 2004

Figure 3.
The incidence of venous thromboembolism in the United States.

GLOBAL BURDEN OF VTE

Raskob et al , 2014: Thrombosis – a major contributor to global disease burden ,

Incidence of VTE / 1,000 population / year (Level 1 data)

REGION	COUNTRY	VTE	SOURCE
WESTERN EUROPE	DENMARK	2.7	*HOLST - 2010*
	NORWAY	1.4	*NAESS - 2007*
NORTH AMERICA	CANADA	1.2	*TAGALAKIS-2013*
	UNITED STATES	2.4	*YUSUF-2012*
ASIA	TAIWAN	0.2	*LEE-2010*
	HONG KONG	0.2	*CHEUK-2004*
	HONG KONG	0.08	*WOO- 1988*

- The "lower"incidence among Asians is a myth!!!
  (*Leung et al.2005;Angchaisuksiri,2011*)
- Hereditary thrombophilia (Protein C/S, antithrombin in Asians)
- Post-op VTE same incidence as Western data (SMART, 2005, 2007)

Figure 4.
The global incidence of venous hromboembolism.

The "four-factor"theory of clotting (1953)

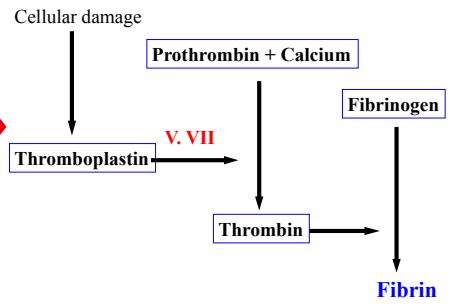


Figure 5.
The "four factor" theory of clotting (1953).

The Global burden of thrombosis is just as staggering (Figure 4)[5]. Take venous thromboembolism (VTE) in Western Europe, Denmark[6] and Norway[7]. The incidence was 1.4 to 2.7 per 1000 population per year. Likewise in North America, it was 1.3 to 2.4 per 1000 population per year[8,9]. In contrast in Asia, the data from Taiwan[10] and Hong Kong[11] shows a lower incidence of 0.16 to 0.20 per 1000 population per year. This widely held belief that thrombosis is not common among Asians can be deceiving and must be clarified and rectified. The misconception was recognized today, such as by Leung et al here in Hong Kong[12], and recently reviewed by Pantep AngChai-suksri[13]. They pointed out that among the factors for the lower incidence, there is a notable difference in the hereditary risk factors (thrombophilia) with Factor V Leiden and prothrombin mutation 20210 being very rare in Asians, but with a higher prevalence of deficiencies in Protein C/S and antithrombin. However, the role of the acquired thrombophilia, such as immobilization, infection, cancer and surgery remains the same as in Western countries. Studies such as SMART 2005[14] and SMART 2007[15] confirmed that postoperative thromboembolic complications are just as high as those reported in Western countries. Hence, the indications for thromboprophylaxis should remain the same for Asian patients.

Turning to clotting, back then 60 years ago in 1953, the concept of clot formation was a fairly simple "four factor" theory (Figure 5). In that year, factor V and VII were added. You might wonder why some clotting factors were designated by Roman numerals. It was necessary because, during World War II, there was a no scientific communication between Europe and North American. As a result, the same clotting factor was often given multiple names. After the war, an International Committee was formed to address this. By 1960, thirteen clotting factors were agreed on (Figure 6). The last one was Factor XIII designated for fibrin stabilizing factor, at a meeting held in Princeton, NJ (Figure 7). Though not a member of this august body, I was honored to be invited to present our data from Hong Kong. Among the attendees was Dr. Armand J. Quick, famous for the Quick Prothrombin Time test, widely used today. Also present was Dr. Oscar D. Ratnoff, who four years later published the article on "Coagulation waterfall". Why did he call this process a waterfall? This diagram, published independently and simultaneously by Prof. Macfarlane from Oxford shows why the clotting process looks like a water fall (Figure 8)[16]. After the start of the clotting when blood made contact with a foreign surface, one factor activates the next in a sequential fashion like a waterfall. Today, this process is commonly referred

"held belief that thrombosis is not common among Asians can be deceiving"

to as the coagulation cascade. As expected, our concept some 50 years later has blossomed (Figure 9). The start of clotting by contact is downplayed. Tissue factor is now the dominant initiating signal. And, with the interactions with clotting factors, thrombin is generated. Thrombin then further developed the clot to a thrombus. Thrombin activates platelets, endothelial cells and white blood cells (Figure 10).

Let's look at a thrombus. Here is a thrombus embolized to the pulmonary artery

(Figure 11). Histologic section demonstrates the presence of mixture of fibrin, red cells, white cells and platelets, often forming layers. The contents of a thrombus vary with the type of thrombus. I would like to share with you a collection of different kinds of thrombi.

In this case of warfarin necrosis, a thrombus is shown here occluding the vessel in the breast adipose tissue. This complication starts as skin necrosis, sometimes with massive loss of skin[17] (Figure 12).

This patient had severe disseminated intravascular coagulation from meningococcemia, with gangrene of the tip of nose and blue toes (Figure 13). She had multiple thrombi in her lungs and renal glomeruli.

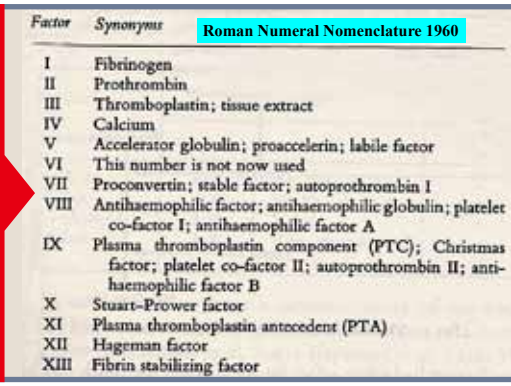
Platelets can be the main component of a thrombus, as in the case of heparin-induced thrombocytopenia[18] (Figure 14). The platelet clumps were confirmed by antiGp Ib immunohistochemical stain. The occluded vessels led to skin necrosis and in this case multiple infarction of the pancreas.

Another example of a platelet rich thrombus can be seen in thrombotic thrombocytopenic purpura, with typical thrombi shown in the myocardium, brain and renal glomeruli, resulting in damages in these affected organs[19] (Figure 15).

Red blood cells (rbc) can also be the main part of a thrombus. Here, aggregated rbc's in a patient with cold hemagglutinin disease with red cell thrombi occluding blood vessels leading to infarction of his ileum and gall bladder[20] (Figure 16). This next example is familiar to all of you – malaria with Plasmodium falciparum infected RBC's, shown in the peripheral blood (Figure 17). The aggregated rbc's occluded small vessels in the skin and muscle. The infected rbc's elaborate an adhesive molecule (P.falciparium erythrocyte membrane protein 1)[21]. This complication may be an important factor in cerebral malaria with brain vasculature occlusions.

Next, white cells can block vessels too. Here is an example in acute myeloid leukemia (FAB classification: M5) of leukemic monoblasts blocking blood vessels in the myocardium and brain (Figure 18).

In cancer patients, tumor cells are often found in thrombi (Figure 19). Most tumor cells express tissue factor, as shown here and are thus highly thrombogenic[22].



Factor	Synonyms	Roman Numeral Nomenclature 1960
I	Fibrinogen	
II	Prothrombin	
III	Thromboplastin; tissue extract	
IV	Calcium	
V	Accelerator globulin; proaccelerin; labile factor	
VI	This number is not now used	
VII	Proconvertin; stable factor; autoprothrombin I	
VIII	Antihaemophilic factor; antihaemophilic globulin; platelet co-factor I; antihaemophilic factor A	
IX	Plasma thromboplastin component (PTC); Christmas factor; platelet co-factor II; autoprothrombin II; anti-haemophilic factor B	
X	Stuart-Prower factor	
XI	Plasma thromboplastin antecedent (PTA)	
XII	Hageman factor	
XIII	Fibrin stabilizing factor	

Figure 6. The nomenclature of clotting factors using Roman numerals.

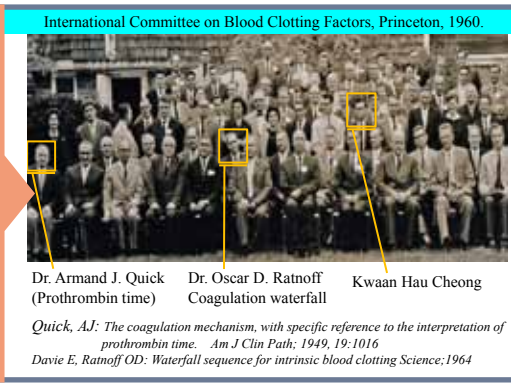


Figure 7. Group photo of the attendees of the meeting of the International Committee on the Nomenclature of Clotting Factors.

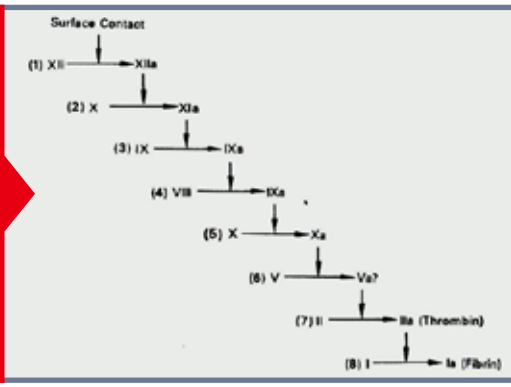


Figure 8. The cascade scheme of blood coagulation.

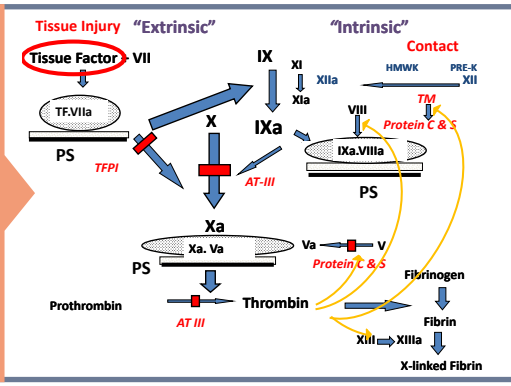


Figure 9. The current scheme of blood coagulation.

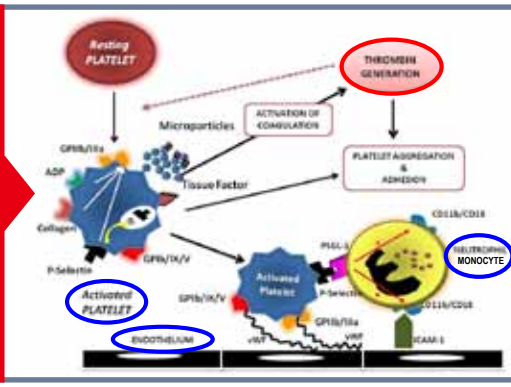


Figure 10. Factors that participate in thrombus formation (see text).

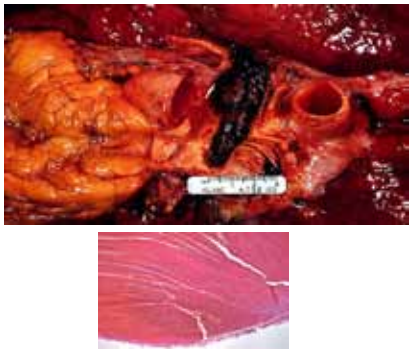


Figure 11. A venous thrombus that had embolized to the pulmonary artery (upper panel), with histologic section stained with hematoxylin and eosin (lower panel).

Figure 12. Thrombus in warfarin necrosis shown in the adipose tissue of the breast (upper left), resulting in skin necrosis (lower left). Similar skin necrosis was found in the abdominal wall (upper right), which extended to wide area (lower right).

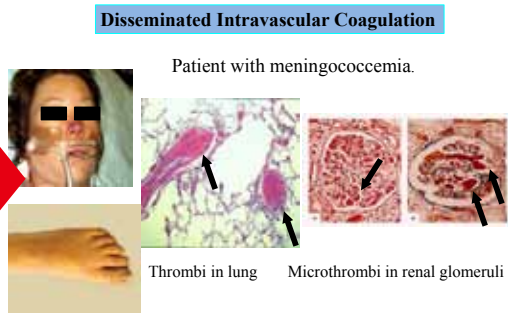


Figure 13. Thrombotic complications in a patient with meningococcemia complicated by severe disseminated intravascular coagulation resulting in wide-spread thrombosis, including vessels in lung and renal glomeruli.

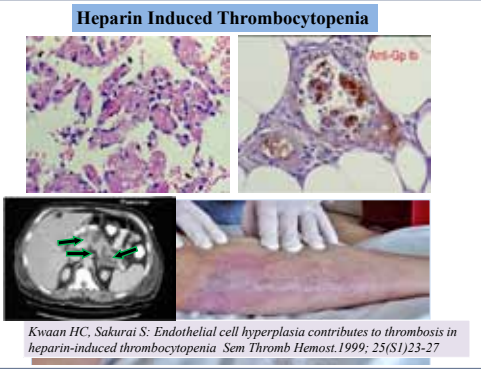


Figure 14. Thrombosis in heparin induced thrombocytopenia, showing thrombi composed of platelet clumps (upper left: hematoxylin and eosin stain and upper right: immunoperoxidase stain with anti-platelet Gp 1b. The thrombosis also resulted in multiple infarcts in the pancreas (arrows) and left leg.

