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PRACTICAL APPROACH TO CLINICAL ETHICS AT THE BEDSIDE FOR GENERAL PHYSICIANS

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Dr Chun Yan TSE CHAIRMAN, HOSPITAL AUTHORITY CLINICAL ETHICS COMMITTEE



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APPROACHES TO CLINICAL ETHICS

When a junior physician is asked about approaches to clinical ethics, the principle based approach raised by Beauchamp and Childress is often quoted.¹ The four principles involved are respect for autonomy, beneficence, non-maleficence, and justice. While the four principles are well known among clinicians, in the academic literature, there are quite a number of other approaches.² These include deontological approaches (e.g. Kantian approach or religious doctrines, in which duties or rules are moral absolutes), utilitarian approaches (consequentialism, looking for the greatest happiness for the greatest number of people), care based approaches (emphasizing moral attention, sympathetic understanding, relationship awareness, accommodation and harmony), virtue ethics approaches (e.g. Confucianism, looking at what a virtuous person would do in the circumstances), case based approach (analogy thinking, using paradigm cases judged to be manifestly right or wrong). The principle based approach of Beauchamp and Childress is often favored by clinicians, possibly because it covers different perspectives, looks rational, and appeals to intuition.

SPECIAL ARTICLES

However, to make good use of the four principles is sometimes not easy. They may be in conflict in some situations. None of the four principles are absolute, and they have to be weighed against each other. There is also no magic formula in weighing the principles. The consideration is context dependent, and is related to the local legal and cultural situations. For example, the respect for autonomy prevails over other considerations when a competent patient refuses a medical treatment. The clinician cannot forcibly give treatment to the patient for beneficence, even though the clinician considers that the treatment will save the patient's life. This is in line with the legal requirement of informed consent. On the other hand, when a patient demands a medical treatment, instead of just respecting autonomy, other principles have to be weighed. If the treatment has minimal benefit to the patient, but is invasive and will definitely cause a lot of harm, the principle of non-maleficence may over-ride autonomy. Similarly, if the expected benefit is very small but the treatment is very expensive, in a public system, the principle of justice has to be considered. Sometimes, there is difficulty in balancing the principles, and ethical dilemma may result.

CONTEXTUAL FACTORS

The balancing of the various principles often depends on the actual situation in an individual case, and there may not be a broad-brush answer for broad categories of clinical situations. Faced with an ethical problem, it is important to have a system to ensure adequate consideration of various contextual factors in an individual case. One useful system is the "four quadrants" approach.³ The four quadrants that must considered are medical indications, patient preferences, quality of life, and contextual features. Going through various issues in the four quadrants systematically, important factors would not be neglected.

CONSENSUS BUILDING AND REASONED DISCUSSION

In many clinical situations, value judgment is involved in the decision making process. Different parties involved in the ethical dilemma may hold different values. In order to arrive at a decision, consensus building among involved parties is important. For bedside situations, it often means consensus building among the patient, the family members and the healthcare professionals. During the discussion process, the goal to achieve should be clearly understood by the involved parties. For bedside situations, the goal is most often to decide "what is in the best interests of the patient", and not what the clinician prefers or what the family members prefer. Conflicts among different parties may be resolved by communication to clarify incorrect information or unrealistic expectation. The clinician should adopt an open, sincere, and empathetic attitude, be a good listener, and be sensitive to emotions experienced by the patient and family members. The clinician should be receptive to the values of the patient in making a judgment. Addressing relevant questions step by step before coming to a final decision may be useful. If there is difficulty in arriving at a consensus, consultation to an ethics committee may be useful.

INSTITUTIONAL OR PROFESSIONAL GUIDELINES

For complex situations, setting up guidelines by healthcare institutions or by professional bodies would help. Such guidelines should delineate the overall approach to the ethical problem, and suggest ways to handle conflicts and disagreements. The guidelines should be evidence based. The drafting process should involve relevant experts of different background, taking into account international trends and local situations. The guidelines are not there to give simple answers to complex ethical problems, but are to guide the clinician through the decision-making process. If the decision-making process recommended by the guidelines is followed appropriately, the guidelines may provide backup to the clinician when faced with challenges to the final decision.

CASE ILLUSTRATIONS

I would like to use two cases to illustrate the approach to clinical ethics problems at the bedside. Both cases were presented in a session chaired by me in the Clinical Ethics Day on 23 April 2016 in the Hospital Authority Head Office Lecture Theatre, co-organized by The Chinese University of Hong Kong Centre for Bioethics and the Hospital Authority Clinical Ethics Committee. Many thanks to Dr. Wong Che Keung of Ruttonjee and Tang Shiu Kin Hospital, and Dr. Frank Wong of Tuen Mun Hospital, for allowing me to use the clinical materials of the two cases respectively. The discussion in this paper is my own, taking into account comments from panelists and members of the audience during the discussion session. Panelists involved in my session were Dr. Doris Tse of Caritas Medical Centre, Professor Chan Ho-mun of City University of Hong Kong, and Ms. Alexandra Lo, lawyer in private practice.

Case 1: Tube feeding in a dying demented patient

The first case was an 84 years old male, with a history of hypertension, diabetes and recurrent ischaemic stroke. His wife died a few years ago. He had two sons and one daughter living in Hong Kong. He was diagnosed to have vascular dementia in 2010, became chair bound and was nursed in a private old aged home from 2012. In 2015, he became bed bound and double incontinent, requiring assisted feeding. He had recurrent admissions in 2015 due to chest infection, and the speech therapist recommended puree diet and thickener in fluid. After an episode of aspiration pneumonia, the speech therapist suggested non-oral feeding due severe oropharyngeal dysphagia. Family conference was held with the second son and the younger daughter, and they both preferred careful hand feeding rather than tube feeding, because the patient voiced out his dislike against tube feeding while in the old age home. They understood the risk of aspiration, pneumonia and death. The patient tolerated careful hand feeding of a few hundred mls per day. At the end of 2015, he developed severe pneumonia. He was kept nil by mouth and given parenteral antibiotics. His relatives were informed of deteriorating clinical condition and imminent death. They understood and agreed to continue conservative management. However, the second son later turned up and requested to start tube feeding. He accepted that his father was dying and agreed to continue comfort care and continue DNACPR order, but he wanted his father to die "with a full stomach", which was a traditional preference among some elderly persons in Hong Kong. The question is whether the clinician should follow the son's request.



SPECIAL ARTICLES



We can approach the problem step by step:

- Was tube feeding going to prolong the patient's life at this stage? The patient was dying from the severe pneumonia. Starting tube feeding would not prolong the patient's life. Inserting a feeding tube was not comfortable, and there could even be risk of further aspiration if tube feeding were started.
- 2. Was the wish to die with a full stomach the wish of the patient?

The request was raised by the patient's son and not the patient. There was no evidence that the patient previously requested, while competent, to have a full stomach in the dying phase. On the contrary, the patient previously voiced out his dislike against tube feeding.

3. Balancing the benefits and harms, should tube feeding be provided?

Decision to tube feed or not should depend on whether the treatment was in the patient's best interests, rather than what the family members preferred. Here, balancing the benefits and harms, and taking into account the wish of the patient, it should be quite clear that tube feeding was not in the patient's best interests and thus should not be provided.

4. If tube feeding should not be provided, how should we handle the son's request?

We should explain to the son that treatment given should be in the patient's best interests, and that tube feeding would do more harm than good to the patient and was not in line with the wish of the patient. It would be better for the patient's son to understand the rationale of the final decision, rather than just telling him that he had no right to request the treatment.

Case 2: HIV disclosure

The second case was a 40 years old male patient. He was newly diagnosed to have a cancer disease, of which AIDS was one of the known etiological factors. Though there was no history suggestive of HIV exposure, screening of HIV was done as part of a standard protocol, after obtaining consent from the patient. His cancer disease deteriorated rapidly, and he became unconscious from a respiratory complication, requiring mechanical ventilation. Then, the HIV result came back positive. The original consent did not mention whether our professional staff could disclose this information to a particular third party or not. The question is whether the HIV information should be disclosed to his wife, who should be at risk of being infected.

Again, we can approach the problem step by step:

1. Ethically, did the risk of HIV infection in his wife justify over-riding a respect for autonomy and allow disclosure without consent?

His wife had a high risk of being infected, and it was important to let her know about the risk. In this particular situation, most people would agree that justice over-rides respect for autonomy, and disclosure without consent could be allowed.

2. Does the local law and code of professional conduct allow such a disclosure?

The Personal Data (Privacy) Ordinance allows an exception to the duty of confidentiality if non-disclosure would likely cause serious harm to another individual.⁴ The Code of Professional Conduct of the Medical Council of Hong Kong also allows such exceptional disclosure.⁵

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3. Should the HIV status be disclosed immediately or should one wait to see if the patient could regain consciousness within a short time to give proper consent?

To avoid the ethical dilemma, an alternative is to wait till the patient regains consciousness and gives consent, if recovery is possible within a short time. But before choosing this alternative, one needs to know whether there are strong indications for immediate disclosure.

4. Regarding risk to his wife, was there strong indication to justify immediate disclosure?

As the patient was hospitalized and the wife was not further exposed, the risk to his wife would not be substantially increased by waiting for a short time before disclosure.

5. Regarding benefit towards the patient, was there strong indication to justify immediate disclosure?

Discussion with family members regarding medical decision in the best interests of the patient could be made effectively without disclosure of HIV status.

6. If recovery within a short time was not likely, what procedural safeguards are required to ensure a prudent decision?

Both the relevant guidelines of the Hospital Authority,⁶ as well as the Advisory Council on AIDS & Scientific Committee on AIDS and STI of the Department of Health,⁷ recommend referral to the institutional ethics committee (or its equivalent) before any decision is made to breach confidentiality. Of course, the decision-making process of the ethics committee should be timely, in order to avoid undue delay in disclosure to the sex partner.

Conclusion

The above two examples illustrate a practical approach to clinical ethics at the bedside. One can see that there are no fixed rules or procedures in solving ethical problems. The approach is context dependent and much depends on common sense. Going through issues step by step may help. Consensus building and reasoned discussion are important in resolving differences in opinion. Institutional or professional guidelines would be useful in complex situations. As new and complex medical technologies are being developed, new ethical issues may emerge. It is important for individual clinicians, healthcare institutions as well as professional bodies to pay attention to ethical problems, in order to ensure medical practice that is not only competent, but also considered to be right in the particular context, and is able to stand against challenges.

REFERENCES

- 1. Beauchamp, Tom L., and James F. Childress. Principles of Biomedical Ethics. 5th ed. New York: Oxford University Press, 2001.
- Kuhse, Helga, and Peter Singer, eds. A Companion to Bioethics. 2nd ed. Chichester, U.K.; Malden, MA: Wiley-Blackwell, 2009.
- Sokol, D. K. The "four quadrants" approach to clinical ethics case analysis; an application and review. J Med Ethics 34 (2008): 513-516. Jonsen, Albert R., Mark Siegler, and William J. Winslade. Clinical Ethics: A Practical Approach to Ethical Decisions in Clinical Medicine. 7th ed. New York: McGraw-Hill Medical, 2010.
- 4. Section 59 of the Personal Data (Privacy) Ordinance Cap 486 of the Government of the Hong Kong S/
- Section 32.3 of the 2016 Code of Professional Conduct of the Medical Council of Hong Kong.
 Para 4 of the 2008 Ethical Guidelines on Disclosure of HIV Status of Mentally Incapacitated Patients without
- 6. Para 4 of the 2008 Ethical Guidelines on Disclosure of HIV Status of Mentally Incapacitated Patients witho a Legal Guardian to the Sex Partners of the Hospital Authority.
- 7. Para 26 of the 2011 Guidelines Principles of Consent, Discussion and Confidentiality Required of the Diagnostic HIV Test of the Advisory Council on AIDS & Scientific Committee on AIDS and STI of the Department of Health.



