HONG KONG COLLEGE OF PHYSICIANS
香港內科醫學院
Sapientia et Humanitas

HONG KONG COLLEGE OF PHYSICIANS

SYNAPSE

RESTRICTED TO MEMBERS ONLY

SEPTEMBER 2017

Professor Sir David Todd
(1928 - 2017)

CONTENTS

THIS ISSUE

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COLLEGE'S ROLE IN ENHANCING PROFESSIONALISM

Dear all Fellows and Members,

It is with extreme sadness that the College mourns the loss of our dearest Professor Sir David Todd, our Founding President. The In Memoriam for Sir David is presented in this issue of Synapse.

It is now 10 months since I took over as the President of the College.

There have been a lot of things happening in the College. I would use **"Enhancing Professionalism"** to sum up all that the College has done.

A College Retreat held on 4th March 2017 has discussed the important areas related to the College development: **"Workforce of Physicians – Manpower (Attraction & Retention)", "Workforce of Physicians – Training (Trainees and Fellows)", "College Affairs – Public Image and Advocacy on public health issues" and "College Affairs – Enhancement of academic activities".** Our College feels that addressing workload and manpower provision are important elements in enhancing training and professionalism in our physician field.

In the recent summer flu season, the stress on the public system in the medical wards has been magnified. This has not only brought about a huge challenge and workload to the public healthcare system, but also exposed an enormous gap and inadequacy in the existing service provision in Internal Medicine for the local population. In fact, the inadequacy has not been an isolated event due to the "summer surge". The bed occupancy of Medical wards in most public hospitals has constantly exceeded 100% in the past years, and the limit has not infrequently been stretched to over 130% in certain hospitals during the peak seasons.

Apart from a stunning growth of inpatient medical bed days of 23% from 2011 to 2016, we have also witnessed a soar of 20% and 34% of Specialist Medical Outpatient Clinic (SOPC) patient headcounts and day-patient attendances respectively during that 5-year period. A glimpse of the subspecialty medical service demand can also be obtained from the dramatic rise of 27% and 33% in the number of haemodialysis sessions and percutaneous coronary interventions (PCI) being carried out respectively in the recent 5 years. In fact, such figures have not completely reflected the actual demand in medical service, since the increasing complexity of medical inpatients and the need to cater for escalating consultations from non-medical patients with medical co-morbidities would not have been reflected in such figures. Unfortunately, such an increase in workload has not been tackled with a parallel injection of medical manpower in physicians from Hospital Authority (HA) (only 15% increase in these 5 years), with an estimated deficit of at least 100 doctors in the public medical workforce in physicians at present.

This has actually caused problems on two areas: training and continuous medical education. In contrast to some other non-physician disciplines, HA physicians do not possess "protected time" for training and teaching. Apart from substantially affecting the career and morale of physicians, this would also potentially affect the quality of service provision towards our patients and materially affect the attractiveness of Internal Medicine to be a career pathway of choice for medical graduates.

Because of this major increased workload to the physicians in the recent months, I, on behalf of the College, have voiced out in the **media briefing** to the public about the stress to the physicians. This was widely reported in the media in August 2017 about the

challenges faced by the physicians on the time spent on each patient, the training time and the potential impact on the quality of care to the patients, both in the acute and chronic as well as inpatient and ambulatory settings.

We have written **a letter to the Secretary for Food and Health Bureau** to elaborate all the above issues. HKCP proposes to the Government to increase resources into the public system to employ more doctors in the public sector. With the increase of medical graduates from the local Universities, our College would urge our Government and HA to prioritize the replenishment of the observed gap in the physician manpower of Departments of Medicine in HA. Experienced and senior physicians are required for the provision of service and training of the junior ones highlighting the importance in retaining them. Lastly, as the professional body in providing, developing, monitoring and assessment of Post-graduate Internal Medicine training in Hong Kong, we would propose having protected training time for HA doctors in Internal Medicine in order to strengthen the quality of training and service provision for our patients.

Another area our College has been working on is the **development of clinical practice guidelines**. Currently we have collaborated with the Central Renal Committee of Hospital Authority on drafting the Clinical Practice Guidelines for renal service in Hong Kong. This was also supported by the Hong Kong Society of Nephrology. A Forum was held on 2 July 2017. A report on this is published in this issue of Synapse.

Our College has set up the **Credentialing Committee** and the Committee has already started to work. The objectives of the Committee are : to identify and prioritize clinical procedures or practice for credentialing, based on considerations of risk level, novelty, volume, and other factors; to propose the procedure and requirements for the credentialing of individuals with regard to specific clinical procedures for endorsement by the Hong Kong College of Physicians. The required standards for credentialing of a clinical procedure are applicable to all irrespective of the organization an individual is practising in, and the standards need to be fair, objective and reliable. The standards should be decided based on the level of competence deemed necessary to ensure patient safety and upholding of professionalism; and to advise and propose to the College Council training opportunities and modes of assessment in line with the credentialing needs.

The enhancement of professionalism cannot be materialized without the injection of new blood with enthusiasm and passion for the practice as a physician. The following two programmes are initiated by the Young Fellows' Committee (YFC) with endorsement from the College Council. We have organized two very successful **Hong Kong College of Physicians Career Talk for Medical Graduates** on 11th June 2017 in Prince of Wales Hospital and on 13th August 2017 in Queen Mary Hospital respectively. Both are very well attended and a report is also published in this Issue of Synapse. Our College does feel the appropriate briefing to young graduates early will attract them to the profession. The early exposure to the wide range of opportunities in the field of physicians would be very beneficial for our medical students to be interested in the career as physicians. The final arrangement of the **HKCP Scholarship for Medical Students** has been worked out. One of the main objectives is to support and encourage medical students in Hong Kong to undertake clinical or laboratory attachment/research pertaining to the practice and advancement of internal medicine at overseas institutions. We will support up to 4 medical students each from the 2 Medical Schools in Hong Kong for such attachment each year. The Deans of the 2 Medical Schools have responded to our College on this Scholarship with great enthusiasm.

Our College has the mandate in enhancing professionalism of Physicians, working through different programmes and voicing out in the appropriate platforms. Once again, we would like to hear your suggestions and comments to us. Please feel free to contact myself and any of the Council members to reflect your views.

Your support is vital for the College to do more for Medicine in Hong Kong.

Best wishes,

Prof Philip K. T. Li
President
Hong Kong College of Physicians

NEPHROLOGY IN THE PRECISION MEDICINE ERA

Patrick H. Maxwell
Regius Professor of Physic and Head of the School of Clinical Medicine, University of Cambridge

(This article is based on the Richard Yu Lecture delivered at the Hong Kong Medical Forum, May 2016)

"Precision Medicine" is commonly used to describe the application of genomic analyses to make a specific molecular diagnosis and then identify effective treatment based on understanding the disease mechanism. This is changing some areas of medicine very rapidly, with longstanding diagnostic categories being subdivided into specific new diseases[1]. For example, a number of cancers can now be subdivided on the basis of key molecular drivers[2].

It is a special honour as a nephrologist to give this lecture named in honour of Richard Yu, an internationally leading renal physician who played a major role in developing renal services in Hong Kong. The evolution of nephrology illustrates that important aspects of "precision medicine" were achievable before the development of modern genomic approaches. A key figure in nephrology was the pioneering English physician Dr Richard Bright (1789-1858), who worked at Guy's Hospital in London, where I undertook some of my postgraduate training in the 1980's. In 1827 Bright described a series of patients in whom the presence of albumen in the urine was associated with kidney disease[3]. Through conducting autopsies on patients with kidney disease he concluded that there were likely to be at least three kinds of kidney disease. Following this, a wide range of developments contributed to the fact that nephrology developed precision approaches before the genomic era. Two of the most important are renal imaging and the renal biopsy. Reliable renal imaging, initially using intravenous urography and now most frequently using ultrasound, can determine renal size accurately, identify the presence of cysts or scars, and diagnose renal obstruction. If the kidneys are not reduced in size then renal disease is often treatable, and renal biopsy will usually achieve a rapid and precise diagnosis, give useful prognostic information, and will guide treatment. Percutaneous renal biopsy entered mainstream use across the world in the 1960's[4]. This was accompanied by the development of immunofluorescent and electron microscopic analysis, leading to a systematic framework for categorising kidney diseases.

A rewarding aspect of nephrology is that some acute inflammatory

processes that previously caused complete renal failure are now treatable with immunosuppression. Good examples are "Goodpasture's disease" in which renal damage is caused by autoantibodies to type IV collagen in the glomerular basement membrane (anti GBM disease), vasculitides associated with antibodies to neutrophil cytoplasmic antibodies (ANCA) and systemic lupus erythematosus (SLE). Of course immunosuppression carries substantial risks, and a major aim over recent years has been to tailor immunosuppressive regimes; seeking maximum efficacy while minimising undesirable consequences. Increasingly, we are moving towards a precision medicine approach to these inflammatory conditions. In some instances, this has involved identifying new genetically determined conditions; an example of this is *CFHR5* nephropathy which I will describe below. In others, multinational collaborative efforts have refined our approach to treatment such as the European Vasculitis Society studies led by my colleague Dr David Jayne from Cambridge[5].

Two other important components of modern nephrology relate to the ability to provide long-term supportive therapy for patients whose kidney function has been lost irretrievably; dialysis and transplantation. These replacement therapies are incredibly effective, and mean that it is almost always possible to live a near normal life in the face of the loss of this critical organ system. I would argue strongly that renal transplantation provides another good example of precision approaches. Steadily improving approaches to organ matching have resulted in improved graft survival, and immunosuppressive regimens have become more effective and less likely to cause harm. Nevertheless, the financial costs are enormous. And although renal replacement therapy is life-sustaining, it is associated with substantially increased morbidity and mortality compared to normal health. Illustrating the scale of the problem, the lifetime risk of end stage renal disease is about 2.1% for Caucasians in the USA and is higher in most other ethnic groups. Renal replacement costs Medicare in the USA approximately $31bn per annum, accounting for 7.1% of paid claims costs in the fee for service system(6). Moreover, chronic kidney diseases stage 3 (GFR < 30 ml/min) to 5 (end stage renal disease requiring renal replacement therapy) are associated with a 3 to 13-fold increase in risk of death compared to controls[6, 7]. Importantly, renal failure becomes much more common as people grow older. In most countries outside the USA, many older people die from renal failure whose lives could be prolonged by renal replacement therapy. This raises major ethical, societal and financial questions. Clearly there would be major benefits if we could prevent renal damage in the early stages of kidney disease, if we could promote regeneration of damaged renal tissue and if we could address the limited availability of well-matched kidneys for transplantation, since transplantation offers substantial advantages over dialysis.