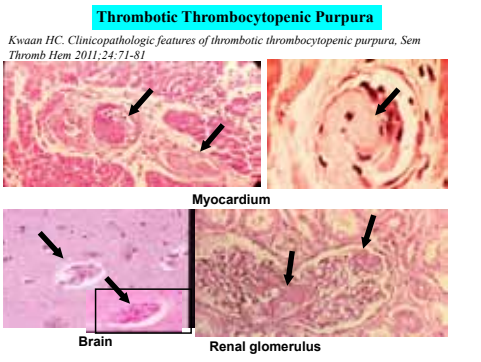


Figure 15. Wide-spread platelet-rich thrombi (arrows) seen in thrombotic thrombocytopenic purpura.

What happens to a thrombus? A thrombus can resolve by lysis or by organization and recanalization. I would like to demonstrate the lysis of a thrombus with this fibrin slide method[23] (Figure 20). Shown here is a venous thrombus, 4 hours after it was induced artificially, with areas of lysis indicated by the white color. The picture on the right shows that there was no lysis if the vessel wall was previously damaged.

"Prof. McFadzean…. exclaimed: "This is fibrinolysis, laddie"!"

The process of lysis of a clot can also be displayed in vitro as shown here in a series of test tubes containing blood clots incubated over a period of time (Figure 21). The clot will dissolve with time resulting in completely fluid blood. In 1954, I was performing the prothrombin time test on a bleeding patient. The endpoint is a clot formation. In the case of this patient's blood, a very small clot formed, but it disappeared right under my eyes within a few seconds. I still remember to this day that when I showed the result to Prof. McFadzean, he exclaimed: "This is fibrinolysis, laddie"! In severe fibrinolysis, the bleeding can be massive, as shown in (Figure 22). Under Prof. McFadzean's guidance, we studied patients at Queen Mary Hospital, with cirrhosis and found that they had increased fibrinolytic activity which could increase by more than 15 fold when undergoing splenectomy[24,25].

Prof. McFadzean decided to find out the source of this fibrinolytic activity, and, in his usual ingenious fashion, designed some in vivo experiments. First, he recruited some volunteers, I myself included, so was David Todd and the Professor himself. A blood pressure cuff was applied to the upper arm. Blood was collected from the antecubital vein and analyzed for fibrinolytic activity (Figure 23). We found that ischemia, and various agents including serotonin, adrenaline and acetylcholine could result in the release of fibrinolytic activity, demonstrating that fibrinolytic activity originated from the veins[26-28]. Some years ago, Virchow observed that blood in small vessels was more likely fluid and incoagulable, i.e. lysed blood, than blood in larger vessels. He deduced that the blood vessels were the origin of fibrinolysis. However, our observations were the first direct demonstration in vivo in man that fibrinolytic activity originates from veins. We were surprised that when the vein in one arm was stimulated, fibrinolytic activity was also released in the other (contralateral) arm (Figure 24). Prof. McFadzean predicted that is due to a signal transmission, conducted by the sympathetic pathway. At time, I felt that this idea was far-fetched. It was not until some 50 years later that an excited James

"our observations were the first direct demonstration in vivo in man that fibrinolytic activity originates from veins"

O'Rourcke came to me at a conference and said that he had just confirmed McFadzean's hypothesis. He was studying the sympathetic innervation of blood vessels and found that tPA is present in the sympathetic endings in the vascular adventitia and can be released into the blood after stress[29]. This is another example of McFadzean's incredible vision.

What is fibrinolysis? The active enzyme in the fibrinolytic system is plasmin, generated from the activation of plasminogen by plasminogen activators, tPA and uPA (Figure 25). Plasmin is a protease and in addition to fibrin, its substrates include extracellular matrix and growth factors. The biologic functions of plasminogen-plasmin system are distinct from fibrinolysis[30-32]. Here are some of the roles that this system plays under physiologic conditions (Figure 26). In addition, it contributes to the pathogenesis of many disorders. The mechanism is complex and will not be discussed in this talk. But as an example, the role of this system in cancer[32] is shown in (Figure 27).

Fibrinolytic enzymes are used in treatment of thrombosis. Thrombolysis was first carried out in human volunteers by Alan Johnson in artificially induced thrombosis with a dental broach in the forearms of healthy human volunteers, a feat that would have difficulty in getting through any IRB today[33]. He used streptokinase, a filtrate from the bacteria streptococcus. Other thrombolytic agents include urokinase, prourokinase, tPA, staphylokinase, Bat saliva activator and snake venom (Figure 28).

There is an interesting anecdote on the pharmaceutical production of urokinase. Urokinase is a plasminogen activator present in the urine. To produce one dose of urokinase, 1,500 liters of urine is required at a cost of US$1000 in 1977[34]. If we take the average of urine output as 2 liters per day, it would require 750 man-urine days. We called these 750 MUDS in jest. As we had by then discovered that urokinase can be found in the culture medium of kidney cell culture[35,36], the manufacturing was quickly switched to the tissue culture method.

Thrombolytic therapy is now widely used. A successful lysis of a venous thrombus in the superficial femoral vein is shown in (Figure 29). Today, the thrombolytic agent is usually administered via a catheter passed to the site of the thrombus[37,38]. In some cases, an ultrasonic device can also be inserted via the catheter allowing the ultrasonic waves to loosen the thrombus[39]. One of the most successful applications of thrombolysis is in the case of ischemic strokes. A recent meta-analysis comparing over 3000 patients treated

"To produce one dose of urokinase, 1,500 liters of urine is required at a cost of US$1000 in 1977[34]"

Figure 16. Thrombi formed by agglutinated red blood cells in a patient with catastrophic cold hemagglutin disease from anti-Pr autoantibody.

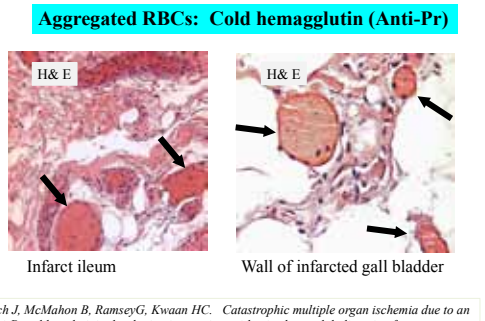


Figure 17. Red blood cells infected by P. falciparum forming microthrombi.

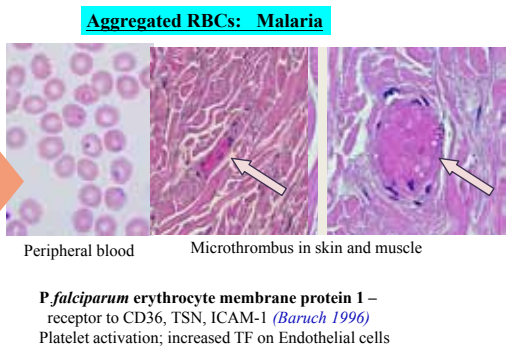


Figure 18. Microthrombi formed by monoblasts in acute myeloid leukemia M5 (FAB classification).

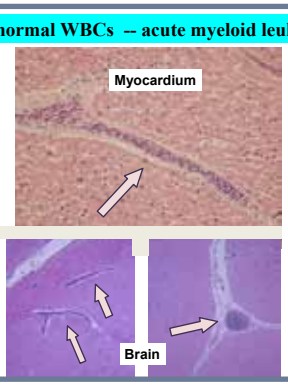


Figure 19. Thrombi containing tumor cells that express tissue factor.

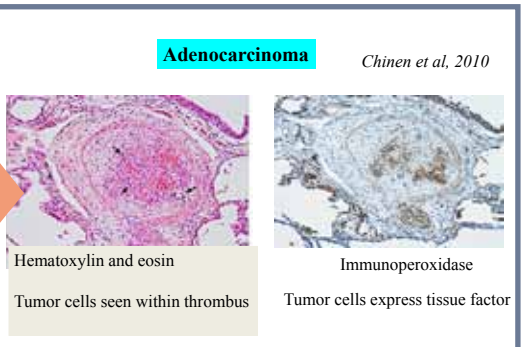
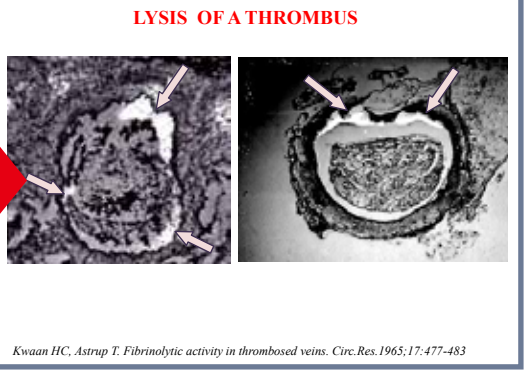


Figure 20. Lysis seen in experimentally produced thrombus shown as white areas (arrows) (fibrin slide method).



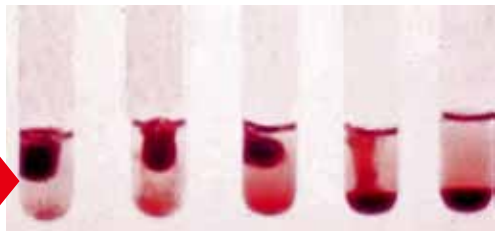


Figure 21.
The process of in vitro fibrinolysis.

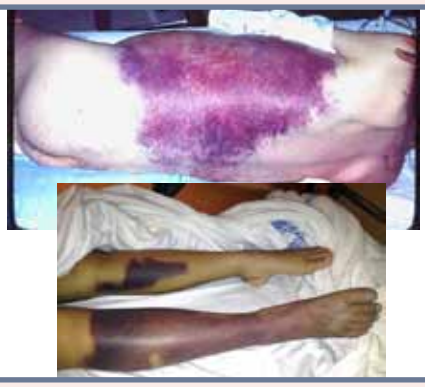


Figure 22.
Massive hemorrhage seen in severe fibrinolysis.

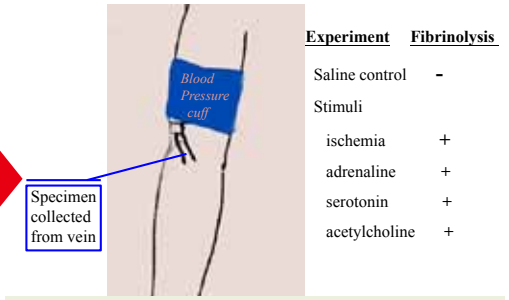


Figure 23.
The investigation of fibrinolysis release from veins in vivo.

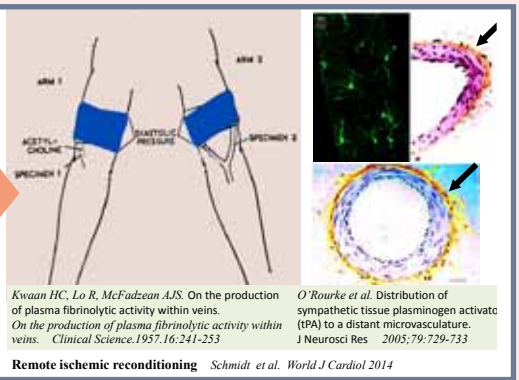


Figure 24.
Signal transmission via the sympathetic neurons in blood vessels.

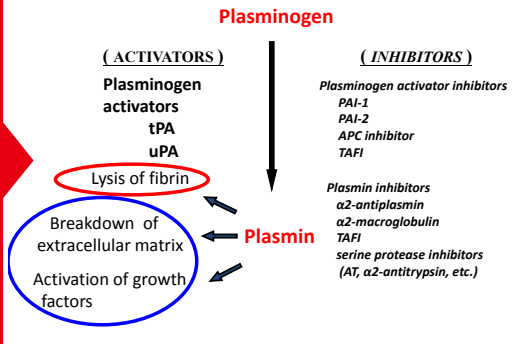


Figure 25.
The scheme of the plasminogen-plasmin system.

with alteplase (tPA) with those that was not given thrombolysis showed a definitely better outcome in the treated patients[40] (Figure 30). The treatment has to be initiated within 6 hours of onset of symptoms, with best results when treated within 3 hours.

Thromboprophylaxis

In the past 30 minutes, I had discussed with you the nature of a thrombus and thrombolysis. Now, I would like to proceed to thromboprophylaxis, either primary or secondary prevention. One notable advance in recent years is the advent of the new oral anticoagulants (NOACs) (Figure 31). These are small molecules, designed to directly inhibit specific steps in the coagulation pathway, with Rivaroxiban (Xarelto ™), Apixiban (Eliquis ™), Edoxaban (Lixiana ™) and Betrixaban being factor Xa inhibitors, and Dabigatran (Praxada™) inhibiting thrombin. They have a number of advantages over warfarin, with predictable bioavailability, less drug interactions, and improved safety, especially intracranial hemorrhage. At first, it was thought that their use requires no monitoring. This has since then proven to be incorrect. Monitoring is needed for those starting or switching anticoagulants, those with extreme body weights, those with change in renal or liver function or receiving medications that may change these functions, prior to invasive procedure, those that develop thrombosis or bleeding while on treatment, when there is a need to reverse the drug action and in particular, the elderly patient and the cancer patient.