COUNCIL NEWS

HKCP Annual General Meeting 2016

The College will be celebrating its 30 th anniversary this year. The Annual Scientific Meeting will be held on 15 – 16 October 2016 at the Hong Kong Academy of Medicine Jockey Club Building, followed by the Annual General Meeting and Annual Dinner on the 15 October 2016.

- Lunch Symposium on New Oral Anti-Coagulants and Atrial Fibrillation
- Symposium on Mosquito-borne infections Zika virus, Dengue, Japanese Encephalitis and Malaria
- Sir David Todd Lecture
- Symposium on Recent therapeutic advances in epilepsy and electrolytes
- Gerald Choa Memorial Lecture
- Best Thesis Award
- Richard Yu Lecture
- Distinguished Research Paper Award for Young Investigators
- Symposium on Clinical Management Updates in psoriatic disorders and IgA Nephropathy

Congratulations

The Council wish to extend heartiest congratulations to our Fellows !

New elected Fellows of the Royal College of Physicians, Edinburgh (2016)

- Dr Chan Chin Pang Ian Department of Medicine & Geriatrics, United Christian Hospital
- 2. Dr Chan Lee, Veronica Department of Medicine & Geriatrics, United Christian Hospital
- Dr Chan Pierre Integrated Medical Service, Ruttonjee Hospital
- Dr Cheung Chun Fong, Jane Department of Medicine & Geriatrics, Pok Oi Hospital
- 5. Dr Chow Bing Fai Integrated Medical Service, Ruttonjee Hospital
- 6. **Dr Chu Yin Yiu Stephanie**, Department of Medicine, Queen Elizabeth Hospital
- 7. **Dr Fan Hon Cheung** Integrated Medical Service, Ruttonjee Hospital
- 8. Dr Lai Wing Kin Andrew Department of Medicine & Geriatrics, Tuen Mun Hospital
- 9. **Dr Lam Sin Man** Intensive Care Unit, Pamela Youde Nethersole Eastern Hospital
- 10. Dr Law Tse Sam, Grace Private Practice

- Dr Law Wai Lam Department of Medicine, Queen Elizabeth Hospital
- 12. **Dr Lee Kwok Kuen Harold** Department of Medicine & Geriatrics, Princess Margaret Hospital
- Dr Lee Wai Chuen, Raymond Department of Critical Care Medicine, Hong Kong Sanatorium & Hospital
- Dr Leung Chi Man Department of Medicine, Pamela Youde Nethersole Eastern Hospital
- 15. **Dr Leung Wah Shing** Department of Medicine & Geriatrics, United Christian Hospital
- Dr Ling Sai On Department of Rehabilitation and Extended Care, Kowloon Hospital
- Dr Lo Ho Yin Department of Medicine, Pamela Youde Nethersole Eastern Hospital
- Dr Lo Hok King Stanley Department of Medicine, Pamela Youde Nethersole Eastern Hospital
- Dr Miu Lui Ling Flora Department of Medicine, Pamela Youde Nethersole Eastern Hospital
- 20. **Dr Mok Wing Yee Winnie** Department of Geriatrics, Fung Yiu King Hospital

- 21. **Dr O Wing Hing** Department of Medicine, Queen Elizabeth Hospital
- 22. **Dr Poon Yik Ning** Department of Respiratory Medicine, Kowloon Hospital
- 23. **Dr Shiu Ka Lock** Department of Medicine & Geriatrics, Pok Oi Hospital
- 24. **Dr Shum Hoi Ping** Intensive Care Unit, Pamela Youde Nethersole Eastern Hospital
- 25. **Dr To Kin Wang** Department of Medicine & Therapeutics, Prince of Wales Hospital
- 26. **Dr Wan Man Choi** Integrated Medical Service, Ruttonjee Hospital
- 27. **Dr Wong Wei Yin** Department of Medicine, Haven of Hope Hospital
- 28. **Dr Yap Yat Hin Desmond** Department of Medicine, Queen Mary Hospital
- 29. **Dr Yim Chie Wai** Department of Rehabilitation Medicine, Kowloon Hospital

CHRONIC HEPATITIS B TREATMENT FOR SPECIAL GROUP OF PATIENTS

Yuen Man Fung

Chair Professor of Gastroenterology and Hepatology Li Shu Fan Medical Foundation Professor in Medicine Department of Medicine, The University of Hong Kong

INTRODUCTION

Chronic hepatitis B (CHB) infection is still of high prevalence in most of the Asia Pacific countries. According to the preliminary data from a recent territory-wide population study in Hong Kong, the CHB infection rate in the general population is around 8 – 9% ⁽¹⁾. Disease knowledge and management of CHB are constantly being updated over time. While the main focus is usually on CHB mono-infected patients, managing CHB patients with special features should not be ignored. This article discusses the features and management issues of CHB in three different special populations, namely, patients with pregnancy, patients co-infected with human immunodeficiency virus (HIV) infection, and patients co-infected with hepatitis C virus (HCV) infection. Because of different special accompanying features inherited in these special populations, unique considerations for the management for the CHB are often required.

PREGNANCY AND CHRONIC HEPATITIS B

Gestational complications in CHB mothers and their infants

According to a study of 253 pregnant CHB mothers and 253 pregnant mothers without CHB infection conducted by Tse et al ⁽²⁾, there were increased threatened preterm labour (p = 0.03), increased chance of gestational diabetes mellitus (p = 0.033), increased chance of ante-partum hemorrhage (p = 0.026), lower infant Apgar score (p = 0.001) as well as increased infant brain hemorrhage (p = 0.007). According to another study, CHB or HCV infection was associated with low birth weight, congenital malformations, and preterm labour ⁽³⁾. However, these findings were not supported by two others studies: one from Germany with 8,193 mothers, 38 of whom were positive for hepatitis B surface antigen (HBsAg) ⁽⁴⁾; and one from Hong Kong with 13,792 mothers, 1,340 of whom were HBsAg-positive ⁽⁵⁾. The potential adverse effects of CHB on pregnant mothers who are otherwise healthy and on their infants therefore require confirmation from more studies.



Nevertheless, the adverse effects of CHB on pregnancy are more obviously observed in patients who are already cirrhotic. In a population-based study of 339 cirrhotic women versus 6,625 non-cirrhotic controls, maternal mortality was increased from 0% to 1.8% (p <0.0001), and fetal mortality was increased from 2.1% to 5.2% (p <0.0001)⁽⁶⁾. From the 15% of mothers who had hepatic decompensation, the maternal and fetal mortality was 6% and 12% respectively.

Post-delivery reactivation of CHB

During the pregnancy period, there is usually no deterioration of liver condition in mothers with CHB infection. Although there are uncommon cases with HBV exacerbation with or without liver failure, most of the HBV pregnant patients have normal liver enzyme levels ⁽⁷⁾. Nevertheless, post-delivery HBV reactivation has been well recognized and first reported by Rawal et al ⁽⁸⁾ and later confirmed by a more recent study by ter Borg et al ⁽⁹⁾. They investigated 38 pregnancies in 31 CHB mothers, 63% of whom were positive for hepatitis B e antigen (HBeAg). Upon six months after delivery, 45% had three fold or more increase in serum alanine transaminase (ALT) levels. In the 13 patients who were treated with lamivudine during the last trimester with the lamivudine withdrawn after delivery, the reactivation rate was even higher at 62%.

During pregnancy, the maternal immune system is altered to prevent rejection of the fetus. This will also enhance the replication of the hepatitis B virus (HBV). Restoration of the immune system post-delivery may lead to enhanced immune mediated attack on the heavily infected hepatocytes. HBV DNA should therefore be monitored for at least 6 months after delivery. Anti-viral treatment started during pregnancy should not be stopped prematurely in order to prevent reactivation.

Maternal-to-child transmission of the HBV

Early studies in the 1970s show that while HBeAg-positive mothers transmitted HBV to 63% of their infants, mothers who were HBeAg-negative could still transmit the infection to 25-30% of their infants ^(10, 11). One of the pioneering studies of the hepatitis vaccine was carried out in 140 HBeAg-positive mothers ⁽¹²⁾. In this study, intrauterine infection occurred in 2.1% of the infants. Seventy-three percent of infants in the control group became infected. The infection rates were reduced to 21% for infants receiving vaccine alone, 6.8% for those receiving vaccine plus one dose of hepatitis B immune globulin (HBIG) and 2.9% for those receiving vaccine plus 2 doses of HBIG (p = 0.0001 for all groups vs controls).



More recent studies have been performed to investigate the levels of viremia which are associated with transmission of the infection to infants. In one study of 869 CHB mothers in China with all infants receiving the hepatitis B vaccine plus one dose of HBIG, 3.1% of the infants were HBsAg-positive at 7-12 months after birth ⁽¹³⁾. The independent risk factors for transmission were cord blood positivity for HBV DNA and maternal HBV DNA levels of $\geq 2 \times 10^5$ IU/mL. Another study from Australia included 138 babies from 313 CHB mothers ⁽¹⁴⁾. Three percent of the infants became HBsAg-positive. None of the infants from mothers with HBV DNA < 2×10^7 IU/mL became infected. From these two studies, infants with maternal HBV DNA levels above \geq 5-7 log IU/mL are at risk of becoming infected despite hepatitis B vaccine and HBIG.

Efficacy and safety of antiviral therapy in the prevention of maternal-to-child transmission of HBV

There have been multiple studies to determine the efficacy of nucleos(t) ide analogues given to mothers during the last trimester in preventing transmission of HBV to the infants. The results of these studies are summarized in Table 1 ⁽¹⁴⁻²⁰⁾. The maternal HBV DNA levels varied from 10⁵ to 10⁸ IU/mL. With the exception of the earliest study with maternal HBV DNA levels of 10⁸ IU/mL before the mothers were treated with lamivudine ⁽¹⁴⁾, all the other studies showed that nucleos(t)ide analogues, whether lamivudine, telbivudine or tenofovir, can reduce the transmission of HBV to the infants to 0-3.7%. (In the earliest study with lamivudine, the high transmission rate to infants of 18% even after the mothers were given lamivudine may be partly related to the very high HBV DNA levels of the mothers, but may also be related to non-compliance with the full three doses of the hepatitis vaccination.)



Nucleos/tides	Controls	Maternal HBV DNA (IU/mL)	Infant HBV infection rate (%)	P values	References
Lamivudine (n=56)	Placebo (n=59)	>10 ⁸	18 vs. 39	0.003	Xu et al(14)
Telbivudine (n=135)	Untreated (n=94)	>106	0 vs. 8	0.002	Han et al (15)
Telbivudine (n=53)	Untreated (n=35)	>10 ⁵	0 vs. 8.6	0.029	Pan et al (16)
Telbivudine (n=233); lamivudine (n=154)	Untreated (n=100)	>10 ⁵	0; 0 vs. 5	0.002*, 0.009+	Yu et al (17)
Telbivudine (n=252); lamivudine (n=51)	Untreated (n=345)	>10 ⁵	1.9; 3.7 vs. 7.6	0.001	Zhang et al (18)
Tenofovir (n=21)	Untreated (n=24)	>106	0 vs. 8.3	0.022	Celen et al (19)
Tenofovir (n=62)	Untreated (n=56)	>10 ^{7.5}	1.5 vs. 10.7	0.048	Chen et al (20)

Table 1 The efficacy of antiviral treatment in preventing maternal to infanttransmission of the hepatitis B virus in seven studies

* p value for the comparison between telbivudine and controls; + p value for the comparison between lamivudine and controls.