Interestingly, genetics and genomics has had a relatively limited impact in nephrology compared to many other specialties. In my view, there are two important reasons for this. First, the great majority of patients with acute

or chronic renal conditions do not have a family history of renal disease and as yet genetic insights are not relevant to most patients with kidney disease. The second issue is that by far the commonest genetic condition causing significant renal pathology, Autosomal Dominant Polycystic Kidney Disease (ADPKD), has been an interesting case study in the challenges of linking an understanding of the genetic cause of a disease to improvements in patient care.

With the exception of ADPKD, monogenic conditions causing renal disease are rare. However, they are important for several reasons. First, individuals value a precise diagnosis. Second, it may influence reproductive choices, permitting preimplantation or *in utero* genetic diagnosis and avoiding passing on the condition to future generations. Third, it will quite often mean that inappropriate treatments – such as immunosuppression – can be avoided. Fourth, it may enable specific treatments. Finally, these rare experiments of nature have provided extraordinary insights into key aspects of physiology and homeostasis. Thus Mendelian conditions have dramatically changed our understanding of how the normal kidney functions. Excellent examples include conditions in which the filtration barrier does not function properly because of a defect in a key structural protein such as podocin, nephrin or type IV collagen[8]. Here I will discuss three genetic conditions which I have worked on – von-Hippel Lindau (VHL) disease, CFHR5 nephropathy and ADPKD – which illustrate how genetic discoveries are leading to progressively more precise treatments in nephrology.

VHL disease is a rare autosomal dominant condition affecting about 1:36,000 individuals at birth and is due to inheriting a mutation in the *VHL* gene[9]. VHL disease is associated with an ~70% lifetime risk of clear cell renal cell carcinoma. Individuals are also at risk for haemangioblastomas in the retina, cerebellum, and spinal cord, and phaeochromocytoma. At a cellular level it is recessive, with the clinical manifestations involving a somatic "second hit" disabling the function of the remaining normal *VHL* gene. Importantly, the great majority of sporadic cases of clear cell renal cell carcinoma (which is the commonest kind of kidney cancer) are also driven by bialleleic inactivation of the *VHL* gene, so this an excellent example of Knudsen's two-hit model of tumour suppressor gene function[10]. From a clinical perspective, isolating the gene had immediate implications for clinical care. This is a small gene of only 3 exons, so analysis is straightforward. In individuals who inherit the defective gene annual screening prevents blindness and death from phaeochromocytoma or metastatic renal cancer. Diagnosis also allows couples to avoid passing the defective gene on to their offspring through pre-implantation genetic diagnosis. Clinical studies of patients with VHL disease have established that small renal masses (<3 cm) can be managed through active surveillance, and that "nephron-sparing" surgery in which a tumour is removed while preserving the rest of the kidney is a safe approach. This means that rather than removing both kidneys as a preventative measure, we can preserve renal function and avoid or postpone renal replacement therapy.

Importantly, these developments based on rare patients with germline *VHL* mutations have influenced the management of sporadic renal tumours.

The VHL protein is centrally involved in how cells sense oxygen. It acts as the recognition component of an ubiquitin E3 ligase that controls a master regulator of gene expression, Hypoxia-Inducible Factor[11, 12]. Cells with a defect in VHL function have constitutively active HIF, which drives expression of a broad range of genes including those encoding angiogenic factors, explaining why the tumors in VHL disease are so vascular. These insights have contributed to the development of strategies to treat VHL defective tumors, including sporadic clear cell renal cell carcinoma, with agents that reduce the action of angiogenic growth factors or interfere with other aspects of HIF signalling[13]. Furthermore, the role of VHL in oxygen sensing led us to identify the PHD enzymes that act as "oxygen sensors"[14]. Rather excitingly, these enzymes are druggable and several companies now have molecules in late stage clinical trials testing the concept that they could be used to treat renal anemia[15].

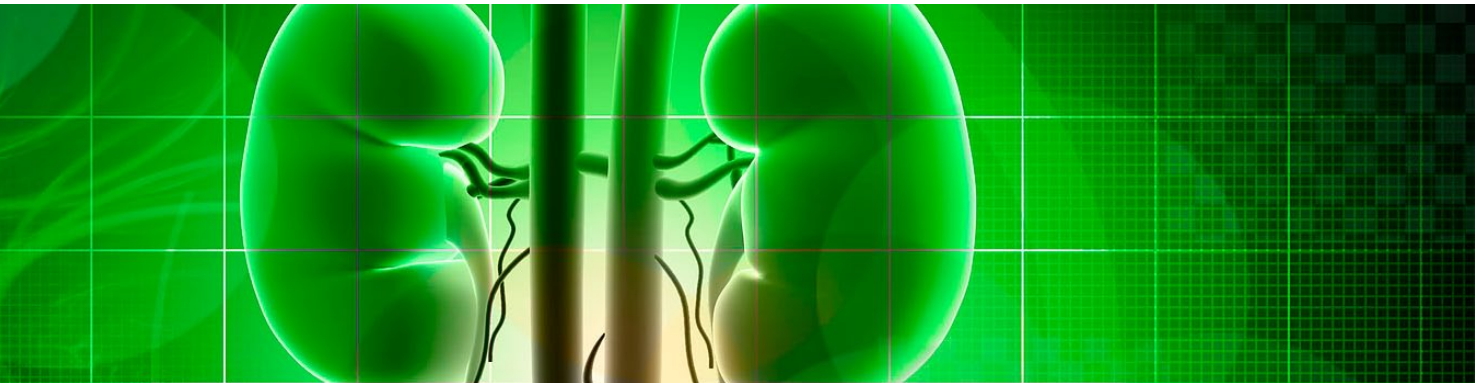
My own scientific career has centred on understanding cellular oxygen sensing, and uncovering the role of the VHL protein was a critical breakthrough for us. It illustrates a very important point that in the human population of 7.4 billion people on the planet there are likely to be informative variants in genes involved in all critical biological pathways. A rather fortunate aspect for me as a nephrologist is that this experiment of nature perturbing causes cysts and cancers in the kidney rather than (say) liver disease.

My second example is CFHR5 nephropathy. We discovered this familial condition through the study of families of Cypriot origin in which affected individuals had glomerular inflammation characterised by deposition of complement without immunoglobulins[16]. This pattern, which is termed C3 glomerulonephritis, is very rare and suggested a defect in regulation of the complement cascade. We found that affected individuals have a duplication of two exons of the *CFHR5* gene, which encodes a protein called complement factor H related 5. Individuals inheriting the mutation have reduced levels of wild type CFHR5 protein, along with the mutated protein which includes duplication of Short Concensus Repeat domains 1 and 2. We found that the mutation is quite common in people of Cypriot origin, and is present on a single haplotype and arose from a single founder at least 400 years ago[17]. As expected for an abnormality of complement regulation in the circulation, we found that following transplantation C3 glomerulonephritis could recur in the transplanted kidney[18]. An unanswered question is whether the glomerular inflammation is due to a reduction in wild type CFHR5 levels (haploinsufficiency) or the mutant CFHR5 protein (dominant negative / gain of function). The case for haploinsufficiency would be greatly strengthened if heterozygosity for a deletion or clear loss-of-function allele also gave rise to CFHR5 nephropathy. An individual with such a mutation has been described, and had persistent C3 glomerulonephritis following an episode of poststreptococcal glomerulonephritis –suggesting increased sensitivity of

complement activation[19]. But very recent evidence supports a gain of function for the fusion protein[20], and for a hybrid CFHR2-CFHR5 protein present in two patients who have a genetic deletion in the region associated with more severe complement dysregulation causing dense deposit disease and depletion of circulating levels of complement C3[21]. In either case, plasma exchange is a logical treatment option, and very limited experience suggests it may be effective. In contrast, immunosuppression with agents such as corticosteroids or cyclophosphamide appears ineffective, consistent with the molecular cause of the disease. Finally, blockade of complement activation with eculizumab (an antibody which binds complement C5) may be a logical precision medicine approach.

The third example is Autosomal Dominant Polycystic Kidney Disease (ADPKD). In contrast to my other examples this is common, affecting approximately 1 in 1000 live births. Approximately 80-85 % of cases are caused by mutations in *PKD1*. It is thus the commonest single gene disorder. Although linkage on Chromosome 16 was reported in 1985[22] and the PKD1 gene was identified in 1994[23], our understanding of why PKD1 mutations lead to cystic kidney disease and renal failure has steadily advanced but remains incomplete[24]. Strikingly, within an individual family the age at which individual members will need renal replacement therapy can vary by ~20 years, demonstrating that other genetic and environmental factors have a major effect. Summarising current knowledge is beyond the scope of the current lecture, but this does illustrate just how challenging it is to move from identifying a gene to understanding disease mechanism.

From a clinical point of view identifying the gene made very little impact for many years, contributing to the nephrology community's scepticism about the utility of genetics in mainstream adult nephrology. Reasons for this include that the gene is very large, that there are adjacent pseudogenes which are highly similar, and mutations tend to be private affecting a single family. The current position is that rather than offering genetic testing to patients and potentially affected offspring, diagnosis is based on family history and the presence of cysts on imaging. Cysts increase in number and size with increasing age; if no cysts are present at age 30 then we can tell an individual that they have not inherited ADPKD. The rationale for this approach includes the following. First, no treatment has been available, other than measuring blood pressure and giving antihypertensives – which can be done regardless of knowing an individual's genetic status. Second, it has been argued that knowledge that there is a 50% risk of passing on ADPKD to one's offspring would not influence reproductive decisions, since the condition only becomes symptomatic in adulthood and is compatible with a relatively normal life. I am uneasy about this because my discussions with patients suggest that some choose not to have children at all in the face of a 50% risk that their child will grow up to have ADPKD. Furthermore, couples made aware of the possibility of pre-implantation genetic diagnosis have been keen to go down this route. Third, it is often expensive



and difficult to pinpoint the disease causing mutation. Excitingly, we are now entering a new era in the management of ADPKD with the demonstration that tolvaptan, an oral inhibitor of the vasopressin V2 receptor, inhibits cyst growth and ameliorates the rate of decline of renal function[25]. Taking tolvaptan is not trivial; it results in inability to concentrate the urine and passing in the region of 7 litres of urine a day. However, my personal experience of looking after patients in London in the three year, phase III TEMPO 3:4 trial is that subjects managed remarkably well. They were prepared to put up with major inconvenience – such as an episode of incontinence when an underground train was held up between stations – in exchange for the possibility that their need for renal replacement therapy would be postponed. A significant safety issue is that some patients develop liver dysfunction. Following the TEMPO 3:4 study, tolvaptan has been licensed for treatment of rapidly progressive ADPKD in many countries including the EU, Switzerland, Japan, Korea, and Canada. In the UK, it is estimated that treatment with tolvaptan could delay end stage renal failure by a mean of 6 years with a cost per quality-adjusted life year of £23,500[26]. This compares favourably with many healthcare interventions. My prediction is that this will totally change the way we look after patients with ADPKD. A significant challenge is to work out a practical definition of "rapidly progressive disease"; the European Working Group on Inherited Genetic Kidney Disease has proposed an algorithm for this, which I think will be very useful[27]. This illustrates that even within a single genetic disorder a personalised, precision approach is required to achieve an appropriate balance between risk, inconvenience and potential benefit for the individual, and to optimise cost effectiveness for society as a whole.