Well, as Shakespeare put it, what is past is prologue *(The Tempest Act 2, Scene I)*; I would like to share with you my dreams on the future. First, we shall have a better understanding of mechanism for thrombosis. It takes much, much more than coagulation to form a thrombus. We have just scratched the surface in understanding these factors, e.g. role of inflammation, complement activation and genetics. For example, most of us have small thrombi in our body: apex of vein valves, etc. Why it extends is the important unanswered question. We may be able to better understand that in the future. We shall know the causes why in some common disorders, such as antiphospholipid syndrome, the rate of thrombosis is high. There will be better ways to detect thrombosis with new diagnostic tools and new laboratory tests. There will be better ways to control hemostasis with improved hemostatic agents. There will be better anticoagulants. They will be designed taking into consideration the role of endothelium, platelets and the thrombus. Issues relevant to efficacy, safety, drug interactions and cost will be solved. There may even be agents that has combined effect on clotting pathways as well as on platelet function, while at the same time may be fibrinolytic. We shall also change our approach to treatment of thrombosis -- with anti-thrombotics rather than anticoagulants. We shall attack each and every step of thrombus formation.

Figure 26.
The biologic role of the plasminogen-plasmin system.

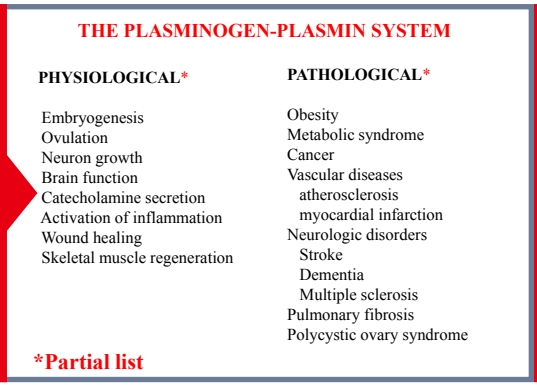


Figure 29.
Venogram showing the successful thrombolysis of a thrombosed superficial femoral vein (upper left: before) and after (upper right). An ultrasonic device is shown in the two lower panels.

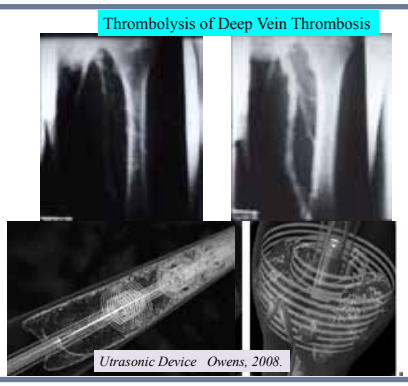


Figure 27.
The complex effects of the plasminogen-plasmin system on tumor cell proliferation.

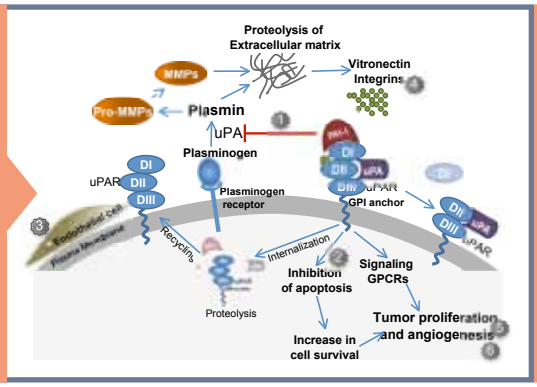


Figure 30.
Meta-analysis of the outcome in ischemic stroke comparing those treated with alteplase with controls.

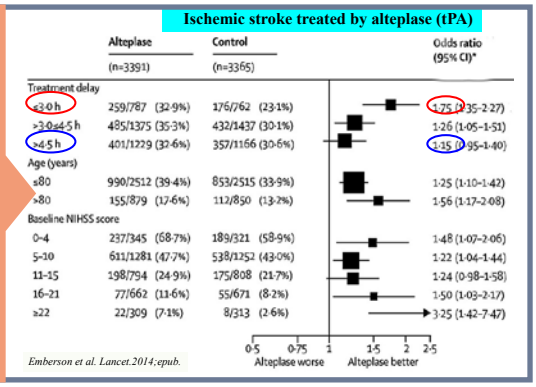


Figure 28.
Thrombolytic agents.

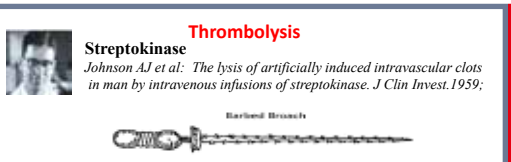
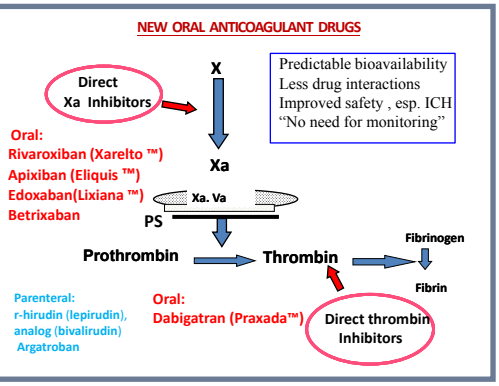


Figure 31.
The new oral anticoagulants.



"We shall also change our approach to treatment of thrombosis — with anti-thrombotics rather than anticoagulants. We shall attack each and every step of thrombus formation."

Acknowledgment

First and foremost, I would like to acknowledge Prof. McFadzean, who started me on the way. I would also like to thank all my past and present colleagues, here in Hong Kong and elsewhere.

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GERALD CHOA MEMORIAL LECTURE 2014

From Good to Great

Francis Ka Leung CHAN
DEPARTMENT OF MEDICINE & THERAPEUTICS
THE CHINESE UNIVERSITY OF HONG KONG

Gerald Choa was internationally known for his distinguished leadership in the medical profession and his dedication to local health services. As Founding Dean of Medicine in CUHK, Professor Choa not only established a second medical school in Hong Kong but also set a life-time example of how he transformed his passion into perfection. Over a century ago, when initiating medical education in Hong Kong, Sir Patrick Manson defined its object as "the spread of medical science, the relief of suffering, the prolongation of life and the increase of comfort during life". At the 20th Anniversary of the Medical Faculty in CUHK, Professor Choa said, "In the present day context, this definition is still valid. The Faculty has certainly succeeded in achieving the object. In a relatively short time, remarkable progress has been made and international recognition gained, as the result of the efforts of the present and past staff who have contributed so much to education, research and community service." Now the Medical Faculty in CUHK is much more than a second medical school to relieve shortage of local doctors, it has emerged as one of the best educational and research institutions in Asia. We are dedicated to nurturing competent and compassionate doctors who will make a difference to the world.

RICHARD YU LECTURE

Physicians and Emerging Infectious Disease Threats

Nelson Lai Shun LEE
DEPARTMENT OF MEDICINE & THERAPEUTICS
PRINCE OF WALES HOSPITAL
THE CHINESE UNIVERSITY OF HONG KONG

In the past decade, the world has experienced continuous threats from various emerging infectious diseases caused by novel pathogens. Avian influenza H5N1, first emerged in 1997 in Hong Kong then re-remerged in 2003, has become widespread and endemic in Asia. As of 2014, 667 cases and 393 (59%) deaths have been reported. Severe Acute Respiratory Syndrome (SARS)-associated coronavirus emerged in 2003 in Guangdong, China, had spread via air travel, and caused a global epidemic in weeks. Worldwide, there were over 8000 infections and 774 (10%) deaths. In Hong Kong alone, it caused over 1700 infections and nearly 300 deaths. About one-fifth of SARS victims were frontline healthcare workers who acquired the infection in the hospitals. In 2009, a swine-origin H1N1 influenza virus resulted in a pandemic. It was estimated that between 151,700 and 575,400 people died from this novel influenza infection globally; a disproportionate number of deaths occurred in Southeast Asia and Africa. Hong Kong was hit hard, resulting in excessive hospitalizations and deaths; fatality rate among hospitalized adults ranged from 3-15%. The H1N1 virus has continued to circulate in the community since.

In late 2012, another coronavirus, Middle-East-Respiratory Syndrome (MERS) coronavirus, emerged in Saudia Arabia and Qatar, which causes lethal pneumonia similar to SARS. To date, over 830 cases and nearly 300 (35%) deaths are recognized, and a more widespread transmission beyond the middle-eastern countries appears imminent. Starting from 2013, there is an on-going epidemic caused by a novel avian influenza H7N9 virus along the east coast of China; a total of 450 cases have been confirmed, with a cumulative death toll of 165 (37%). These emerging virus infections are all zoonotic in origin, and have acquired the ability to infect humans, escaping the innate host-defense. They mimic other causes of community-acquired pneumonia seen in daily practice, but typically run a rapidly deteriorating course. Standing at the forefront, physicians are managing these new diseases with fresh information generated through concurrent clinical/translational research on rapid diagnosis, antiviral treatment, pathogenesis, nosocomial transmission, and means of prevention. Some of our research efforts, which will be presented in this lecture, have equipped healthcare professionals in Hong Kong and elsewhere to meet the challenges posed by emerging infectious diseases.

The ASM 2014


Dr Kenneth Tsang, Prof TM Chan, Sir David and Dr Matthew Ng


Prof Yu


Prof Carol Pollock, from University of Sydney lectured on new diabetic therapies.


Dr Andrew Wong, Dr Matthew Tong, Dr ST Lai, Dr Doris Tse

ANNUAL SCIENTIFIC MEETING
(18-19 October 2014)

The College's annual scientific meeting at the Hong Kong Academy of Medicine Building featured three core symposiums on cancer screening, the role of physicians in rehabilitation and stroke prevention, preceded by a lunch symposium on new therapies in diabetes.

Highlights included the three prestigious named lectures. Professor Kwaan Hau Cheong, our distinguished AJS McFadzean orator was introduced by Sir David Todd. He delivered a fascinating account of thrombosis from the early work led by Professor McFadzean to key developments through the decades. The Gerald

Symposium on Cancer Screening


Chaired by Dr Pun Hui


Dr SC Ng

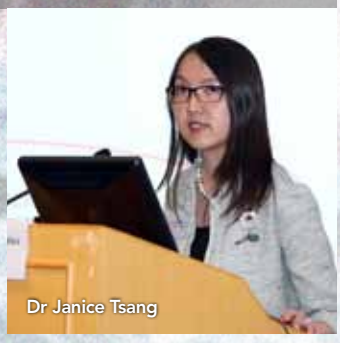

Dr James Ho

Symposium on Rehabilitation - The Role of Physicians


Chaired by Dr Leonard Li


Dr ST Lau


Dr KS Chan


Dr Janice Tsang

Symposium on Stroke Prevention


Chaired by Dr PW Ng


Dr Yannie Soo


Dr KH Chan

Choa Memorial Lecturer was Professor Francis Chan, who paid tribute to Professor Choa as the founding Dean of Medicine at the Chinese University of Hong Kong, with a lecture titled "From Good to Great". The Richard Yu Lecture by Professor Nelson Lee focused on emerging infectious diseases.

The Sir David Todd Lecture Medal was presented to Dr David Siu for his research in cardiac arrhythmias. Winners of the College's prizes for the Best Thesis Award and the Distinguished Research Paper Award for Young Investigators 2014 presented their work on the second day of the meeting.


Prof TM Chan
Chairman, Scientific Committee, HKCP

27TH ANNUAL GENERAL MEETING
16TH CONGREGATION AND ANNUAL COLLEGE DINNER

At the AGM held on the 18 October 2014, Dr Patrick Li delivered the presidential report which summarized the work and achievements of the HKCP during the past year. He addressed patient safety during invasive medical procedures, credentialing of specialists and feasibility of incorporating simulation training. He paid tribute to members of the various subcommittees for their continuing contributions rendered to the College. The official ceremony proceeded with the conferral of Fellowships and Memberships in the presence of a dignified platform party.

New Fellows were admitted during the annual Conferment ceremony held at the Annual College Dinner. This occasion was well attended by new and old Fellows alike, along with their family members. During the annual dinner, Professor Kwaan Hau Cheong delivered the 19th AJS McFadzean Oration. The President presented all the College awards for best academic achievements in the year 2013-2014.

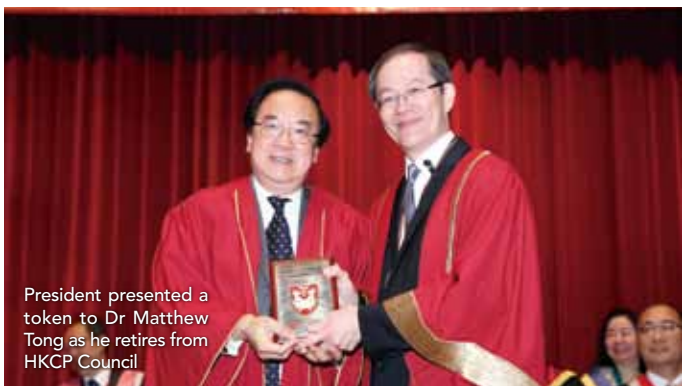

Past and Present Presidents, HKCP

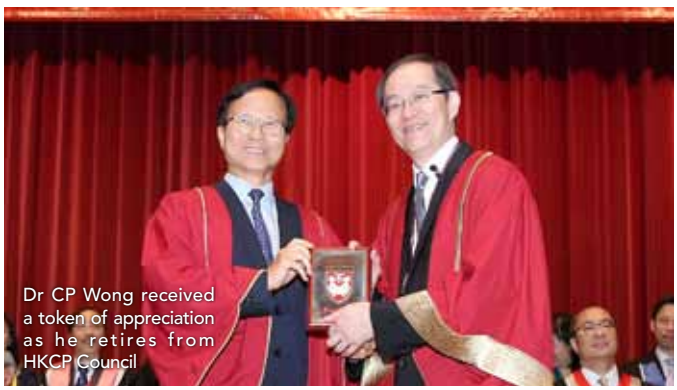

Dr Patrick Li and Prof Anthony Chan at the AGM


Sir David with Dr Jane Chan


Prof Kwaan with Dr Chew, Sir David and Prof WK Lam


Dr Patrick Li, Prof Yu and Prof Kwaan


President presented a token to Dr Matthew Tong as he retires from HKCP Council


Dr CP Wong received a token of appreciation as he retires from HKCP Council

The AJS McFadzean Oration 2014

FROM BLOOD CLOTS TO THROMBOSIS AND BEYOND - *A REMARKABLE GROWTH OF KNOWLEDGE IN SIX DECADES*

Professor Hau Cheong KWAAN
Marjorie C. Barnett Professor of Hematology-Oncology
Professor of Medicine
Northwestern University Feinberg School of Medicine,
Chicago, U.S.A.