As far as the safety to the fetus is concerned, telbivudine and tenofovir both belong to category B according to the Food and Drug Administration of the United States, i.e., the drugs are not associated with fetal abnormalities in animal studies. Lamivudine, adefovir and entecavir are classified as category C in which adverse effects were demonstrated in animal studies. According to the Antiretroviral Pregnancy Registry (APR)(www.apregistry.com) recording the possible teratogenic effects of drugs in women who were positive for the human immunodeficiency virus (HIV) from January 1989 to July 2015, of the 16,699 live births from patients exposed to anti-retroviral agents at any time during pregnancy, there were 473 outcomes with birth defects i.e. 2.8 birth defects/ 100 births. This rate is comparable to the birth defect rates of normal pregnancy of 2.7% (data from Center for Disease Control). Birth defect rate of infants in lamivudine-treated women was 143 out of 4566 pregnancies (3.1%) and in tenofovir-treated women was 60 out of 2608 pregnancies (2.3%). While the Registry cautions that there may be potential under reporting, there are already two studies showing that there are no differences in the congenital abnormalities and infant growth between HBV pregnant mothers with and without tenofovir treatment ^(21, 21). A systemic review of telbivudine in 1,693 HBV infected pregnant women showed a birth defect rate of 2.5/1,000 compared to non-antiviral controls of 3.4/1,000 births ⁽²³⁾. There is insufficient data for entecavir and adefovir.

The duration of therapy post-delivery is not clearly defined. Maternal serum ALT levels should be carefully monitored for reactivation in mothers in whom antivirals are stopped. The incidence of reactivation varying for 5% ⁽¹⁸⁾ to 62% ⁽⁹⁾. For mothers whose ALT and HBV DNA levels during pregnancy fall within the recommended levels for initiation of antiviral treatment, the nucleos(t)ides should preferably be continued on a long-term basis.

Data on the safety for infants breastfed by mothers on antiviral treatments are lacking. There were cases where HIV or HBV infected mothers continued with tenofovir, showing that there was a minimal if not negligible level of tenofovir in the breast milk ^(24, 25). According to several cases with HBV mothers on tenofovir, there were no short term adverse effects and the infants remained HBsAg negative ⁽²⁶⁾. It has been shown that different feeding methods do not increase the risk of transmission of the virus to the infants as long as they have received HBIG and the first dose of the hepatitis B vaccine ⁽²⁷⁾.

CO-INFECTION WITH HIV AND HCV

HBV and HIV co-infection

Concomitant HBV and human immunodeficiency virus (HIV) infection is not an uncommon disease because of the shared common routes of the virus transmission. It is estimated that 5-15% of the 40 million HIV infected patients in the world are co-infected with HBV^(28, 29). With the effective treatment by the highly active anti-retroviral therapy (HARRT), the outcome of HIV infection improves dramatically. These patients are now suffering from the morbidity and mortality from CHB infection.

HBV/HIV co-infected patients have higher rates of occurrence of hepatitis flares and liver disease progression compared to patients with HBV mono-infection ⁽³⁰⁾. For example, patients with HBV/HIV co-infection has 8 and 18 folds increased chance of liver-related mortality compared to patients with mono-HIV infection and mono-HBV infection respectively ⁽³¹⁾.

To date, treating CHB in HBV/HIV co-infected patients is of prime consideration to improve their life expectancy. While the criteria and duration of the treatment of CHB in patients with mono-infection of HBV are still under active debate, treatment decision and regimen for CHB in HBV/ HIV co-infection further add the complexity as the activity of the HIV needs to be taken into consideration. It is mainly because some anti-HBV agents have anti-HIV activity which may lead to the development of resistance of HIV.

The criteria of treatment initiation for CHB should be at least the same as, if not less stringent, as those for CHB mono-infection. In this case, significant HBV viraemia (HBV DNA > 2,000 IU/mL) with evidence of liver inflammation as indicated by elevated ALT levels should be the treatment initiation criteria. Although there are still arguments on whether the treatment should be given for long-term or stopped after achieving certain goals in CHB monotherapy, treatment for HBV/HIV co-infection is generally accepted to be long-term. It is because of the comparatively lower response rates and the necessity of continuous HIV treatment, which may provoke immune-mediated attack against the HBV disease after the immune reconstitution.

As mentioned above, HIV activity has a major determinant role on the treatment regimen for HBV/HIV co-infection. For patients who do not need HIV treatment e.g. CD4 count > 500 cells/mm³ and low HIV RNA levels (<100,000 copies/mL), anti-HBV agents without activity against the HIV should be considered. This is to avoid the potential emergence of HIV resistance if these patients need anti-HIV therapy in the future. In this setting, pegylated interferon, adefovir (at 10 mg daily dose), and telbivudine are the options.

It has been shown that the treatment response in HBV/HIV is poorer compared with that of HBV mono-infection when conventional interferon is used ⁽³²⁾. The short-term response to interferon-alpha therapy was found in 28% of patients with HBV/HIV co-infection compared to 51% of patients HBV mono-infected patients (p=0.06). The response was even poorer in HBV/ HIV co-infected patients with low CD4 counts. The HBV reactivation rate was also higher in HBV/HIV co-infected patients. Miailhes and colleagues studied the efficacy of 48 weeks of pegylated interferon in 51 HBV/HIV co-infected patients. Twenty percent of patients had loss of HBeAg at week 72 and only 8% of patients had sustained response (33). Concerning the side effects of interferon, the potential negative effect on the CD4 counts secondary to its bone marrow suppression needs special attention in HIV infected patients.







Because of the absence of anti-HIV activity when used at 10 mg daily, adefovir may be considered to treat the CHB in HBV/ HIV co-infected patients with quiescent HIV disease. It is however noteworthy that adefovir has only modest HBV suppressive effect with a moderately high rate of development of HBV resistance (20% after 3 years and 29% after 5 years for HBeAg-positive and – negative patients respectively)⁽³⁴⁾. Another possible agent with more profound HBV DNA suppression is telbivudine. It is however, hindered again by the considerably higher rate of resistance (22% and 8.6% after 2 years of treatment for HBeAg-positive and –negative patients respectively).

The two most potent antiviral agents, namely entecavir and tenofovir for CHB infection are not recommended for HBV/HIV treatment in patients who do not require HIV treatment at the time of assessment of both diseases. For entecavir, it has been reported to have mild anti-HIV activity (1 log copies/ml reduction in HIV RNA)⁽³⁵⁾. In addition, M184V, the HIV-1 variants with the lamivudine resistant mutation which has cross resistance profile with entecavir has been found in one out of three HIV/ HBV co-infected patients who have HIV RNA reduction on entecavir monotherapy without HARRT⁽³⁶⁾. Therefore, entecavir may potentially increase the risk of HIV resistance in patients who are not receiving HARRT.

For tenofovir, it has potent viral suppression in both HBV and HIV monoinfections. In HBV/ HIV co-infection, an average of 4 logs reduction of HBV DNA has been demonstrated in several studies ⁽³⁷⁻⁴¹⁾. According to a study conducted in the Netherlands ⁽⁴²⁾, undetectable HBV DNA was achieved in more than 95% of HBV/HIV patients after 5 years of tenofovir treatment.

It also has an outstanding viral mutation profile with no development of clinically significant resistance mutations to HBV. Whether A194T mutation found in patients with suboptimal viral suppression confer actual phenotypic resistance needs further exploration ⁽⁴³⁾. However, it is not recommended in patients who do not require HIV treatment because of its anti-HIV effect.

In a clinical setting where treatments for both HBV and HIV infections are necessary, anti-viral agents with both anti-HBV and anti-HIV activities are recommended. HAART regimen with intrinsic potent anti-HBV activities e.g. tenofovir with lamivudine or with emtricitabine should be considered. Lamivudine or emtricitabine monotherapy for HBV should be strictly avoided because of the great concern of HBV resistance mutations. For instance, according to the study conducted in France ⁽⁴⁴⁾, more than 90% of patients developed lamivudine resistance after 4 years of lamivudine therapy. In a situation where tenofovir is relatively contraindicated e.g. patients with renal insufficiency, HAART without tenofovir with addition of entecavir should be considered to control the HBV infection.

In patients who are already under lamivudine-containing HARRT, continuous and close monitoring of HBV viraemia is mandatory. If there are viral breakthroughs suggestive of the emergence of lamivudine resistance, tenofovir or adefovir should be added to control the viral replication. It has been shown that 63% and 25% of these patients can achieve undetectable HBV DNA after 48 weeks of tenofovir and 144 weeks of adefovir respectively^(45, 46).

In the final scenario, where only HIV infection is required to be treated and the HBV disease is quiescent without the need of treatment, two drugs active against HBV in a fully potent HAART regimen e.g. tenofovir with lamivudine or with emtricitabine is the recommended treatment regime ^(47, 48). The aim is to prevent HBV exacerbation due to immune reconstitution syndrome after the initiation of HARRT treatment.

HBV and HCV co-infection

Similar to HBV/HIV co-infection, certain percentages of patients have coinfection of HBV and HCV because of the common route of parenteral transmission. Prevalence of HBV/HCV co-infection varies in different countries. It is generally found that 5-10% of CHB patients are positive for anti-HCV whereas 2-10% of chronic hepatitis C (CHC) patients are positive for HBsAg ⁽⁴⁹⁻⁵²⁾. Intravenous drug users, patients on haemodialysis and patients with organ transplantation are considered to be the high risk populations for HBV/ HCV coinfection ⁽⁵³⁻⁵⁵⁾. Several studies have shown that compared with mono-infection of HBV or HCV, HBV/HCV co-infection carried a significantly higher rate of disease progression and development of hepatocellular carcinoma ⁽⁵⁶⁻⁵⁹⁾.

Unlike HBV/HIV co-infection, there is an interaction between HBV and HCV in term of the virus replication. HCV is found to exert negative effects on HBV replication as shown by several animal and human studies (49, 60-62). This may be related to the finding that the core protein of HCV is able to inhibit the HBV replication ⁽⁶³⁻⁶⁶⁾. It is therefore not surprising to observe that compared to HBV mono-infected patients, HBV/HCV co-infected patients have a higher rate of HBsAg seroconversion (2.1% vs. 0.43% respectively) ^(63, 67). With the same principle, because of the higher activity of the HCV, treatment fir CHC infection is relatively more urgently needed. It is however noteworthy that this viral dominance is dynamic over time and continuous monitoring of the viral levels of both viruses is necessary.

According to the study conducted in Taiwan using pegylated interferon, the sustained virological responses (SVRs) of different HCV genotypes were similar between HCV mono-infected patients and HBV/HCV co-infected patients ⁽⁶⁸⁾. In particular, the SVRs for genotype 1 were 72.2% and 77.3% and for genotype 2/3 were 82.8% and 84% for patients with HBV/HCV co-infection and HCV mono-infection respectively. Because of the anti-HBV effects of the pegylated interferon, it may be associated with some clinical outcome in CHB. For example, a high rate (5%) of annual rate of HBsAg seroclearance was observed in HBV/HCV co-infected patients when the treatment indication using pegylated interferon is for HCV^(69, 70).

Interferon free regime for CHC is now the commonly adopted treatment regime globally because of the rapid licensing of various highly effective direct-acting antivirals (DAA). Practically, nearly all CHC will be cured by this form of treatment in the near future. HBV/HCV co-infected patients should therefore be closely monitored by HBV DNA measurement for the possibility of HBV reactivation after the removal of the inhibitory effects to HBV by HCV. It has been shown that as high as 36-62% of co-infected patients with previously undetectable HBV DNA have detectable HBV DNA after successful CHC treatment ^(68, 69, 71). Oral nucleos(t)ide analogs e.g. entecavir and tenofovir should be considered if HBV reactivation occurs because severe flares of hepatitis have been reported.