Overall, I believe these three examples of monogenic kidney diseases provide clear illustrations of the potential for genomic approaches to provide decisive insights into human biology, and how they are contributing to precision approaches which will improve the lives of patients. Combining these with nephrology's impressive track record of precise diagnosis and effective treatments will be very powerful. Importantly, there is a balance to be struck between grouping different diseases into large enough populations to achieve incremental improvement (such as better cardiovascular outcomes on dialysis) versus focussing on small groups with a specific molecular or immunological defect. Like many aspects of medicine this isn't an either - or; to achieve the best for our patients we should adopt both.

References, acknowledgements and declarations for this article are available on the online version of Synapse which can be accessed http://www.hkcp.org/synapse.htm.

The Forum was well attended by doctors and nurses of both public and private sector. Prof Philip Li (Front row L-5) and Dr WL Cheung (Front row L-4) made the opening speeches.

Forum on Clinical Practice Guidelines for Renal Service in Hong Kong

This was organized by the Hong Kong College of Physicians, and the Central Renal Committee, Hospital Authority. The Hong Kong Society of Nephrology was the co-organiser.

Held on July 2, 2017 in the lecture theatre, Hospital Authority Building, the forum started with the opening speeches from Prof Philip Li, our President and Dr WL Cheung, Director (Cluster Services) of Hospital Authority. There were 8 presentations comprising topics on hemodialysis, peritoneal dialysis, renal nursing, infection control, transplantation, general nephrology, renal registry and accreditation of renal dialysis units.

The meeting was well attended by 357 participants who participated in the discussion of the Clinical Practice Guidelines. The Guidelines will later be published in the "Nephrology" journal.

Chairpersons, speakers and Nephrology Board Members of our College, Members of Central Renal Committee and Council members of Hong Kong Society of Nephrology in the Forum.

HKCP Annual Scientific Meeting and Annual General Meeting 2017

The Annual Scientific Meeting will be held on 21 – 22 October 2017 at the Hong Kong Academy of Medicine Jockey Club Building, followed by the Annual General Meeting and Annual Dinner on the 21 October 2017.

Program includes:

- Symposium on Transplantation in Hong Kong: past, present and future
- Symposium on Immunotherapy for Cancers
- Symposium on Update on Endocrine Disorders
- Symposium on Cardiovascular Benefits of New Anti-diabetic Medications
- Sir David Todd Lecture
- Richard Yu Lecture
- Gerald Choa Memorial Lecture
- Distinguished Research Paper Awards for Young Investigators
- Best Thesis Awards

WHAT'S NEW – The HKCP Scholarship for Medical Students

This new initiative aims to stimulate medical students towards an interest in internal medicine through elective attachments of at least 6 weeks.

Up to eight annual awards of $ 20000 per award will be open for application by medical students through the Faculty offices of University of Hong Kong or Chinese University of Hong Kong. Applications will be accepted from 1 September to 31 October of each year. The selection process will be adjudicated by a designated HKCP selection panel. Results will be announced in January of the following year

The overseas attachment which may involve clinical or research or humanitarian activities must be related to internal medicine and be endorsed by the respective medical Faculties. Upon completion of the attachment, the awardee will submit a formal report to the College within three months of return.

For more information about this scholarship, please refer http://www.hkcp.org.

Fellowship, Membership and Joint HKCPIE/ MRCP applications

At our recent Council Meeting, the Council decided that with immediate effect, trainees, who wish to apply for Joint HKCPIE/MRCP examinations (including Part I, Part II and PACES), College Memberships and Fellowships, are advised to send the relevant application forms to the College Secretariat via double registered mails, should they wish the documents to be duly delivered to the College. Our College will not be responsible for loss of mails in the application process, including delivery failures due to insufficient postage fees.

Dr Chan Wai Man Johnny
Hon Secretary

Synapse Editorial Board Meeting with our President

(Left to right) Dr Heyson CH Chan, Dr Francis CK Mok, Dr KK Chan (Deputy Editor), Dr John Mackay (Assistant Editor), Dr Carolyn PL Kng (Editor), Professor Philip Li (President, HKCP), Dr Alexander MH Leung, Ms Gloria Ng (Secretary, HKCP) and Dr Terrence PS Yip

Newly Elected FRCP(London) 2017

1. **Dr Lam Cheuk Sum**
   Department of Medicine & Geriatrics, Pok Oi Hospital
2. **Dr Yung Chun Yu**
   Department of Medicine, Pok Oi Hospital
3. **Dr Yap Yat Hin Desmond**
   Department of Medicine, Queen Mary Hospital
4. **Dr Cheng Boron Cheung-wah**
   Private Practice
5. **Dr Cheung Siu Fai**
   Department of Medicine, Yan Chai Hospital
6. **Dr Leung Chi Man**
   Department of Medicine, Pamela Youde Nethersole Eastern Hospital
7. **Dr Ko Wai San Fanny**
   Department of Medicine & Therapeutics, Prince of Wales Hospital
8. **Dr Kung Kam Ngai**
   Department of Medicine & Geriatrics, United Christian Hospital
9. **Dr Chak Wai Leung**
   Department of Medicine, Queen Elizabeth Hospital
10. **Dr David CL Lam**
    Department of Medicine, Queen Mary Hospital
11. **Dr Chung Tin Hei**
    Private Practice

INTERNATIONAL COLLABORATION

Royal Australasian College of Physicians Congress 2017, Melbourne, 7-9 May, 2017

Professor Philip Li represented the College to attend the Royal Australasian College of Physicians Congress 2017 held in Melbourne from 7-9 May, 2017.

He participated in the College Convocation Ceremony as well as a meeting with the presidents of the Royal Australasian College of Physicians, Royal College of Physicians, London and the American College of Physicians on areas of collaboration in the near future.

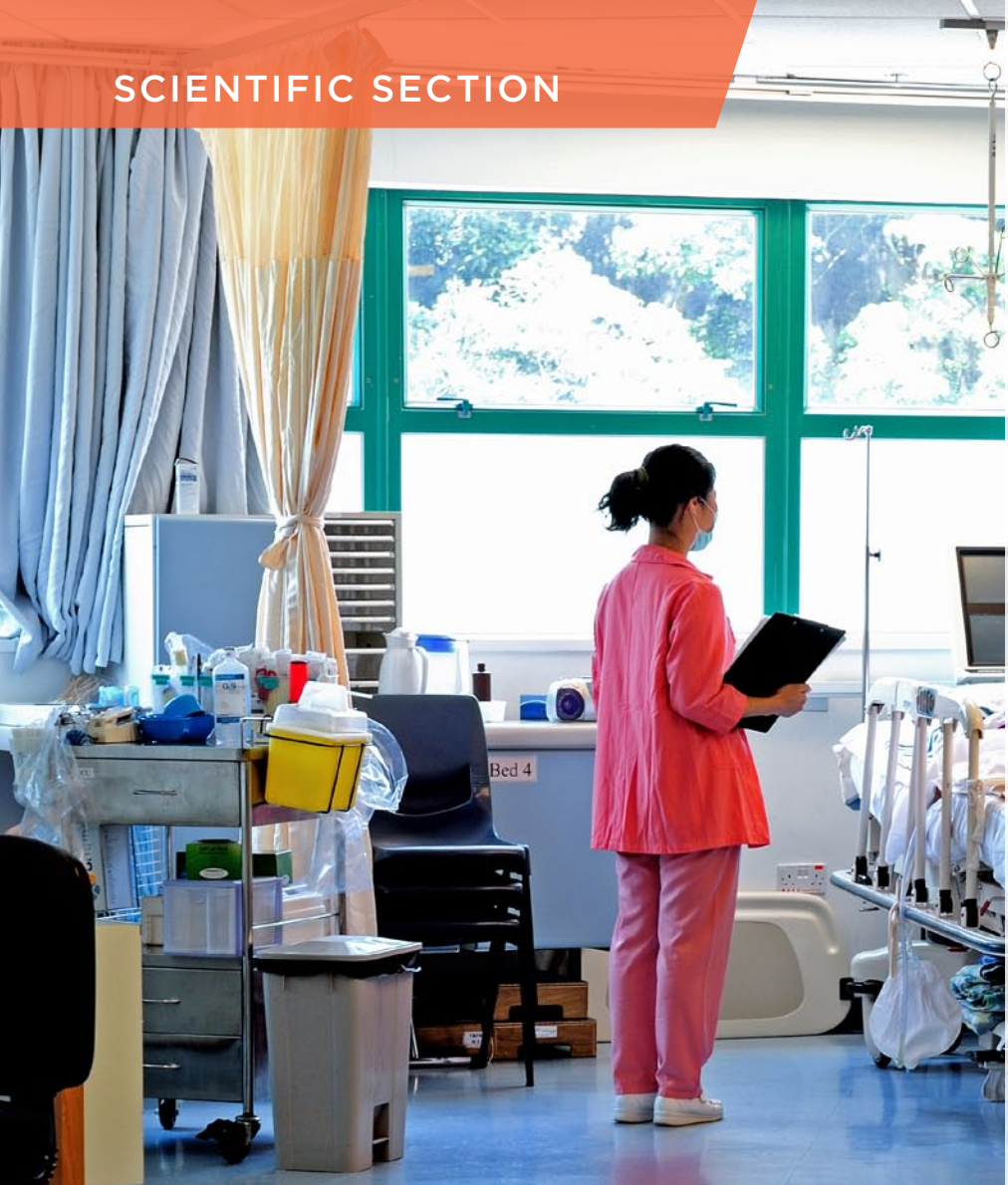
Professor Philip Li (L-2) with Prof Catherine Yelland, President of Royal Australasian College of Physicians (L-3) and Prof Jane Dacre, President of Royal College of Physicians (L-4) in the RACP Congress.

Prof Philip Li with Prof Jack Ende, President of American College of Physicians, in the RACP Congress.

51st Singapore — Malaysia Congress of Medicine and Diamond Jubilee celebration of the Singapore Academy of Medicine, July 21-23, 2017

On July 21-23, Prof Philip Li represented the College at the 51st Singapore — Malaysia Congress of Medicine which also coincided with the Diamond Jubilee celebration of the Singapore Academy of Medicine. He also attended the Joint Council Meeting as organized by the three Academies of Medicine of Singapore, Malaysia and Hong Kong for further collaboration. He was conferred Fellow of the Academy of Medicine of Singapore as well as Fellow of the Singapore College of Physicians during this Congress.