The Gerald Choa Memorial Lecture 2014

FROM GOOD TO GREAT

Professor Francis Ka Leung CHAN
Department of Medicine & Therapeutics
The Chinese University of Hong Kong

Sir David Todd Lecture 2014

CARDIAC ARRHYTHMIAS : FROM FAST TO SLOW, FROM PATIENTS, GENE TO STEM CELLS, FROM A CLINICIAN TO A BASIC SCIENTIST

Dr David Chung Wah SIU
Department of Medicine,
Queen Mary Hospital
The University of Hong Kong

Richard Yu Lecture 2014

PHYSICIANS AND EMERGING INFECTIOUS DISEASE THREATS

Professor Nelson Lai Shun LEE
Department of Medicine and Therapeutics
Prince of Wales Hospital
The Chinese University of Hong Kong

Distinguished Research Paper Award for Young Investigators 2014

	**Dr Siew Chien NG** Department of Medicine & Therapeutics, Prince of Wales Hospital **Winner for the Best Oral Presentation Award**	Incidence and phenotype of inflammatory bowel disease based on results from the Asia-Pacific Crohn's and Colitis Epidemiology Study	Ng SC et al; Asia–Pacific Crohn's and Colitis Epidemiologic Study (ACCESS) Study Group Gastroenterology. 2013 Jul;145(1):158-165.
	**Dr Walter Wai Kay SETO** Department of Medicine, Queen Mary Hospital	Reduction of hepatitis B surface antigen levels and hepatitis B surface antigen seroclearance in chronic hepatitis B patients receiving 10 years of nucleoside analogue therapy	Seto WK, Wong DK, Fung J, Huang FY, Lai CL, Yuen MF. Hepatology. 2013 Sep;58(3):923-31.
	**Dr Grace Lai Hung WONG** Department of Medicine & Therapeutics, Prince of Wales Hospital	On-treatment alpha-fetoprotein is a specific tumor marker for hepatocellular carcinoma in patients with chronic hepatitis B receiving entecavir	Wong GL, Chan HL, Tse YK, Chan HY, Tse CH, Lo AO, Wong VW. Hepatology. 2014 Mar;59(3):986-95.
	**Dr Thomas Chung Cheung YAU** Department of Medicine, Queen Mary Hospital	Development of Hong Kong Liver Cancer staging system with treatment stratification for patients with hepatocellular carcinoma.	Yau T, Tang VY, Yao TJ, Fan ST, Lo CM, Poon RT. Gastroenterology. 2014 Jun;146(7):1691-700.
	**Dr Kai Hang YIU** Department of Medicine, Queen Mary Hospital	Prognostic value of preoperative right ventricular geometry and tricuspid valve tethering area in patients undergoing tricuspid annuloplasty	Yiu KH, Wong A, Pu L, Chiang MF, Sit KY, Chan D, Lee HY, Lam YM, Chen Y, Siu CW, Lau CP, Au WK, Tse HF. Circulation. 2014 Jan 7;129(1):87-92.

Young Investigator Research Grant 2014

The following doctors received a research grant from the HKCP to complete their respective projects as named. Selection was decided by a scientific panel headed by Professor KS Wong. Applications for 2015 will be advertised in the College website around April-May of each year.

The grant was established in 2012, to encourage young members of fellow who are aged 40 years or below to conduct research in Hong Kong. Up to five Grants of up to $50000 each are awarded annually.

Early diagnosis of frontotemporal dementia (FTD) with arterial spin labeling perfusion MRI (ASL-MRI)
Dr Lisa Wing Chi AU

A feasibility study correlating molecular signatures between initial biopsies and resected specimens in patients with unresectable gastrointestinal stromal tumours who become resectable after neoadjuvant imatinib mesylate and exploring potential treatment implications
Dr Herbert Ho Fung LOONG

24-hour thrombolysis for acute ischaemic stroke with telemedicine. A pioneer project in Hong Kong
Dr Yannie Oi Yan SOO

Vitamin D deficiency in cancer patients receiving palliative care
Dr Raymond Kam Wing WOO

Renal tubular epithelial cell-binding immunoglobulin G in nephritis impact on clinical activity, renal histopathology and immunopathogenesis
Dr Desmond Yat Hin YAP

Award for obtaining the highest score in AIM Exit Assessment (2014)

Dr Thomas Sau Yan CHAN
DEPARTMENT OF MEDICINE
QUEEN MARY HOSPITAL

Dr Gill Harinder SINGH
DEPARTMENT OF MEDICINE
QUEEN MARY HOSPITAL

Award for obtaining the highest score in PACES (2014)

Dr Clara Ka Chung SIN
DEPARTMENT OF HEALTH

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SIR DAVID TODD LECTURE

Cardiac Arrhythmias: From Fast to Slow, From Patients, Gene to Stem cells, From a Clinician to a Basic Scientist

David Chung Wah SIU
DEPARTMENT OF MEDICINE, QUEEN MARY HOSPITAL
THE UNIVERSITY OF HONG KONG

The heart beats more than 2 billion times in this highly coordinated fashion during a normal human lifespan. Cardiac arrhythmias including both brady-arrhythmias and tachy-arrhythmias as a result of abnormalities in the generation and/or conduction of these electrical impulses can disrupt this essential, life-sustaining physiological process, and result in symptom or even sudden cardiac death. As a result, the management as well as research of cardiac arrhythmias can be both challenging and rewarding. My long-term goal of research is to improve the clinical outcomes of patients with cardiac arrhythmias and to develop novel therapeutic strategy for these conditions.

CLINICAL RESEARCH

Atrial Fibrillation

My research interest in cardiac arrhythmias stems from the difficulties and challenges in managing patients with hyperthyroidism related atrial fibrillation (AF) soon after my graduation. The clinical management has been largely based on expert-opinions. Albeit lacking clinical data, beliefs such as hyperthyroid AF is highly reversible and requires no specific treatment such as anticoagulation, have been widely prevailed. In order to better serve my patients, with the support from Prof. A Kung, I have established a large cohort of hyperthyroid AF patients to understand their clinical outcomes. This leads to a series of publications in high-ranking journals in endocrinology and cardiology and provides clinical evidences to support our management of hyperthyroid AF. These works have been put together to my thesis of Medical Doctor, which eventually leads to the prestigious Patrick Mansion Gold Medal.

Nonetheless, hyperthyroid patients constitute only a small fraction of AF population. The management of ordinary Chinese AF patients is even more challenging, due to the scarcity of data in our ethnic group. For instance, the utilization of oral anticoagulant, the cornerstone in AF management, is extremely low amongst Chinese. This reflects the challenge of balancing the benefit of stroke risk reduction with the risk of intracranial hemorrhage consequent to the therapy in Chinese as there are widespread perceptions that Chinese might have a lower risk of stroke attributable to AF, but a higher baseline rate of intracranial hemorrhage. Over the last 3 years, I have established the world largest cohort of Chinese AF patients (~10,000 patients) and through this registry we have been able to provide solid clinical data to answer many of these burning questions in the everyday management of AF.

BASIC AND TRANSLATIONAL RESEARCH

Bio-artificial Pacemakers

Sick sinus syndrome, a common cardiac arrhythmia, can result from pacemaker cell loss, conduction block between pacemaker cells and surrounding cardiomyocytes. Patients usually experience symptom such as syncope necessitating electronic pacemaker implantation. Important shortcomings of electronic pacemakers including limited battery lifespan, physiological non-responsiveness, and safety concerns as reflected by frequent recalls and safety alerts have motivated my quest of biological alternatives during my 2-year post-doctoral fellowship training at the University of California, Davis supported by the Croucher Fellowship Award.

Using protein engineering to modify the biophysical properties of hyperpolarization-activated cyclic nucleotide-modulated (HCN) channels underlying the funny current (I_f), our research team has successfully exploited adenoviral vector-mediated gene-transfer of a gating-engineered version of HCN channels to non-pacemaker cardiomyocytes to over-express I_f, conferring them pacemaker activity, which responds to physiological signals. This phenotypic shift induced pacemaker activities has been shown in our in vivo large animal model of sick sinus syndrome to reduce electronic pacemaker dependence.

Pluripotent Stem Cells for Modeling of Inherited Cardiomyopathy

While advances in the pharmacological management of heart failure have improved the clinical outcomes of patients with heart failure due to ischemic cause, the outcome of patients with heart failure secondary to inherited form of cardiomyopathy remains poor. This is because in stark contrast to ischemic cause, the pathophysiology of many inherited cardiomyopathy remains elusive, thereby specific therapeutic strategies targeting the key pathogenic process is not available for most inherited cardiomyopathy. Theoretically, direct functional electrophysiological characterization of human cardiomyocytes obtained from patients with the syndrome will provide much-needed insight to the diagnosis, pathogenesis and pharmacological responsiveness of individual patients, but is limited by the current technology.

Human induced pluripotent stem cells (iPSCs) can be generated from dermal fibroblasts by the transduction of a defined set of transcription factors to reprogram them back to an equivalent of the early embryonic state and can then be directed to cardiomyocytes, offering virtually an unlimited ex vivo source for patient-specific cardiomyocytes for in vitro experimentation, bypassing the need to obtain living human cardiac tissue. In the past decade, we have been focusing to optimize iPSC technology; notably, my laboratory is one of the first three laboratories worldwide to generate human induced pluripotent stem cells using an animal-product free protocol. Using this approach, we have provided important pathophysiological insights as well as to identify potential therapeutic targets for patients with various cardiomyopathies such as Friedreich ataxia and lamin-related cardiomyopathy. Our data has been selected by the European Society of Cardiology as the hotline session (equivalent to late-breaking in the US) in 2011.

BEST THESIS AWARD
Gold Award Winner

Prospective Study into the Risk of Colorectal Neoplasms in Asymptomatic Individuals with a Family History of Advanced Adenomas

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DEPARTMENT OF MEDICINE & THERAPEUTICS
PRINCE OF WALES HOSPITAL

BACKGROUND AND AIM

The risk of colorectal neoplasms among siblings of individuals with advanced adenomas is unclear. We assessed the prevalence of advanced adenomas and cancers in siblings of individuals with advanced adenomas compared with siblings with no such lesions.

METHODS

Colonoscopies were performed in 188 asymptomatic siblings to subjects with advanced adenomas (adenomas ≥10mm, high grade dysplasia, villous/tubulo-villous; cases, age 58±6.4 years) and 323 age- and sex-matched siblings of healthy subjects who had normal colonoscopies (controls, 57.6± 5.9 years). Primary outcome was the prevalence of advanced neoplasms defined as cancers or advanced adenomas.

RESULTS

The prevalence of advanced neoplasms was 11.7% among siblings of patients and 2.8% among controls (matched odds ratio [mOR], 4.58; 95% confidence interval [CI], 2.07-10.14; P≤0.001). The prevalence of adenomas ≥10 mm (11.2% vs. 1.9%; mOR, 6.62; 95% CI, 2.61-16.8; P<0.001), villous adenomas (5.9% vs. 1.2%; mOR, 5.01; 95% CI, 1.57-16.04; p=0.007) and all colorectal adenomas (38.3% vs. 21.7%; mOR, 2.27; 95% CI, 1.51- 3.41; P<0.001) was higher among siblings of patients. Two cancers were detected among siblings of patients; no cancer was detected in controls. The prevalence of advanced neoplasms among siblings of patients was higher when their index case was female (mOR, 6.4; 95% CI, 2.0–20.46) and had large adenomas (mOR, 4.6; 95% CI, 1.9–11.3) or villous adenomas (mOR, 7.6; 95% CI, 1.6-36.1).

CONCLUSIONS

Siblings of patients with advanced adenomas have a more than four-fold increased risk of developing advanced neoplasms. This high risk group should be offered screening colonoscopy.

BEST THESIS AWARD
Silver Award Winner

Diaphragmatic Ultrasonography for the Mechanically Ventilated Patients

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ADULT INTENSIVE CARE UNIT
QUEEN MARY HOSPITAL

INTRODUCTION

Mechanical ventilation (MV) is a crucial intensive care unit (ICU) tool, however its use can cause diaphragmatic atrophy and dysfunction. Previous studies described ultrasound measurements of diaphragm thickness or excursion.

OBJECTIVE

We hypothesize that diaphragmatic ultrasonography is a safe and effective tool for predicting ventilator weaning outcome in critically ill patients requiring MV.

METHODS

We performed a prospective observational pilot study to evaluate the relationship of ultrasonographically measured percentage respiratory change of diaphragmatic thickness (Δtdi%) and diaphragmatic excursion with clinical outcome. We recruited 31 ICU patients upon necessitating MV. We performed serial ultrasound on bilateral diaphragm from start until end of MV period.