If HBV is the dominant virus in HBV/HCV co-infected patients and the HBV activity is indicated for treatment, oral nucleos(t)ide analogs should be considered. In patients with high viraemic levels for both HBV and HCV, it is recommended to use pegylated interferon and ribavirin. If undetectable HBV DNA is not achieved, follow-up treatment by entecavir or tenofovir should be given. Alternatively, entecavir or tenofovir may be given concomitantly with the CHC treatment. The use of DAAs in these situations has not been assessed and it will be interesting to determine the best regime and their outcome for HBV/ HCV co-infected patients treated with all oral agents.



References:

- Liu SHK, Seto WK, Wong DKH et al. A territory-wide study on the seroprevalence of hepatitis B virus in the general population after universal hepatitis B immunization era in Hong Kong, China. J Hepatol 2016 (Suppl 2):5378. Tse KY, Ho LF, Lao T. The impact of maternal HBsAg carrier status on pregnancy outcomes: a case-control study. J Hepatol 2005;3:771-5. Safir A, Levy A, Sikuler E, Sheiner E. Maternal hepatitis B virus or hepatitis C virus carrier status as an independent risk factor for adverse perinatal outcome. Liver Int 2010;30:765-70. Lobstein S, Faber R, Tillmann HL. Prevalence of hepatitis B among pregnant women and its impact on pregnancy and newborn complications at a tertiary hospital in the eastern part of Germany. Digestion. 2011;83:76-82. To WW, Cheung W, Mok KM. Hepatitis B surface antigen carrier status and its correlation to gestational hypertension. Aust N Z J Obstet Gynaecol 2003;43:119-22.
- 2.
- 3.
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- 5.

- hospital in the eastern part of Germany. Digestion. 2011;83:76-82.
 To WW, Cheung W, Mok KM. Hepatitis B surface antigen carrier status and its correlation to gestational hypertension. Aust N Z J Obstet Gynaecol 2003;43:119-22.
 Shaheen AA, Myers RP. The outcomes of pregnancy in patients with cirrhosis: a population-based study. Liver Int 2010;30:275-83.
 Sinha S, Kumar M. Pregnancy and chronic hepatitis B virus infection. Hepatol Res 2010;40:31-48.
 Rawal BK, Parida S, Watkins RP, Ghosh P, Smith H. Symptomatic reactivation of hepatitis B in pregnancy. Lancet 1991;337: 364.
 ter Borg MJ, Leemans WF, de Man RA, Janssen HL. Exacerbation of chronic hepatitis B infection after delivery. J Viral Hepat 2008;15:37-41.
 Anderson KE, Stevens CE, Tsuei JJ, Lee WC, Sun SC, Beasley P. Hepatitis B antigen in infants born to mothers with chronic hepatitis B antigenemia in Taiwan. Am J Dis Child 1975;129:1389-92.
 Stevens CE, Beasley RP, Tsui J, Lee WC. Vertical transmission of hepatitis B antigen in Taiwan. NE ngl J Med 1975;292 ;771-4.
 Wong VC, Ip HM, Reesink HW, Lelie PN, et al. Prevention of the HBsAg carrier state in newborn infants of mothers who are chronic carriers of HBsAg and HBeAg by administration of hepatitis B vaccine and hepatitis-B immunoglobulin. Double-blind randomised placebo-controlled study. Lancet 1984;1:921-6.
 Zou H, Chen Y, Duan Z, Zhang H, Pan C. Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat 2009;16:94-103.
 Han GR, Cao MK, Zhao W, et al. A prospective and open-label study for the efficacy and Safety of telbivudine in pregnancy for the prevention of perinatal transmission of hepatitis B virus infection. J Hepatol 2012;10:520-6.
 Yu MM, Cui YT, Wang L, et al. Comparison of telbivudine versus lamivudine in interrupting mother-to-infant transmission of hepatitis B virus
- in the ANRS 12109 TEmAA Study, Step 2. Antimicro Agents Chemother 2011;55:1315–7.
 25. Mak LY, Seto WK, Lai CL, Yuen MF. DNA polymerase inhibitors for treating hepatitis B: a safety evaluation. Expert Opin Drug Saf 2016;15:383-92.
 26. Pan CQ, Mi LJ, Reddy KR et al. Tenofovir disoproxil fumarate for prevention of vertical transmission of hepatitis B virus infection by highly viremic pregnant women: a case series. Dig. Dis. Sci. 2012; 57: 2423-9.
 27. Zhang L, Gui X, Fan J, et al. Breast feeding and immunoprophylaxis efficacy of mother-to-child transmission of hepatitis B virus. J Matern Fetal Neonatal Med 2014-27182-4.
- Murphy MJ, Wilcox RD. Management of the coinfected patient: human immunodeficiency virus/hepatitis B and human immunodeficiency virus/ hepatitis C. Am J Med Sci 2004;328:26-36.
 Matthews G. The management of HIV and hepatitis B coinfection. Curr Opin Infect Dis 2007;20:16-21.

- Colin JF, Cazals-Hatem D, Loriot MA, et al. Influence of human immunodeficiency virus infection on chronic hepatitis B in homosexual men. Hepatology 1999;29:1306-10.
 Thio CL, Seaberg EC, Skolasky R Jr, et al. HIV-1, hepatitis B virus, and risk of liver-related mortality in the Multicenter Cohort Study (MACS). Lancet 2002;360:1921-6.
 Di Mating V Theorem 1.5 Colina For the formation of the state of the stat
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 Di Martino V, Thevenot T, Colin JF, et al. Influence of HIV infection on the response to interferon therapy and the long-term outcome of chronic hepatitis B. Gastroenterology 2002;123:1812-22.
 Miailhes P, Maynard-Muet M, Lebossé F, et al. Role of a 48-week pegylated interferon therapy in hepatitis B e antigen positive HIV-co-infected patients on cART including tenofovir: EMVIPEG study. J Hepatol 2014;61:761-9
 Fung J, Lai CL, Yuen MF. New paradigms for the treatment of chronic hepatitis B. Jastroenterol Hepatol 2008;23:1812-92.
 McMahon MA, Jilek BL, Brennan TP, et al. The HBV drug entecavir effects on HIV-1 replication and resistance. N Engl J Med 2007;356:2614-21.
 Sasadeusz J, Audsley J, Mijch A, et al. The anti-HIV activity of entecavir: a multicentre evaluation of lamivudine-experienced and lamivudine-naive patients. AIDS 2008;22:1947-55.
 van Bömmel F, Wünsche T, Schürmann D, Berg T. Tenofovir treatment in patients with lamivudine-resistant hepatitis B mutants strongly affects viral replication. Hepatology 2002;36:507-8.

- Núñez M, Pérez-Olmeda M, Díaz B, Ríos P, González-Lahoz J, Soriano V. Activity of tenofovir on hepatitis B virus replication in HIV-co-infected patients failing or partially responding to lamivudine. AIDS 2002;16:2352-4.
 Ristig MB, Crippin J, Aberg JA, et al. Tenofovir disoproxil fumarate therapy
- for chronic hepatitis B in human immunodeficiency virus/hepatitis B virus-coinfected individuals for whom interferon-alpha and lamivudine therapy have
- coinfected individuals for whom interferon-alpha and lamivudine therapy have failed. J Infect Dis 2002;186:1844-7.
 40. Nelson M, Portsmouth S, Stebbing J, et al. An open-label study of tenofovir in HIV-1 and Hepatitis B virus co-infected individuals. AIDS 2003;17:F7-10.
 41. Benhamou Y, Tubiana R, Thibault V. Tenofovir disoproxil fumarate in patients with HIV and lamivudine-resistant hepatitis B virus. N Engl J Med 2003;348:177-8.
 42. de Vries-Sluijs TE, Reijnders JG, Hansen BE, et al. Long-term therapy with tenofovir is effective for patients co-infected with human immunodeficiency visus and hopatitis B virus.
- tenofovir is effective for patients co-infected with human immunodeficiency virus and hepatitis B virus. Gastroenterology 2010;139:1934-41.
 43. Amini-Bavil-Olyaee S, Herbers U, Sheldon J, Luedde T, Trautwein C, Tacke F. The rtA194T polymerase mutation impacts viral replication and susceptibility to tenofovir in hepatitis B e antigen-positive and hepatitis B e antigennegative hepatitis B virus strains. Hepatology 2009;49:1158-65.
 44. Benhamou Y, Bochet M, Thibault V, et al. Long-term incidence of hepatitis B virus resistance to lamivudine in human immunodeficiency virus-infected patients. Hepatology 1999;30:1302-6.
 45. Benhamou Y, Thibault V, Vig P, et al. Safety and efficacy of adefovir dipivoxil in patients infected with lamivudine-resistant hepatitis B and HIV-1. J Hepatol 2006;44:62-7.
 46. Cheruvu S, Marks K, Talal AH. Understanding the pathogenesis and

- Cheruvu S, Marks K, Talal AH. Understanding the pathogenesis and management of hepatitis B/HIV and hepatitis B/hepatitis C virus coinfection. Clin Liver Dis 2007;11:917-43.
 Mendes-Corrêa M, Núñez M. Management of HIV and hepatitis virus
- coinfection. Expert Opin Pharmacother 2010;11:2497-516. 48. Lacombe K, Rockstroh J. HIV and viral hepatitis coinfections: advances and

- Lacombe K, Rockstroh J. HIV and viral hepatitis coinfections: advances and challenges. Gut 2012;61 Suppl 1:i47-58.
 Crespo J, Lozano JL, de la Cruz F, et al. Prevalence and significance of hepatitis C viremia in chronic active hepatitis B. Am J Gastroenterol 1994;89:1147-51.
 Fattovich G, Tagger A, Brollo L, et al. Hepatitis C virus infection in chronic hepatitis B virus carriers. J Infect Dis 1991;163:400-2.
 Gaeta GB, Stornaiuolo G, Precone DF, et al. Epidemiological and clinical burden of chronic hepatitis B virus/hepatitis C virus infection. A multicenter Italian study J Hepatol 2003;39:1036-41.
 Dai CY, Yu ML, Chuang WL, et al. Influence of hepatitis C virus on the profiles of patients with chronic hepatitis B virus infection. L Gastroenterol Hepatol
- of patients with chronic hepatitis B virus infection. J Gastroenterol Hepatol 2001;16:636-40.
- Pallás JR, Fariñas-Alvarez C, Prieto D, Delgado-Rodríguez M. Coinfections by HIV, hepatitis B and hepatitis C in imprisoned injecting drug users. Eur J by HIV, hepatitis C in a manufacture of the second sec
- Epidemiol 1999;15:699-704.
 54. Reddy GA, Dakshinamurthy KV, Neelaprasad P, Gangadhar T, Lakshmi V. Prevalence of HBV and HCV dual infection in patients on haemodialysis. Indian J Med Microbiol 2005;23:41-3.
- Aroldi A, Lampertico P, Montagnino G, et al. Natural history of hepatitis B and C in renal allograft recipients. Transplantation 2005;79:1132-6. 56. Benvegnù L, Fattovich G, Noventa F, et al. Concurrent hepatitis B and C virus
- infection and risk of hepatocellular carcinoma in cirrhosis. A prospective study. Cancer 1994;74:2442-8.