Before the Induction Comita and Conferment Ceremony of the 51st Singapore -- Malaysia Congress of Medicine. (L-R: Prof Chan Choong Meng, President, Singapore College of Physicians, Prof Frank Murray, President, Royal College of Physicians of Ireland, Prof Philip Li, Prof Donald Li, Prof Derek Bell, President, Royal College of Physicians, Edinburgh, Prof S R E Sayampanathan, Master, Academy of Medicine, Singapore and Prof CS Lau)



SPECIALTY UPDATE: CRITICAL CARE MEDICINE

An Introduction to Extracorporeal Membrane Oxygenation — A Life Support Therapy

K C Sin, G WY NG
Department of Intensive Care,
Queen Elizabeth Hospital

Introduction

Extracorporeal Life Support (ECLS), or commonly referred to as Extracorporeal Membrane Oxygenation (ECMO) is considered as one of the rescue therapy for patients who fail to respond to conventional treatment. ECLS was initially developed as a means of Cardiopulmonary Bypass in cardiothoracic operation in 1953.[1] When ECLS is used outside the operation theatre, it is generally referred as ECMO. The first report of successful ECMO use for an adult patient with acute post-traumatic respiratory failure was published by Hill in 1972.[2,3] The use of ECMO, however, was mostly limited to neonatal and paediatric group of patients in earlier years, particularly after a randomized controlled trial in 1979 showed no additional mortality benefit in adult patients with severe respiratory failure receiving ECMO treatment.[4]

Hong Kong experienced two large-scale infectious disease outbreaks: 2003 Severe Acute Respiratory Syndrome (SARS) outbreak and 2009 Swine H1N1 influenza outbreak. In 2003, SARS patients with severe respiratory failure were supported by conventional mechanical ventilation[5], with overall case fatality rate of 17%. In 2009 novel swine H1N1 influenza pandemic, there were large number of patients admitted to intensive care units worldwide due to respiratory failure. Advances in ECMO technology and ventilatory management strategy led to a significant improvement in outcomes, with overall case fatality rate of 0.03%. The CESAR trial and experiences from international research showed convincing evidence that ECMO was an effective supportive treatment for patients

with severe respiratory failure and acute respiratory distress syndrome (ARDS) due to H1N1 influenza.[6,7,8,9] ECMO is now used as a rescue therapy for patients who fail to respond to conventional mechanical ventilation[10,11].

In Hong Kong, five ECMO centres were established in 2010 under the governance of Hospital Authority, with an aim to provide ECMO services for patients with severe respiratory failure and prepare for severe respiratory disease emergency outbreaks. With improvement in skills and accumulation of clinical experience in ECMO management, the indications for ECMO support have been expanding for patients with potentially reversible causes of cardiac and/or respiratory failure. According to data registry from the Hospital Authority Intensive Care Coordinating Committee ECMO workgroup, the number of patients receiving ECMO therapy has been growing since 2010. (Figure 1).

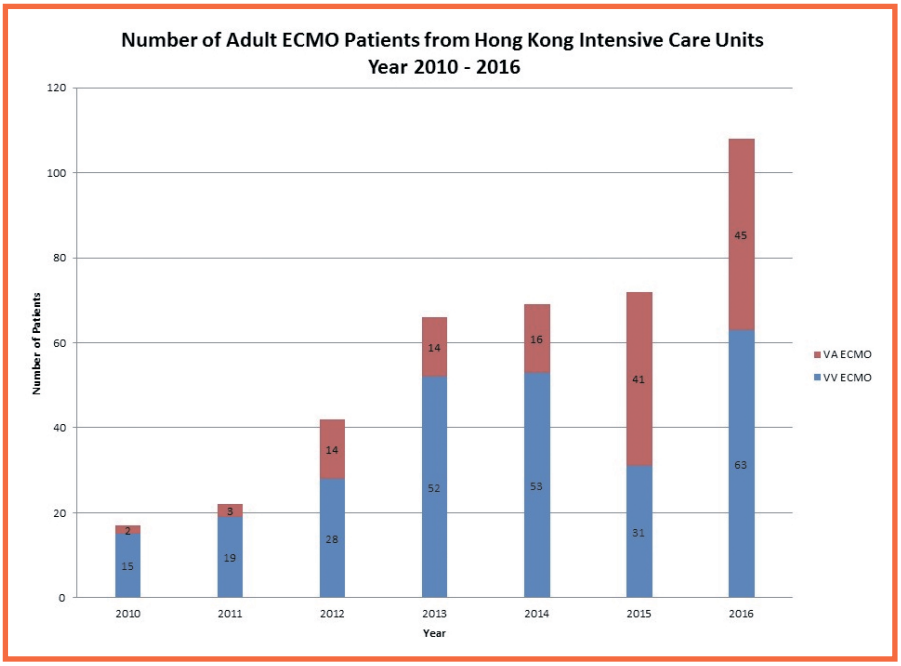
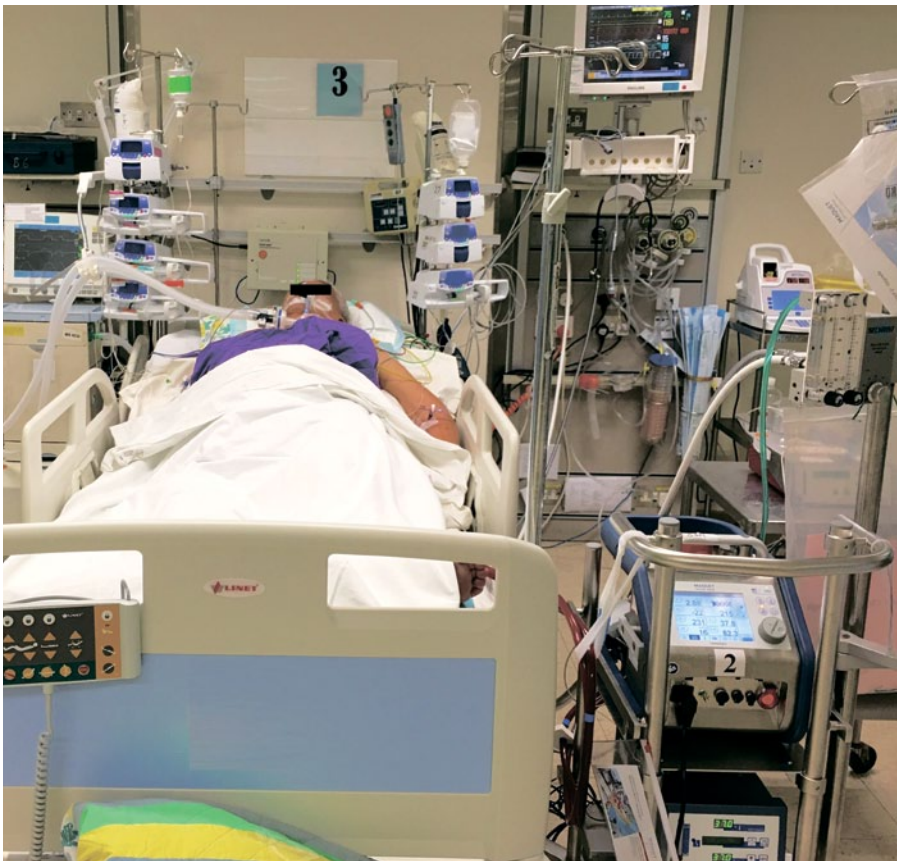


Figure 1. Number of adult ECMO patients from four Hong Kong Intensive Care Units (PMH, PYNEH, QEH & QMH)

What is Extracorporeal Membrane Oxygenation?

An ECMO system consists of access cannula & return cannula, a motor pump, and a membrane oxygenator. Deoxygenated blood is drained from the venous circulation by motor pump, and passes through the oxygenator that provides oxygen and removes carbon dioxide. The oxygenated blood then returns to the body via return cannula.

ECMO is basically classified into veno-venous (VV) and veno-arterial (VA) mode. VV ECMO provides solely lung support, whereas VA ECMO provides both heart and lung support. The difference lies in the position of return cannula. For VV ECMO, the return cannula is placed in the venous system with no intention to support the hemodynamics. While for VA ECMO, the return cannula is placed in the arterial system, working as an external pump to support the failing heart.



Clinical photo showing a patient on ECMO therapy

How Effective is the Support?

For VV ECMO, gaseous exchange occurs at the blood gas interface in the membrane oxygenator. Similar to the native alveoli, oxygen diffuses in and carbon dioxide diffuses out. The amount of oxygen delivered is influenced by the membrane surface area, membrane thickness, haemoglobin level, and ECMO blood flow. For carbon dioxide removal, the membrane oxygenator allows more efficient carbon dioxide flow than oxygen due to better solubility and diffusion property. Theoretically, 0.5–1.5 Liter/minute of ECMO blood flow is sufficient for the membrane oxygenator to clear up 100% of the metabolic carbon dioxide production.

VA ECMO primarily aims at providing support to heart for end-organ perfusion and allows time for possible heart recovery. The motor pump can generate a flow rate up to 7 Litres/minute and creates positive pressure in the arterial system. The total cardiac output is composed of both native cardiac output and VA ECMO flows. As a result, the mean arterial pressure (MAP) is directly related to the summation of the native cardiac output and the VA ECMO pump flow.

Patient Selection and Clinical Evidence

VV ECMO

Mechanical ventilation is the mainstay of supportive therapy for patients with respiratory failure. However, mechanical ventilation is associated with lung alveolar injury secondary to elevated trans-pulmonary pressures and/or over-distension. Lung protective ventilation is proven to reduce ventilator-associated lung injury and mortality in ARDS patients.[12] This can be achieved by limiting the tidal volume and plateau pressure with controlled ventilation modes. However, uncontrolled hypercarbia and respiratory acidosis are commonly encountered due to the poor compliance of the pathological lungs.

With the external source of gaseous exchange in the membrane oxygenator, VV ECMO allows ARDS patients to adopt lung protective strategy without the above problems. VV-ECMO is currently considered as a rescue therapy for ARDS patients who have failed conventional mechanical ventilation. Favorable results of H1H1 patients from local and international ECMO centres in recent years have led to an exponential use of the technology to other respiratory disease entities [6,7,8,9,10,11].

Nevertheless, careful selection of suitable candidates to receive ECMO is critical as ECMO is a high risk and costly procedure. Different ECMO centres have individual selection criteria. In general, a pre-treatment predicted mortality of higher than 80% is widely accepted as potential ECMO candidates. Table 1 shows the common indications and contraindications of VV ECMO.