RESULTS

For predicting weaning failure, decrease in Δtdi% and excursion at the end of MV period were the best predictors [ROC AUC Left 0.979, Right 1.000; excursion AUC Left 0.986, Right 0.861]. Even first day values showed high negative predictive values (≥88.9%). For predicting MV duration, at least one side of the diaphragm showed increase Δtdi% and excursion to be associated with shorter duration.

CONCLUSIONS

The diaphragmatic ultrasound measurements of Δtdi% and excursion are safe to perform and feasible predictors of MV outcome. The best predictor of weaning failure is reduction of Δtdi% and diaphragmatic excursion on last day. Application of these ultrasound parameters may help assess readiness for weaning.

BEST THESIS AWARD
Bronze Award Winner

Comparison of three comorbidity indices as predictors of survival in Chinese incident peritoneal dialysis patients

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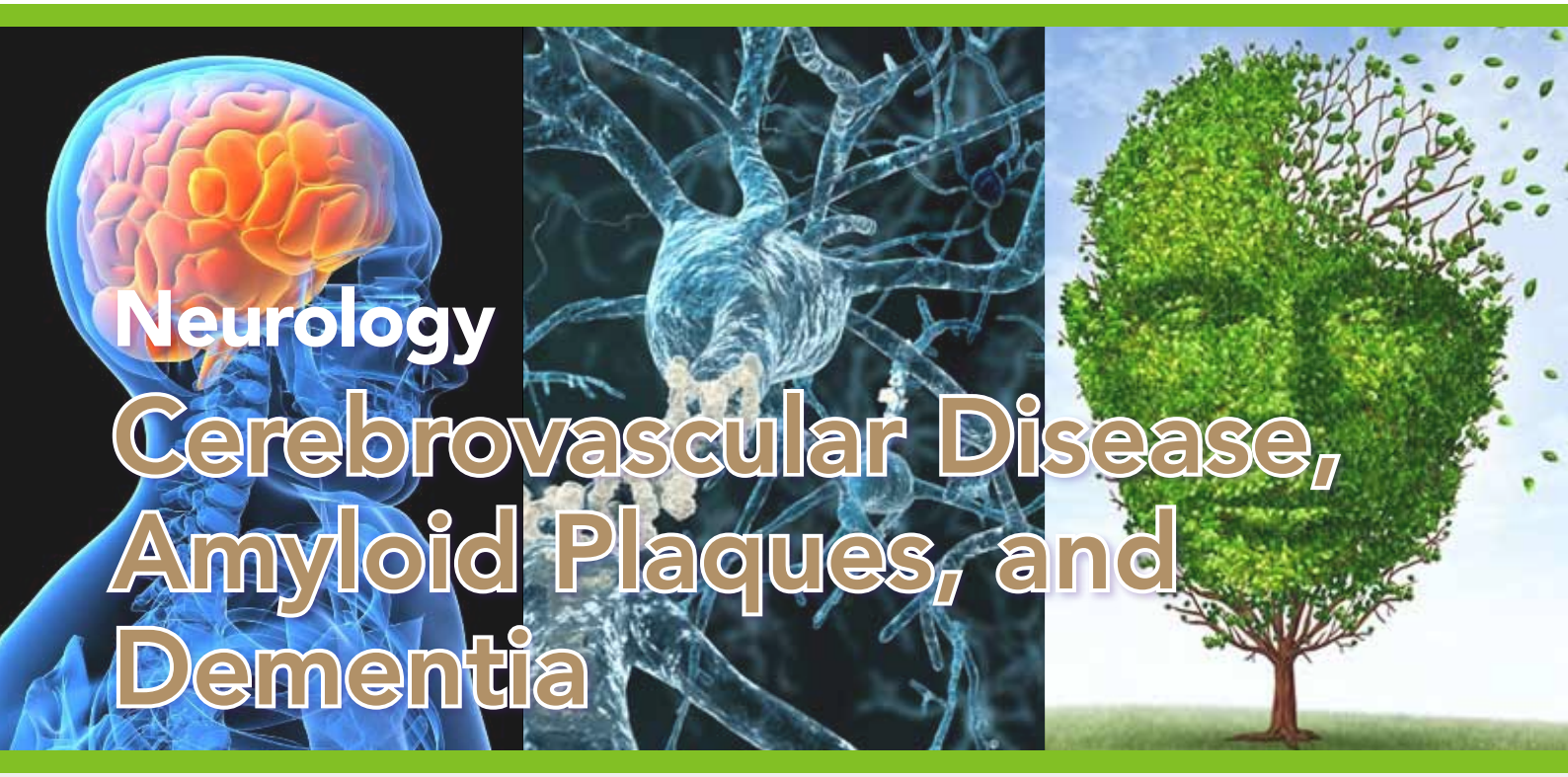
PRINCE OF WALES HOSPITAL

BACKGROUND
A number of comorbidity indices have been developed to quantify the burden of comorbid conditions and predict survival in dialysis patients, but data on the use of these indices in Chinese peritoneal dialysis (PD) patients are limited. The aim of this study is to compare the performance of the Charlson Comorbidity Index (CCI), Hemmelgarn index and Liu index in predicting survival in incident Chinese PD patients.

METHODS
In this retrospective study, 461 incident PD patients were recruited. Clinical information and presence of comorbid conditions were obtained by chart review and cross-checking. Cox regression models were used to compare the prognostic value of the three indices.

RESULTS
The mean age was 57.7 ± 13.7 years. The median Charlson, Hemmelgarn, and Liu scores were 4 (inter-quartile range [IQR] 2 to 5), 1 (IQR 0 to 2), and 4 (IQR 2 to 5), respectively. Patients were followed for 45.5 ± 33.0 months. Cox regression analysis showed that after adjusting for confounding factors, CCI was the best predictor of patient survival, with each point increase in Charlson score associated with 31% increased risk of mortality. The Hemmelgarn index was the best one to predict technique survival and peritonitis-free survival. However, over 70% of patients scored 0 or 1 by this system, limiting its role as a prognostic marker.

CONCLUSIONS
The CCI was the best predictor of patient survival in incident Chinese PD patient. Further prospective studies are required to determine the role of comorbidity indices on hospitalization and medical expenditure in Chinese PD patients, as well as the application of comorbidity index for serial monitoring of PD patient.



Neurology
Cerebrovascular Disease, Amyloid Plaques, and Dementia

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Introduction

Amyloid plaques, as one of the neuropathological diagnostic hallmarks of Alzheimer's disease (AD), frequently co-exist with CVD.[1] Neuropathological examinations show that between 6% and 47% individuals with dementia had co-existing AD and CVD pathologies (Table 1). Indeed, mixed pathologies may account for the majority of dementia cases (38%).[2] Over the last 2 decades, there has been a surge in interest in the study of the potential relationship or interaction between amyloid plaques and CVD. Does CVD induce amyloid plaques? Will CVD interact synergistically with amyloid plaques to increase the severity of cognitive impairment?

In the past, research on the relationship and interaction between amyloid plaques and CVD relied mostly on autopsy study. Although autopsy study is considered the gold standard for determining the underlying pathology of dementia, it has inherent limitations with correlating pathological and clinical findings. In particular, uncertainties exist with regard to the temporal relationship between the development of brain lesions and dementia syndrome because of the time lag between dementia onset and autopsy. Moreover, the exact severity or extent of certain manifestations of CVD, in particular, white matter changes (WMC), which is a manifestation of cerebral small vessel disease, is difficult to quantify in autopsy studies.[3]

Clinical in-vivo study investigating the relationship between amyloid plaques and cognitive impairment has only been made possible a decade ago with the advent of amyloid imaging such as Carbon-11-labeled Pittsburgh Compound B (PiB) positron emission tomography (PET). Other amyloid ligands, such as flutemetamol (GE-067), florbetaben (BAY-94-9172, AV-1) and florbetapir (AV-45) have also become available to the market in recent years.[4] Amyloid PET can detect comorbid amyloid deposition in patients with high vascular burden. In this article, we reviewed the potential interactions between CVD and amyloid plaques and their relationships with cognitive impairment, with reference to in-vivo clinical studies using amyloid PET imaging[5-21] and autopsy studies measuring amyloid plaques.[22-25] Understanding the mechanisms for elderly dementia syndrome will provide insight in devising preventive or curative strategy of this highly prevalent and disabling disorder.

Prevalence of amyloid plaques in subjects with CVD

It is only in recent years that researchers began to study in-vivo

Table 1: Pathology findings in dementia

Author	Setting	Number	Mean Age (years)	AD+ CVD (%)	Pure AD (%)	Pure VaD (%)	LBD (%)	FTD (%)	Others (%)
Victoroff (1995)	Hospital	196 d	76.6	25 (12.8%)	88 (44.9%)	9 (4.6%)	13 (6.6%)	NA	61 (31.1%)
Snowdon (1997)	Convents	45 d	76-100	21 (47%)	NA	NA	NA	NA	NA
Nolan (1998)	Hospital	87 d	NA	32 (37%)	44 (50.6%)	-	NA	NA	11 (12.6%)
Seno (1999)	Nursing home	122 d	83.6±8.3	14 (11%)	41 (34%)	42 (35%)	NA	NA	25 (20%)
Barker (2002)	Hospital	382 d	79±13	43 (11%)	159 (42%)	12 (3%)	99 (25.9%)*	21 (5.5%)†	48 (12.6%)
Akatsu (2002)	Geriatric hospital	158 d	81.2±8.3	9 (6%)	73 (46%)	34 (22%)	28 (18%)	-	14 (8%)
Riekse (2004)	Community	124 d	65+	18 (14.5%)	30 (24.2%)	8 (6.5%)	NA	NA	124 (54.8%)
Petrovitch (2005) (HAAS)	Community	333 (120 d)	78	22 (18.3%)‡	37 (30.8%)	35 (29.2%)	–	–	26 (21.7%)
Schneider (2007) (the Rush MAP)	Community	141 (50 d)	87.8±5.6	19 (38%)	15 (30%)	6 (12%)	NA	-	10 (20%)
Brunnstrom (2008)	Hospital	524 d	80 (39-102)	113 (21.6%)	220 (42%)	124 (23.7%)	1 (0.2%)	21 (4%)	45 (8.6%)
Schneider (2009) (the Rush ROS & MAP)	Community	179 d	86.9	54 (30.2%)	76 (42.4%)	8 (4.5%)	30 (16.8%)*	NA	11 (6.2%)
Brayne (2009) (CC75C study)	Community	213 (113 d)	91.1 (81-101)	25 (22.1%)	76 (67.2)	4 (3.5%)	1 (1%)	NA	7 (6%)
White (2009) (HAAS)	Community	443 (183 d)	72-90+	26 (14.2%)‡	34 (18.6%)	62 (33.8%)	20 (10.9%)	-	41 (22.4%)
Jellinger (2010)	Hospital	1700 d	84.3±5.4	473 (27.8%)	775 (45.6%)	209 (12.3%)	158 (9.3%)	NA	85 (5%)
Sinka (2010)	Hospital	93 (86 d)	90-103	17 (19.7%)	48 (55.8%)	21 (24.4%)	-	-	-
Echavarri (2012)	Hospital	200 d	78.7 (15-100)	31 (16%)	52 (26%)	4 (2%)	1 (0.5%)	4 (2%)	113 (56.5%)
Grinberg (2013) (BBBABSG)	Community	1291 (113 d)	78.3±9.7	15 (13.3%)	40 (35.4%)	24 (21.2%)	NA	NA	34 (30.1%)
Magaki (2014)	Hospital	218 d	50-100	24 (11%)	123 (56.4%)	1 (0.5%)	31 (14.2%)*	14 (6.4%)	25 (11.5%)

*Including LBs+ others; † Including FTD+ others; ‡ AD+ others, but most are AD + CVD

Abbreviations: AD, Alzheimer's disease; CVD, cerebrovascular disease; VaD, vascular dementia; LBD, Lewy body disease; FTD, frontotemporal dementia; LBs, Lewy bodies; HAAS, Honolulu-Asia Aging Study; MAP, Rush Memory and Aging Project; ROS, Religious Order Study; CC75C, Cambridge City over-75s Cohort; BBBABSG, Brain Bank of the Brazilian Aging Brain Study Group; d, number of patients with dementia; NA, not available.

the prevalence of amyloid plaques in subjects with vascular dementia (VaD). Using amyloid PET, AD-like PiB retention was found in 29.7% of patients with incident dementia after stroke or transient ischemic attack (TIA) and 31.1% of patients with clinical diagnosis of subcortical vascular dementia.[5, 6] These findings are in keeping with the prevalence reported in few neuropathological studies showing that AD pathology, including both amyloid plaques and neurofibrillary tangles, was found between 25.8% and 55.6% of patients diagnosed with possible vascular dementia (VaD).[26-28]

Interactions between CVD and amyloid plaques

Being the two commonest causes for dementia, mounting evidence shows that CVD and AD share risk factors including age, hypertension, diabetes mellitus, hypercholesterolaemia, ischemic heart disease, and smoking. Research findings on the relationship between CVD manifestation and amyloid accumulation are discussed below.

Atherosclerosis and amyloid plaques

Atherosclerosis in large cerebral arteries can compromise CBF and cause cerebral hypoperfusion. AD

patients had a higher proportion and more pronounced atherosclerosis in the circle of Willis than those patients with non-AD dementias (except VaD) as shown in autopsy examination.[22] Through [18F] AV-45 PET examination, the distribution of the increased amyloid disposition was found to be lateralized to the side of stenosis in patients with dementia.[7] Together, these clinical and pathological findings suggested that there was a shared etiology between atherosclerosis and amyloid deposition.