- Cancer 1994;74:2442-8.
 57. Chiaramonte M, Stroffolini T, Vian A, et al. Rate of incidence of hepatocellular carcinoma in patients with compensated viral cirrhosis. Cancer 1999;85:2132-7.
 58. Weltman MD, Brotodihardjo A, Crewe EB, et al. Coinfection with hepatitis B and C or B, C and delta viruses results in severe chronic liver disease and responds poorly to interferon-alpha treatment. J Viral Hepat 1995;2:39-45.
 59. Kaklamani E, Trichopoulos D, Tzonou A, et al. Hepatitis B and C viruses and their interaction in the origin of hepatocellular carcinoma. JAMA 1991;265:1974-6.
 60. Brotman B, Prince AM, Huima T, Bichardson L, van den Ende MC, Pfaifer.
- Brotman B, Prince AM, Huima T, Richardson L, van den Ende MC, Pfeifer U. Interference between non-A, non-B and hepatitis B virus infection in chimpanzees. J Med Virol 1983;11:191-205.
- Bradley DW, Maynard JE, McCaustland KA, Murphy BL, Cook EH, Ebert JW. Non-A, non-B hepatitis in chimpanzees: interference with acute hepatitis A
- virus and chronic hepatitis II climinatzes. Interference with active hepatitis A virus and chronic hepatitis B virus infections. J Med Virol 1983;11:207-13.
 62. Fong TL, Di Bisceglie AM, Waggoner JG, Banks SM, Hoofnagle JH. The significance of antibody to hepatitis C virus in patients with chronic hepatitis B. Hepatology 1991;14:64-7.
 62. Clinic Virus Virus C C C M D Le Charter Virus C C L L L L Virus Virus C C L L L Virus Virus C C L L Virus Virus
- Sheen IS, Liaw YF, Lin DY, Chu CM. Role of hepatitis C and delta viruses in the
- Sheen IS, Liaw YF, Lin DY, Chu CM. Role of hepatitis C and delta viruses in the termination of chronic hepatitis B surface antigen carrier state: a multivariate analysis in a longitudinal follow-up study. J Infect Dis 1994;170:358-61.
 Shih CM, Lo SJ, Miyamura T, Chen SY, Lee YH. Suppression of hepatitis B virus expression and replication by hepatitis C virus core protein in HuH-7 cells. J Virol 1993;67:5823-32.
 Shih CM, Chen CM, Chen SY, Lee YH. Modulation of the trans-suppression activities of the protein of the trans-suppression.
- activity of hepatitis C virus core protein by phosphorylation. J Virol 1995;69:1160-71.
- Schüttler CG, Fiedler N, Schmidt K, Repp R, Gerlich WH, Schaefer S. Suppression of hepatitis B virus enhancer 1 and 2 by hepatitis C virus core protein. J Hepatol 2002;37:855-62.
 Hamzaoui L, El Bouchtili S, Siai K, Mahmoudi M, Azzouz MM. Hepatitis B virus and hepatitis C virus co-infection: a therapeutic challenge. Clin Res Hepatol Gastroenterol 2013;37:e16-20.
- Liu CJ, Chuang WL, Lee CM, et al. Peginterferon alfa-2a plus ribavirin for the treatment of dual chronic infection with hepatitis B and C viruses. Gastroenterology 2009;136:496-504.
- Yu ML, Lee CM, Chen CL, et al. Sustained hepatitis C virus clearance and increased hepatitis B surface antigen seroclearance in patients with dual chronic hepatitis C and B during posttreatment follow-up. Hepatology 2013;57:2135-42.
- Yeh ML, Hung CH, Huang JF, et al. Long-term effect of interferon plus ribavirin on hepatitis B surface antigen seroclearance in patients dually infected with hepatitis B and C viruses. PLoS One 2011;6:e20752.
- Yu ML, Lee CM, Chuang WL, et al. HBsAg profiles in patients receiving peginterferon alfa-2a plus ribavirin for the treatment of dual chronic infection with hepatitis B and C viruses. J Infect Dis 2010;202:86-92.

Passing rate for the Joint HKCPIE/MRPC(UK) Part II (Written) examination for the past years:

	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%)

Passing rate for the Part I examination for the years 2002 – 2016:

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January 20104528 (62%)September 20106239 (63%)January 20114423 (52%)September 20116449 (77%)January 20124528 (62%)September 20128059 (74%)January 20134122 (54%)September 20137660 (79%)January 20143020 (67%)September 20152920 (69%)September 201510071 (71%)January 20163318 (55%)	September 2009	47	28 (60%)
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%) January 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 2010	45	28 (62%)
January 20114423 (52%)September 20116449 (77%)January 20124528 (62%)September 20128059 (74%)January 20134122 (54%)September 20137660 (79%)January 20143020 (67%)September 20152920 (69%)September 201510071 (71%)January 20163318 (55%)	September 2010	62	39 (63%)
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 2015 29 20 (69%) January 2015 29 20 (69%) September 2015 100 71 (71%) January 2016 33 18 (55%)	January 2011	44	23 (52%)
January 20124528 (62%)September 20128059 (74%)January 20134122 (54%)September 20137660 (79%)January 20143020 (67%)September 20148464 (76%)January 20152920 (69%)September 201510071 (71%)January 20163318 (55%)	September 2011	64	49 (77%)
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%)	January 2012	45	28 (62%)
January 20134122 (54%)September 20137660 (79%)January 20143020 (67%)September 20148464 (76%)January 20152920 (69%)September 201510071 (71%)January 20163318 (55%)	September 2012	80	59 (74%)
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%)	January 2013	41	22 (54%)
January 20143020 (67%)September 20148464 (76%)January 20152920 (69%)September 201510071 (71%)January 20163318 (55%)	September 2013	76	60 (79%)
September 2014 84 64 (76%) January 2015 29 20 (69%) September 2015 100 71 (71%) January 2016 33 18 (55%)	January 2014	30	20 (67%)
January 2015 29 20 (69%) September 2015 100 71 (71%) January 2016 33 18 (55%)	September 2014	84	64 (76%)
September 2015 100 71 (71%) January 2016 33 18 (55%)	January 2015	29	20 (69%)
January 2016 33 18 (55%)	September 2015	100	71 (71%)
	January 2016	33	18 (55%)

Passing rates for the 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%
March 2015	36/75 = 48%
October 2015	35/75 = 47%
March 2016	40/75 = 53%

Joint HKCPIE/MRCP (UK) Part II PACES Examination March 2016 Pass List

Au Chi Kin Chan Chiu Wai Shirley Chan Hoi Kei Iki Chan Lap Shing Chan Yee Lok Chau Siu Kwan Chris Cheng Yiu Fai Cheung Pak Kin Chi Wai Kin Chiu Karen Hiu Ching Fong Ka Man Fung Kok Leung Ho Cheuk Bong Ho Hoi Lung Ho Yee Ting Christina Kan Kau Yue Andre Lam Cheuk Ting Lam Hoi Yee Lam Hon Sin Lam Luk Ping Lau Wai Pan Lee Tsui Yin Jaime Li Ka Shu Justin Ling Wood Hay lan Ma Kei Chuen Mak Kwan Ping Ng Kam Man Shum Tung Sen So Hay Man Suen To Lam Tam King Wai Tsang Wing Yan Josephine Un Ka Chun Wong Ho On Leo Wong Wing Yu Wu Tsz Yuen Yan Ka Shing Yau Ho Tuen Yip Chi Yuen Yong Jason Xern E

Revised Interim Assessment Format in Advanced Internal Medicine

With the purpose of improved testing of trainees' capability in diagnostic process and management, there will be changes in Examination format in Interim Assessment (IA) of Advanced Internal Medicine, with *implementation starting in December 2016*.

Details of the IA format will be as follows:

- 1. The current format of Case Reports and Supervisor's Scoring will maintain unchanged. This score contributes 10 marks out of the total of 40.
- 2. The clinical viva will be conducted in 30 minutes for each candidate. It consists of three parts.

The first part will be standardized clinical scenarios examining the candidate's ability in (a) making diagnosis, (b) organizing appropriate investigations and (c) setting good management plan.

The second part shall include 3 areas of investigation interpretation: (a) non-blood investigation (e.g. ECG, lung function test), (b) data/blood investigation, (c) imaging interpretation.

Both parts carry equal time and weight of markings. The maximum score is 30 marks out of the total of 40.

The third part comprises two short questions from the topics in Hong Kong College of Physicians Annual Scientific Meeting, Hong Kong Medical Forum and Advances in Medicine. Each question carries 0.5 mark as bonus or deduction. In other words, 1 bonus mark will be added to the total score when the two questions are answered correctly. The total marks will be unchanged when one answer is correct while the other is wrong. Failing to answer both questions correctly will have 1 mark deducted from the total score.

For confidentiality of standardized questions, a short period of quarantine may be applied to candidates.

Criteria for Trainer Status

The Council at its 297th Meeting of 23 June 2016 decided the following procedures for accreditation of trainer status:

- (1) The Chief of Service (COS) of each clinical department should submit to the respective Specialty Board the list of Fellows who are eligible to be appointed as trainers. The List would then be circulated to all Board Members and Programme Directors for comment and agreement before submission to E&AC for endorsement.
- (2) The respective COS and Programme Directors would be informed about Fellows who had clinical viva score of ≤ 32 in both the specialty and the broad-based specialty. They should take note of and assess the Fellows' performance regarding their competence to serve as trainer during the two years after completion of training. The concerned Fellows should demonstrate that they are actively engaged in full-time institutional practice of both the specialty and the broad-based specialty, be able to conduct training in accredited training programmes and are recognized to be actively contributing to the discipline.
- (3) For Fellows who have only undergone single specialty training, the respective COS and Programme Director will be informed if their clinical viva score of the single specialty is ≤ 32.
- (4) The procedures for nomination of Fellows to be appointed as trainers would be streamlined. The respective COS and Programme Directors will indicate their support for the concerned Fellows to be appointed as trainers. If the COS and Programme Directors do not recommend a Fellow with clinical viva score of more than 32 marks in either the specialty or the broad-based specialty, an independent reviewer would be appointed to assess the candidate.
- (5) Fellows who are not recommended to be appointed as trainer in the specialties can re-apply one year later.

The implementation date for the above regulations will be 1 January 2019. The new procedures will be applicable to those trainees who will take the Exit Assessment of the 1st specialty in November/December 2016.