TABLE 1. Common indications for and contra-indications to VV-ECMO

Indications	Contra-indications
• Hypoxic respiratory failure due to any cause when the predicted mortality risk is >80% (PaO_2/FiO_2 <100 on FiO_2 >90% and/or Murray score[118] 3-4 despite optimal care for ≥6 hours) • CO_2 retention on mechanical ventilation despite high Pplt (>30 cm H_2O) e.g. Acute severe asthma • Pulmonary embolism • Acute airway compromise	• Unwitnessed cardiac/respiratory arrest • Disseminated malignancy • Major pharmacological immunosuppression • Recent CNS haemorrhage • Non recoverable co-morbidity such as major CNS damage or terminal malignancy • Age: no specific age contraindication but consider increasing risk with increasing age

Abbreviations: CNS = central nervous system; CO_2 = carbon dioxide; FiO_2 = fraction of inspired oxygen; Pplt = plateau pressure; PaO_2 = partial pressure of oxygen

VA ECMO

VA ECMO is a modified form of Cardiopumonary Bypass used in open heart surgery, and has been used as supportive therapy for post cardiotomy cardiogenic shock. Recently, its uses have been extended outside operation theatre particularly after intra-aortic balloon pump was shown to have no survival benefit over conventional medical treatment alone.[13,14] Acting as a mechanical circulatory support, ECMO therapy is not an ultimate treatment. It is only a temporary bridge to potential recovery, transplantation, or long-term cardiac support therapies. Careful selection of suitable candidates for VA ECMO is essential for a favorable outcome. Table 2 illustrates the common indications and contra-indications of VA ECMO.

TABLE 2. Common indications for and contra-indications to VA-ECMO

Indications	Contra-indications
In patients with cardiogenic shock: • Post-cardiotomy shock • Acute myocarditis • Refractory life-threatening cardiac arrhythmia • Drug overdose/toxicity Acute coronary syndrome • Peripartum cardiomyopathy • Acute anaphylaxis • Pulmonary embolism • Sepsis related cardiomyopathy • Periprocedural support for high-risk percutaneous cardiac interventions (e.g. Primary PCI, TAVI) • As a bridge to a more definitive treatment: o VAD support o Transplant	• Progressive and non-recoverable cardiac and respiratory disease, and not a candidate for transplant or VAD support • Advanced malignancy • Known severe central nervous system injury • Cardiac arrest of unknown duration • Prolonged CPR without adequate tissue perfusion • Unrepaired acute aortic dissection • Severe aortic regurgitation • Severe chronic organ dysfunction • Anticoagulation • Peripheral VA-ECMO is contra-indicated in severe peripheral vascular disease

Abbreviations: PCI = Percutaneous Coronary Intervention; TAVI = Transcatheter Aortic Valve Implantation; CPR = cardiopulmonary resuscitation; VAD = ventricular assist device

Possible Complications

ECMO is a complex and high risk procedure. Complications are not common but can be life-threatening (Table 3). The outcome of patients treated with ECMO is dependent on two factors: patient selection and complications prevention.

Table 3. Possible complications common to VV and VA ECMO

Air embolism	Broken three-way/ pigtail Cannula is dislodged or its side holes are exposed
Oxygenator failure	Mechanical breakdown: when the oxygenator membrane is broken or wore off Functional breakdown: When there are clots/ thrombus formation on the surface that hinders gaseous exchange
Vascular injury	Injury: Trauma to adjacent organs e.g. localized hematoma, pneumothorax, dissection of vessel, esophageal perforation, cardiac perforation Malposition: Recirculation occurs when the tips of the return cannula and access cannula are too close so that oxygenated blood from the return cannula drain directly into the access cannula
Motor pump failure	Motor failure Broken pump head
Bleeding	Bleeding may occur at the cannula site, surgical site, or site of previous invasive procedure Gastrointestinal bleeding, pulmonary hemorrhage, and spontaneous intracranial bleeding may occur

Specific Issue related to VV ECMO

Recirculation

Recirculation only happens exclusively to VV ECMO, in which oxygenated blood from the return cannula is drained into the access cannula directly instead of systemic circulation.[15]

Recirculation is suspected when blood in both access and return cannula appear bright red (oxygenated blood). Close proximity of the access and return cannula, high pump speed and ECMO blood flow, larger cannula size correlates with increase amount of recirculation. The tips of the return & access cannulae should be more than 10 cm apart to minimize recirculation.

Specific Issues related to VA ECMO

Systemic Thromboembolism

The risk of systemic thromboembolism is considered higher in VA ECMO compared to VV ECMO. Patients requiring VA ECMO support usually have severe left ventricular dysfunction and / or arrhythmia. In peripheral VA ECMO, where the return cannula is placed in the femoral artery, the retrograde blood flow generated from the motor pump can oppose the opening of the aortic valve if myocardial contractility is poor. This can lead to ventricular thrombus formation. Systemic thromboembolism may occur when the left ventricle start to recover and the aortic valve opens. A higher anticoagulation intensity is advocated to minimize the risk of thrombus formation.

Hydrostatic Pulmonary Edema

Patients with severe left ventricular failure receiving peripheral VA ECMO are prone to develop acute hydrostatic pulmonary edema. The retrograde pressure from the peripheral VA ECMO increases the left ventricular afterload. Distension of left ventricle and left atrium occurs followed by hydrostatic pulmonary edema. The size of the heart chambers and aortic valve opening should be regularly monitored by echocardiography. Restrictive fluid strategy should be adopted. Concomitant use of intra-aortic balloon pump and arterial vasodilating medications can reduce afterload and facilitate aortic valve opening.[16] Decompression of over-distended left ventricle and left atrium should be considered in refractory cases. It can be done by percutaneous technique or open surgical approach.[17,18]

Harlequin Syndrome

Differential hypoxia (or Harlequin syndrome) is a phenomena with relative hypoxemia of the upper body due to competition of blood flow between peripheral VA ECMO and the native heart. It occurs when the failing heart recovers, with co-existing bad lung condition. In patients with respiratory failure, poorly oxygenated blood returns to the native heart and is pumped into the systemic circulation. At the same time, peripheral VA ECMO forces the fully oxygenated blood into the aorta in a retrograde manner. When the left ventricular function improves, a high portion of poorly oxygenated blood is ejected into the aorta. As a result, the upper body (including the brain) is perfused mainly by the poorly oxygenated blood from the heart while the lower part of the body is supplied by fully oxygenated blood from the ECMO. The point where the two opposing flows meet varies and depends on the ECMO blood flow and the myocardial function. The oxygen saturation should therefore be routinely measured on right upper limb in peripheral VA ECMO patients. Differential hypoxia is strongly suspected if there is a significant oxygen saturation discrepancy between the two hands. If differential hypoxia occurs, measures should be taken in order to optimize the lung condition. In refractory case, reconfiguration of VA ECMO should be considered. Examples include conversion to VV ECMO, relocation of the arterial cannula to right subclavian artery, or insertion of an additional return cannula to the venous system (V-AV ECMO).

Limb ischaemia

Lower extremity ischemia may occur if common femoral artery is used for peripheral VA ECMO cannulation. To reduce the risk of distal limb ischemia, a perfusion cannula can be inserted into the ipsilateral superficial femoral artery in antegrade direction by Seldinger technique.[19] Distal limb circulation should be monitored by either reperfusion cannula blood flow or lower limb tissue oxygenation with near-infrared spectroscopy.[20]

Anticoagulation

Inflammatory reaction and coagulation cascades are triggered when circulatory blood is exposed to non-biologic surface of the circuit during ECMO therapy. Anticoagulation is used to minimize risk of clot formation in the systemic circulation and ECMO circuit, especially in patients with poor left ventricular function that has high tendency of thrombus formation.

Unfractionated heparin (UFH) is the choice of anticoagulation widely used in ECMO patients. It interacts with anti-thrombin III to form heparin anti-thrombin complex, which inhibits coagulation by inactivating thrombin and factor Xa. UFH has a short half-life and its action is reversible by protamine sulfate. Potential complications of heparin include bleeding, heparin-induced thrombocytopenia, and tachyphylaxis.[21] Factor Xa inhibitors (e.g. argatroban)[22,23] and direct thrombin inhibitors (e.g. bivalirudin) have been reported to be safe alternatives in ECMO therapy.[24]

The Activated Partial Thromboplastin Time (aPTT) and Activated Clotting Time (ACT) are routinely used to monitor coagulation status.[25] The suggested optimal anticoagulation target in ECMO patients is ACT 180 – 220 seconds or aPTT 50-70. However, ACT does not have a linear correlation to APTT, and the relationship of paired ACT and APTT samples is poor.[26] Moreover, study comparing ACT and APTT showed that APTT correlated slightly better with heparin concentration than ACT.[27]

Conclusion

As a rescue therapy, ECMO provides powerful respiratory and / or circulatory support to the critically ill patients with cardiopulmonary collapse. With the expansion of clinical indications, there are emerging discussion on disease futility and treatment withdrawal. Like other organ support systems, ECMO is never a definitive treatment nor risk-free. It serves as a bridge to disease recovery, or transplantation. Appropriate patient selection with meticulous monitoring to prevent complications is crucial to the success of this novel treatment.

Acknowledge:

We thank Hospital Authority Intensive Care Coordinating Committee ECMO Workgroup, Intensive Care Units of Princess Margaret Hospital (PMH), Queen Elizabeth Hospital (QEH), Queen Mary Hospital (QMH), and Pamela Youde Nethersole Eastern Hospital (PYNEH) for contributing data.

References, acknowledgements and declarations for this article are available on the online version of Synapse which can be accessed http://www.hkcp.org/synapse.htm.