Infarcts and amyloid plaques

Most amyloid PET studies did not find significant relationships between infarcts and amyloid deposition.[8, 9] In fact a study showed that in patients with subcortical vascular cognitive impairment, the number of lacunes was negatively related with the PiB retention ratio.[10] In patients with recent ischemic stroke, although a relatively increased PiB retention was found in peri-infarct region comparing to the contralateral mirror region, this increased regional PiB retention did not translate into a higher global PiB retention.[11] Moreover, the possibility that the observed increase in local PiB retention being a result of leakage of free PiB due to the BBB damage could not be excluded. Overall, thus far there is no solid evidence of a significant relationship between cerebral infarcts and global amyloid burden.

WMC and amyloid plaques

Findings regarding the relationship between WMC and amyloid accumulation appear to be inconsistent. In general, most studies showed a lack of correlation between WMC and amyloid load.[8-10, 12, 13] However, there were also studies showing positive relationships between WMC and amyloid.[14, 15] For instance, in sporadic AD patients, WMC at baseline was significantly related to the progression of amyloid deposition in 28 months, suggesting that WMC might accelerate the aggregation of amyloid protein produced in situ.[14] However, WMC appeared to be more closely linked to vascular amyloid deposition associated with cerebral amyloid angiopathy (CAA) rather than parenchymal amyloid deposition characteristic of AD.[15, 16] Noteworthy is that a study among cognitively normal individuals showed that gray matter regions with more WMC had lower amyloid deposition.[17] It was hypothesized that WMC might disrupt the entry of misfolded proteins from white matter tracts and thus resulted in lower amyloid load in gray matter regions.[17] In view of these conflicting results, further investigation between WMC and amyloid is warranted.

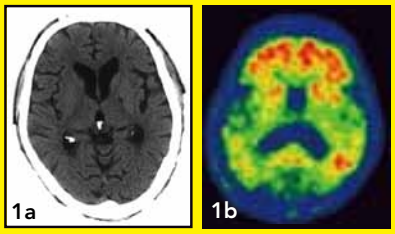
Interactions between CVD and amyloid plaques upon cognition

Comorbid CVD and amyloid plaques upon expression of dementia

The Nun study was a landmark study that revealed a concerted effect of lacunes and AD pathologies upon clinical expression of dementia. It suggested that concurrent cerebral infarcts might significantly amplify the risk and severity of dementia in patients with neuropathologically confirmed AD.[29] (Figure 1) More recent studies using amyloid PET also showed converging findings. For example, PiB- positive (PiB+) subcortical VaD patients had lower cognitive performance than those who were PiB- negative (PiB-).[6] Interestingly, there appears to be a dynamic balance between amyloid deposition and CVD pathologies upon the expression of dementia because on a given level of cognitive impairment, less amount of one kind of pathology is found when the amount of other kind of pathology increases. For example, in neuropathological studies, amyloid plaques were found to be fewer in those with CVD lesions.[23-25] Among patients with poststroke dementia, PiB+ patients had fewer old infarcts than their PiB- counterparts.[5, 6]

Figure 1 Clinical case illustrating potential synergistic interaction between CVD and amyloid plaques upon cognition

A 78-year-old gentleman presented with acute mild left hemiparesis. CT brain showed small left basal ganglia lacunar infarct. (Figure 1a) He complained of progressive memory decline and difficulty in handling money shortly after the episode. 3 months later he was diagnosed to have mild dementia (mini-mental state examination score of 22). PIB PET showed retention over bilateral frontal, parietal, temporal, occipital lobes, precuneus and posterior cingulate gyrus typical of AD. (Figure 1b)



Although findings from above studies raised the possibility of a synergistic effect between the two pathologies, other study suggested that CVD largely contributed an additive, rather than synergistic, effect upon the expression of dementia against the background of amyloid burden.[10] Note that synergistic effects between the two pathologies might be specific to cognitive domains such as visuospatial functions.[20]

Comorbid CVD and amyloid plaques upon cognitive profile of dementia

In AD, amyloid has most robust effects on episodic memory among the cognitive domains. In contrast, executive dysfunction tends to be typically more prominent in patients with subcortical vascular disease, in which their effects are more pronounced on the frontosubcortical system. Such dissociations have been demonstrated in patients with mixed pathologies. For example, in patients with subcortical vascular dementia and mild cognitive impairment, amyloid burden appeared to be more related to episodic memory

decline, whereas WMC was associated with poor executive function.[6, 10, 20] A path analysis study showed that memory deficits related to amyloid burden was mediated by hippocampal atrophy while WMC affected executive function via frontal thinning.[21]

However, the relationships between cognitive domains and pathologies may not be straightforward. For example, WMC could also affect episodic memory by affecting working memory and executive retrieval with frontal dysfunction.[21] In addition, amyloid and CVD might influence memory and executive function independent of brain atrophy.[21]

Several possibilities may explain the overlaps of the cognitive domains associated with amyloid deposition and CVD. First, cerebral regions affected by amyloid deposition and CVD may overlap. Distribution of amyloid deposition is typically observed in the frontal cortex, posterior cingulate, precuneus, caudate, parietal cortex, and lateral temporal cortex, which are brain regions involved in a wide range of cognitive processes including executive functions, attention and memory. Locations of cerebrovascular lesions in individuals are variable in CVD. In addition to executive dysfunction, cerebrovascular lesions in posterior cerebral artery territory may also affect the medial temporal lobe with consequential memory failure.[30] Second, distinct cerebral regions are associated with multiple cognitive processes rather than a single cognitive function. For instance, both executive functions and memory are subserved by the frontal lobes and related pathways.[31] Third, in addition to brain atrophy, functional network disruptions may also impair cognition.[32] Therefore, given that differences in neuropsychological profiles between AD, VaD and mixed dementia are less distinct than expected, it may be clinically difficult to distinguish pathologies based on cognitive profile alone.

Conclusion and future directions

Autopsy and amyloid PET studies showed the existence of comorbid CVD and amyloid deposition in patients with cognitive impairment. Some studies suggest that certain cerebrovascular lesions are associated with amyloid plaques accumulation. Overall, the co-existence of the two pathologies is associated with a higher prevalence of dementia and more severe cognitive impairment. In addition, differences in neuropsychological profiles between CVD and amyloid plaques are less distinct than we once thought.

Although some studies hinted that CVD may be a triggering factor for amyloid plaques accumulation, relationships between amyloid deposition and different kinds of cerebrovascular lesions are still unclear. Longitudinal studies with repeated amyloid and MR imaging in young subjects with high risks of CVD or AD will be helpful to study the evolution in the development and interactions between CVD and amyloid deposition. In clinical practice, aggressive control of risk factors of CVD may likely prevent or delay onset of dementia in elderly subjects harboring amyloid plaques. Clinical trials are needed to confirm such hypothesis.

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Joint HKCPIE/MRCP(UK) Part I examination for the years 2002 – 2014:

	Sitting	Pass
Sep 02	100	33 (33%)
Jan 03	124	55 (44%)
May 03 (SARS Special)	21	7 (33%)
Sep 03	54	29 (54%)
Jan 04	93	39 (42%)
Sep 04	29	16 (55%)
Jan 05	96	68 (70.8%)
Sep 05	24	15 (62.5%)
Jan 06	95	74 (80%)
Sept 06	21	13 (62%)
Jan 07	87	67 (77%)
Sep 07	23	12 (52%)
Jan 08	56	38 (68%)
Sept 08	47	32 (68%)
Jan 09	59	47 (80%)
Sept 09	47	28 (60%)
Jan 10	45	28 (62%)
Sept 10	62	39 (63%)
Jan 11	44	23 (52%)
Sept 11	64	49 (77%)
Jan 12	45	28 (62%)
Sept 12	80	59 (74%)
Jan 13	41	22 (54%)
Sept 13	76	60 (79%)
Jan 14	30	20 (67%)
Sep 14	84	64 (76%)

Joint HKCPIE/MRPC(UK) Part II (Written) examination for the years 2002 –2014:

	Sitting	Pass
2 Jul 2002	53	27 (51%)
13 Nov 2002	50	24 (48%)
13 Aug 2003	110	62 (56%)
10 Dec 2003	54	31 (57%)
28 Jul 2004	65	42 (65%)
8 Dec 2004	46	32 (70%)
13 Apr 2005	32	15 (47%)
27 Jul 2005	76	56 (74%)
7 & 8 Dec 2005	26	16 (62%)
12&13 Apr 2006	29	13 (45%)
26 & 27 Jul 2006	91	68 (75%)
6 & 7 Dec 2006	33	18 (55%)
11 & 12 Apr 2007	34	22 (65%)
25 & 26 Jul 2007	80	70 (88%)
5 & 6 Dec 2007	19	13 (68%)
9 & 10 Apr 2008	21	13 (62%)
30 & 31 Jul 2008	47	36 (77%)
3 & 4 Dec 2008	17	10 (59%)
8 & 9 Apr 2009	32	25 (78%)
29 & 30 Jul 2009	50	43 (86%)
25 & 26 Nov 2009	12	7 (58%)
7 & 8 April 2010	41	34 (83%)
28 & 29 July 2010	25	19 (76%)
24 and 25 Nov 2010	8	2 (25%)
6 and 7 April 2011	45	35 (78%)
23 and 24 Nov 2011	32	25 (78%)
28 and 29 March 2012	55	43 (78%)
12 and 13 December 2012	57	44 (77%)
10 and 11 April 2013	60	52 (87%)
11 and 12 December 2013	48	34 (71%)
9 and 10 April 2014	54	46 (85%)

Pass rates for PACES over the past years:

October 2001	36/72 = 50%
February 2002	34/74 = 46%
October 2002	29/72 = 40%
February 2003	30/69 = 43%
October 2003	27/59 = 46%
March 2004	39/64 = 61%
October 2004	26/69 = 38%
March 2005	35/75 = 47%
October 2005	28/75 = 37%
March 2006	36/75 = 48%
October 2006	16/73 = 22%
March 2007	44/74 = 59%
June 2007	44/74 = 59%
October 2007	36/55 = 65%
March 2008	36/74 = 49%
October 2008	29/65 = 45%
February 2009	39/75 = 52%
October 2009	24/72 = 33%
March 2010	33/75 = 44%
October 2010	40/74 = 54%
February 2011	23/66 = 35%
October 2011	34/70 = 49%
February 2012	32/74 = 43%
October 2012	32/74 = 43%
March 2013	28/75 = 37% (for HK Local candidates)
October 2013	28/74 = 38%
February 2014	29/74 = 39% (for HK Local candidates)
October 2014	21/74 = 28%

Pass list for October PACES 2014

Au Wing Han
Chan Zi
Cheung Yan Ki
Dao Ho Yi
Fok Yuen Tung Vanessa
Huang Hiu Fung
Ip Tsz Hung
Kwok Wing Tung
Kwong Wai Lun
Lam Ching Pong
Luk Ching On Vivi-Anne
Ma Chun Kit
Ma Hei Yee
Ng Hoi Yan Alexandra
Ng King Man Kevin
Ng Sin Hang Stephanie
Tam Yuen Yee Ellen Maria
Tam Tin Chak Aston
Wan Ching Kit
Wong Darren Jat-Lon
Yiu Yuen Fung

MANDATORY SCIENTIFIC MEETINGS

Philip Kam Tao LI , Chairman, Education and Accreditation Committee

At its 280th Meeting of 26 January 2015, the Council decided the requirement for attendance of the 3 mandatory scientific meetings (Annual Scientific Meeting of HKCP, Advances in Medicine and Hong Kong Medical Forum) during Basic Physician Training as shown below:

Mandatory Scientific Meetings

Trainees should attend the mandatory Scientific Meetings during the course of their Basic Physicians Training. The 3 recognised Scientific Meetings are the Annual Scientific Meeting (ASM) of the HKCP, Advances in Medicine (AIM) organized by CUHK and Hong Kong Medical Forum (HKMF) organized by HKU. Trainees are required to attend:

a. *at least one meeting a year and a total of 6 out of these 9 meetings during the 3 years of their training (or calculated on a pro-rata basis if the required training duration as registered with the College is below 3 years. If the training period is more than 3 years, the trainees should attend 2 out of 3 meetings per additional year.)*

b. *at least one HKCP ASM over any 2-year period*

c. *Trainees who have a deficit of 3 or less of such conferences/forums will be allowed to make up for the deficiency during their Advanced Internal Medicine/Geriatric Medicine (as single specialty or broad-based specialty) training. Trainees who have a deficit of more than 3 meetings will be subject to possible penalty at the discretion of the E&AC.*

The above regulation will be applicable to all new basic physician trainees who will be commencing their training from 1 July 2015 onwards.