Statistics on No. of Trainees in all Specialties Updated in May 2016

							TRAINEES							
		HONG KONG EAST CLUSTER HONG KONG WEST CLUSTER												
SPECIALTY	TRAINEES	PYNE	н	RH		TWEI	н	FYKH	GH	_	QMI	ł	TWH	
	TOTAL (DH/HA/			YEAF	2					YE	AR			
		1	1	1		1	0	1 0	1		1 1	-	1	
CARDIOLOGY	19	$^{1}_{2-I}$	1	1 2—I	2	1 2	0	1 0 2	$\begin{vmatrix} 1\\2\\2 \end{vmatrix}$	0	1 - 1 2 - 1	4	1 2	0
		3 4	8	3—1 4	4	3 4	0	4^{3} 0	3	5	3—11 4	11	3 4	0
CLINICAL PHARMACOLOGY &	0	1	0	1	0	1	0	1 0	1	0	1	0	1	0
THERAPEUTICS		$\frac{2}{3}$		23		23		$\frac{2}{3}$	$\begin{vmatrix} 2\\ 3 \end{vmatrix}$		$\frac{2}{3}$		2 3	
		4	0	4	0	4	0	4 0	4	0	4	1	4	0
CRITICAL CARE MEDICINE	9	1 2—I	1	$\frac{1}{2}$	0	$\frac{1}{2}$	0	$\frac{1}{2}$ 0	$\frac{1}{2}$	0	1 2I	2	$\frac{1}{2}$	0
		3	6	$\overline{3}_{4}$	0	$\overline{3}_{4}$	0	$\frac{1}{3}$	$\overline{3}_{4}$	0	3—I	7	$\overline{3}_{4}$	0
DERMATOLOCY & VENEREOLOCY	8	1	0	1	0	1	0	1 0	1	0	1_I	1	1	0
	0	23	0	2	U	2	U	$\frac{1}{2}$	23	0	2	1	2	Ŭ
		4	0	4	0	4	0	4 0	4	0	4	1	4	0
ENDOCRINOLOGY, DIABETES &	15	1	0	1	1	1	0	1 0	1	0	1	0	1	0
METABOLISM		3		3^{2-1}		3	_	$\frac{2}{3}$	3		3	-	23	
		4	3	4	2	4	1	4 0	4	0	4	6	4	0
HEPATOLOGY &	25	1—1 2—II	3	1	0	1 2	0	1 0 2	$\begin{bmatrix} 1\\2 \end{bmatrix}$	0	1—11 2	2	1—1 2	1
		3 4	5	3 4	2	3 4	0	4^{3} 0	3	0	3	8	3 4	0
GERIATRIC MEDICINE	13	1	1	1—I	1	1—I	1	1 0	1	0	1	2	1	0
		2 3—I		2 3		23		23	23		2—I 3—I		2 3	
		4	6	4	9	4	2	4 4	4	3	4	1	4	1
HAEM/HAEM ONCOLOGY	10	1 2—I	1	$\frac{1}{2}$	0	$\frac{1}{2}$	0	$\frac{1}{2}$ 0	$\begin{vmatrix} 1 \\ 2 \end{vmatrix}$	0	1—II 2	2	$\frac{1}{2}$	0
		3	3	3	0	3	0	$\frac{1}{3}$	$\overline{3}_{4}$	0	3	7	3	0
IMMUNOLOCY & ALLERCY	0	1	0	1	0	1	0	1 0	1	0	1	0	1	0
	0	23	U	23	U	23	U	$\frac{1}{2}$	$\frac{1}{2}$	0	$\frac{1}{2}$	0	23	Ŭ
		4	0	4	0	4	0	³ 4 0	4	0	4	0	4	0
INFECTIOUS DISEASE	4	$\frac{1}{2}$	0	$\frac{1}{2}$	0	1	0	$\frac{1}{2}$ 0	1	0	1	0	1	0
		$\frac{2}{3}$	n	$\frac{2}{3}$	0	3	0	$\frac{2}{3}$	$\frac{2}{3}$	0	3	1	3	0
	1.01	4 1 II	14	1	4	4	0	4 0	1	0	4 1 VIII	24	-+ 1 I	1
INTERNAL MEDICINE	191	2 - VII	14	$\frac{1}{2}$ —III	4	$\frac{1}{2}$	0	$\frac{1}{2}$	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	0	$\begin{vmatrix} 1 - v \\ 2 - V \\ 2 - V \end{vmatrix}$	24	$\frac{1-1}{2}$	1
		4—III	43	3—1 4	15	4	9	4 4	4	7	4-V	67	4	9
MEDICAL ONCOLOGY	3	1	0	1	0	1	0	1 0	1	0	1	1	1	0
		$\frac{2}{3}$	0	$\frac{2}{3}$	0	3	0	$\frac{2}{3}$	$\begin{vmatrix} 2\\3\\4 \end{vmatrix}$	0	3^{2-1}	2	$\frac{2}{3}$	
	10	4	0	4	0	4	0	4 0	4	0	4	2	4	0
NEPHROLOGY	13	2	0	1 2	0	$\frac{1}{2}$	0	2 0	$\begin{vmatrix} 1\\2\\2 \end{vmatrix}$	0	2	0	2	0
		3 4	5	3 4	0	3 4	0	4^{3} 0	$\frac{3}{4}$	0	3 4	6	3 4	3
NEUROLOGY	14	1	0	1	0	1	0	1 0	1	0	1	1	1	0
		23	_	23	_	23	_	23	$\begin{vmatrix} 2\\ 3\\ \end{vmatrix}$	_	2 3—I	_	23	
		4	5	4	2	4	0	4 0	4	0	4	7	4	0
PALLIATIVE MEDICINE	5	$\frac{1}{2}$	0	$\frac{1}{2}$	0	$1 \\ 2$	0		$\begin{vmatrix} 1\\ 2 \end{vmatrix}$	0	$1 \\ 2$	0	$\frac{1}{2}$	0
		3 4	0	3 4	2	34	0		3 4	3	34	0	3 4	0
REHABILITATION	3	1	0	1	0	1—I	2	1 0	1	0	1	0	1	0
		2 3		2 3		2 3		2 3	$\frac{2}{3}$		2 3		2 3	
		4	0	4	2	4—I	3	4 1	4	0	4	0	4	5
RESPIRATORY MEDICINE	16	1—I 2—I	2	$^{1}_{2-I}$	1	$\frac{1}{2}$	0	$\frac{1}{2}$ 0	$1 \\ 2$	0	1—I 2	2	$\frac{1}{2}$	0
		3	4	3	5	3	0	3	$\overline{3}_{4}$	8	3—I	5	3	0
RHEUMATOLOGY	9	1	2	1	0	1	0	1 0	1	0	1	0	1	0
		2—I	2	23	0	$\frac{1}{2}$	0	$\frac{2}{3}$	$\frac{1}{2}$	0	$\frac{1}{2}$	0	23	0
		4	3	4	1	4	1	4 0	4	0	4	5	4	1

		TRAINEES																						
		KOV	VLO	DON C CLUST	EN R	ITRAL		KOW	VI CI	LOON I LUSTEF	EAS R	ST			I	(OW	/L(DON WI	EST (CLU	ST	ER		
SPECIALTY	TRAINEES	BH		KH		QEH	ŀ	юнн	1	гкон	i	исн	Ī	СМС	KW	н	0)LMH	PM	1H	V	VTSH	YC	Н
	TOTAL (DH/HA/ OTHERS)			YEAR						YEAR								YEA	4R					
CARDIOLOGY	19	$\frac{1}{2}$	0	1	0	1—II 2	$\frac{2}{2}$	0	$\frac{1}{2}$	_I 2	$\frac{1}{2}$	0		1—I 1 2	1	0	$\frac{1}{2}$	0	$\frac{1}{2}$	1	$\frac{1}{2}$	0	1	0
		3 4	0	3 4	0	3 4 10	3	о	3 4	_I 2	3 4	6	4	3 4 1	3 4	3	3 4	о	3—I 4	9	$\begin{vmatrix} \overline{3} \\ 4 \end{vmatrix}$	0	3 4	3
CLINICAL PHARMACOLOGY & THERAPEUTICS	0	1 2 3 4	0 0	1 2 3 4	0 0	$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$) 1 2 3 0 4	0 0	1 2 3 4	0	1 2 3 4	0		$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0	1 2 3 4	0 0	1 2 3 4	0 0
CRITICAL CARE MEDICINE	9	1 2 3 4	0 0	1 2 3 4	0 0	$\begin{array}{ccc}1&0\\2\\3\\4&5\end{array}$) 1 2 3 4	0 0	1 2 3 4	0	1 - 2 3 4	—I 1 5		$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	1—I 2 3—I 4	2 3	1 2 3 4	0 0	1 2 3 4	0	1 2 3 4	0 0	1 2 3 4	0 0
DERMATOLOGY & VENEREOLOGY	8	1 2 3 4	0 0	1 2 3 4	0 0	$ \begin{array}{ccc} 1 & 0 \\ 2 \\ 3 \\ 4 & 0 \end{array} $) 1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0		$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0	1 2 3 4	0 0	1 2 3 4	0 0
ENDOCRINOLOGY, DIABETES & METABOLISM	15	1 2 3 4	0 0	1—I 2 3 4	1 0	1 3 2—I 3—II 4 8	8 1 2 3 4	0 0	1 2 3 4	0	1 2 3- 4	1 —I 3		1—I 1 2 3 4 1	1—I 2—I 3—I 4	3	1 2 3 4	0	1 2—I 3 4	1	1 2 3 4	0 0	1 2 3 4	0
GASTROENTEROLOGY & HEPATOLOGY	25	1 2 3 4	0 0	1 2 3 4	0 0	1 3 2 3—III 4 5	3 1 2 3 4	0 0	1 2 3 4	1 —I 3	1 2 3- 4	2 —II 4		1—I 3 2—I 3 4—I 3	1 2 3—I 4	1 7	1 2 3 4	0	1 2—I 3—I 4	2	1 2 3 4	0 0	1 2—I 3 4	1 5
GERIATRIC MEDICINE	13	1—I 2 3 4	1 1	1 2 3 4	0 5	1 1 2 3 4—I 3	1 - 2 3 4	—I 2 —I 3	1 2 3 4	1 —I 2	1 2- 3 4	_I 1 6		$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	1 2 3 4	0	1 2 3 4	0	1 2 3 4—I	1	1 2 3 4	0 5	1 2 3 4	0 5
HAEM/HAEM ONCOLOGY	10	1 2 3 4	0 0	1 2—I 3 4	1 0	$ \begin{array}{ccc} 1 & 0 \\ 2 \\ 3 \\ 4 & 3 \end{array} $) 1 2 3 4	0 0	1 2 3 4	0	1 2 3 4	0 2		$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$	1 2 3 4	0 0	1 2 3 4	0 0	1—I 2—I 3 4	I 3 4	1 2 3 4	0 0	1 2 3 4	0 0
IMMUNOLOGY & Allergy	0	1 2 3 4	0 0	1 2 3 4	0 0	$ \begin{array}{ccc} 1 & 0 \\ 2 \\ 3 \\ 4 & 0 \end{array} $) 1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0		$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0
INFECTIOUS DISEASE	4	1 2 3 4	0 0	1 2 3 4	0 0	1 1 2 3—I 4 4	1 2 3 4 4	0 0	1 2 3 4	0 0	1 2 3 4	0		$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	1 2 3 4	0 0	1 2 3 4	0 0	1—I 2 3 4	1	1 2 3 4	0 0	1 2 3 4	0 0
INTERNAL MEDICINE	191	1—II 2 3—I 4—I	4 2	1—II 2—II 3 4	4 4	1—II 19 2—III 3-VIII 4-VI 58) 1 2 3 3 4	-I 4 $-I -II 3$	1 2 3 4	—I 7 —I —III —II 23	1- 2- 3- 4-	—II 10 —I —III —IV 40		$ \begin{array}{c} 1 - VI & 8 \\ 2 - I \\ 3 \\ 4 - I & 22 \end{array} $	1—I 2—I 3—I 4—I	V 11 II II 46	1 2 3 4	0	1—V 2—V 3—I 4—V	√ 19 √ √ √ 48	1 2 3 4	0 2	1 2—II 3 4—I	4 11 24
MEDICAL ONCOLOGY	3	1 2 3 4	0 0	1 2 3 4	0 0	$ \begin{array}{ccc} 1 & 0 \\ 2 \\ 3 \\ 4 & 2 \end{array} $) 1 2 3 2 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0		$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 1	1 2 3 4	0 0	1 2 3 4	0 0
NEPHROLOGY	13	1 2 3 4	0 0	1 2 3 4	0 0	$ \begin{array}{ccc} 1 & 1 \\ 2 \\ 3-I \\ 4 & 7 \end{array} $	1 2 3 7 4	0 0	1 2 3 4	_I 1	1- 2 3 4	—I 1 4		$\begin{array}{ccc}1&0\\2\\3\\4&2\end{array}$	1 2—I 3 4	1 8	1 2 3 4	0 0	1 2—I 3 4	1 6	1 2 3 4	0 0	1 2—II 3 4	2 [2
NEUROLOGY	14	1 2 3 4	0 0	1 2 3 4	0 0	1—I 3 2—I 3—I 4 5	3 1 2 3 4	0 0	1 2 3 4	1 —I 2	1 2 3 4	0		1—I 1 2 3 4 1	1 2 3 4	0	1 2 3 4	0 0	1—I 2—I 3—I 4	3	1 2 3 4	0 0	1 2 3 4	0 3
PALLIATIVE MEDICINE	5	1 2 3 4	0 0	1 2—I 3 4	1 0	$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$) 1 2 3 4	1 —I _3	1 2 3 4	0 0	1 2 3 4	0		$\begin{array}{ccc}1&0\\2\\3\\4&2\end{array}$	1 2 3 4	0 0	1 2 3 4	0	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0
REHABILITATION	3	1 2 3 4	0 0	1 2 3 4	0 6	$ \begin{array}{ccc} 1 & 0 \\ 2 \\ 3 \\ 4 & 0 \end{array} $) 1 2 3 4	0 1	1 2 3 4	0 0	1 2 3 4	0 3		$\begin{array}{ccc}1&0\\2\\3\\4&0\end{array}$	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 0	1 2 3 4	0 4	1 2 3 4	0 0
RESPIRATORY MEDICINE	16	1 2 3—I 4	1 1	1—I 2 3 4	1 6	$ \begin{array}{ccc} 1 & 0 \\ 2 \\ 3 \\ 4 & 7 \end{array} $) 1 2 3 7 4	_I _I _4	1 2 3 4	2 —I —I 3	1 2 3 4	0		$\begin{array}{ccc}1&0\\2\\3\\4&2\end{array}$	1—I 2 3 4	1 3	1 2 3 4	0 0	1—I 2 3 4	1 5	1 2 3 4	0	1 2 3 4	0
RHEUMATOLOGY	9	1 2 3 4	0 0	1 2 3 4	0 0	$ \begin{array}{cccc} 1 & 1\\ 2-I\\ 3\\ 4 & 4 \end{array} $	1 2 3 4	0 0	1 2 3 4	0	1 2 3 4	0		$\begin{array}{c c} 1 & -I & 1 \\ 2 & & \\ 3 & & \\ 4 & 2 \end{array}$	1—I 2—I 3 4	2	1 2 3 4	0 0	1 2 3—I 4	1	1 2 3 4	0 0	1 2 3 4	0