PASSING RATES : JOINT HKCPIE/MRCP (UK) PART II (WRITTEN) EXAMINATION – 2002 - 2017

	Sitting	Pass
2 July 2002	53	27 (51%)
13 November 2002	50	24 (48%)
13 August 2003	110	62 (56%)
10 December 2003	54	31 (57%)
28 July 2004	65	42 (65%)
8 December 2004	46	32 (70%)
13 April 2005	32	15 (47%)
27 July 2005	76	56 (74%)
7 & 8 December 2005	26	16 (62%)
12 & 13 April 2006	29	13 (45%)
26 & 27 July 2006	91	68 (75%)
6 & 7 December 2006	33	18 (55%)
11 & 12 April 2007	34	22 (65%)
25 & 26 July 2007	80	70 (88%)
5 & 6 December 2007	19	13 (68%)
9 & 10 April 2008	21	13 (62%)
30 & 31 July 2008	47	36 (77%)
3 & 4 December 2008	17	10 (59%)
8 & 9 April 2009	32	25 (78%)
29 & 30 July 2009	50	43 (86%)
25 & 26 November 2009	12	7 (58%)
7 & 8 April 2010	41	34 (83%)
28 & 29 July 2010	25	19 (76%)
24 & 25 November 2010	8	2 (25%)
6 & 7 April 2011	45	35 (78%)
23 & 24 November 2011	32	25 (78%)
28 & 29 March 2012	55	43 (78%)
12 & 13 December 2012	57	44 (77%)
10 & 11 April 2013	60	52 (87%)
11 & 12 December 2013	48	34 (71%)
9 & 10 April 2014	54	46 (85%)
10 & 11 December 2014	26	25 (96%)
25 & 26 March 2015	53	45 (85%)
9 & 10 December 2015	68	65 (96%)
6 & 7 April 2016	29	28 (97%)
7 & 8 December 2016	62	50 (81%)
29 & 30 March 2017	25	21 (84%)

PASSING RATES : PART I EXAMINATION – 2002 - 2017

	Sitting	Pass
September 2002	100	33 (33%)
January 2003	124	55 (44%)
May 2003 (SARS Special)	21	7 (33%)
September 2003	54	29 (54%)
January 2004	93	39 (42%)
September 2004	29	16 (55%)
January 2005	96	68 (70.8%)
September 2005	24	15 (62.5%)
January 2006	95	74 (80%)
September 2006	21	13 (62%)
January 2007	87	67 (77%)
September 2007	23	12 (52%)
January 2008	56	38 (68%)
September 2008	47	32 (68%)
January 2009	59	47 (80%)
September 2009	47	28 (60%)
January 2010	45	28 (62%)
September 2010	62	39 (63%)
January 2011	44	23 (52%)
September 2011	64	49 (77%)
January 2012	45	28 (62%)
September 2012	80	59 (74%)
January 2013	41	22 (54%)
September 2013	76	60 (79%)
January 2014	30	20 (67%)
September 2014	84	64 (76%)
January 2015	29	20 (69%)
September 2015	100	71 (71%)
January 2016	33	18 (55%)
September 2016	84	63 (75%)
January 2017	36	19 (53%)

PASSING RATES : PACES – 2001 - 2017

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%
March 2015	36/75 = 48%
October 2015	35/75 = 47%
March 2016	40/75 = 53%
October 2016	36/74 = 49%
March 2017	26/74 = 35%

PASS LIST (2017) : JOINT HKCPIE/MRCP (UK) PART II PACES EXAMINATION MARCH

Chan Jun Yi Abram
Cheng Wilson Wai Shun
Ching Shing
Chiu Hei Yeung Kelvin*
Chow Tsz Shan
Hui Ka Yin
Lai Pak Yin
Lee Ka Chun Kevin
Lee Pok Him
Leung Ka Chun
Leung Yat Kai
Li Cheuk Him
Lie Davina Ngoi Wah
Ma Ming Yao
Ma Sin Kwan Tammy
Mak Man Yin
Mak Yiu Hang Peter
Ng Wing Sze
Sin Chun Ming
So Jacqueline
Tam Yiu Sum
To Hau Man Harmony
Wong Chun Kit
Wong Ho Sum Cally
Wong Tsun Yee Jenny
Wu Hiu Fung Jerome
Yeung Ka Ho

* Dr Chiu is not our College's registered trainee. He is working in the Department of Microbiology of QMH.

CAREER TALK

Heyson CH Chan
Chairman, Young Fellows' Committee, Hong Kong College of Physicians

The Young Fellows' Committee organized the 1st Hong Kong College of Physicians Career Talk for Medical Graduates on 11th June 2017 in Prince of Wales Hospital. The response was overwhelming with more than 230 medical graduates from both Chinese University of Hong Kong and University of Hong Kong attending the talk.

The aims of the career talk were as follows:

- To attract potential trainees to internal medicine
- To help medical graduates better prepare for medical internship
- To equip medical graduates with essential skills in job application process in medicine

The College President, Professor Philip Li, delivered an opening address to the audience. He shared his experience during his internship and the reason he chose internal medicine as his training specialty.

Members from the Young Fellows' committee presented four seminars during the talk. The first seminar was "Introduction to Internal Medicine" where the audience was given an overview of the training and examination structure of internal medicine. It was followed by "Life as a Physician" with focus on the job satisfaction of being a physician and the ways to strike the optimal work-life balance in the current environment. During "Tips of CV Writing and Interview Skills", samples of CV and important interview skills were explained. Finally, practical tips on navigating through medical internship with real-life case scenarios illustrating common pitfalls of interns were shared. Special emphasis on attitude and professionalism were also made in the final topic "Houseman Survival Guide".

Apart from the seminars, there was an informal peer sharing session during the tea break. Representatives from various specialties (gastroenterology, neurology, nephrology, hematology, respiratory, dermatology, medical oncology, geriatrics, infectious disease, rehabilitation, palliative medicine and critical care medicine) of internal medicine were present. Light refreshments and a relaxed ambience allowed participants to freely chat with our representatives. This provided opportunities for interactive discussion and peer advice including how to master medical internship, sharing on career development and in-depth understanding of the various specialties.

Evaluation from the participants showed that the talk was very well received. Most found the talk, in particular, the informal sharing session with specialty representatives useful for their career planning. They also appreciated the real-life scenarios as useful in translating previously learnt medical knowledge into clinical practice, especially for common clinical encounters during on-calls. Some participants reflected that they were not aware of CV writing and interview skills before this event, and this talk introduced essential techniques vital for their future job applications.

With the experience from this round, we are planning to organize the second round in the next few months. The target audience will be extended to final year medical students with priority given to medical graduates who could not attend the talk in June. In the second round, it is hoped that a more extensive coverage of different specialties will be covered and a refinement on the rundown based on the feedback from the first round will be made.

The event would not be a success without the unfailing support from different parties, partioulary our President, College council and the secretariat. The Young Fellows' committee members made spirited contributions in delivering interesting and informative talks, designing the run-down and ironing out the logistics. Last but not least, appreciations to representatives from different specialties for their active participation and for giving valuable advice to the attendees.

The President, Prof Philip Li, addressing the audience

Dr Heyson Chan explaining the rundown

Over 230 medical graduates from the University of Hong Kong and the Chinese University of Hong Kong attended the talk

Prof Li with members of the Young Fellows' Committee (from left to right: Dr WS Mak, Dr WT Kwok, Dr Iki Chan, Dr Angeline Lo, Dr Jacqueline So, Prof Philip Li, Dr Heyson Chan, Dr Thomas Chan, Dr WH Chan, Dr Kelvin Tsoi)

Representatives from various specialties were giving peer advice to the attendees in the informal sharing session

Statistics on No. of Trainees in all Specialties

Updated in July 2017

SPECIALTY	TRAINEES TOTAL (DH/HA/ OTHERS)	TRAINEES						
		HONG KONG EAST CLUSTER			HONG KONG WEST CLUSTER			
		PYNEH	RH	TWEH	FYKH	GH	QMH	TWH
		YEAR			YEAR			
CARDIOLOGY	26	1 4 2—III 3 4—I *7*	1 0 2 3 4 *4*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *4*	1 4 2—II 3—II 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 0 2 3 4 *1*	1 0 2 3 4 *0*
CRITICAL CARE MEDICINE	12	1 2 2—I 3—I 4 *6*	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 3 2—I 3—I 4—I *7*	1 0 2 3 4 *0*
DERMATOLOGY & VENEREOLOGY	7	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	14	1 0 2 3 4 *5*	1 1 2—I 3 4 *2*	1 1 2—I 3 4 *1*	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*
GASTROENTEROLOGY & HEPATOLOGY	23	1 4 2—I 3—I 4—II *4*	1 0 2 3 4 *1*	1 1 2 3—I 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 4 2—I 3—III 4 *7*	1 0 2 3 4 *0*
GERIATRIC MEDICINE	15	1 0 2 3 4 *6*	1 0 2 3 4 *9*	1 2 2 3—II 4 *1*	1 0 2 3 4 *4*	1 0 2 3 4 *3*	1 0 2 3 4 *1*	1 0 2 3 4 *1*
HAEM/HAEM ONCOLOGY	13	1 2 2—I 3 4—I *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 3 2—I 3—II 4 *7*	1 0 2 3 4 *0*
IMMUNOLOGY & ALLERGY	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—I 3 4 *0*	1 0 2 3 4 *0*
INFECTIOUS DISEASE	4	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 *0*	1 0 2 3 4 *1*	1 0 2 3 4 *0*
INTERNAL MEDICINE	190	1 16 2—VII 3—II 4—VII *42*	1 5 2—II 3 4—III *17*	1 3 2—I 3—II 4 *8*	1 0 2 3 4 *6*	1 0 2 3 4 *7*	1 23 2—VIII 3—X 4—V *66*	1 0 2 3 4 *8*
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 0 2 3 4 *2*	1 0 2 3 4 *0*
NEPHROLOGY	17	1 1 2—I 3 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 *7*	1 0 2 3 4 *3*
NEUROLOGY	10	1 0 2 3 4 *5*	1 1 2—I 3 4 *3*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2—I 3 4 *7*	1 0 2 3 4 *0*
PALLIATIVE MEDICINE	4	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 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
REHABILITATION	3	1 0 2 3 4 *0*	1 0 2 3 4 *1*	1 1 2—I 3 4 *3*	1 0 2 3 4 *1*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *5*
RESPIRATORY MEDICINE	11	1 1 2 3—I 4 *5*	1 1 2 3 4—I *5*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *8*	1 1 2 3—I 4 *6*	1 0 2 3 4 *0*
RHEUMATOLOGY	8	1 0 2 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 1 2 3—I 4 *4*	1 0 2 3 4 *1*