Statistics on No. of Trainees in all Specialties

Updated in January 2015

SPECIALTY	TRAINEES TOTAL (PP/DH/HA/ OTHERS)	TRAINEES						
		HONG KONG EAST CLUSTER			HONG KONG WEST CLUSTER			
		PYNEH	RH	TWEH	FYKH	GH	QMH	TWH
		YEAR			YEAR			
CARDIOLOGY	17	1—I 1 2 3 4 ***5***	1—I 2 2—I 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***5***	1 3 2—II 3—I 4 ***11***	1 0 2 3 4 ***0***
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3 4—I ***1***	1 0 2 3 4 ***0***
CRITICAL CARE MEDICINE	7	1—I 1 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***7***	1 0 2 3 4 ***0***
DERMATOLOGY & VENEREOLOGY	10	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
ENDOCRINOLOGY, DIABETES & METABOLISM	13	1 1 2 3 4—I ***3***	1—I 1 2 3 4 ***1***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***6***	1 0 2 3 4 ***0***
GASTROENTEROLOGY & HEPATOLOGY	24	1—I 2 2 3—I 4 ***6***	1 1 2 3 4—I ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***8***	1 0 2 3 4 ***0***
GERIATRIC MEDICINE	15	1 1 2—I 3 4 ***6***	1 0 2 3 4 ***11***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***4***	1 0 2 3 4 ***2***	1—I 2 2—I 3 4 ***1***	1 0 2 3 4 ***1***
HAEM/HAEM ONCOLOGY	7	1—I 1 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***7***	1 0 2 3 4 ***0***
IMMUNOLOGY & ALLERGY	0	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INFECTIOUS DISEASE	3	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***
INTERNAL MEDICINE	186	1—VII 15 2—II 3—III 4—III ***38***	1—II 6 2—I 3 4—III ***14***	1 0 2 3 4 ***8***	1 0 2 3 4 ***4***	1 0 2 3 4 ***7***	1—III 23 2—VI 3—V 4—IX ***58***	1 0 2 3 4 ***10***
MEDICAL ONCOLOGY	2	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***2***	1 0 2 3 4 ***0***
NEPHROLOGY	15	1 1 2 3—I 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***6***	1 0 2 3 4 ***3***
NEUROLOGY	17	1 1 2 3—I 4 ***5***	1 0 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***7***	1 0 2 3 4 ***0***
PALLIATIVE MEDICINE	5	1 0 2 3 4 ***0***	1 1 2 3 4—I ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
REHABILITATION	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 1 2 3—I 4 ***3***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***6***
RESPIRATORY MEDICINE	13	1—I 1 2 3 4 ***4***	1 0 2 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***8***	1 1 2—I 3 4 ***5***	1 0 2 3 4 ***0***
RHEUMATOLOGY	9	1—II 3 2—I 3 4 ***3***	1 0 2 3 4 ***1***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***5***	1 0 2 3 4 ***1***

		TRAINEES										
		KOWLOON CENTRAL CLUSTR		KOWLOON EAST CLUSTER			KOWLOON WEST CLUSTER					
SPECIALTY	TRAINEES TOTAL (PP/ DH/HA/ OTHERS)	KH	QEH	HOHH	TKOH	UCH	CMC	KWH	OLMH	PMH	WTSH	YCH
		YEAR		YEAR			YEAR					
CARDIOLOGY	17	1 0 2 3 4 ***0***	1 1 2 3—I 4 *10*	1 0 2 3 4 ***0***	1 2 2 3—I 4—I ***2***	1 0 2 3 4 ***6***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***3***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***8***	1 0 2 3 4 ***0***	1 1 2 3 4—I ***3***
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
CRITICAL CARE MEDICINE	7	1 0 2 3 4 ***0***	1 0 2 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 1 2 3—I 4 ***5***	1 0 2 3 4 ***2***	1 1 2—I 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
DERMATOLOGY & VENEREOLOGY	10	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
ENDOCRINOLOGY, DIABETES & METABOLISM	13	1—I 1 2 3 4 ***0***	1 2 2—II 3 4 ***8***	1 0 2 3 4 ***0***	1 0 2 3 4 ***4***	1 1 2—I 3 4 ***2***	1 1 2 3 4—I ***2***	1—I 2 2—I 3 4 ***4***	1—I 1 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***
GASTROENTEROLOGY & HEPATOLOGY	24	1 0 2 3 4 ***0***	1 5 2—III 3—II 4 ***5***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***3***	1 3 2—II 3 4—I ***3***	1 1 2 3—I 4 ***3***	1 1 2—I 3 4 ***6***	1 0 2 3 4 ***0***	1—I 2 2—I 3 4 ***5***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***5***
GERIATRIC MEDICINE	15	1 0 2 3 4 ***4***	1 1 2 3—I 4 ***3***	1 1 2 3—I 4 ***3***	1 1 2 3—I 4 ***2***	1 1 2 3 4—I ***8***	1 0 2 3 4 ***7***	1 0 2 3 4 ***8***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***13***	1 0 2 3 4 ***4***	1 2 2 3—I 4—I ***5***
HAEM/HAEM ONCOLOGY	7	1—I 1 2 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
IMMUNOLOGY & ALLERGY	0	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INFECTIOUS DISEASE	3	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INTERNAL MEDICINE	186	1—III 3 2 3 4 ***4***	1-II *22* 2-VIII 3-VI 4-VI *54*	1—I 3 2—II 3 4 ***3***	1 8 2—III 3—II 4—III *22*	1 *10* 2—III 3—IV 4—III *41*	1 ***3*** 2 3—II 4—I *22*	1—III *8* 2—III 3—I 4—I *41*	1 0 2 3 4 ***3***	1—V *17* 2—IV 3—V 4—III *50*	1 0 2 3 4 ***2***	1—III 8 2—I 3—I 4—III *23*
MEDICAL ONCOLOGY	2	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
NEPHROLOGY	15	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***4***	1 1 2 3—I 4 ***2***	1—I 1 2 3 4 ***8***	1 0 2 3 4 ***0***	1—I 3 2—I 3—I 4 ***5***	1 0 2 3 4 ***0***	1—II 2 2 3 4 ***2***
NEUROLOGY	17	1 0 2 3 4 ***0***	1—I 3 2—I 3—I 4 ***4***	1 0 2 3 4 ***0***	1 2 2—I 3 4—I ***2***	1 2 2 3—II 4 ***4***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1—I 2 2—I 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***
PALLIATIVE MEDICINE	5	1—I 1 2 3 4 ***0***	1 0 2 3 4 ***0***	1 2 2—I 3 4—I ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
REHABILITATION	1	1 0 2 3 4 ***6***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***
RESPIRATORY MEDICINE	13	1 0 2 3 4 ***5***	1 0 2 3 4 ***7***	1—I 2 2—I 3 4 ***4***	1 2 2—I 3—I 4 ***3***	1 0 2 3 4 ***4***	1 0 2 3 4 ***2***	1 0 2 3 4 ***3***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***5***	1 0 2 3 4 ***2***	1 0 2 3 4 ***1***
RHEUMATOLOGY	9	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***3***	1 0 2 3 4 ***2***	1—I 1 2 3 4 ***2***	1 0 2 3 4 ***0***	1 2 2 3—I 4—I ***3***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***2***

		TRAINEES						
		NEW TERRITORIES EAST CLUSTER					NEW TERRITORIES WEST CLUSTER	
SPECIALTY	TRAINEES TOTAL (PP/DH/HA/ OTHERS)	AHNH	NDH	PWH	SH	TPH	POH	TMH
		YEAR					YEAR	
CARDIOLOGY	17	1 0 2 3 4 ***2***	1 1 2—I 3 4 ***4***	1 1 2 3—I 4 ***8***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1—I 2 2—I 3 4 ***5***
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
CRITICAL CARE MEDICINE	7	1 1 2 3 4—I ***2***	1 0 2 3 4 ***2***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***2***
DERMATOLOGY & VENEREOLOGY	10	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
ENDOCRINOLOGY, DIABETES & METABOLISM	13	1 1 2 3—I 4 ***1***	1 0 2 3 4 ***3***	1 1 2 3—I 4 ***8***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***3***
GASTROENTEROLOGY & HEPATOLOGY	24	1 0 2 3 4 ***1***	1—I 2 2 3 4—I ***4***	1—I 2 2—I 3 4 ***5***	1 0 2 3 4 ***0***	1—I 2 2—I 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***6***
GERIATRIC MEDICINE	15	1 0 2 3 4 ***2***	1 0 2 3 4 ***2***	1 0 2 3 4 ***4***	1 0 2 3 4 ***6***	1—I 1 2 3 4 ***3***	1 0 2 3 4 ***2***	1—I 3 2 3 4—II ***6***
HAEM/HAEM ONCOLOGY	7	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***2***
IMMUNOLOGY & ALLERGY	0	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INFECTIOUS DISEASE	3	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1—II 2 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***
INTERNAL MEDICINE	186	1 5 2 3—II 4—III ***12***	1—I 5 2—I 3—I 4—II ***19***	1—VII 20 2—V 3—III 4—V ***54***	1 4 2—II 3 4—II ***6***	1—II 6 2—II 3 4—II ***6***	1 0 2 3 4 ***12***	1—VII 18 2—I 3—V 4—V ***41***
MEDICAL ONCOLOGY	2	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***14***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
NEPHROLOGY	15	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 3 2 3—II 4—I ***6***
NEUROLOGY	17	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***1***	1 1 2—I 3 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—II 3 2 3—I 4 ***3***
PALLIATIVE MEDICINE	5	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
REHABILITATION	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***1***	1 0 2 3 4 ***2***
RESPIRATORY MEDICINE	13	1 0 2 3 4 ***2***	1 0 2 3 4 ***4***	1—I 1 2 3 4 ***6***	1 1 2—I 3 4 ***0***	1 1 2—I 3 4 ***1***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***5***
RHEUMATOLOGY	9	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***2***

** Total No. of trainees is shown in upper right corner of each hospital*
*** No. of trainers is shown in italics & bold in lower right corner of each hospital*

SPECIALTY	TRAINEES TOTAL (PP/DH/HA/OTHERS)	TRAINEES	
		DH	
DERMATOLOGY & VENEREOLOGY	10	1—I 2—II 3—II 4—III	8 ***10***
INFECTIOUS DISEASE	3	1 2 3 4	0 ***2***
RESPIRATORY MEDICINE	13	1 2 3 4	0 ***9***

** Total No. of trainees is shown in upper right corner of each hospital*
*** No. of trainers is shown in italics & bold in lower right corner of each hospital*

Statistics on No. of Fellows in all Specialties
Updated in January 2015

		FELLOWS									
		HONG KONG EAST CLUSTER				HONG KONG WEST CLUSTER					HONG KONG EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/OTHERS)	PYNEH	RH	TWEH	Subtotal	FYKH	GH	QMH	TWH	Subtotal	
CARDIOLOGY	246	9	7	0	**16**	0	6	18	0	**24**	**41**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	**0**	0	0	2	0	**2**	**2**
CRITICAL CARE MEDICINE	90	12	2	0	**14**	0	0	11	0	**11**	**25**
DERMATOLOGY & VENEREOLOGY	101	0	0	0	**0**	0	0	1	0	**1**	**1**
ENDOCRINOLOGY, DIABETES & METABOLISM	107	5	2	3	**10**	0	0	10	1	**11**	**21**
GASTROENTEROLOGY & HEPATOLOGY	180	5	1	2	**8**	0	0	13	0	**13**	**21**
GERIATRIC MEDICINE	184	6	13	2	**21**	5	2	3	2	**12**	**33**
HAEM/HAEM ONCOLOGY	59	4	0	0	**4**	0	0	11	0	**11**	**15**
IMMUNOLOGY & ALLERGY	6	0	0	0	**0**	0	0	0	0	**0**	**0**
INFECTIOUS DISEASE	41	3	0	0	**3**	0	0	2	0	**2**	**5**
INTERNAL MEDICINE	1294	58	25	11	**94**	4	14	98	12	**128**	**222**
MEDICAL ONCOLOGY	47	0	0	0	**0**	0	0	9	0	**9**	**9**
NEPHROLOGY	129	7	0	0	**7**	0	0	9	3	**12**	**19**
NEUROLOGY	112	5	3	0	**8**	0	0	8	2	**10**	**18**
PALLIATIVE MEDICINE	26	0	2	0	**2**	0	2	2	0	**4**	**6**
REHABILITATION	54	0	3	3	**6**	1	0	1	6	**8**	**14**
RESPIRATORY MEDICINE	183	11	6	1	**18**	0	9	11	0	**20**	**38**
RHEUMATOLOGY	75	4	2	1	**7**	0	0	9	1	**10**	**17**

		FELLOWS															
		KOWLOON CENTRAL CLUSTER				KOWLOON EAST CLUSTER				KOWLOON WEST CLUSTER							KOWLOON CENTRAL + EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/OTHERS)	BH	KH	QEH	Subtotal	HOHH	TKOH	UCH	Subtotal	CMC	KWH	OLMH	PMH	WTSH	YCH	Subtotal	
CARDIOLOGY	246	0	0	16	**16**	0	3	7	**10**	2	7	1	10	0	6	**26**	**52**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
CRITICAL CARE MEDICINE	90	0	0	7	**7**	0	4	6	**10**	4	4	0	7	0	1	**16**	**33**
DERMATOLOGY & VENEREOLOGY	101	0	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
ENDOCRINOLOGY, DIABETES & METABOLISM	107	0	0	8	**8**	0	5	4	**9**	2	5	2	5	0	2	**16**	**33**
GASTROENTEROLOGY & HEPATOLOGY	180	0	0	6	**6**	0	4	4	**8**	5	10	1	9	0	8	**33**	**47**
GERIATRIC MEDICINE	185	1	6	4	**11**	3	2	13	**18**	9	11	2	14	6	5	**47**	**76**
HAEM/HAEM ONCOLOGY	59	0	0	6	**6**	0	2	2	**4**	0	0	0	6	0	0	**6**	**16**
IMMUNOLOGY & ALLERGY	6	0	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
INFECTIOUS DISEASE	41	0	0	7	**7**	0	0	1	**1**	0	0	0	5	0	0	**5**	**13**
INTERNAL MEDICINE	1296	2	11	79	**92**	7	29	52	**88**	32	50	8	71	10	26	**197**	**377**
MEDICAL ONCOLOGY	47	0	0	3	**3**	0	0	2	**2**	0	1	0	1	0	0	**2**	**7**
NEPHROLOGY	129	0	0	9	**9**	1	2	6	**9**	2	8	0	9	0	2	**21**	**39**
NEUROLOGY	112	0	3	9	**12**	0	2	4	**6**	1	5	1	4	1	2	**14**	**32**
PALLIATIVE MEDICINE	27	1	0	0	**1**	4	0	2	**6**	4	0	1	0	1	0	**6**	**13**
REHABILITATION	54	0	7	1	**8**	1	0	4	**5**	1	0	1	2	4	0	**8**	**21**
RESPIRATORY MEDICINE	184	1	6	7	**14**	6	4	8	**18**	5	3	0	5	7	1	**21**	**53**
RHEUMATOLOGY	75	0	2	5	**7**	0	2	3	**5**	3	4	0	3	0	2	**12**	**24**