		TRAINEES																
		NEW TERRITORIES EAST CLUSTER										NEW TERRITORII WEST-CLUSTER						
SPECIALTY	TRAINEES	AHNH NDH PWH SH TPH									PO	H	ТМН					
	TOTAL (DH/HA/					YEA	R						YE	AR				
CARDIOLOGY	19	1	0	1	1	1—I	1	1	0	1	0	1	1	1	3			
		2 3		2 3—I		23		23		23		2—I 3		2—I 3—II				
	0	4	1	4	4	4	8	4	0	4	0	4	2	4	5			
THERAPEUTICS	0	2	0	$\begin{bmatrix} 1 \\ 2 \\ 3 \end{bmatrix}$	0	$\frac{1}{2}$	0	$\begin{bmatrix} 1 \\ 2 \\ 3 \end{bmatrix}$	0	$\begin{bmatrix} 1 \\ 2 \\ 3 \end{bmatrix}$	0	$\frac{1}{2}$	0	$\frac{1}{2}$	0			
		4	0	4	0	4	3	4	0	4	0	4	0	4	0			
CRITICAL CARE MEDICINE	9	1 2	0	1 2	0	1 2	0	1 2	0	1 2	1	1 2	0	1 2—I	1			
		3 4	2	3 4	3	3 4	2	3 4	0	3—I 4	0	3 4	0	3 4	2			
DERMATOLOGY & VENEREOLOGY	8	$\frac{1}{2}$	0	1	0	1	1	1	0	1	0	1	0	1	0			
		$\frac{2}{3}$	0	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0	3 - I	1	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0	3	0	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0			
ENDOCRINOLOGY, DIABETES &	15	1	1	1	0	1	0	1	0	1	0	1	0	1—I	3			
METABOLISM		23	_	23	_	23	_	23		23		2 3		2—11 3				
GASTROENTEROLOGY &	25	4—I 1	1	4	2	4	2	4	0	4	1	4	 	4	3			
HEPATOLOGY	20	2—I 3	-	2—II 3	-	2—I 3—I	-	23	U	2 3—I		23	U	23	U			
	10	4	2	4	4	4	5	4	0	4	0	4	3	4	7			
GERIAIRIC MEDICINE	13	1 2 2	0	$\begin{vmatrix} 1\\2\\2 \end{vmatrix}$	0	$\begin{vmatrix} 1\\2\\2 \end{vmatrix}$	0	$\begin{vmatrix} 1\\2\\2 \end{vmatrix}$	0	$\begin{bmatrix} 1\\ 2 \\ -I \end{bmatrix}$	1	$^{1}_{2-I}$	1	$\begin{array}{c}1\\2-I\\2\end{array}$	1			
		3 4	2	3 4	2	3 4	4	3 4	7	3 4	3	3 4	3	3 4	7			
HAEM/HAEM ONCOLOGY	10	1 2	0	1 2	0	1—I 2—I	2	1 2	0	1 2	0	1 2	0	1 2—I	1			
		3 4	0	3 4	0	3 4	3	3 4	0	3 4	0	3 4	0	3 4	3			
IMMUNOLOGY & ALLERGY	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0			
		$\frac{2}{3}$	0	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0	$\begin{vmatrix} 2\\ 3\\ 4 \end{vmatrix}$	0	$\begin{vmatrix} 2\\ 3\\ 4\end{vmatrix}$	0	$\frac{2}{3}$	0	$\frac{2}{3}$	0			
INFECTIOUS DISEASE	4	1	0	1	0	1	2	1	0	1	0	1	0	1	0			
		2 3		2 3		2—I 3—I		2 3		2 3		2 3		2 3				
INTERNAL MEDICINE	1.91	4	2	4 1I	5	4 1VII	2	4	0	4	0	4	0	4 1_IV	2			
	151	$\frac{1}{2}$ —II	4	2—II 3—I	5	$\begin{vmatrix} 1 - VII \\ 2 - IV \\ 3 - V \end{vmatrix}$	1 20	2—I 3—II	5	2—I 3—II	5	2—II	5	2-X 3-II	20			
		4−II	14	4—Î	23	4—III	55	4	7	4	5	₫—I	15	4—IV	48			
MEDICAL ONCOLOGY	3	1 2	0	$\begin{vmatrix} 1 \\ 2 \\ 2 \end{vmatrix}$	0	$\begin{vmatrix} 1 - I \\ 2 \\ 2 \\ - I \end{vmatrix}$	2	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	0	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	0	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	0	$\begin{vmatrix} 1\\ 2\\ 2 \end{vmatrix}$	0			
		3 4	0	3 4	0	3-1 4	14	3 4	0	3 4	0	3 4	0	3 4	0			
NEPHROLOGY	13	1 2—I	1	1 2	0	1—II 2	2	1 2	0	1 2	0	1 2	1	1—I 2	3			
		3 4	3	3 4	1	3 4	7	3 4	0	3 4	0	3 4	0	3—I 4—I	7			
NEUROLOGY	14	1	0	1	1	1—I	2	1	0	1	0	1	0	1 2 II	2			
		$\frac{2}{3}$	1	2 3 4I	1	3 - I	8	$\begin{bmatrix} 2 \\ 3 \\ 4 \end{bmatrix}$	0	$\begin{bmatrix} 2 \\ 3 \\ 4 \end{bmatrix}$	0	$\frac{2}{3}$	0	3^{2-11}_{4}	3			
PALLIATIVE MEDICINE	5	1	0	1	0	1—I	1	1	2	1	0	1	0	1	0			
		23		23		23		2—I 3—I		23		2 3		23				
REHABILITATION	3	4	0	4	0	4	0	4	2	4	0	4	0	4 1—I	1			
		2 3	Ū	23	Ū	23	U	23	Ū	23	U	23	U	23				
	16	4	0	4	0	4	0	4	0	4	1	4	0	4	3			
RESPIRATORY MEDICINE	16	$\frac{1}{2}$	0	$\begin{vmatrix} 1\\ 2\\ 2 \end{vmatrix}$	0	$\begin{bmatrix} 1\\ 2 \\ -I \end{bmatrix}$	1	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	1	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	0	$\begin{bmatrix} 1 \\ 2 \\ 2 \end{bmatrix}$	0	$\begin{bmatrix} 1 \\ 2 \\ -I \end{bmatrix}$	1			
		3 4	2	3 4	5	3 4	5	$\frac{3-1}{4}$	0	3 4	3	4	2	4	4			
RHEUMATOLOGY	9	1 2	0	1—I 2	1	1—I 2	1	$\frac{1}{2}$	0	$1 \\ 2$	0	1 2	0	$1 \\ 2$	0			
		3 4	0	3 4	0	3 4	3	3 4	0	3 4	2	3 4	1	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	8	1—III 6						
		2—I						
		3—II						
		4 11						
INFECTIOUS DISEASE	4	1 0						
		2						
		3						
		4 4						
RESPIRATORY MEDICINE	16	1 0						
		2						
		3						
		4 8						

* 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 May 2016

							FELLOWS							
		HONG	KON	G EAST	CLUSTER	НО	NG I	KONG	WEST C	CLUSTER	HONG KONG			
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/ OTHERS)	PYNEH	RH	TWEH	Subtotal	FYKH	GH	QMH	TWH	Subtotal	EAST + WEST CLUSTER			
CARDIOLOGY	258	9	7	0	16	0	6	18	0	24	40			
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	0	0	0	2	0	2	2			
CRITICAL CARE MEDICINE	94	11	2	0	13	0	0	12	0	12	25			
DERMATOLOGY & VENEREOLOGY	107	0	0	0	0	0	0	2	0	2	2			
ENDOCRINOLOGY, DIABETES & Metabolism	111	6	2	3	11	0	0	11	1	12	23			
GASTROENTEROLOGY & HEPATOLOGY	188	6	2	2	10	0	0	14	0	14	24			
GERIATRIC MEDICINE	192	6	11	3	20	7	2	4	0	13	33			
HAEM/HAEM ONCOLOGY	61	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	40	3	0	0	3	0	0	2	0	2	5			
INTERNAL MEDICINE	1349	59	26	11	96	6	14	101	10	131	227			
MEDICAL ONCOLOGY	47	0	0	0	0	0	0	9	0	9	9			
NEPHROLOGY	135	8	0	0	8	0	0	8	4	12	20			
NEUROLOGY	121	6	4	0	10	0	0	11	1	12	22			
PALLIATIVE MEDICINE	28	0	2	0	2	0	2	1	0	3	5			
REHABILITATION	55	0	1	3	4	1	0	1	5	7	11			
RESPIRATORY MEDICINE	191	11	6	1	18	0	9	10	0	19	37			
RHEUMATOLOGY	80	4	2	1	7	0	0	9	1	10	17			

		FELLOWS															
		ŀ	CEN CEN CLU	LOON TRAL ISTER	1	KOV	VLOON CLUSTI	EAST ER			KOW	LOON	WEST	CLUST	'ER		KOWLOON CENTRAL + EAST
SPECIALTY	FELLOWS TOTAL (PP/ DH/HA/ OTHERS)	вн	КН	QEH	Subtotal	нонн	ткон	UCH	Subtotal	СМС	KWH	OLMH	РМН	WTSH	ҮСН	Subtotal	+ WEST CLUSTER
CARDIOLOGY	258	0	0	15	15	0	5	8	13	2	9	2	12	0	6	31	59
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
CRITICAL CARE MEDICINE	94	0	0	6	6	0	4	7	11	5	5	0	6	0	1	17	34
DERMATOLOGY & VENEREOLOGY	107	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
ENDOCRINOLOGY, DIABETES & Metabolism	111	0	0	8	8	0	5	4	9	2	4	2	5	0	3	16	33
GASTROENTEROLOGY & HEPATOLOGY	188	0	0	8	8	0	5	5	10	4	8	2	9	0	7	30	48
GERIATRIC MEDICINE	192	1	8	4	13	3	2	13	18	8	12	2	13	5	7	47	78
HAEM/HAEM ONCOLOGY	61	0	0	7	7	0	2	3	5	0	0	0	6	0	0	6	18
IMMUNOLOGY & ALLERGY	6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
INFECTIOUS DISEASE	40	0	0	7	7	0	0	1	1	0	0	0	4	0	0	4	12
INTERNAL MEDICINE	1349	2	12	82	96	8	32	54	94	35	53	10	70	6	30	204	394
MEDICAL ONCOLOGY	47	0	0	3	3	0	0	2	2	0	0	0	1	0	0	1	6
NEPHROLOGY	135	0	0	10	10	1	2	6	9	2	9	0	9	0	2	22	41
NEUROLOGY	121	0	3	10	13	0	3	5	8	1	5	1	4	1	2	14	35
PALLIATIVE MEDICINE	28	1	0	0	1	5	0	2	7	5	0	1	0	1	0	7	15
REHABILITATION	55	0	9	1	10	1	0	3	4	1	1	1	1	3	0	7	21
RESPIRATORY MEDICINE	191	2	7	7	16	6	4	8	18	5	6	0	7	4	3	25	59
RHEUMATOLOGY	80	0	1	6	7	0	2	4	6	2	5	0	4	0	2	13	26