		TRAINEES											
		KOWLOON CENTRAL CLUSTR						KOWLOON EAST CLUSTER			KOWLOON WEST CLUSTER			
SPECIALTY	TRAINEES TOTAL (DH/HA/ OTHERS)	BH	KH	KWH	OLMH	QEH	WTSH	HOHH	TKOH	UCH	CMC	NLTH	PMH	YCH
		YEAR						YEAR			YEAR			
CARDIOLOGY	26	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2–I 3 4 *3*	1 0 2 3 4 *0*	1 4 2–II 3–II 4 *9*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2 3–I 4 *2*	1 0 2 3 4 *6*	1 1 2 3–I 4 *1*	1 0 2 3 4 *0*	1 2 2–II 3 4 *8*	1 0 2 3 4 *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*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
CRITICAL CARE MEDICINE	12	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2 3–I 4 *4*	1 0 2 3 4 *0*	1 0 2 3 4 *4*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *3*	1–I 3 2 3–II 4 *5*	1 2 2–I 3–I 4 *3*	1 0 2 3 4 *0*	1 0 2 3 4 *4*	1 0 2 3 4 *1*
DERMATOLOGY & VENEREOLOGY	7	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*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
ENDOCRINOLOGY, DIABETES & METABOLISM	14	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 2 2 3–I 4–I *4*	1 1 2 3–I 4 *2*	1 1 2 3–I 4 *8*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *4*	1 1 2–I 3 4 *3*	1 2 2 3–I 4 *3*	1 0 2 3 4 *0*	1 1 2 3 4–I *4*	1 0 2 3 4 *2*
GASTROENTEROLOGY & HEPATOLOGY	23	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 2 2–II 3 4 *8*	1 0 2 3 4 *2*	1 0 2 3 4 *4*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *4*	1 1 2 3–I 4 *3*	1 1 2 3–I 4 *3*	1 0 2 3 4 *0*	1 1 2–I 3 4 *7*	1 1 2 3 4–I *5*
GERIATRIC MEDICINE	15	1 1 2 3–I 4 *1*	1 0 2 3 4 *5*	1 0 2 3 4 *8*	1 0 2 3 4 *1*	1 0 2 3 4 *3*	1 0 2 3 4 *5*	1 2 2–I 3–I 4 *3*	1 0 2 3 4 *1*	1 3 2–II 3 4–I *8*	1 0 2 3 4 *8*	1 0 2 3 4 *1*	1 0 2 3 4 *9*	1 1 2 3–I 4 *5*
HAEM/HAEM ONCOLOGY	13	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–I 3 4 *3*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *2*	1 1 2–I 3 4 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 2 2 3–II 4 *4*	1 0 2 3 4 *0*
IMMUNOLOGY & ALLERGY	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*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
INFECTIOUS DISEASE	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 0 2 3 4 *6*	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 2 2–I 3–I 4 *3*	1 0 2 3 4 *0*
INTERNAL MEDICINE	190	1 2 2–I 3–I 4 *2*	1 0 2 3 4 *7*	1 10 2–IV 3–IV 4–II *42*	1 1 2 3–I 4 *8*	1 12 2–II 3–V 4–V *60*	1 0 2 3 4 *6*	1 4 2–II 3–I 4–I *5*	1 4 2–II 3–II 4 *24*	1–I 13 2–VI 3–V 4–I *37*	1 9 2–III 3–VI 4 *25*	1 0 2 3 4 *0*	1 15 2–V 3–V 4–V *50*	1 3 2 3–I 4–II *24*
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 *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 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
NEPHROLOGY	17	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2–I 3 4 *7*	1 0 2 3 4 *0*	1 0 2 3 4 *8*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 2 2–II 3 4 *2*	1 3 2–II 3–I 4 *4*	1 1 2–I 3 4 *2*	1 0 2 3 4 *0*	1 2 2–I 3 4–I *6*	1 1 2 3 4–I *2*
NEUROLOGY	10	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 1 2 3–I 4 *5*	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 1 2 3–I 4 *1*	1 0 2 3 4 *0*	1 2 2 3–I 4–I *3*	1 0 2 3 4 *3*
PALLIATIVE MEDICINE	4	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 1 2 3 4–I *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 1 2–I 3 4 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
REHABILITATION	3	1 0 2 3 4 *0*	1 0 2 3 4 *7*	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 1 2–I 3 4 *1*	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*
RESPIRATORY MEDICINE	11	1 0 2 3 4 *0*	1 0 2 3 4 *6*	1 1 2 3–I 4 *3*	1 0 2 3 4 *0*	1 1 2 3–I 4 *7*	1 0 2 3 4 *2*	1 1 2 3 4–I *5*	1 0 2 3 4 *3*	1 0 2 3 4 *5*	1 0 2 3 4 *2*	1 0 2 3 4 *0*	1 1 2 3–I 4 *5*	1 0 2 3 4 *1*
RHEUMATOLOGY	8	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2 3–I 4 *4*	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 1 2 3–I 4 *2*	1 0 2 3 4 *3*	1 1 2 3–I 4 *2*	1 1 2 3 4 *0*	1 0 2 3 4 *3*	1 0 2 3 4 *2*

SPECIALTY	TRAINEES TOTAL (DH/HA/ OTHERS)	TRAINEES — NEW TERRITORIES EAST CLUSTER — AHNH (YEAR)	NDH (YEAR)	PWH (YEAR)	SH (YEAR)	TPH (YEAR)	NEW TERRITORIES WEST CLUSTER — POH (YEAR)	TMH (YEAR)
CARDIOLOGY	26	1 0 2 3 4 ***1***	1 1 2—I 3 4 ***4***	1 3 2—II 3—I 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***2***	1—II 4 2—I 3—I 4 ***6***
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3—I 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	12	1 0 2 3 4 ***2***	1 0 2 3 4 ***4***	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 1 2 3 4—I ***3***
DERMATOLOGY & VENEREOLOGY	7	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 ***0***
ENDOCRINOLOGY, DIABETES & METABOLISM	14	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1—I 4 2—I 3 4—II ***4***
GASTROENTEROLOGY & HEPATOLOGY	23	1 3 2—I 3 4—II ***2***	1 0 2 3 4 ***3***	1 2 2—II 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 3 2 3—III 4 ***7***
GERIATRIC MEDICINE	15	1 0 2 3 4 ***2***	1 0 2 3 4 ***2***	1 1 2—I 3 4 ***5***	1 0 2 3 4 ***6***	1 1 2 3 4—I ***3***	1 1 2—I 3 4 ***3***	1 3 2—II 3 4—I ***7***
HAEM/HAEM ONCOLOGY	13	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 3 2—I 3 4—I ***3***
IMMUNOLOGY & ALLERGY	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***
INFECTIOUS DISEASE	4	1 1 2—I 3 4 ***2***	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 ***2***
INTERNAL MEDICINE	190	1 5 2—II 3 4—III ***14***	1 4 2—I 3—I 4—II ***20***	1 21 2—VII 3—X 4—IV ***57***	1 0 2 3 4 ***6***	1 3 2 3 4—III ***7***	1—II 8 2—II 3 4—IV ***18***	1—V 29 2—X 3—V 4—IX ***55***
MEDICAL ONCOLOGY	2	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 2 2 3—II 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	17	1 1 2 3 4—I ***3***	1 0 2 3 4 ***1***	1 2 2 3—II 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3 4—I ***2***	1 2 2—I 3—I 4 ***6***
NEUROLOGY	10	1 0 2 3 4 ***1***	1 0 2 3 4 ***1***	1 2 2—I 3—I 4 ***8***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***0***	1 1 2 3—I 4 ***4***
PALLIATIVE MEDICINE	4	1 0 2 3 4 ***0***	1 1 2 3 4—I ***0***	1 1 2 3—I 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 ***1***
REHABILITATION	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 ***1***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***3***
RESPIRATORY MEDICINE	11	1 0 2 3 4 ***2***	1 0 2 3 4 ***4***	1 2 2—I 3 4—I ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***3***	1—I 2 2 3 4—I ***5***
RHEUMATOLOGY	8	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***0***	1 1 2 3—I 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1—I 1 2 3 4 ***1***	1 1 2—I 3 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 (DH/HA/OTHERS)	TRAINEES
		DH
DERMATOLOGY & VENEREOLOGY	7	1 7 2—II 3—V 4 ***12***
INFECTIOUS DISEASE	4	1 0 2 3 4 ***3***
RESPIRATORY MEDICINE	11	1 0 2 3 4 ***7***

** 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 July 2017

		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	265	6	6	0	**12**	0	6	17	0	**23**	**35**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	**0**	0	0	2	0	**2**	**2**
CRITICAL CARE MEDICINE	108	8	2	0	**10**	0	0	12	0	**12**	**22**
DERMATOLOGY & VENEREOLOGY	108	0	0	0	**0**	0	0	0	0	**0**	**0**
ENDOCRINOLOGY, DIABETES & METABOLISM	115	6	2	2	**10**	0	0	11	1	**12**	**22**
GASTROENTEROLOGY & HEPATOLOGY	196	5	3	1	**9**	0	0	13	0	**13**	**22**
GERIATRIC MEDICINE	192	6	10	2	**18**	7	3	5	0	**15**	**33**
HAEM/HAEM ONCOLOGY	63	3	0	0	**3**	0	0	10	0	**10**	**13**
IMMUNOLOGY & ALLERGY	6	0	0	0	**0**	0	0	0	0	**0**	**0**
INFECTIOUS DISEASE	42	3	0	0	**3**	0	0	2	0	**2**	**5**
INTERNAL MEDICINE	1424	56	26	9	**91**	6	15	101	10	**132**	**223**
MEDICAL ONCOLOGY	47	0	0	0	**0**	0	0	8	0	**8**	**8**
NEPHROLOGY	139	8	0	0	**8**	0	0	8	4	**12**	**20**
NEUROLOGY	127	6	5	0	**11**	0	0	12	1	**13**	**24**
PALLIATIVE MEDICINE	28	0	2	0	**2**	0	1	1	0	**2**	**4**
REHABILITATION	58	0	1	4	**5**	1	0	1	5	**7**	**12**
RESPIRATORY MEDICINE	192	10	6	1	**17**	0	9	7	0	**16**	**33**
RHEUMATOLOGY	81	4	2	1	**7**	0	0	7	1	**8**	**15**

		FELLOWS															
		KOWLOON CENTRAL CLUSTER							KOWLOON EAST CLUSTER				KOWLOON WEST CLUSTER					KOWLOON CENTRAL + EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/ DH/HA/ OTHERS)	BH	KH	KWH	OLMH	QEH	WTSH	Subtotal	HOHH	TKOH	UCH	Subtotal	CMC	NLTH	PMH	YCH	Subtotal	
CARDIOLOGY	265	0	0	7	2	14	0	23	0	5	8	13	3	0	13	6	22	58
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
CRITICAL CARE MEDICINE	108	0	0	5	0	6	0	11	0	4	7	11	6	0	6	1	13	35
DERMATOLOGY & VENEREOLOGY	108	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
ENDOCRINOLOGY, DIABETES & METABOLISM	115	0	0	4	2	10	0	16	0	5	4	9	3	0	6	3	12	37
GASTROENTEROLOGY & HEPATOLOGY	196	0	0	9	2	11	0	22	0	5	5	10	4	0	9	6	19	51
GERIATRIC MEDICINE	192	1	8	8	2	4	6	29	4	2	13	19	8	1	12	7	28	76
HAEM/HAEM ONCOLOGY	63	0	0	0	0	7	0	7	0	2	3	5	0	0	6	0	6	18
IMMUNOLOGY & ALLERGY	6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
INFECTIOUS DISEASE	42	0	0	0	0	8	0	8	0	0	1	1	0	0	4	0	4	13
INTERNAL MEDICINE	1424	3	12	48	10	95	9	177	7	31	57	95	35	2	79	32	148	420
MEDICAL ONCOLOGY	47	0	0	0	0	2	0	2	0	0	2	2	0	0	0	0	0	4
NEPHROLOGY	139	0	0	9	0	11	0	20	1	2	6	9	3	0	10	2	15	44
NEUROLOGY	127	0	3	4	1	10	1	19	0	2	6	8	1	0	5	3	9	36
PALLIATIVE MEDICINE	28	1	0	0	1	0	1	3	5	0	2	7	5	0	0	0	5	15
REHABILITATION	58	0	9	0	1	0	5	15	1	0	4	5	1	0	1	0	2	22
RESPIRATORY MEDICINE	192	1	7	5	0	8	4	25	6	3	8	17	5	1	7	3	16	58
RHEUMATOLOGY	81	0	1	3	0	6	0	10	0	3	4	7	2	0	5	2	9	26