		FELLOWS									
		NEW TERRITORIES EAST CLUSTER						NEW TERRITORIES WEST CLUSTER			NEW TERRITORIES EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/OTHERS)	AHNH	NDH	PWH	SH	TPH	Subtotal	POH	TMH	Subtotal	
CARDIOLOGY	246	5	4	14	1	1	**25**	3	11	**14**	**39**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	5	0	0	**5**	0	0	**0**	**5**
CRITICAL CARE MEDICINE	90	4	5	2	0	0	**11**	0	6	**6**	**17**
DERMATOLOGY & VENEREOLOGY	101	0	0	3	0	0	**3**	0	0	**0**	**3**
ENDOCRINOLOGY, DIABETES & METABOLISM	107	1	5	16	1	0	**23**	0	4	**4**	**27**
GASTROENTEROLOGY & HEPATOLOGY	180	2	4	12	0	0	**18**	1	14	**15**	**33**
GERIATRIC MEDICINE	184	2	2	6	9	3	**22**	4	11	**15**	**37**
HAEM/HAEM ONCOLOGY	59	0	0	5	0	0	**5**	0	6	**6**	**11**
IMMUNOLOGY & ALLERGY	6	0	0	0	0	0	**0**	0	0	**0**	**0**
INFECTIOUS DISEASE	41	3	1	3	0	0	**7**	0	2	**2**	**9**
INTERNAL MEDICINE	1294	24	26	87	11	8	**156**	16	72	**88**	**244**
MEDICAL ONCOLOGY	47	0	0	18	0	0	**18**	0	0	**0**	**18**
NEPHROLOGY	129	5	1	10	0	0	**16**	2	6	8	**24**
NEUROLOGY	112	2	1	12	1	0	**16**	2	4	**6**	**22**
PALLIATIVE MEDICINE	26	0	0	0	2	0	**2**	0	1	**1**	**3**
REHABILITATION	54	0	1	2	1	1	**5**	1	3	**4**	**9**
RESPIRATORY MEDICINE	183	4	7	7	0	3	**21**	2	9	**11**	**32**
RHEUMATOLOGY	75	3	0	5	0	3	**10**	1	3	**4**	**14**

CHIN HIN CHEW

PPA(E), MB, FAMS, FRCP (Edin, Lond, Glasg)
FRACP, MACP, Hon FHKCP, Hon FCPS

John MACKAY

Dr Chew's relationship with Hong Kong goes back a long way, ever since he arrived in Hong Kong in 1949 from Singapore to begin his medical training at Hong Kong University.

Chew Chin Hin was born in 1931 in Singapore. His father Benjamin was born in Malacca and came to Singapore with his family at the age of ten, graduated in 1929 from Singapore's King Edward VII College of Medicine, and in 1931 was on the staff at the Singapore General Hospital. Chin Hin had two brothers and a sister, none of whom became doctors.

His most vivid childhood memory was imprinted at the age of ten when the first Japanese bombs dropped on Singapore in December 1941: Chin Hin remembers bodies lying in the grounds of the Singapore General Hospital, placed there because the mortuary was overflowing with casualties from the near-by Chinatown. There were bodies in the grounds of the hospital again, just before Singapore surrendered, when shells killed eleven medical students.

After the Japanese took control of the island they occupied the Singapore General Hospital, so Chin Hin and his family were moved with the whole staff of the hospital and 500 patients to Yio Chu Kang, now the old Woodbridge Hospital, and moved again nine months later to the Tan Tock Seng Hospital, (TTSH). TTSH and Kandang Kerbau Hospital (KKH) became the main general hospitals for the civilians. Conditions were grim with tuberculosis, dysentery, malaria and other diseases common, and only scarce medical supplies available. He himself had several bouts of malaria. Chin Hin's father was the first Singapore physician to administer penicillin in 1945, giving it to save the life of a colleague suffering from pneumonia.

CHC decided at an early age to become a doctor, having seen the selfless service and compassion of his father and other doctors who coped with the cruel effects of war.

The same spirit that enabled the staff at TTSH to cope with the casualties of war was called upon again in 2003 when the hospital bore the brunt of treating patients of the SARS outbreak.

His schooling was at the Victoria School, and the Anglo-Chinese School where he excelled in sports, representing the school in hockey and cricket. Because of his disrupted war-time schooling Chin Hin had to work hard at school, completing the syllabus for the Cambridge school-leaving exams in one and a half years instead of three and a half.

In 1949 he applied for a place at the medical schools in Singapore and Hong Kong. Hong Kong University accepted him straight away whereas Singapore University put his application on a waiting list because at that time its student intake covered Malaya as well. To get to Hong Kong in time for the start of term he was obliged to take the only available cabin in the next ship, First Class on the P&O liner Corfu. He had only just settled in Hong Kong when the medical school in Singapore offered him a place – too late. Professor A J S McFadzean had been appointed the year before as Head of Department and Professor of Medicine in Hong Kong.

Singapore's Medical School had been founded by the British Colonial Government in 1905. There had been a policy before the war of not giving specialty training to local doctors, so from 1942 to 1945 during the Japanese occupation, education practically ceased because all the expatriate doctors had left or been interned. The remaining doctors had to do their best to educate themselves in specialty medicine. Likewise the Medical School in Hong Kong had closed during the Japanese occupation.

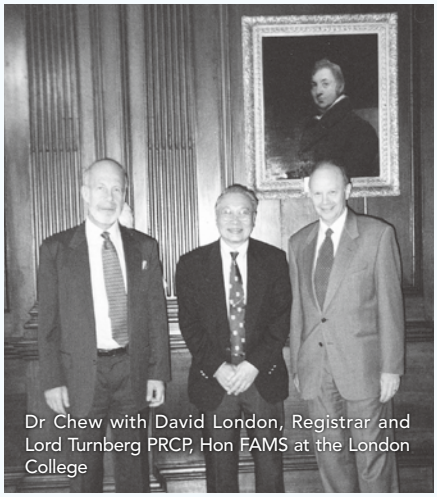
He spent six busy and happy years at university, staying first at Lugard Hall for three years then Morrison Hall. He was a keen sportsman so played hockey and cricket and integrated well with other students, many from Malaya and Singapore. He integrated particularly well with another medical student from Hong Kong, Anna Hui who became his wife in 1956.

After qualification in 1955 Dr Chin Hin Chew worked for six months as a preregistration Medical Officer in surgery at the Queen Mary Hospital, then moved back to Singapore General Hospital as a House Physician under Professor Gordon Arthur Ransome, later to be knighted for his contribution to Medicine in Singapore.

Starting in 1957 he spent two years at the Tan Tock Seng Hospital (founded 1844). His father had by now moved into private medical practice.

The next fifteen months were spent in Britain. He completed the Internal Medicine Course in Edinburgh, the Chest Diploma in Cardiff, the Clinical Medicine Course back in Edinburgh, and passed the Membership Examinations at Glasgow, FRFPS and Edinburgh, MRCPE. From this time onwards he enjoyed an enduring relationship with Professor Sir John Crofton and shared his interest in the study of tuberculosis. Professor Sir Derrick Dunlop was another well-remembered tutor and friend.

Coming back to Singapore he rejoined the TTSH under Head of Medicine Dr Seang Aun Yeoh; while his wife was appointed Medical Officer in Charge of the Kallang Outpatient Clinic. They settled into a house in 15 Akyab Road where they were to spend the next 31 years until he retired from Government service at 60. He and his wife raised four children, one of whom qualified with a Double First degree from Stirling University and now lives in Toronto; a daughter became a medical administrator then studied for two years at Cambridge University before being ordained as a minister of the Church of England. There are now two grandchildren.



Dr Chew with David London, Registrar and Lord Turnberg PRCP, Hon FAMS at the London College


Gerald Choa Memorial Lecture 2002

By 1963 he was a consultant in the department of medicine at the TTSH, beginning a long career in medical education, becoming Chief Physician from 1965 to 1979. He was a member of the Singapore Medical Council from 1972 to 1993. In 1979 he became Medical Director of the TTSH, and two years later Deputy Director of Medical Services for the Ministry of Health, a post he held till his retirement in 1991. At the Ministry he initiated Rheumatology and Geriatric Medicine as separate disciplines.

Dr Chew's research over many years concerning tuberculosis at TTSH, which was the main tuberculosis treatment centre in Singapore, lead to a Research Committee which in 1966 was elevated to a Ministry of Health Committee and carried out joint research with the British Medical Research Council led by Wallace Fox a World Health Organisation (WHO) consultant. He has published over sixty papers on TB and other topics.

In 1986 as Chairman of this committee Dr Chew presided over the World Conference of TB and Lung Disease held in Singapore. At this meeting his committee presented several landmark papers on short course regimes of fully supervised treatment, now recognized by WHO as Directly Observed Treatment (DOT). Thirty years later the DOT regime remains the standard protocol.

Research was just one of Dr Chew's main interests. Medical education was also a passion. In 1963 he was invited to become a Member of the Singapore Academy of Medicine (founded 1957), became a Master of the Academy in 1973, and from 1996 to 2007 was a member of the Specialist Accreditation Board. Dr Chew was appointed Hon. Post Graduate Advisor to the Postgraduate Medical School, and Emeritus Consultant at TTSH. In 2004 the Singapore College of Physicians was founded and one year later Dr Chew was proud to receive the first Honorary Fellowship. The President of Singapore, Devan Nair, bestowed on him the Public Administration Gold Medal in 1983.

In recognition of his work in raising the standards of medical education in Singapore he has received many international awards; the Gold Medal for Emeritus Advisor of the RCPE, the Australasian College of Physicians Medal, the Mastership of the American College of Physicians (MACP) in 2010, the only Singaporean so honoured; and the Honorary Fellowship of the College of Physicians of Hong Kong. This last honour must be an especially warm memory because it was presented to him by one of his many friends from Morrison Hall days, Professor Richard Yu. He is the only Singaporean to be elected to the Association of Physicians of Great Britain and Ireland.

Medical ethics was another interest; he was the first Chairman of the National Ethics Committee from 1994 till 2000, during this time he initiated the Advanced Medical Directive allowing people to state they do not want extraordinary life-sustaining measures if they become terminally ill. In 2002 he chose as his subject 'Ethical Medicine' when he gave the Inaugural Gerald Choa Memorial Lecture at the Annual General Meeting of the Hong Kong College of Physicians.

In this lecture he gave two memorable quotations: Sir William Osler wrote,"The practice of medicine is an art, not a trade; a calling, not a business; a calling in which your heart will be exercised equally with your head". And the prayer of a physician enunciated by Moses Maimonides: "Endow me with strength of heart and mind so that both may be ready to serve rich or poor, the good and wicked, friend and enemy. And may I never see in the patient anything else but a fellow creature in pain".

In his own words Dr Chew stated, "Central to the delivery of health care is the physician-patient relationship and the principles that govern this. These include beneficence, honesty, confidentiality and trust. Our first responsibility will always be to our patients. The primary goals are therefore to treat and cure where possible, to help the patient cope with illness, disability and death; and to bring relief to suffering; in all instances the dignity of patients must be upheld."

Prof. Chin Hin Chew returned to Hong Kong in October this year to attend the Hong Kong College of Physicians Annual Meeting and renew his many old acquaintances. It was also in respect of his first Professor of Medicine, Alex McFadzean, whose centenary was being commemorated. He is still active as Honorary Postgraduate Advisor and Adjunct Professor, Division of Graduate Medical Studies, National University of Singapore; and also holds the post of emeritus Consultant, Tan Tock Seng Hospital. The Minister for Health Gan Kim Yong in tribute to Singapore health pioneers said "Professor Chew has always been passionate about medical education…as Master of the Academy of Medicine, he was instrumental in the development of local Postgraduate qualifications. He demanded excellence and insisted that these be as robust as those in the UK and Australasia."

In his leisure time he enjoys playing golf and watching the sports at which he was once proficient; hobbies included chess and still include photography and stamp collecting.

Looking back he recalls with humility and satisfaction the role he played in tuberculosis control, in the establishment of specialist geriatrics, and recalls with joy the successful careers of the students and young doctors that he taught. Professor Chin Hin Chew was keen to pass on his advice that to teach and educate others is a lifelong process and the best way to enhance your own learning. Listening to his eloquent and kindly conversation it is easy to imagine the pleasure with which his students enjoyed his ward rounds.


AGM 2002