		FELLOWS									
		NE	W TERI	RITORI	ES EA	ST CLU	NEW WI	V TERR EST CL	NEW TERRITORIES		
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/ OTHERS)	AHNH	NDH	PWH	SH	TPH	Subtotal	РОН	ТМН	Subtotal	EAST + WEST CLUSTER
CARDIOLOGY	258	3	6	13	0	0	22	3	11	14	36
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	5	0	0	5	0	0	0	5
CRITICAL CARE MEDICINE	94	5	6	2	0	0	13	0	5	5	18
DERMATOLOGY & VENEREOLOGY	107	0	0	3	0	0	3	0	0	0	3
ENDOCRINOLOGY, DIABETES & Metabolism	111	1	5	17	1	0	24	1	4	5	29
GASTROENTEROLOGY & HEPATOLOGY	188	3	5	10	0	0	18	6	10	16	34
GERIATRIC MEDICINE	192	2	2	7	8	4	23	3	12	15	38
HAEM/HAEM ONCOLOGY	61	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	40	3	0	3	0	0	6	0	2	2	8
INTERNAL MEDICINE	1349	27	27	87	11	10	162	21	72	93	255
MEDICAL ONCOLOGY	47	0	0	18	0	0	18	0	0	0	18
NEPHROLOGY	135	5	1	11	0	0	17	2	7	9	26
NEUROLOGY	121	2	2	12	1	0	17	2	5	7	24
PALLIATIVE MEDICINE	28	0	0	0	2	0	2	0	1	1	3
REHABILITATION	55	0	1	2	1	1	5	1	3	4	9
RESPIRATORY MEDICINE	191	4	6	7	0	3	20	3	10	13	33
RHEUMATOLOGY	80	3	0	4	0	3	10	1	4	5	15



PROFESSOR JOSEPH JAO-YIU SUNG SBS JP

MB BS (HKU); PhD (Calgary); MD (CUHK); FRCP (London); FRCP (Edinburgh); FRCP (Glasgow); FRACP; FAGA; FACG; FHKCP; FHKAM (Medicine)

John Mackay

Joseph Sung has been a well-known and respected figure in medical circles since he was appointed Chair Professor of Medicine and Therapeutics at the Chinese University of Hong Kong in 1989, and became even more widely known after his achievements in leading the Prince of Wales Hospital team during the SARS epidemic in 2003.

The Hong Kong College of Physicians had every good reason to bestow on him an Honorary Fellowship in 2015. It was my pleasure to meet him on that occasion and to be invited to write this 'Profile' of him for Synapse. To find out more about his life I interviewed him at his Vice-Chancellor's office at the Chinese University in January 2016.

Professor Joseph Sung's forebears came from Ningbo in Zhejiang province. His father moved just up the coast to Shanghai where he had a practice as an optometrist. He left Shanghai when the Japanese invaded in 1937 to go to Hong Kong, where he continued to work through the Japanese occupation. His first child, Joseph, was born in 1959.

Joseph went to school at Queen's College, founded by the government in 1862, and remaining one of the most prestigious schools in Hong Kong.

At Hong Kong University he entered the Medical School qualifying MB BS in 1983. His internship year was spent at the Queen Mary Hospital, six months each of medicine and of orthopaedic surgery. He remembers Professor David Todd as an outstanding influence, hugely knowledgeable, and dedicated to patient care. At Professor Todd's inaugural briefing for new interns he announced that there were no fixed working hours; doctors were expected to finish their work however long it took. Professor Sung commented that senior members of staff also came in to attend patients during off duty hours.



PROFILE DOCTOR



In 1984 he joined the newly opened Prince of Wales Hospital in Shatin, as a Registrar in pathology for the first year, and in the medical department for the next two years, and passed the MRCP examination. During the following three years he was a Fellow, specialising in gastroenterology and hepatology.

Despite the long hours he still found time to develop an interest and do research on gall-stones, and sometimes came into the hospital when he was off duty to see patients with particularly interesting and challenging problems. He also found time to court the fellow student who was to become his wife in 1989.

Intent on getting further training overseas, he was given a scholarship in 1989 from the Croucher Foundation to study under Dr Bill Costerton in the University of Calgary, Canada. The original one year scholarship was extended by six months after which an Izaak Walton Killan Memorial Scholarship, the most prestigious graduate award from the University of Alberta, was awarded to cover the second part of his three year stay. These years were important for him scientifically as he learned about bacterial biofilms and their importance to medicine, for instance gall stone formation on which he had already done research, and colonization of catheters. His work was rewarded with the grant of a PhD in biomedical science. The years were also important because they introduced him to a scientific establishment where coffee-break casual conversations were on a wide range of subjects, including art, not just about medical matters as he had experienced in Hong Kong.

He returned to the PWH in 1992 as a Lecturer in Medicine and has stayed with the medical school ever since, climbing the ranks and earning an MD. In 1989 he was appointed to his present position as Chair Professor of Medicine and Therapeutics.

All was not plain sailing. In 2003 the Severe Acute Respiratory Syndrome (SARS) swept into Hong Kong. The Prince of Wales hospital had to accept the first rush of patients though not equipped with facilities to isolate infectious disease. As Chair of the medical department Professor Sung was in charge of the team though not a respiratory physician. When patients and staff were dying there was a call from staff for him to close the hospital, but this was a decision only the Hospital Authority could take. There were 1,750 patients admitted to hospitals in Hong Kong of whom 286 died this included 386 medical workers of whom eight died. This period of four months until the epidemic was controlled must have been an enormous strain and cause for sadness for Professor Sung and his colleagues.

The aftermath brought scrutiny of the performance of the handling of the epidemic by a government appointed, independent, international committee chaired by Prof. Sian Griffiths, and a second committee appointed by the Hospital Authority, both of whom made on the whole favourable and constructive reports. A committee set up by the Legislative Council found fault where the others did not, and lead to the resignation of a number of eminent doctors, administrators and clinicians.

PROFILE DOCTOR

Following the SARS epidemic he was named "Asian Hero" by the *Time* magazine in recognition of his outstanding leadership. Underscoring his significant services to the Hong Kong community, Professor Sung was awarded, among others, the Distinguished Award for Scientist and Medical Professional in the Fighting Against SARS (Medical Technology Personnel Category), and the Leader of the Year 2003 (Community and Public Affairs Category) by the media in Hong Kong. He was presented with a Silver Bauhinia Star in 2004.

A sabbatical in 2004 was a welcome break from Hong Kong. A Croucher Senior Research Fellowship allowed him to take six months off to conduct research in Hong Kong or overseas.

"I decided to take this opportunity to go to one of the best schools of public health in the US, the John Hopkins University, Bloomberg School of Public Health. I spent 3 months there both as a student as well as a teacher because I was asked to share the experience of SARS management in Hong Kong as part of the Master of Public Health teaching", he says.

"On the other hand, I learned about other aspects of public health from them, and more about infectious disease control. It was a very useful period for me that allowed me to bring back a lot of knowledge and experience in infectious disease and establish the Centre for Emerging Infectious Diseases at CUHK when I returned in 2004."

The Stanley Ho Centre for Emerging Infectious Diseases at the Chinese University of Hong Kong is the first of its kind in Hong Kong. Professor Sung serves as its Founding Director and Advisor. It is an interdisciplinary group with expertise in epidemiology, microbiology, clinical medicine and health economics to strengthen infectious disease preparedness of the society. The Centre studies the relationship of emerging infectious diseases and the public health implications in surveillance and management.

In 2007 he was appointed 2007 Mok Hing Yiu Professor of Medicine at Chinese University. Dr Mok, his father and grandfather before him have been very generous donors to Hong Kong Universities.

Research and teaching have always been important to Professor Sung. His original work on gastrology and hepatology and cancer prevention has resulted in over 800 articles in peer-reviewed journals. He has reviewed and contributed chapters to 25 books, including the Oxford Textbook of Medicine. (5th edition)

Professor Sung has received many scientific awards in recognition of his achievements, including eleven Fellowships. In 2008 he was honoured by the Prevent Cancer Foundation of the United States with the Laurel Award for his work in cancer screening and prevention. He was made an Academician of the Chinese Academy of Engineering in 2011, and an Academician of the Eurasian Academy of Science in 2012.

He is a Member of 13 Professional bodies in Hong Kong and abroad.

In 2010 he was appointed Vice Chancellor and President of the Chinese University of Hong Kong, only the second doctor of the seven to be appointed so far.



PROFILE DOCTOR

To this position he has brought a philosophy developed from his own negative experiences as a student in Hong Kong where,"It was all studying and exams. Worse still, the medical school was segregated from the main campus". In stark contrast was his time in Calgary where he experienced a more holistic education.

He has said that his favourite film is 'The Dead Poets Society' where a schoolteacher inspires his students to explore the meaning of life through poetry. "A civic society's most important quality is to be people-orientated. In a far and just society everybody lives harmoniously. I believe the arts are a diverse expression of the value of life and freedom".

In 2011 he initiated 'I-Care' to increase student civic and personal awareness, a series of events on diverse topics that have been attended enthusiastically by students. 1,500 students turned out in October 2013 to listen to He Weifang on China's constitutional development. A film was shown on the Scholarism movement, other speakers talked on dance, poetry, pop music, all designed to enhance students' civic awareness. He is popular for his relaxed and easygoing demeanour, and ability to maintain close relations with his students, a habit he says he picked up while being the head of Shaw College at CUHK from 2008 to 2010.

A major project for the university has been the setting up of a Chinese University of Hong Kong Shenzhen research Institute with the help of the Shenzhen authorities. The first students were admitted in 2014, to learn engineering science and business.

Professor Sung's other main project is the Chinese University Medical Centre to be completed in 2019 at the University, a private teaching hospital of 600 beds, with 70% of the beds reserved for treating local patients at affordable package charges.

Asked for his opinion on the medical scene in Hong Kong Professor Sung commented that the standards are good. He regrets that the public/private

divide is growing; the Hospital Authority can ill afford the numbers of doctors moving into the private sector. He recommends his own philosophy that doctors should regard a medical career as more than just a way to earn money.

His second term as Vice Chancellor will end in 2018 at which time Professor Sung looks forward to returning to full time clinical work, teaching and research. He also looks forward to more time to enjoy his study of calligraphy and other areas of art. His offices are testament to his art interests with the walls hung with pictures and calligraphy, two of the pieces his own calligraphy, and the tables covered with books on art such as impressionist photography.

He is looking forward to spending more time with his family, his wife who is a hospital specialist and his two daughters both studying medicine at the Chinese University of Hong Kong.

He has one particular continuing medical interest outside Hong Kong. Since 2012 Professor Sung has been the Li Dak Sum Professor at Ningbo University, involved in planning the curriculum of the medical school. Li Dak Sum is well-known in Hong Kong as a major funder of our universities; he has also given \$100 million to Ningbo University, being a native of Ningbo: as was Joseph Sung's grandfather.