		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	265	3	7	13	0	0	23	3	11	14	37
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	5	0	0	5	0	0	0	5
CRITICAL CARE MEDICINE	108	6	5	2	0	0	13	0	5	5	18
DERMATOLOGY & VENEREOLOGY	108	0	0	3	0	0	3	0	0	0	3
ENDOCRINOLOGY, DIABETES & METABOLISM	115	2	4	16	0	0	22	2	4	6	28
GASTROENTEROLOGY & HEPATOLOGY	196	3	4	11	0	0	18	4	12	16	34
GERIATRIC MEDICINE	192	1	2	8	7	4	22	4	10	14	36
HAEM/HAEM ONCOLOGY	63	0	0	5	0	0	5	0	7	7	12
IMMUNOLOGY & ALLERGY	6	0	0	0	0	0	0	0	0	0	0
INFECTIOUS DISEASE	42	3	0	3	0	0	6	0	3	3	9
INTERNAL MEDICINE	1424	26	27	91	10	9	163	24	78	102	265
MEDICAL ONCOLOGY	47	0	0	19	0	0	19	0	0	0	19
NEPHROLOGY	139	5	1	11	0	0	17	3	8	11	28
NEUROLOGY	127	2	2	10	2	0	16	3	5	8	24
PALLIATIVE MEDICINE	28	0	0	0	2	0	2	0	1	1	3
REHABILITATION	58	0	1	2	1	1	5	1	3	4	9
RESPIRATORY MEDICINE	192	5	5	8	0	3	21	3	9	12	33
RHEUMATOLOGY	81	1	0	3	0	2	6	1	4	5	11

PROFESSOR PETER MATHIESON

MBBS FRCP, PhD, FMedSci
PRESIDENT and VICE –CHANCELLOR OF
THE UNIVERSITY OF HONG KONG

John Mackay

The Kidney is the Centre of the Universe

This was the title of Prof Mathieson's McFadzean oration in 2015 to the Hong Kong College of Physicians. The kidney has certainly been at the centre of Prof Mathieson's clinical career. His interest in nephrology was first sparked by an admired clinician who taught him during his third year as a university student at the London Hospital. He qualified MBBS with First Class Honours and went on to do training jobs in London hospitals, passing the MRCP in 1986. The following year he was appointed a Registrar in Nephrology at the Hammersmith Hospital where one of his colleagues was Philip Li the current President of the HKCPhys.

At the time he first focussed on nephrology it was thought to be a difficult, esoteric, subject which put some people off. However, he found it intellectually attractive, both the care of acute illness and the fact that many people have a chronic disease which allows for the building up of a long-term relationship. The treatment also requires team-work, a fact that may be relevant to the fact that so many nephrologists that he knows are confident team managers, such as Richard Yu and Philip Li in Hong Kong and the present President of the National University of Singapore.

In 1987 he received a Medical Research Council Fellowship to do research in Cambridge which resulted in a PhD in 1992. He continued working in Cambridge on a further MRC Fellowship at the Addenbrooke's Hospital and as Director of Studies for Clinical Medicine at Christ's College, Cambridge.

In 1995 he was appointed the foundation professor of renal medicine at University of Bristol and honorary consultant nephrologist, North Bristol NHS Trust. In 2008 Peter Mathieson was appointed Dean of the Faculty of Medicine and Dentistry at the University of Bristol. Following the formation of Bristol Health Partners, Peter was appointed as the founding Director in May 2012, a role which he undertook alongside the role of Dean.

In recognition of his achievements, Mathieson was elected Fellow of the Academy of Medical Sciences in 1999. Between 2003 and 2007 he chaired the Research Grants Committee of Kidney Research UK. He was a member of the Renal Association Clinical Trials committee starting 1996, chairman from 2000 – 2003, resigning in 2007 at which date he was elected as the youngest ever president of the Renal Association.

After the six years that he was in Bristol, taking on progressively more responsibility, Prof Mathieson could look back on the considerable improvements he had made in the administration, which had been on his arrival, in a poor state financially, academically and regarding staff morale.

When he was offered the position of Principal and Vice-Chancellor of Hong Kong University he was more than ready to accept a complete change of scene.

Peter Mathieson's family background was not academic. His father was an officer in Trinity House, an organisation founded by King Henry VIII of England in 1514 which has the responsibility of maintaining navigational aids, such as lighthouses, buoys and radio and satellite systems round the coast of England; piloting ships, and distributing charitable donations for the welfare of retired seamen and the training of cadets.

Although his father was Scottish he was posted to various locations, so Peter was born in Colchester in England, spent four years in Swansea in Wales, and was in Penzance, at the south-westerly tip of England when his father died. Peter was only seven. His mother was a nurse and he had one elder brother.

He went to the Humphry Davy Grammar School in Penzance where he did well, being Head Boy for his final year. He enjoyed languages but came to realise that the most likely career option would be as a teacher, so switched to

sciences with which he could train as a doctor. He remarked, ironically, that after becoming a doctor he has enjoyed much of his career as a teacher. At school, he was very keen on sports, playing on the wing for his school Rugby team, which was unbeaten for seven years. He excelled also in athletics, sprinting and high jump. A more leisured pastime was photography.

Having achieved the necessary 'A' level qualifications he was accepted to study medicine at London University Hospital. It was there during their first year that he and his future wife met. She was studying dentistry. They married when she finished her four-year course and he was still a student. They had two children, a son who is a trader, married, and has just presented the Mathiesons with their first grandchild; and a daughter who is a clinical psychologist.

In Hong Kong, he and his wife have found much to enjoy; the companionship of friends in the medical community, the students who are hard-working, respectful and polite, though some have been distracted by politics which is regrettable. One particular pleasure was being awarded a Fellowship of the Hong Kong College of Physicians. Although the hospital in Shenzhen was described to him by some when he arrived as a "problem", he feels that following clarification of their respective roles and expectations, The University of Hong Kong and the Shenzhen authorities are agreed on a way forward, and the patient numbers are increasing.

Professor Matheson is leaving Hong Kong one year before the end of his term as President despite the fact that there is much that he would still like to contribute here. He is confident that he will be revisiting Hong Kong in the future in his capacity as Principal and Vice-Chancellor of The University of Edinburgh. He feels that his knowledge of Hong Kong and China was a factor in his appointment to Edinburgh, where the University has always had a very international outlook, even more so these days when the relationship with European Union countries is becoming less assured.

He is looking forward to reexperiencing the city of Edinburgh. He remembers being taken by his Scottish father to the heights of Edinburgh Castle when he was a small child, to look over the city. A city to which he will now return as Principal and Vice- chancellor of the university founded in 1583, a community with 45,000 students and staff. He already has the Clan Matheson tartan tie to wear.

In Memoriam: Professor Sir David Todd (1928 - 2017)

Dear Fellows and Members,

The Hong Kong College of Physicians mourns with immense sadness the passing away of Professor Sir David Todd on 16th August 2017.

Sir David has been the Giant of Medicine in Hong Kong and was beloved by all his students, colleagues and friends. He has been a role model for all of us as a most distinguished physician, scientist and medical educationist. Sir David's tremendous respect and affection for his patients became the life force of his career. Even more extraordinary was his ability to influence those around him, his students and colleagues, to care for their patients with passion, compassion and humility and at the same time exercising clinical skills and scientific advancement.

Sir David's grand vision in fostering and upholding professionalism in medicine led to the creation of the Hong Kong College of Physicians. He founded our College in the year 1986 with a legion of senior physicians and became the Founding President. Sir David's work has contributed enormously to advancing the science and art of Medicine, developing and maintaining professional standards and ethical integrity, and promoting and monitoring advanced medical education. He remained as President of our College until 1992 and has served as an ex-officio Council Member ever since. Under Sir David's leadership, a 1985 agreement with the Royal Colleges of Physicians of United Kingdom was made to hold the entire MRCP examinations in Hong Kong. This was the first time such a course was pursued, and history attests to the trust the Royal Colleges have placed on Sir David as Chief Organiser and Examiner, and their confidence in the professional training Sir David had been able to develop and uphold. The David Todd lectureship was created by the College in 1996 to award and honor distinguished young physician scientist. In recognition of all the groundbreaking contributions that Sir David made to internal medicine in Hong Kong, the College conferred Honorary Fellowship to him in 1997.

The success of our College has prompted the establishment of other specialties in internal medicine after 1993. Under the leadership of Sir David, such good work was extended to specialist practitioners of medicine at large. After serving as Chairman of the Preparatory Committee and President of the Interim Council (1990-1992), Sir David became Founding President of the Hong Kong Academy of Medicine in 1992 and served till 1996. He was instrumental in introducing continuing medical education as a mandatory requirement for specialist registration, proving to the community that professional competence to be safe guarded among the Academy's Fellows. Sir David was conferred Honorary Fellow of the Academy in 1997.

Professor Todd ("Tat Yeh" as his fond nickname to all of us) was the Professor of Medicine, University of Hong Kong (1972-1974), and succeeded Prof AJS McFadzean as Head in 1974-1989. He served as Sub-Dean of Medicine (1976-78) and Pro-Vice Chancellor (1978-80). He retired as Emeritus Professor in 1994. All his students, regardless of what field they go into, have very fond memories of him. Countless numbers of medical leaders in Hong Kong in the past 40-50 years, be they in public clinical service, academia, health administration or private practice, certainly felt the honor of being able to be his students at times.

Sir David's contribution to public service was equally illustrious. He was Chairman of the Research Grants Council (1991-93), a member of the first Hospital Authority Board in 1990 and Chairman of its Health Services Research Committee (1993-97), Chairman of the AIDS Trust Fund (1993-97). He also served in the Trustee of Croucher Foundation, SK Yee Medical Foundation and Chevening Scholarship Board of Advisors. The list is too numerous to be comprehensive. With all his contributions Sir David was awarded OBE (1982) and CBE (1990), and enjoyed the distinction of being the first person in Hong Kong to be knighted for his contribution to Medicine in 1995.

To many of us, Sir David was a very powerful man in the field of Medicine; but he never acted like one. He was a gentleman, a pioneer, a legend, a great human, and a great humanitarian act with humbleness and humility. His unsurpassed intellect, passion and courage has changed the landscapes of Medicine of Hong Kong. Our College offers the greatest salute to this icon of Medicine of Hong Kong and mourns over the loss of him. Sir David will always be in our heart and our College offers the deepest condolences to Sir David's family.

Professor Philip KT Li
President
Hong Kong College of Physicians

Professor Richard YH Yu
Senior Advisor
Hong Kong College of Physicians